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6

The Life History Framework and the FSD Model

The concepts discussed in Chapter 5 can be used to analyze specific symptoms and disorders in evolutionary terms (Brüne, 2015; McGuire & Troisi, 1998; Nesse, 2015). However, they still fall short of providing a coherent theoretical framework for the discipline. A compelling framework should meet four main challenges: (a) explain observed patterns of comorbidity between disorders; (b) address the problem of heterogeneity within diagnostic categories; (c) bridge psychopathology with normative individual differences in personality and cognition; and (d) make sense of the developmental features of mental disorders, including their life course trajectories and early risk factors. The life history framework I present in this chapter meets all these challenges and offers an alternative taxonomy of disorders based on functional criteria. In earlier versions of the framework I introduced the distinction between *fast spectrum* and *slow spectrum disorders* (Del Giudice, 2014*a*, 2014*b*, 2016*c*, 2016*d*). Here, I update and revise that initial formulation in a number of ways. In particular, I supplement the fast–slow spectrum distinction with the novel category of *defense activation disorders.* The tripartite distinction between fast spectrum, slow spectrum, and defense activation disorders is the foundation of the fast-slow-defense (FSD) model of psychopathology. I describe the FSD model and discuss its predictions about sex differences, developmental patterns, and risk factors for the three categories of disorders. At the end of the chapter, I explore some additional implications of the model, consider the role of general intelligence in the origin of psychopathology, and compare the FSD taxonomy with the structural models based on the transdiagnostic approach.

HOW TO READ THIS CHAPTER

This chapter presents a large amount of information, including a bird's eye view of the FSD model in its complete form. It is important to keep in mind that the reasons for sorting disorders into the three FSD categories and splitting them

into functional subtypes are explained in detail in the analytical chapters of Part III (Chapters 7–20). The chapters of Part III also discuss limitations, anomalies, and unknowns with respect to the classification of each disorder. There are two reasons for introducing the full model before examining individual disorders in depth. First, many readers will likely find it useful to get a sense of the big picture before delving into the intricacies of diagnostic categories and their subtypes. Second, there is a practical advantage in placing all the information about the life history framework in a single reference chapter instead of dispersing it throughout the book. At the same time, some of the information presented here may become easier to appreciate when one is more familiar with the details of individual disorders. This is why I recommend coming back to this chapter after finishing Part III for a synthesis and recapitulation of the framework.

A FUNCTIONAL TAXONOMY OF MENTAL DISORDERS

Fast and Slow Spectrum Disorders

In Chapter 4, I discussed how life history strategies organize broad functional patterns of individual and sex differences. By coordinating variation across multiple traits, life history strategies also set the stage for the development of psychopathology. Different strategies and profiles are associated with differences in motivation, self-regulation, personality, cognition, and neurobiological functioning (Figure 4.3; Table 4.1), which in turn increase (or decrease) the risk of developing different types of mental disorders. As one moves along the fast–slow continuum, some symptoms and disorders should become more frequent, while others should become less likely to occur. This is the functional basis for the distinction between fast spectrum (or *F-type*) disorders and slow spectrum (or *S-type*) disorders—that is, disorders that cluster at the fast or slow end of the life history continuum (Del Giudice, 2014*a*). For example, fast strategists are more at risk for disorders that involve impulsivity and antisocial behavior; in contrast, slow strategists tend to develop disorders involving behavioral constraint, exaggerated self-control, or reduced sexual motivation. Moreover, alternative profiles within fast and slow strategies are associated with specific constellations of disorders; for example, schizophrenia and other psychotic disorders are characterized by exaggerated mentalistic cognition and arise mainly in the context of seductive/creative strategies; conversely, autism risk is associated with reduced mentalistic skills and other traits that define the skilled/provisioning profile (see Del Giudice et al., 2010, 2014*a*).

In sum, the core proposition of the life history framework is that risk for different types of mental disorders partly depends on broader patterns of individual differences, which in turn can be functionally understood as manifestations of alternative life history strategies. This correspondence makes it possible to classify disorders based on their connections with different

strategies and profiles within those strategies. Interestingly, the distinction between fast and slow spectrum disorders recovers some classic ideas about *undercontrolled* versus *overcontrolled* profiles of self-regulation and their links to different clusters of mental disorders (e.g., Block, 2002; Block & Block, 1980; Huey & Weisz, 1997). However, the life history perspective has a wider scope and situates individual differences in self-regulation within a much larger network of interconnected traits that spans personality, cognitive abilities, and sexual maturation (Chapter 4).

A common misconception about the life history framework is that, since fast and slow spectrum disorders are functionally linked to adaptive patterns of individual differences, they should also be understood as adaptive strategies in their own respect. While this may apply to some particular conditions, the distinction between F-type and S-type disorders is much more general and does not depend on the assumption that mental disorders are adaptive. By modulating the expression of multiple traits and the functioning parameters of key psychological mechanisms, life history allocations can increase or decrease the risk for *all* kinds of broad-sense disorders, from harmful dysfunctions to adaptive strategies. To restate: the life history framework does not assume that all or even most mental disorders are adaptations; both the fast and slow spectrum of psychopathology include plenty of maladaptive and/or dysfunctional conditions alongside adaptive or potentially adaptive strategies.

Multiple Causal Pathways

A more detailed analysis based on the taxonomy presented in Chapter 5 suggests the existence of several potential pathways from life history strategy to psychopathology. To begin, adaptive life history–related traits may be regarded as symptoms because of their undesirable features. This is most likely to occur in relation to antisocial, exploitative, or socially devalued strategies (e.g., psychopathic traits, mild autistic symptoms). Other times, life history–related traits that are adaptive on average may yield individually maladaptive outcomes. An especially interesting case occurs when potentially adaptive traits (e.g., mentalistic cognition) are expressed at maladaptive levels—for example as a result of assortative mating between two individuals high (or low) on the same trait or as a maladaptive side effect of intragenomic conflict. Consistent with this view, a number of mental disorders—most notably autism, schizophrenia, and attention-deficit/hyperactivity disorder (ADHD)—show fairly high rates of assortative mating, with correlations between partners in the .40–.50 range (Nordsletten et al., 2016). Finally, life history–related traits may directly or indirectly increase a person's vulnerability to specific types of dysfunctions. Upregulation of serotonergic signaling may render the system especially sensitive to mutations in serotonin-related genes; increased motivation for affiliation and reciprocity may make people vulnerable to developing harmful levels of guilt and shame (Oakley et al., 2012). In the etiology of dysfunctions, individual traits and vulnerabilities interact with a range of environmental insults such as infections, nutritional deficits, and psychosocial stressors. Intriguingly, fast traits such as risk proneness and sexual

promiscuity may increase an individual's exposure to environmental risk factors (e.g., pathogens, injuries, traumatic events), further increasing the risk of harmful dysfunctions.

Because individual differences in life history affect the risk of psychopathology through multiple causal pathways, the life history framework does not apply only to "trait-like" disorders characterized by developmentally stable configurations of symptoms (e.g., autism, personality disorders), but also to "state-like" disorders whose onset reflects a distinct change in psychological functioning (e.g., eating disorders, depression; see Kennair, 2014). Importantly, the fact that stable life history–related traits (e.g., impulsivity, overcontrol) predispose people to develop some pathological conditions does not mean that treatments for those conditions should necessarily target the predisposing traits. Treatments that address specific symptoms and the psychological processes that maintain them can be highly effective in resolving a disorder (Kennair, 2007). However, it is also possible to design broadband interventions that seek to modify aspects of the patient's personality instead of focusing on a particular symptom or condition (Barlow, Sauer-Zavala et al., 2014; Norton & Paulus, 2016; Roberts et al., 2017). The evidence indicates that cognitive-behavioral treatments based on the two approaches are about equally effective (Pearl & Norton, 2017). This is not surprising: if mental disorders arise from complex causal pathways, it should often be possible to intervene at different levels with similar clinical results.

Defense Activation Disorders

As discussed in Chapter 5, defensive mechanisms are a major source of psychiatric symptoms. Many negative emotions—such as fear, anxiety, shame, and disgust—can be construed as self-protective reactions to social and nonsocial threats (Gilbert, 1992, 1995; Nesse, 2005*a*, 2015; Nesse & Jackson, 2006). To some degree, these emotions contribute to the phenomenology of many if not most mental disorders; for example, autism is often associated with social anxiety, and shame is a pervasive correlate of eating disorders (e.g., Allan & Goss, 2012). However, some disorders *primarily* consist of symptoms that reflect the activation of a defensive mechanism. I propose to group those conditions into a distinct functional category, that of defense activation or *D-type* disorders. Conditions such as phobias, panic, posttraumatic stress, generalized anxiety, and depression can be understood as manifestations of strongly activated defenses; of course, this does not mean that these conditions are necessarily beneficial to fitness. While there is no doubt that aversive reactions such as panic and anxiety *can* be adaptive, defense activation errors in absence of a threat may also yield maladaptive outcomes. Defensive mechanisms may also become damaged or dysregulated, giving rise to harmful dysfunctions. In short, D-type disorders may belong to any of the biological categories listed in Figure 5.1.

Defenses and the Fast–Slow Continuum

Danger, threats, and mortality risk are among the aspects of the environment that most strongly contribute to determine life history allocations. There is no doubt that life history strategies have broad implications for the regulation of defenses; however, the relations between life history variation and specific patterns of defense activation are far from straightforward. This is why the list of life history–related features in Table 4.1 is notably lacking in defense-related traits, with the exception of disgust sensitivity. The adaptive calibration model (ACM) model of stress physiology (introduced in Chapter 4) helps illustrate this concept. According to the model, hyperreactive profiles tend to develop at both the slow end (sensitive pattern) and the fast end of variation (vigilant pattern), while a subset of fast strategists develop hyporeactive profiles (unemotional pattern; Figure 4.2). Even if stress physiology has deep functional connections with life history strategies, those connections do not translate into a simple fast–slow gradient of stress reactivity.

More generally, there are reasons to predict that patterns of upregulated defenses should become more common at both ends of the fast–slow continuum (Del Giudice, 2014*a*; Lienard, 2011). In the context of fast life histories, hair-trigger defenses contribute to protect the person from immediate danger in a threatening, unpredictable environment. In the context of slow strategies, upregulated defenses may help prevent dangerous events and avoid potentially risky situations in the future, even if the present environment is reasonably safe and predictable. Protecting oneself against even minor sources of harm contributes to the long-term maintenance of somatic investment—a key priority for slow strategists, who make major investments in embodied capital. Also, when the environment is safe and threats are rare, the benefits of sensitive defenses can be afforded without paying the costs of hyperactivation. Chronic defense activation is much more likely to occur in stressful and chaotic environments that expose people to repeated, high-intensity threats.

These considerations suggest that, all else being equal, D-type conditions should arise relatively more often in dangerous, stressful environments—which is to say, contexts that also favor the development of fast life history strategies. Moreover, the behavioral traits associated with fast strategies (e.g., risk-taking, impulsivity) increase the chance of experiencing stressful events such as accidents, romantic breakups, and job losses; the cumulative effect of such events further contributes to increase defense activation and with it the risk of D-type disorders. At the level of personality traits, neuroticism is the principal marker of upregulated defense activation. Neuroticism covaries with low agreeableness and conscientiousness as part of metatrait alpha, and some of its facets measure antagonistic negative emotions such as anger and hostility. This pattern of correlations contributes to predict a stronger association between defense activation and F-type disorders compared with S-type disorders. In general, females have more to lose than men from both physical damage and social rejection (Archer, 2009; Benenson, 2013, 2014; Campbell, 2004; Martel, 2013); accordingly, D-type disorders can be expected to be markedly more

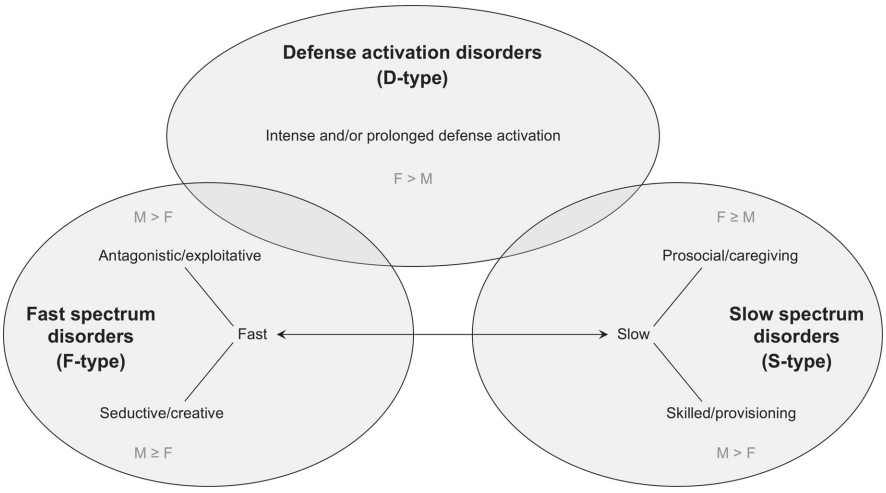

Figure 6.1. Conceptual structure of the FSD classification model. Overlap between disorder categories represents comorbidity. While defense activation disorders may arise at both ends of the fast-slow continuum, they are expected to occur more frequently in associations with fast spectrum disorders. F = females. M = males.

common in females, especially as one moves toward the fast end of the continuum. Figure 6.1 illustrates the relation between defense activation disorders and fast–slow spectrum disorders in the FSD model. As shown in the figure, D-type disorders occur at both ends of the fast–slow continuum; however, their specific symptoms may vary to some extent depending on the person's life history profile and comorbid conditions. For example, symptoms of worry involve a preoccupation with potential events in the future (e.g., losing one's job, becoming ill) rather than a focus on immediate threats (Chapter 15). Interestingly, the facets of neuroticism associated with worry tend to predict better physical health and increased longevity, in contrast with those associated with anxiety (Hill, Weiss et al., 2017). It is reasonable to hypothesize that D-type conditions involving intense worry may co-occur relatively more often with disorders in the slow spectrum given their future-oriented focus on harm prevention (see Fernandes et al., 2018).

The concept of defense activation disorders calls attention to the specular possibility that some conditions may primarily reflect a *lack* of activation of adaptive defenses in contexts that would require a vigorous response (pain insensitivity and immunosuppression are medical examples). Instances of systematic defense downregulation are more likely to occur in association with fast strategies, especially with behavioral profiles characterized by high levels of risk-taking. The underlying logic is that, in order to be successful, high-risk strategies require outright insensitivity to threats and dangers, including powerful social threats such as disapproval and rejection. Moreover, unresponsive strategies can mitigate the costs of inappropriate or excessive defense activation when the environment becomes too unpredictable or threatening (Del Giudice et al., 2011; Korte et al., 2005). In

general, males benefit from risk-taking more than females; for this reason, they should also be more vulnerable to pathological forms of defense downregulation.

Evolutionary scholars have long pointed out that standard diagnostic systems may be neglecting the existence of conditions marked by a dangerous lack of defense activation (Horwitz & Wakefield, 2012; Nesse, 1990). Conditions of this kind could be labeled *defense inactivation disorders* (abbreviated as lowercase *d-type* disorders, to distinguish them from D-type disorders marked by exaggerated activation). The *Diagnostic and Statistical Manual* (DSM) includes many disorders characterized by defense hyperactivation, but virtually no conditions whose core symptoms reflect defense hypoactivation. The only potential exception is "disinhibited social engagement disorder," a childhood condition whose key symptom is lack of fear and reticence in approaching unfamiliar adults (American Psychiatric Association, 2013). Of course, several common disorders are characterized by a degree of defense inactivation—for example, insensitivity to fear is an important component of psychopathy (Fowles & Dindo, 2006; Hughes et al., 2012). However, failures of defense activation are not as dramatic and subjectively aversive as strong defensive reactions; as a result, even legitimate dysfunctions of this kind may not be regarded as pathological when they occur on their own (that is, they may fail to meet Wakefield's harm criterion). Given the dearth of information at this time, it would be premature to explicitly include this type of condition in the FSD model; still, this remains a fascinating area for future research.

Applying the Framework

The classification scheme summarized in Figure 6.1 can be employed with two distinct but complementary goals. The first goal is to map the large-scale structure of psychopathology by describing broad patterns of comorbidity between disorders (Del Giudice, 2016*d*). The second is to identify functionally heterogeneous conditions within existing diagnostic categories. Importantly, the FSD classification is not meant to replace specific evolutionary models of individual disorders; rather, the goal is to integrate and connect them under the theoretical umbrella of the life history framework.

Patterns of Comorbidity

The life history framework maps the structure of psychopathology on two main axes of variation: a primary axis aligned with the fast–slow continuum (risk for F-type versus S-type disorders) and a secondary axis that captures the tendency to display strong and/or sustained defense activation (risk for D-type disorders). In addition to these two axes of comorbidity, low cognitive ability contributes to increase the risk for most mental disorders, as I discuss in more detail later. Females are generally at higher risk for defense activation conditions than are males, while fast and slow spectrum disorders may show different associations with sex depending on their relations with specific life history profiles (e.g.,

disorders linked to the skilled/provisioning profile should be more common in males).

It is important to appreciate that, at this level of analysis, comorbidity is explained by the existence of underlying patterns of individual differences in the function and/or functionality of psychological processes. These differences are reflected in personality traits and profiles of motivation and cognitive ability (Table 4.1). From this perspective, two disorders may co-occur within individuals and families even in the absence of a shared underlying dysfunction (as postulated by the Research Domain Criteria [RDoC] approach) or a direct causal "bridge" between their symptoms (as assumed by the network approach). The idea that large-scale patterns of comorbidity stem from broad dimensions of individual variation rather than disorder-specific risk factors is in line with the spirit of the transdiagnostic approach. At the same time, the FSD model carves up the landscape of psychopathology in a way that only partially matches the internalizing–externalizing distinction and other categories of transdiagnostic models. In a later section, I compare the two models in detail and highlight the advantages of the FSD taxonomy.

While the broad dimensions described by the FSD model offer a useful first approximation of the structure of psychopathology, they are hardly exhaustive. Within each of the three main FSD categories (F-type, S-type, D-type) one finds smaller clusters of disorders that share particular traits or risk factors. Comorbidity clusters may arise from specific combinations of life history–related traits; for example the combination of increased mentalizing, high openness to experience, and disinhibition in the seductive/creative profile may increase the risk for a number of related conditions in the schizophrenia and bipolar spectrum. On an even finer scale, two disorders may co-occur because they share a specific causal factor—a particular dysfunction in the same psychological mechanisms or a certain set of genetic/epigenetic variants. This level of analysis is the one emphasized by the RDoC approach, which seeks to identify dysfunctions at the level of specific neurobiological mechanisms.

Functional Heterogeneity

Because diagnostic categories in the DSM are mainly based on symptom similarity, they may fail to capture important distinctions between conditions that have different functional underpinnings but show similar constellations of symptoms. The problem of heterogeneity is widely recognized in the discipline; however, there are many possible ways to draw distinctions within a diagnostic category, and without sound theoretical criteria the task of subtyping can become overwhelming. The FSD model can be used as a lens to identify meaningful variants within existing diagnostic categories. The life history correlates reviewed in Chapter 4 can be used as convergent "markers" to locate a condition (and its subtypes) within the functional space of Figure 6.1. For example, it is possible to describe fast and slow spectrum subtypes of eating disorders that are primarily characterized by differences in risk factors and associated personality features rather than specific symptoms (Chapter 13; Del Giudice, 2014*a*).

The task of identifying variants or subtypes within existing categories is not disconnected from that of describing large-scale comorbidity—in fact, the former is a precondition for the latter. As it turns out, not all disorders in the DSM can be assigned unambiguously to the fast or slow spectrum; in a number of cases, a life history analysis suggests the existence of functionally distinct conditions whose symptoms are similar enough that they are diagnosed as the same disorder in the DSM taxonomy. This is fully expected—the whole point of a functional approach is to redraw the boundaries between disorders based on their underlying organization *rather than* just symptom similarity. However, splitting existing categories into subtypes based on alternative criteria remains a radical move, considering that symptom similarity has played a central role in psychiatric classification for most of recent history. For example, arguing that attention and hyperactivity symptoms are best regarded as manifestations of at least three functionally distinct conditions instead of a unitary ADHD category (Chapter 11) may strike some as wildly unparsimonious (e.g., Martel, 2014). However, it is important to realize that—to the extent that DSM categories are internally heterogeneous—this is the inevitable consequence of any approach to classification based on functional rather than purely symptomatic criteria. In the life history framework, the parsimony lost at the level of individual categories is recovered at the level of broad functional distinctions (Figure 6.1) and the resulting large-scale model of comorbidity.

Contemporary approaches to psychopathology differ in how they deal with existing DSM categories. For example, most applications of the transdiagnostic approach are based on symptom scores derived from DSM checklists (e.g., Caspi et al., 2014; Lahey et al., 2015; for a recent exception, see Carragher et al., 2016). At least implicitly, then, they assume the validity and internal consistency of the corresponding criteria. What differentiates transdiagnostic models from the DSM taxonomy is that symptom scores are used to define broader, continuous dimensions of variation (e.g., internalizing, externalizing, thought disorders). As a consequence, this approach has much to say about comorbidity but is virtually silent on the issue of heterogeneity *within* existing diagnostic categories.

The RDoC and computational approaches do not share this limitation; in fact, an explicit goal of RDoC is to reconstruct the taxonomy of mental disorders by identifying specific etiological processes that may cut across existing diagnostic categories and groups. To the extent that this approach is successful, it will inevitably generate distinctions that are not well captured by standard criteria of symptom similarity. Still, there is an important difference between the RDoC approach and the life history framework. From the standpoint of RDoC, new diagnostic boundaries are expected to emerge from the bottom up as knowledge of neurobiological mechanisms and their potential dysfunctions accumulates; in contrast, the life history approach starts with broad, functionally meaningful distinctions (e.g., between fast and slow spectrum disorders) and uses them as heuristics to identify heterogeneity within existing categories. While the two approaches rest on different methodological premises, they are not incompatible—the focus on

specific mechanisms promoted by RDoC can be a useful complement to the kind of top-down analysis encouraged by the FSD model.

Nomenclature

Applying the life history framework to specific disorders raises the issue of how to deal with existing diagnostic labels. In general, evolutionary models of mental disorders have focused on DSM categories such as schizophrenia and ADHD, usually taking them at face value (see Brüne, 2015; McGuire & Troisi, 1998). However, a life history perspective suggests that at least some of these categories contain distinct subtypes that belong to different functional spectra. A possible option would be to discard DSM labels or even reject the standard diagnostic criteria as invalid. I believe this would be a mistake: while the DSM has several well-known limitations, many of its categories have substantial validity and descriptive power. Even radical bottom-up attempts inspired by the network approach end up recovering clusters of symptoms that closely resemble the major conditions described in the DSM. Similarly, computational methods applied to brain imaging data often converge with classic diagnostic distinctions (e.g., Bansal et al., 2012; Boschloo et al., 2015; Haubold et al., 2012). Moreover, from a pragmatic standpoint there are simply no realistic alternatives, since most of the empirical evidence available today has been collected using criteria from the various editions of the DSM.

For these reasons, in the remainder of the book I will use the standard nomenclature to refer to individual conditions (see Box 6.1 for a list of acronyms). To identify putative subtypes of a given disorder, I will simply add the relevant FSD descriptor to the label, as in "fast spectrum ADHD," "F-type ADHD," or "F-ADHD." For variants that seem to fall outside the FSD classification, I will use the descriptor "O-type" (for "other"), as in "O-type ADHD" or "O-ADHD." Whenever necessary, I will point to limitations and weaknesses in the current DSM criteria for certain disorders. This solution should be regarded as provisional; one should always remember that boundaries between conditions might not be so clear-cut as implied by existing classification systems, and that future research will likely suggest new categories and labels that may eventually replace the ones in use today. Figure 6.2. shows the classification of common mental disorders in the current version of the FSD model.

Scope of the FSD Model

As discussed in Chapter 5, mental disorders form a highly heterogeneous collection, with many possible evolutionary explanations and myriad proximate causes. No single set of taxonomic criteria can possibly capture all the important distinctions within psychopathology. Moreover, it is reasonable to expect that different criteria will prove more or less useful depending on the specific goal at hand. The primary goal of the FSD model is to map the large-scale structure of psychopathology on functionally significant dimensions of individual variation and to do so at the broadest level of analysis, that of life history strategies (Figure 6.2).

Box 6.1

Acronyms for Common Mental Disorders

ADHD: attention-deficit/hyperactivity disorder
AN: anorexia nervosa
APD: avoidant personality disorder
ASD: autism spectrum disorder
ASPD: antisocial personality disorder
BD: bipolar disorder
BED: binge-eating disorder
BN: bulimia nervosa
BPD: borderline personality disorder
CD: conduct disorder
ED: eating disorder
GAD: generalized anxiety disorder
MDD: major depressive disorder
NPD: narcissistic personality disorder
OCD: obsessive-compulsive disorder
OCPD: obsessive-compulsive personality disorder
ODD: oppositional-defiant disorder
PDD: persistent depressive disorder
PTSD: posttraumatic stress disorder
SAD: social anxiety disorder
SPD: schizotypal personality disorder
SSD: schizophrenia spectrum disorder
SUD: substance use disorder

It is easy to see how this classification approach can be supplemented by narrower distinctions, for example based on the involvement of specific motivational or self-regulatory mechanisms. Some of the resulting categories would be similar to those of the DSM (e.g., eating disorders), though others would not (e.g., disorders of mating and status). Indeed, other evolutionary scholars have suggested that motivational domains can be used heuristically to classify mental disorders (e.g., McGuire & Troisi, 1998; Stevens & Price, 2000). Another interesting approach would be to classify disorders based on their genetic structure, for example by distinguishing conditions that depend mainly on the effects of rare and de novo mutations from those that are largely influenced by common genetic variants. Given that different criteria may offer different and complementary insights into the nature of psychopathology, a pluralistic attitude toward classification is most likely to be successful in the long run. In this book I explore the potential of the FSD model, while mapping out its boundaries and taking note of its inevitable limitations.

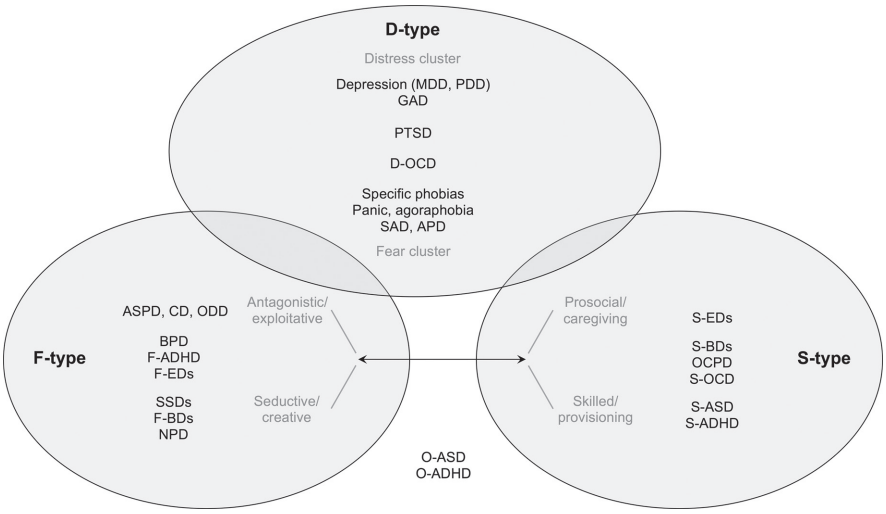

Figure 6.2. Fast spectrum (F-type), slow spectrum (S-type), and defense activation (D-type) disorders in the current version of the FSD model. Overlap between disorder categories represents comorbidity. Conditions that fall outside the FSD classification (O-type disorders) are shown at the bottom of the figure. ADHD = attention-deficit/hyperactivity disorder. APD = avoidant personality disorder. ASD = autism spectrum disorder. ASPD = antisocial personality disorder. BDs = bipolar disorders. BPD = borderline personality disorder. CD = conduct disorder. EDs = eating disorders. GAD = generalized anxiety disorder. MDD = major depressive disorder. NPD = narcissistic personality disorder. OCD = obsessive-compulsive disorder. OCPD = obsessive-compulsive personality disorder. ODD = oppositional-defiant disorder. PDD = persistent depressive disorder. PTSD = posttraumatic stress disorder. SAD = social anxiety disorder. SSDs = schizophrenia spectrum disorders.

FAST SPECTRUM DISORDERS

Fast spectrum disorders arise in connection with fast life history strategies and the corresponding behavioral, cognitive, and neurobiological traits. The idea that fast strategies predispose to psychopathology was proposed decades ago (Belsky et al., 1991; Draper & Harpending, 1982, 1988; MacMillan & Kofoed, 1984). Over the years, a number of conditions have been framed as either adaptive manifestations or maladaptive outcomes of fast-related traits: externalizing disorders (most notably psychopathy and its DSM counterpart, antisocial personality disorder), ADHD, borderline personality disorder, schizophrenia, and eating disorders (e.g., Barr & Quinsey, 2004; Brüne et al., 2010; Del Giudice et al., 2010; Figueredo & Jacobs, 2010; Frederick, 2012; MacMillan & Kofoed, 1984; Mealey, 1995; Salmon et al., 2009). In Part III of the book, I discuss individual disorders and their evolutionary origins in detail; here, I briefly summarize their classification according to the FSD model. In the current version of the model, the fast spectrum of psychopathology comprises the following conditions:

- A cluster of disorders marked by disruptive and antisocial behaviors, namely antisocial personality disorder (ASPD), conduct disorder (CD), and oppositional-defiant disorder (ODD).
- Most instances of schizophrenia spectrum disorders (SSDs).
- A high-frequency, fast spectrum subtype of bipolar disorders (F-BDs).
- A high-frequency, fast spectrum subtype of ADHD (F-ADHD) associated with antisocial/conduct disorders and psychosis risk.
- Personality disorders marked by high levels of antagonism, disinhibition, and/or psychoticism—most notably antisocial personality disorder (ASPD, also part of the conduct/antisocial cluster); schizotypal personality disorder (SPD, also part of the schizophrenia spectrum); borderline personality disorder (BPD); and narcissistic personality disorder (NPD).
- A fast spectrum subtype of eating disorders (F-EDs) marked by high impulsivity and neuroticism.

Among these conditions, antisocial and conduct disorders (ASPD, CD, and ODD) fit the prototype of the antagonistic/exploitative profile, whereas SSDs, F-BDs, and NPD are linked to the seductive/creative profile (Figure 6.2). The constellation of traits that characterizes BPD is intermediate between the antagonistic/exploitative and seductive/creative profiles. It is worth stressing that strategic profiles are not meant as rigidly separated categories, and some people may combine aspects of both. F-type disorders associated with different profiles (e.g., ASPD and BDs) may co-occur within individuals and families, though not as frequently as disorders that share the same functional correlates (e.g., SSDs and BDs).

Fast Spectrum Markers

A working list of fast spectrum markers is presented in Table 6.1. The table shows two largely overlapping sets of markers corresponding to the two strategic profiles discussed in Chapter 4. The main correlates of F-type disorders are low conscientiousness, agreeableness, and honesty-humility; high impulsivity and risk-taking; precocious and unrestricted sexuality; reduced long-term mating and stability of romantic attachments; low levels of disgust sensitivity (especially in the sexual and moral domains); and earlier/faster sexual maturation (especially in females). Markers such as high imagination and a verbally biased pattern of cognitive ability are typical of the seductive/creative profile. This list is not intended to be definitive and will have to be expanded and refined as further research details the neurobiological, hormonal, and genetic bases of human life history strategies and their variants. Tentative neurobiological correlates of the fast spectrum include reduced serotonergic activity, (typically) upregulated mesolimbic dopamine, reduced activity of the dorsolateral prefrontal cortex (DLPFC), and high androgen levels. The seductive/creative profile should be associated with elevated oxytocinergic

Table 6.1 Markers of Fast Spectrum Disorders (F-Type)

F-type markers	Antagonistic/exploitative profile	Seductive/creative profile
Personality factors	Low agreeableness Low conscientiousness Low honesty-humility	Low agreeableness Low conscientiousness Low honesty-humility High openness (imagination/ aesthetics)
Motivation	Precocious sexuality Unrestricted sociosexuality Unstable romantic attachments Reduced long-term mating orientation Low disgust sensitivity (especially sexual/moral)	Precocious sexuality Unrestricted sociosexuality Unstable romantic attachments Reduced long-term mating orientation Low disgust sensitivity (especially sexual/moral)
Decision-making, self-regulation	High impulsivity High risk-taking and sensation seeking	High impulsivity High risk-taking and sensation seeking
Cognitive ability		High mentalistic cognition Low mechanistic cognition Verbal > visuospatial ability
Sexual maturation	Early, fast maturation (especially females)	Early, fast maturation (especially females)

function, increased activity of the default mode network, and a more complex pattern with respect to androgens and other sex hormones (Chapter 4).

A crucial point to keep in mind is that the life history markers listed in Table 6.1 may or may not play a *causal* role in the etiology of any given disorder. For example, impulsivity and risk-taking have a direct causal effect on the development of some externalizing symptoms (e.g., Lahey & Waldman, 2003). However, the robust association between externalizing symptoms and early sexual maturation (Chapter 7) does not mean that sexual maturation per se is a cause of externalizing behavior. The goal of Table 6.1 is not to detail the etiology of F-type disorders, but to present a list of convergent markers that can be used to classify a condition as belonging to the fast spectrum of psychopathology. Also note that certain life history markers—such as high mentalistic cognition or precocious sexuality—may show stronger associations with the less severe forms of a disorder, emerge only during remission phases, or appear more clearly in the unaffected relatives of diagnosed individuals. This is most likely to happen in disorders that reflect harmful dysfunctions or maladaptive outcomes, to the extent that they are associated with impairments (either temporary or permanent) in the functionality of neural and cognitive processes. To illustrate, openness/imagination and creativity

are elevated in people with schizotypal personalities and in relatives of schizophrenic patients, but *not* in the patients themselves (Chapter 8). This does not make these traits less useful as markers of a functional connection between schizophrenia and the seductive/creative profile of the fast spectrum.

Sex Differences

The life history approach helps make predictions about systematic patterns of sex differences in psychopathology. On the whole, disorders linked to the antagonistic/exploitative profile should be more common in males; in females, the same underlying dispositions (such as impulsivity and risk-taking) may give rise to different behavioral manifestations because of the different costs and benefits of traits such as physical aggression and avoidant attachment. Consistent with this prediction, the prevalence of externalizing disorders is considerably higher in males (Martel, 2013). As noted in Chapter 4, theoretical considerations suggest a more or less balanced sex ratio in the seductive/creative profile. However, males may still be somewhat more at risk for the corresponding disorders, both because they show higher variability in personality and behavior (and are thus more likely to develop extreme levels of fast-related traits) and because they are generally more susceptible to dysfunctions, owing to stronger sexual selection and sensitivity to X-linked mutations. Disorders in the psychosis spectrum (SSDs and BDs) have similar prevalence in males and females (with slightly higher male risk for schizophrenia), while men are more likely to be diagnosed with NPD than are women (Aleman et al., 2003; Diflorio & Jones, 2010; Oltmanns & Powers, 2012; Räsänen et al., 2000; Stinson et al., 2008).

Borderline personality disorder combines features of both the antagonistic/exploitative and seductive/creative profiles and shows considerable genetic and phenotypic overlap with externalizing behaviors and some aspects of psychopathy (Crowell et al., 2013; Hunt et al., 2015; Oltmanns & Powers, 2012; Chapter 12). BPD is diagnosed more often in females, although some studies of community samples have found a balanced sex ratio, suggesting that self-selection may partly explain the female bias in clinical settings (Grant et al., 2004, 2008; Martel, 2013; Oltmanns & Powers, 2012; Silberschmidt et al., 2015). Finally, eating disorders (EDs) tap into sex-specific aspects of female physiology and psychology (Chapter 13) and are overwhelmingly more common in females. Not coincidentally, ED risk in males is strongly associated with non-heterosexual orientations (e.g., Feldman & Meyer, 2007; Russell & Keel, 2002; Yean et al., 2013).

Developmental Patterns

While mental disorders occur throughout the whole life course, different conditions show different developmental patterns—including typical age of onset, age-related peaks in prevalence, and trajectories of remission. In a life history perspective, the developmental characteristics of a given disorder should depend—at

least in part—on its motivational underpinnings and the maturation trajectory of the relevant motivational systems. Within the fast spectrum, a useful distinction can be drawn between disorders that primarily involve social competition for dominance and status and those that relate more directly to courtship and mating. This distinction overlaps with that between the antagonistic/exploitative and seductive/creative profiles, but the correspondence is not exact (e.g., NPD is linked to the seductive/creative profile, but its core motivational signature is exaggerated competition for status; Russ et al., 2008). Competition-related disorders are likely to develop earlier, with a peak in middle childhood (Del Giudice, 2014*c*; Del Giudice et al., 2009). Middle childhood is a critical phase for status competition; both the status and aggression systems undergo important developmental shifts, likely under the influence of adrenal androgens. Other important changes occur in the attachment system as close relations with peers start to become salient alongside those with parents. While sexual and mating motivations begin to awaken in middle childhood, they only become fully active at puberty (following maturation of the HPG axis) and peak with the attainment of reproductive maturity, between adolescence and young adulthood (Ellis et al., 2012; Forbes & Dahl, 2010; Mundy et al., 2016). This developmental window is when courtship- and mating-related disorders are expected to arise most often. In general, F-type disorders can be expected to improve or remit starting from the third decade of life, after the peak of social and reproductive competition has ended and sex hormone levels begin to decline.

There are multiple reasons why the onset of competition-related disorders should first peak in middle childhood. To begin, the switch point of adrenarche amplifies individual and sex differences by activating a number of mechanisms and pathways involved in social competition. The resulting individual profiles may include adaptive strategies—some of which may be regarded as broad-sense disorders—as well as biologically maladaptive variants (e.g., detrimentally high or low levels of competition-related traits). Second, rapid changes in the activity of the relevant psychological mechanisms can make them temporarily vulnerable to dysfunctions. Third, competition with peers is likely to expose some children to stage-specific stressors and failures (e.g., peer rejection), which may trigger defensive responses or even result in behavioral and neurobiological dysregulation. Fourth, the intense social learning that takes place in middle childhood opens the way to potentially maladaptive learning at all levels—ineffective social skills, pathological levels of self-esteem, or "deviancy training" by antisocial peers (Dishion & Patterson, 2016). Finally, modern cultural practices may contribute to both developmental and evolutionary mismatches; for example, age segregation between children and adolescents both in schools and in out-of-school activities seems to exacerbate aggression and other problematic behaviors (Ellis et al., 2012). Similar arguments apply to the onset of mating-related disorders during puberty and adolescence.

These developmental predictions are supported by the empirical data. The onset of conduct/antisocial disorders such as CD and ODD shows a first peak in

middle childhood and another around puberty, although there is a subgroup of children who follow a trajectory of elevated, persistent aggression starting from infancy (see Chapter 7). Eating disorders (which have deep connections with mating competition) typically emerge in adolescence, and the risk for psychosis spectrum disorders such as schizophrenia and BDs peaks in young adulthood (Costello et al., 2003; Hulvershorn & Nurnberger, 2014; Khandaker et al., 2014; Mitchell & Bulik, 2014). Developmental data on personality disorders are extremely scarce due to the long-standing assumption that these conditions should only be diagnosed in adults. However, narcissistic traits can be measured reliably starting from middle childhood; also, the constellation of traits that define BPD is already in place at age 12, indicating that this condition likely develops before puberty (Belsky et al., 2012; Sharp et al., 2012; Thomaes & Brummelman, 2016). As a rule, F-type conditions tend to remit or become milder in the third and fourth decades of life, although severe disorders such as schizophrenia may result in chronic or even lifelong impairment.

The distinction between competition- and mating-related disorders is heuristically useful, but does not fully explain the developmental course of F-type disorders. Another important factor to consider is the maturation schedule of specific psychological mechanisms. For example, the development of executive functions accelerates dramatically with the transition to middle childhood; in many cultures, the sophisticated self-control skills that emerge during this phase enable children to contribute significantly to family subsistence (Best et al., 2009; Del Giudice, 2014*c*). The rapid maturation of executive functions in middle childhood amplifies both adaptive and maladaptive individual differences; because of the increasing importance of self-control in children's daily life, latent dysfunctions that previously had little impact on behavior may grow into full-fledged disorders. The confluence of these developmental factors likely contributes to explain why the onset of ADHD peaks at the boundary between early and middle childhood (Kessler et al., 2005).

Differences in the developmental course of fast spectrum disorders intersect with sex differences in prevalence, with the result that childhood-onset conditions also tend to be more common in males. One reason noted earlier is that disorders that peak in childhood tend to be functionally linked to status competition. A complementary reason is that human males have been under stronger sexual selection than females and tend to mature later, a common pattern in species where males need to build up resources and competitive skills to succeed in mating competition. Traits that function as fitness indicators are more vulnerable to developmental disruptions, and slower maturation implies a broader window of susceptibility to stressors and insults experienced in early life (Geary, 2002, 2015; Martel, 2013). This kind of explanation is unlikely to apply to disorders that reflect adaptive or potentially adaptive behavioral strategies (e.g., conduct disorders) but may help explain the early onset of some male-biased conditions that seem to involve genuine dysfunctions, such as childhood-onset schizophrenia.

The Definition of "Childhood-Onset" Disorders

The preceding discussion highlights the need to draw clear distinctions between life stages in order to make sense of the developmental features of disorders. Early childhood, middle childhood, and adolescence are characterized by different functional and endocrine profiles and have widely different implications for the biological meaning of psychological symptoms. From this vantage point, the loose definitions of childhood found in the psychiatric literature are potentially problematic. In this area, most authors use the label "childhood-onset" for any disorder arising by 12 years of age; in doing so, they pool together individuals at very different stages of development, from children who have yet to undergo adrenarche to adolescents who are experiencing the initial phase of puberty. Some adopt an even more inclusive definition that stretches to 18 years of age (e.g., Khandaker et al., 2014). To avoid conceptual confusion, it is important to keep in mind that standard conventions in the literature do not necessarily map on the biological boundaries between developmental stages.

A subtler but important point is that individual differences in life history strategy do not only affect the risk of developing S-type or F-type disorders at certain developmental stages, but also the timing of those stages and the transitions between them. For this reason, life history–related differences in maturation timing may further confound the conventional distinction between childhood- and adolescent-onset disorders when age is used as a proxy for the person's stage of development. Consider the hypothetical case of two disorders, an S-type condition A that emerges in middle childhood and an F-type condition B with onset in adolescence (Figure 6.3). Because fast strategies are associated with accelerated puberty (especially in females), condition B will peak early, and many cases will fall within the conventional age range for middle childhood; moreover, the cases with the earliest onset may also be the most severe as they tend to be linked to more extreme versions of fast strategies. The prediction is reversed for condition A: children will develop the disorder comparatively late, overlapping with the conventional age range for adolescence. Following standard conventions, a minority of cases of condition A would be classified as "adolescent-onset," while a majority of those of condition B (including the more severe ones) would be classified as "childhood-onset." If the two conditions had similar symptoms, they might be regarded as a single disorder despite their different functional underpinnings and hormonal correlates (e.g., adrenal androgens for disorder A, gonadal hormones for disorder B). Both the "childhood-onset" and "adolescent-onset" forms of this putative disorder would be functionally inconsistent, and would comprise mixtures of individuals with different underlying conditions and life history profiles.

Risk Factors

To make predictions about the probable risk factors for a disorder, it is crucial to consider its place in the evolutionary taxonomy of Figure 5.1. When dysfunctions play a major role in the etiology of a certain condition, one can

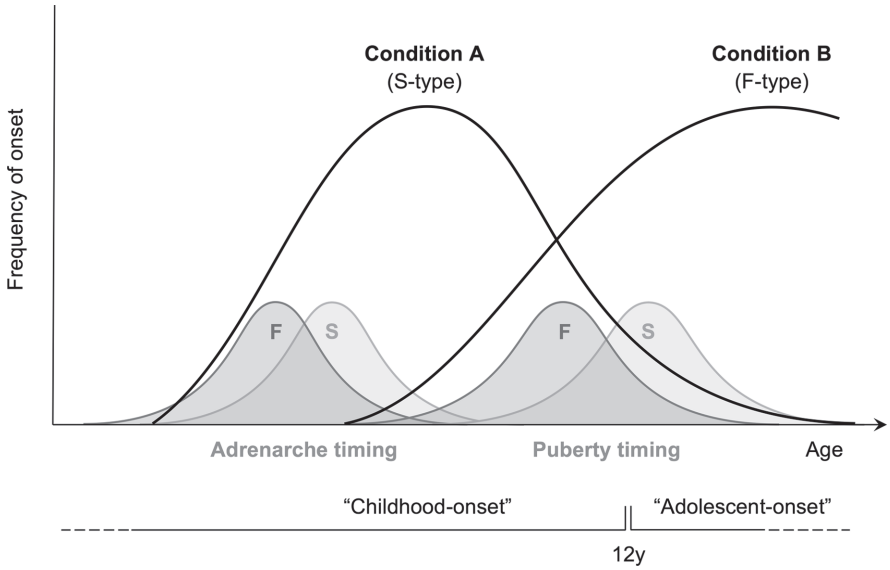

Figure 6.3. Conventional criteria for age of onset may be confounded by life history-related differences in maturation timing. Condition A is a slow spectrum disorder with onset in middle childhood, whereas condition B is a fast spectrum disorder with onset in adolescence. Following conventional age-based criteria, a minority of cases of condition A would be classified as "adolescent-onset," while a substantial proportion of cases of condition B would be classified as "childhood-onset." F = fast life history strategies. S = slow life history strategies.

reasonably expect that risk for that condition will be associated with deleterious mutations and environmental insults such as early infections, malnutrition, or exposure to toxins and other harmful chemicals. Even in the absence of narrow-sense dysfunctions, mutations and developmental stressors may also increase the probability that adaptive processes will yield individually maladaptive outcomes. There is considerable evidence that all the factors I just listed are implicated in the etiology of ADHD, schizophrenia, and BDs (e.g., Ahn et al., 2014; Benros et al., 2012; D'Onofrio et al., 2014; Ehli et al., 2012; Khandaker et al., 2014; Mitchell & Goldstein, 2014). For disorders that plausibly represent adaptive strategies on the fast spectrum, the life history framework predicts correlations with variables associated with harshness and unpredictability—including stressful life events, low socioeconomic status (SES), prenatal stress, negative family relationships, maltreatment, trauma, and peer victimization. These are the main environmental risk factors emphasized in the literature on developmental psychopathology, and they correlate with increased prevalence of fast spectrum conditions, from antisocial/conduct disorders and BPD to schizophrenia (Caspi et al., 2014; Fonagy & Luyten, 2016; Green J. G. et al., 2010; Hulvershorn & Nurnberger, 2014; Larsson et al., 2014; McMahon et al., 2003; Russell et al., 2014; Wadsworth et al., 2016; Waldman & Lahey, 2013). Since socioeconomic disadvantage (e.g., low SES) also increases

exposure to developmental insults such as infections and malnutrition, risk factors of different kinds will typically occur together rather than in isolation. Moreover, some environmental factors (e.g., prenatal infections, exposure to maternal stress hormones) may increase the risk for F-type disorders by acting both as cues of harshness/unpredictability and as risk factors for harmful dysfunctions; internal cues of somatic damage may also contribute to the development of faster life history strategies by feeding into mechanisms of facultative calibration.

While environmental stressors can increase the risk for fast spectrum disorders, their influence should not be overestimated. Life history strategies are at least moderately heritable and may be partly maintained by balancing selection; accordingly, some individuals will follow canalized trajectories and develop the full suite of fast life history–related traits—and associated risk for psychopathology—even without being exposed to cues of harshness or unpredictability. Chronic stress is likely to have a stronger and longer lasting impact on infants and children who are highly susceptible to the quality of their environment. Another important caveat is that empirical correlations between contextual variables and mental disorders are not necessarily causal, but may be partially or fully explained by confounding factors such as family-level effects and genotype–environment correlations. For example, epidemiological studies that control for relatedness patterns indicate that the associations between antisocial behavior, schizophrenia, and neighborhood deprivation are driven to a considerable extent by self-selection, as families with a higher shared risk for criminality and/or psychosis also tend to live in more disadvantaged neighborhoods (Sariaslan et al., 2013, 2015).

Extending this line of reasoning, a life history perspective suggests that some associations between putative risk factors and psychopathological outcomes may arise from shared genetic effects on the strategies of parents and offspring. For example, low birth weight reliably predicts earlier sexual maturation (Hochberg, 2008, 2010; Nettle et al., 2013) as well as increased risk for F-type disorders including antisocial/conduct disorders and schizophrenia. In the standard view, low birth weight is an indicator of prenatal stress and poor nutrition, which in turn increase the risk of pathological dysregulation in the child. However, low birth weight can also be interpreted as an outcome of reduced energetic and metabolic investment by the mother; if mothers with faster strategies curtail parental investment in the growing fetus, they will tend to have smaller babies *and* transmit them heritable factors that predispose to earlier maturation and F-type disorders. Associations between birth weight, early maturation, and fast spectrum psychopathology may thus be explained—at least in part—by the effect of shared genetic and/or epigenetic variants. Conflicts about fetal nutrition between maternally and paternally expressed genes may further exacerbate this pattern (see Chapters 8–10).

Paternal age is another potential risk factor liable to be confounded with adaptive life history variation. Because sperm production involves frequent and

continual cell divisions, the number of de novo mutations in sperm increases steadily as men get older. For this reason, paternal age is often used as an indicator of mutation load in the offspring and is a robust risk factor for several psychiatric conditions, from autism to schizophrenia and BDs (D'Onofrio et al., 2014; Francioli et al., 2015; Malaspina et al., 2015). However, men with faster strategies are also likely to become fathers earlier in life (e.g., Moffitt & Caspi, 2005), suggesting that *low* paternal age should be a predictor of F-type disorders. From this vantage point, the overall association between paternal age and fast spectrum psychopathology is the sum of two contrasting effects: a risk-increasing effect of older age mediated by mutation load and a risk-increasing effect of younger age mediated by life history strategy. Consistent with this prediction, epidemiological data show a curvilinear relation between age and risk: in addition to older fathers, younger fathers (and mothers) show increased rates of mental disorders in their offspring—sometimes even more so, as in the case of ADHD and SSDs. Restricting the analysis to paternal siblings (which automatically controls for variation in the father's life history strategy) shows a linear increase of risk with advancing paternal age, which can be interpreted as the "purified" effect of mutation load (Chudal et al., 2015; D'Onofrio et al., 2014; Merikangas et al., 2017).

SLOW SPECTRUM DISORDERS

Slow spectrum disorders arise in connection with slow life history strategies and the corresponding phenotypic traits. The idea that not only fast strategies, but also slow strategies may set the stage for mental disorders is a recent development in evolutionary psychopathology (Del Giudice, 2014*a*; Del Giudice et al., 2010). In the current version of the FSD model, the slow spectrum of psychopathology comprises the following conditions:

- A high-functioning subtype of autism spectrum disorder, typically associated with normal or high intelligence (S-ASD).
- A low-frequency, slow spectrum subtype of bipolar disorders (S-BDs).
- A low-frequency, slow spectrum subtype of ADHD (S-ADHD) that overlaps with S-ASD.
- Personality disorders marked by high conscientiousness and/or agreeableness, including obsessive-compulsive personality disorder (OCPD).
- A slow spectrum subtype of obsessive-compulsive disorder (S-OCD) in which symptoms are primarily motivated by feelings of incompleteness/imperfection.
- A slow spectrum subtype of eating disorders (S-EDs), with two distinct personality variants characterized by high versus low levels of neuroticism.

Within the slow spectrum, S-ASD and S-ADHD match the features of the skilled/provisioning profile, whereas the low-neuroticism variant of S-EDs is linked to the prosocial/caregiving profile (Figure 6.2). Consistent with the idea that life history profiles are not rigid categories but extremes of a distribution of traits, all S-type disorders show a substantial amount of comorbidity with one another.

Slow Spectrum Markers

Table 6.2 shows a working list of slow spectrum markers. The main correlates of S-type disorders are high conscientiousness and honesty-humility, low impulsivity and risk-taking, increased long-term mating and romantic attachment stability, high levels of disgust sensitivity (especially in the sexual and moral domains), delayed and restricted sexuality, and slower sexual maturation (especially in females). Agreeableness is high in the prosocial/caregiving profile, but moderate to low in the skilled/provisioning profile because of reduced empathy and social compliance. Other correlates of the skilled/provisioning profile are low openness/imagination, enhanced visuospatial abilities, and a bias toward mechanistic reasoning. Tentative neurobiological correlates of the slow spectrum include increased serotonergic activity, (typically) downregulated mesolimbic

Table 6.2 Markers of Slow Spectrum Disorders (S-Type)

S-type markers	**Prosocial/caregiving profile**	**Skilled/provisioning profile**
Personality factors	High agreeableness	Moderate/low agreeableness (empathy, compliance)
	High conscientiousness	High conscientiousness
	High honesty-humility	High honesty-humility
		Low openness (imagination/aesthetics)
Motivation	Delayed sexuality	Delayed sexuality
	Restricted sociosexuality	Restricted sociosexuality
	Stable romantic attachments	Stable romantic attachments
	High long-term mating orientation	High long-term mating orientation
	High disgust sensitivity (especially sexual/moral)	High disgust sensitivity (especially sexual/moral)
Decision-making, self-regulation	Low impulsivity	Low impulsivity
	Low risk-taking and sensation seeking	Low risk-taking and sensation seeking
Cognitive ability		Low mentalistic cognition
		High mechanistic cognition
		Visuospatial > verbal ability
Sexual maturation	Late, slow maturation (especially females)	Late, slow maturation (especially females)

dopamine, low androgen levels, and heightened DLPFC activity. Moreover, the skilled-provisioning profile should be associated with reduced oxytocinergic function, dampened activity of the default mode network, and a pattern of sex hormones capable of accounting for the combination of male-typical traits such as high visuospatial skills and female-typical traits such as restricted sociosexuality (Chapter 4).

Sex Differences

The skilled/provisioning profile is a male-typical variant of slow strategy; accordingly, the risk for disorders that fit this profile (S-ASD and S-ADHD) is substantially higher in males, with sex rations of at least 3:1 and up to 10:1 (Agnew-Blais & Seidman, 2014; Baxter et al., 2015). All else being equal, more females than males should display a prosocial/caregiving profile; at the same time, males are generally more vulnerable to disorders and dysfunctions. The combination of these effects should result in a balanced or female-biased prevalence for conditions associated with this profile. In the current version of the FSD taxonomy, the only disorder with a clear link to the prosocial/caregiving profile is a variant of S-type eating disorders (S-EDs); as noted earlier, the distribution of EDs is strongly female-biased at both ends of the continuum. OCPD is not strongly associated with a particular strategic profile; some studies report similar OCPD rates in males and females, while others find a slightly higher prevalence in men (de Reus & Emmelkamp, 2012; Grant et al., 2004; Samuels & Costa, 2012). Finally, the slow spectrum subtype of OCD is associated with both OCPD and ASD and is more common in males, though not as male-biased as autism (Chapter 20).

Developmental Patterns

The developmental trajectory of S-type disorders is harder to predict than that of their F-type counterparts. Slow strategies are less intensely focused on competition and mating and allocate the organism's resources over a longer time frame with substantial investment in parenting and/or nepotistic effort. All else being equal, then, the risk for this type of disorders should be more broadly distributed across the life span. This is certainly the case of OCPD, which peaks in the third decade of life and becomes more prevalent during the fourth and fifth decades (Albert et al., 2004; Diedrich & Volderholzer, 2015; Grant et al., 2004; Samuels & Costa, 2012). The slow spectrum subtype of OCD also has a wide risk window that extends from childhood throughout adulthood (Chapter 20).

This general prediction can be refined for disorders linked to the skilled/provisioning profile. An important aspect of this profile is a protracted phase of reproductive immaturity devoted to the accumulation of somatic capital through learning, exploration, and skills practice. Accordingly, disorders associated with this profile should involve delays or interruptions in the acquisition of age-typical

skills, especially those relating to mating and social competition. The exact timing of onset is then going to depend on the specific mechanisms involved in the disorder. For example, mentalizing abilities undergo a major developmental transition in early childhood; this is when autism is typically diagnosed, although developmental delays in language and communication may appear already in the first 1–2 years (Bernier & Dawson, 2016). A counterintuitive prediction is that, all else being equal, disorders associated with the skilled/provisioning profile should tend to become *less* severe as mating competition gets more intense, in marked contrast with mating-related conditions within the fast spectrum. For slow strategists, the peak of mating effort should also begin somewhat later, often closer to adulthood than adolescence. The available evidence indicates that autism symptoms are stable across childhood but tend to improve in adulthood, with recovery rates estimated between 3% and 25% (Helt et al., 2008; Seltzer et al., 2004; Wu et al., 2016).

Risk Factors

Because slow life history strategies are more likely to develop in safe, predictable contexts, there should be a general tendency for S-type disorders to be associated with favorable family conditions, low levels of early stress, and high SES—all aspects of the environment that are usually regarded as *protective* factors against psychopathology. These counterintuitive associations should be more apparent for conditions that reflect alternative strategies rather than dysfunctional processes. Within the slow spectrum, OCPD is the best candidate for a potentially adaptive strategy (Chapter 12). Consistent with predictions, the risk of OCPD increases at higher levels of income and education and is associated with the lowest rates of exposure to traumatic events, neglect, and abuse among the personality disorders (Battle et al., 2004; Coid, 1999; Grant et al., 2004; Torgersen et al., 2001; Walsh et al., 2013; Yen et al., 2002). As in F-type disorders, the correlation between environmental stress and risk for psychopathology should be attenuated by genetic variation in life history strategies and individual differences in susceptibility.

The picture becomes more complicated for S-type disorders that involve harmful dysfunctions. On the one hand, socioeconomic disadvantage and chronic stress influence life history development by acting as cues of harshness/unpredictability; on the other hand, they can be expected to increase the risk of dysfunction regardless of the individual's position on the fast–slow continuum. In total, high SES should work as a risk factor for some aspects of slow spectrum psychopathology and as a protective factor for others; the net effect is going to depend on the relative weight of dysfunctions in the etiology of any given disorder. Some diagnostic categories comprise a spectrum of conditions, from functional and possibly adaptive variants to clearly dysfunctional ones, as well as different life history profiles. In those cases, epidemiological patterns with respect to SES may become quite complex. This is the case of ASD, which spans a broad range of severity—from mild symptoms at the boundary of normal personality to extremely dysfunctional

cases with major intellectual disability. Autism is associated with many genetic and environmental risk factors, from deleterious mutations and advanced paternal age to prenatal infections and perinatal complications (Bernier & Dawson, 2016; Mandy & Lai, 2016). The prevalence of severe autism with intellectual disability—which, as I argue in Chapter 10, is a cluster of dysfunctional conditions outside the FSD classification—is either unrelated to SES or more common at lower socioeconomic levels. In contrast, several studies (particularly in the United States) have found associations between higher SES and risk for autism *without* intellectual disability, which comprises the bulk of the slow spectrum subtype of ASD. In part, this effect is explained by high-income, educated parents having better access to healthcare; however, there are indications that differential access does not fully account for the epidemiological findings (e.g., DiGuiseppi et al., 2016; Durkin et al., 2010; Hill et al., 2014; Leonard et al., 2011; Rai et al., 2012; Wu et al., 2016). The key point is that the pattern of socioeconomic risk for ASD is complex and nuanced, in contrast with the consistent negative association with SES found in F-type disorders.

As noted in a previous section, the effect of paternal age on mutation load is confounded with its functional role as a life history–related trait. In the fast spectrum, these two effects go in opposite directions; in the slow spectrum, they tend to reinforce one another. Unsurprisingly, higher paternal age is consistently associated with increased risk of ASD; a more interesting finding is that advancing *maternal* age is an equally strong predictor of autism spectrum conditions in the offspring (Idring et al., 2014; Lee & McGrath, 2015; Leonard et al., 2011; Sandin et al., 2013). From a purely genetic standpoint, this is a puzzling finding. The eggs of older mothers are more likely to carry chromosomal abnormalities, and there is recent evidence that de novo mutations increase slightly with maternal age (Wong et al., 2016). However, the overall effect on mutation load is much smaller than in the case of sperm; moreover, this explanation does not fit with the small or even negative correlations observed between maternal age and F-type disorders such as schizophrenia. Of course, the link between maternal age and ASD is easily explained from a life history perspective if women who engage in slower strategies tend to mature later, delay childbirth, *and* transmit risk factors for S-type disorders to their offspring.

DEFENSE ACTIVATION DISORDERS

Defense activation disorders are conditions that arise primarily from the intense and/or prolonged activation of evolved defensive mechanisms. There are many possible reasons for exaggerated defense activation, including undesirable but adaptive responses, neurobiological dysfunctions, evolutionary and developmental mismatches, and maladaptive learning. In previous versions of the life history framework, depression and generalized anxiety disorder (GAD) were described as nonspecific conditions that could arise at both ends of the fast–slow continuum (Del Giudice 2014*a*, 2014*b*; 2016*c*, 2016*d*). The FSD model builds on

this idea by adding a distinct category for disorders of defense activation (Figure 6.2). These disorders share common predisposing traits and form a broad comorbidity cluster that connects with both F-type and S-type disorders. In the current version of the FSD model, D-type disorders comprise:

- Depressive disorders, including major depressive disorder (MDD) and persistent depressive disorder (PDD) or *dysthymia*.
- Generalized anxiety disorder (GAD).
- Posttraumatic stress disorder (PTSD).
- Specific phobias.
- Panic attacks, panic disorder, and agoraphobia.
- Social anxiety disorder (SAD, also known as "social phobia") and the largely overlapping diagnosis of avoidant personality disorder (APD).
- A defense activation subtype of obsessive-compulsive disorder (D-OCD) in which symptoms are primarily motivated by harm prevention.

In total, D-type disorders include most of the conditions classified as internalizing in the transdiagnostic approach; the main exceptions are eating disorders and "interstitial" conditions such as bipolar disorders and BPD, which correlate with multiple transdiagnostic factors (more on this later). In the DSM-5, D-type disorders are split into three main categories, namely "anxiety disorders," "depressive disorders," and "obsessive-compulsive and related disorders." Within the broader defense activation family it is possible to identify two smaller clusters of disorders: a *distress* cluster marked by anxious or depressed affect (depression and GAD) and a *fear* cluster in which the primary emotional experience is one of fear (panic disorder, most specific phobias, SAD, and APD). These labels were introduced by Watson (2005) and are often employed in the transdiagnostic approach to describe the lower level structure of the internalizing spectrum (e.g., Carragher et al., 2015; Kotov et al., 2017). The distinction between distress and fear disorders is useful but should not be employed too rigidly: anxiety-related symptoms such as avoidance and worry in response to a potential threat (e.g., public speaking in SAD) can easily coexist with intense fear or even panic when the threat materializes (Boyer & Bergstrom, 2011). While some authors include PTSD in the distress cluster, its symptoms reflect a mixture of distress and fear, suggesting that an intermediate classification may be more appropriate (Watson, 2005). Also, the central emotion in some phobias seems to be disgust rather than fear (Chapter 17).

The classification of a subtype of OCD as a D-type disorder is a major change from earlier versions of the taxonomy and deserves special comment. In the original description of the framework (which did not include defense activation disorders), I suggested that OCD could be subdivided into fast and slow spectrum subtypes based on the particular kind of symptoms shown by patients (specifically *reactive* vs. *autogenous* obsessions; see Chapter 20). However, the distinction turned out to be less clear and functionally meaningful than anticipated (Del Giudice, 2014*b*; Del Giudice et al., 2014*b*). In most patients, the symptoms of

OCD are motivated by the desire to prevent harm and reflect the activation of the security and/or disgust systems. In the updated FSD model, this variant of OCD is an obvious candidate for inclusion in the defense activation category (Berle & Phillips, 2006; Boyer & Liénard, 2006; Szechtman & Woody, 2004; Woody & Szechtman, 2011). Like other defense-related conditions, D-OCD occurs in conjunction with both F-type and S-type disorders, possibly with somewhat different constellations of obsessive symptoms (Chapter 20; Del Giudice et al., 2014*b*).

Markers of Defense Activation Disorders

Elevated neuroticism is the core personality correlate of D-type disorders (Hong & Cheung, 2015; Watson & Naragon-Gainey, 2014; Watson & Stasik, 2014). Neuroticism describes the tendency to experience frequent, intense negative emotions in response to challenging events, coupled with a pervasive appraisal of the world as threatening or beyond one's coping abilities. People high in neuroticism tend to be intolerant of uncertainty, pessimistic, sensitive to negative evaluations, and prone to rumination and worry. Evolutionary and neurobiological models of personality converge on the idea that neuroticism reflects heightened sensitivity of defensive psychological mechanisms designed to deal with multiple types of social and nonsocial threats (Allen & DeYoung, 2017; Barlow, Ellard et al., 2014; Denissen & Penke, 2008; DeYoung, 2015; Nettle, 2011*a*; Patrick & Bernat, 2006). These mechanisms include the behavioral inhibition system (BIS) and the security, fear, and disgust systems, but also components of other motivational systems (e.g., status, affiliation) that mediate the individual's response to potentially catastrophic social threats such as humiliation, rejection, and exclusion. From the standpoint of the smoke detector principle, neuroticism indicates a readiness to activate defenses so as to avoid false negatives—even at the cost of many false alarms and an increased risk of maladaptive outcomes (Bateson et al., 2011). Consistent with this interpretation, all D-type disorders share a background of elevated neuroticism; this pattern is so robust that the internalizing factor of the transdiagnostic model is almost perfectly correlated with the neuroticism factor of the Big Five (Barlow, Ellard et al., 2014; Griffith et al., 2010; Hengartner et al., 2016; Rodriguez-Seijas et al., 2015).

Neurobiological Correlates

In the brain, defensive responses are mediated by a complex network of anatomical structures and neurochemical pathways. The amygdala is often singled out as a neural "gateway" in the regulation of vigilance and responsivity to threats. Individual differences in neuroticism have been shown to correlate with the volume of the amygdala; there is also some evidence for systematic differences in amygdala reactivity, although the findings are not as consistent (Allen & DeYoung, 2017; Mincic, 2015). Other regions showing systematic anatomical and functional associations with neuroticism are the hippocampus and parahippocampal gyrus, the striatum, the dorsal prefrontal cortex (DPFC), the medial prefrontal

cortex (MPFC), and the anterior cingulate cortex (ACC) (Bjørnebekk et al., 2013; Holmes et al., 2012; Servaas et al., 2013).

At the molecular level, glutamate and GABA exert important regulatory influences on anxiety, fear, and approach/avoidance motivation. Many defense activation symptoms—such as anxiety, depression, and panic—are associated with a physiological profile of reduced GABAergic activity as well as alterations in glutamatergic transmission. Moreover, one of the few genomic regions that have shown reliable associations with neuroticism includes the gene for a glutamate receptor (Kalueff & Nutt, 2007; Lener et al., 2016; Möhler, 2012; Sanacora et al., 2012; Smith, Anderson et al., 2015). Serotonin plays multiple and contrasting roles in the modulation of defenses as it inhibits fear but tends to *potentiate* the BIS, the security system, and the disgust system. Serotonergic activity is thus unlikely to show global correlations with neuroticism—and indeed, the evidence in this regard is mixed and inconsistent (Ormel et al., 2013). This is not to say that serotonin is not involved in the etiology of D-type disorders; however, its role is probably complex and nuanced, in contrast with the popular idea that depression and anxiety are caused by low serotonin levels. As I discuss in Chapter 14, a life history approach may help make sense of some contradictory findings concerning the role of serotonin in depression.

The stress response system is a key neurobiological node in the regulation of defensive processes. It mediates fight-flight-freeze responses (mainly through the sympathetic and parasympathetic nervous system), mobilizes energetic and cognitive resources in the face of sustained threats (mainly through the HPA axis), and contributes to the physiology of disgust (through parasympathetic activation). Despite its manifold contributions to defensive processes, it is important to remember that the stress response system is not a specialized defensive mechanism; rather, its main function is to coordinate physiological and behavioral allocations across a wide range of domains and in response to both positive and negative events. Defensive mechanisms recruit this system precisely because of its ability to rapidly and effectively regulate the state of the whole organism. There is evidence that both norepinephrine levels and HPA reactivity are implicated in negative emotionality, anxiety, and depression; also, variation in the gene for the CRH receptor 1 is reliably associated with neuroticism (Allen & DeYoung, 2017; Barlow, Sauer-Zavala et al., 2014; Charney & Nestler, 2009; Smith, Escott-Price et al., 2015). However, findings in this area are often contradictory and indicate that neuroticism and defense activation can be associated with both hyper- and hypoactivity of the HPA axis (Charney & Nestler, 2009; Ormel et al., 2013). A plausible solution to this paradox lies in the temporal dynamics of the HPA response: periods of sustained activation are usually followed by phases of hyporeactivity and low cortisol production, to promote recovery and/or to protect the organism from the severe side effects of prolonged exposure to glucocorticoids (Barlow, Sauer-Zavala et al., 2014; Miller, Chen et al., 2007). For this reason, the combination of intense stress and high HPA reactivity does not necessarily translate into a stable pattern of elevated cortisol. Instead, the expected trajectory is cyclical, with fluctuations between high and low HPA activity and cortisol levels—especially in chronically stressful contexts. In the language of the ACM model, it is important to distinguish between the temporary hypoactivity

that follows response peaks in vigilant or sensitive patterns and the persistent lack of reactivity that characterizes stable unemotional patterns (Figure 4.2).

Sex Differences

With respect to sex differences, the general prediction is that D-type disorders should arise more often in females than in males. This is consistent with the higher level of neuroticism observed in women, which indicates a lower threshold for the activation of defenses and a generalized tendency toward risk avoidance. In line with this prediction, females are more at risk for all the disorders in this category. The largest sex differences are found in depression, GAD, PTSD, panic disorder, agoraphobia, and specific phobias (with sex ratios raging from 1.5:1 to 3:1), the smallest ones in OCD and SAD. While epidemiological studies in the general population consistently find higher female risk for OCD, clinical samples tend to be male-biased, suggesting that severe cases of this disorder may occur more often in males (Arch et al., 2013; Calamari et al., 2011; Fontenelle & Hasler, 2008; McLean & Anderson, 2009; McLean et al., 2011; Tolin & Foa, 2006; Valentiner et al., 2014).

The prediction of higher female risk for D-type disorders can be refined in at least two ways. First, males and females differ in their vulnerability to different kinds of threats; as a consequence, they may show somewhat different patterns of symptoms and risk factors even when they develop the same disorder. For example, loss of status has a stronger impact on depression in males, whereas females are more sensitive to social rejection and loss of social support; obsessions with contamination themes (linked to pathogen disgust) are more common in females than in males; and so on (de Mathis et al., 2011; Kendler et al., 2005; Martel, 2013; Nolen-Hoeksema & Hilt, 2009). Second, sex differences in defense activation should be small in infancy and early childhood, when boys and girls face the same threats and share essentially the same vulnerabilities. Starting from middle childhood, however, boys and girls begin to take part in different social worlds with specific sets of challenges; moreover, sex differences in muscle mass and strength, bone density, and adiposity become more pronounced, giving boys a definite advantage in dealing with physical danger (Del Giudice et al., 2009; Wells, 2007). These trends culminate in adolescence and young adulthood, which is when D-type disorders are expected to show the largest and most robust sex differences. As mating competition becomes more intense, males engage in more physical risk-taking and aggression; in contrast, sexually mature females become especially vulnerable to injuries and diseases that may compromise their fecundity or interfere with pregnancy and nursing. They are also physically weaker and thus less able to defend themselves from assaults and other threats. Consistent with this prediction, young boys and girls show similar levels of neuroticism and negative emotionality; sex differences in these traits appear in middle childhood, peak in adolescence and young adulthood, and decline starting from the third decade of life (Else-Quest et al., 2006; Soto et al., 2011).

The epidemiology of defense activation clearly reflects these developmental trends—for example, sex differences for strongly female-biased disorders such as PTSD and depression are either absent or attenuated during childhood (Costello et al., 2003; Nolen-Hoeksema & Hilt, 2009; Tolin & Foa, 2006). Note that many studies follow standard conventions and define "childhood" as the first 12 years of life, a window that includes middle childhood and the initial phase of puberty; thus, sex differences in early childhood are probably even smaller than the existing data indicate.

Developmental Patterns

The timing of onset of D-type disorders should mirror the development of the defensive mechanisms involved in each particular condition. In turn, the developmental trajectory of psychological defenses tracks the rise and fall of different kinds of threats over the life cycle, as well as the shifting balance between the costs and benefits of defense activation. Many social challenges related to status, mating, and affiliation become salient in middle childhood and even more so after puberty. Accordingly, conditions such as depression and SAD are rare in early childhood, become more common after puberty, and peak either in adolescence or young adulthood (Costello et al., 2011; Ledley et al., 2013; Nolen-Hoeksema & Hilt, 2009). The prevalence of panic disorder and agoraphobia also increases through adolescence and adulthood, with a secondary rise after age 40. These female-biased disorders are rooted in perceptions of physical and social vulnerability, and their developmental pattern seems to track the trajectory of the *actual* risk for physical diseases and other potential dangers (e.g., assault by strangers). Indeed, both panic and agoraphobia show sizable correlations with poor physical health, from cardiovascular and gastrointestinal conditions to diabetes and respiratory illnesses. In contrast, specific phobias—especially for animals, blood, and injections—have a distinct onset peak in childhood, which is the phase of maximal vulnerability to physical dangers of this kind. Childhood phobias tend to improve or remit as children grow up, although people who suffered from early phobias remain at higher risk for other D-type disorders later on (American Psychiatric Association, 2013; Arch et al., 2013; Beesdo-Baum & Knappe, 2014; Costello et al., 2011; Goodwin et al., 2014; Valentiner et al., 2014).

The developmental features of OCD are especially interesting when considered from a functional perspective. Overall, the risk of OCD peaks in young adulthood; however, more than one-third of cases develop much earlier, before 10–12 years of age (Calamari et al., 2011; Nakatani et al., 2011; Ruscio et al., 2010). As I argue in Chapter 20, early onset is particularly common in the slow subtype of OCD (S-OCD), which is marked by a high frequency of symptoms with themes of symmetry, ordering, repeating, hoarding, and cleaning. All these behaviors are fairly common in children; in nonclinical samples, they increase through early childhood and peak at the beginning of middle childhood (Boyer & Liénard, 2006; Laing et al., 2009). Disgust also plays a prominent role in OCD, and the disgust system only becomes fully mature in middle childhood, when children

begin to eat the same food as adults (thanks to the eruption of permanent teeth) and become able to forage on their own. These new capabilities expose children to a whole new range of pathogen-related threats and emerge in synchrony with a dramatic increase in disgust sensitivity—which in turn may contribute to trigger the onset of OCD symptoms (Berle & Phillips, 2006; Del Giudice, 2014*c*; Rozin, 1990). In addition to disgust, middle childhood witnesses the rapid maturation of other psychological mechanisms, including executive functions and the acquisition system (collecting and hoarding behaviors are very common at this age). These developmental changes contribute to explain the emergence of benign compulsive-like behaviors in most children and the initial peak in the onset of OCD (Chapter 20).

A generalization that is often made in the developmental literature is that internalizing disorders show a first peak in adolescence, in contrast with the typical childhood onset of externalizing disorders (e.g., Martel, 2013). While this is clearly the case for depression and GAD, other internalizing disorders fail to conform to this simple pattern. Most specific phobias and a substantial proportion of OCD and SAD cases develop in childhood, while the prevalence of agoraphobia and panic disorder keeps increasing through adulthood with no adolescent peak. Reframing (most) internalizing conditions as disorders of defense activation encourages a richer and more sophisticated understanding of their developmental features, and helps avoid rigid generalizations by calling attention to the changing nature of threats and vulnerabilities throughout the life course.

Risk Factors

If D-type conditions are rooted in the upregulation of adaptive defensive mechanisms, the risk for this family of disorders should increase in environments that are dangerous, threatening, or particularly challenging. This is consistent with a large literature showing that D-type and F-type disorders share many important risk factors, from low SES and early stress to negative family relations, maltreatment, and trauma (Beesdo-Baum & Knappe, 2014; Furmark, 2002; Goodwin et al., 2014; Groh et al., 2012; Hammen, 2005; Hudson, 2005; Kendler et al., 2004; Madigan et al., 2013; McMahon et al., 2003; Rudenstine, 2014). In addition, experiencing a severe stressor at one point in life seems to amplify the risk that later challenging events will trigger the onset of disorders such as depression and GAD (Hammen, 2005; Kendler et al., 2011). Such "sensitization" or "kindling" effects are entirely consistent with the logic of the smoke detector principle. In Bayesian terms, experiencing adversity should lead people to increase their estimated probability that other threats will occur in the future. (As noted in Chapter 2, such estimates are most likely computed as a unconscious regulatory variables.) When the probability of encountering threats is high, it is optimal to lower the threshold for the activation of defenses (Figure 2.1); in turn, a lower threshold makes it more likely that a defensive mechanism will respond strongly when faced with new stressors or even in the absence of actual threats (false positives). Importantly, many common types of stressors—from accidents

to divorce—do not occur at random but are influenced by the person's own behavior. Thus, fast personality traits such as impulsivity and risk-taking can indirectly increase vulnerability to D-type conditions (e.g., Hamilton et al., 2015; Kendler & Gardner, 2016; Snyder & Hankin, 2016).

While the data show a clear pattern of increased risk for D-type disorders in adverse and stressful environments, there are also some exceptions and caveats to consider. First of all, not all the disorders in this category fit the general pattern; in particular, the prevalence of OCD is largely independent from socioeconomic factors, and some studies have found increased risk at higher levels of SES (Brander et al., 2016; Fontenelle & Hasler, 2008; Goodwin et al., 2014). As I argue in Chapter 20, this may be partly explained by the existence of two distinct subtypes of OCD, a defense activation subtype and a slow spectrum subtype. Second, epidemiological studies have shown a nonlinear relation between stressful life events and depression: prevalence increases steeply at very high levels of stress but flattens out at low levels, so that even people growing up in extremely safe and protected environments suffer a nontrivial risk of depression (Kendler et al., 2004; see also Hudson, 2005). Third, the correlation between insecure attachment in childhood and internalizing disorders (which overlap significantly with D-type disorders) is reliably lower than that with externalizing disorders (about .15 versus .30; Groh et al., 2012). Fourth, symptoms of anxiety, depression, and stress are elevated in the offspring of older mothers, suggesting a functional link with slow life histories (Tearne et al., 2016). Taken together, these data converge on the idea that D-type disorders occur at both ends of the fast–slow continuum. At the same time, harshness and unpredictability are expected to increase the risk of both D-type and F-type disorders and amplify their observed comorbidity.

In contrast with the robust effects of adversity and early stress, mutations and developmental disturbances do not seem to have a major influence on the risk for D-type disorders (Doherty & Owen, 2014). The few available data suggest a somewhat elevated risk of being diagnosed with depression in the offspring of both younger and older fathers; however, the severity of depressive and anxious symptoms seems to be largely unrelated to paternal age. The data on OCD are inconclusive, as OCD risk has been associated with advancing paternal age in one study and advancing maternal age in another (Buizer-Voskamp et al., 2011; Steinhausen et al., 2013; Tearne et al., 2016; Wu, Liu et al., 2012). The relatively minor role played by this type of risk factors suggests that, as a rule, D-type disorders arise from the expression of functional defensive mechanisms rather than harmful dysfunctions. It does not follow that these conditions necessarily represent adaptive responses. Even properly functioning defenses are subject to activation errors, evolutionary/developmental mismatches, and maladaptive learning; moreover, mechanisms that are initially functional may become damaged or dysregulated following periods of chronic hyperactivation. In other words, conditions that start as adaptive response may sometimes morph into dysfunctions along the way. These complications make it especially difficult to adjudicate between alternative evolutionary explanations of depression and other defense-related conditions (Chapters 14–20).

FURTHER CONSIDERATIONS ON THE FSD MODEL

Curvilinear Effects of Adversity

An interesting feature of the FSD model is its ability to explain some puzzling empirical findings on the relation between exposure to adversity and psychological outcomes such as distress, well-being, and reactivity to stress and pain. While subjective distress and psychological disturbances predictably increase at high levels of adversity, they are also moderately elevated in people exposed to very *low* levels of adversity, giving rise to a J-shaped curve (Figure 6.4). Other data show that the probability of attempting suicide increases in people who report many stressful life events, but also in those who report very few (McFeeters et al., 2015; Seery, 2011; Seery et al., 2010). These findings are typically taken to mean that experiencing moderate levels of stress and adversity builds up resilience, promotes effective coping, and generally "inoculates" people against the impact of future stressors.

The FSD model suggests a deeper interpretation of the same findings based on life history concepts. As illustrated in Figure 6.4, higher levels of adversity favor the development of faster strategies and progressively increase the risk of F-type as well as D-type disorders (genotype–environment correlations may also contribute to this pattern). While low levels of adversity reduce the risk of

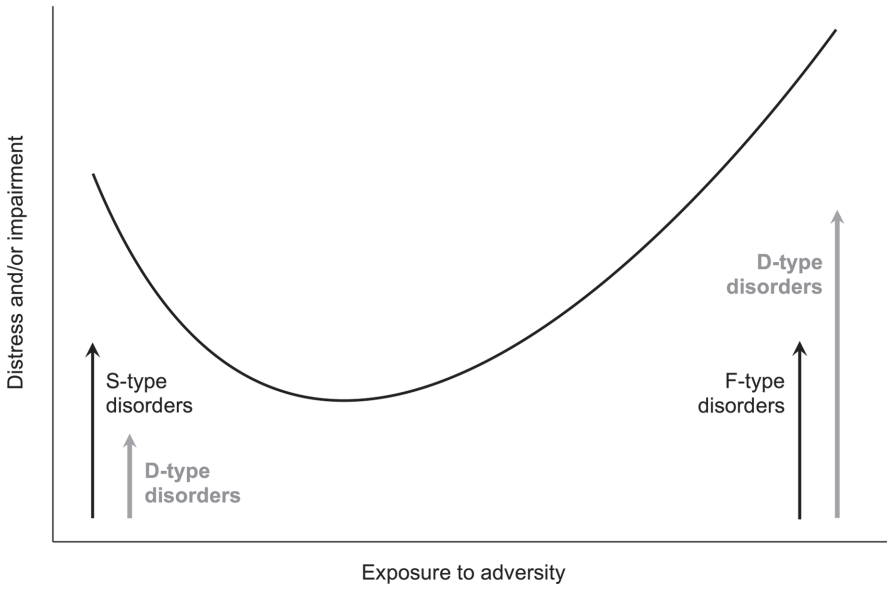

Figure 6.4. The FSD model explains the curvilinear effects of adversity on psychological distress. High levels of adversity tend to increase the risk of fast spectrum and defense activation disorders; low levels of adversity tend to increase the risk of slow spectrum disorders and, to a lesser degree, defense activation disorders. Note that both life history strategies and individual differences in defense activation are substantially heritable, and only partly influenced by contextual factors such as stress and adversity.

F-type disorders, they tend to entrain slower strategies and thus increase the risk of both S-type and D-type disorders; still, the overall risk for defense activation disorders in safe, protected environments remains lower than in severely stressful contexts. The combined result of these effects (represented as upward pointing arrows in Figure 6.4) is a J-shaped curve linking adversity to measures of distress, well-being, and psychiatric symptoms. This interpretation is supported by studies showing that both high and low exposure to adversity predict stronger physiological reactions to laboratory stressors, heightened pain perception, and more posttraumatic symptoms following distressing events such as the 9/11 attack (Seery, 2011; Seery et al., 2013). All these variables indicate upregulation of defensive mechanisms, which can be adaptive at both ends of the fast–slow continuum. Also consistent with this interpretation, studies have shown an inverted J-shaped relation between conscientiousness and well-being. Specifically, well-being increases with higher conscientiousness, but only up to a point; as one approaches the upper end of the scale, extreme conscientiousness morphs into "pathological" obsessive-compulsive personality and well-being drops again, though not as steeply (Carter et al., 2016).

The Role of General Intelligence

The emphasis placed by the FSD model on strategic variation in motivation and self-regulation should not obscure the role of cognitive ability in the origin of mental disorders. Some aspects of cognitive functioning do appear in the FSD model as life history markers; in particular, the seductive/creative and skilled/provisioning profiles are partially defined by different patterns of mentalistic/mechanistic skills and cognitive ability factors (verbal vs. perceptual/rotation). However, the chief dimension of variation in cognitive ability—general intelligence or g—is largely unrelated to individual differences in life history strategy and does not have a specific place in the classification model.

This does not mean that variation in g is unimportant for psychopathology—quite the opposite. To begin, low levels of intelligence are associated with high mutation load, which is a risk factor for many mental disorders (Arslan & Penke, 2015*b*; Penke & Jokela, 2016). Even variation in the middle range of g can be expected to affect the risk for psychopathology in a number of ways. In general, people with lower intelligence are less able to take care of their health and less likely to engage in prevention behaviors; as a result they are more exposed to multiple risk factors for mental and physical disorders (Gottfredson, 2004; Wraw et al., 2015). Low intelligence also increases the likelihood of maladaptive learning and may further reduce the viability of antisocial or socially devalued strategies, making it more likely that they will be diagnosed as disorders. For example, cognitive ability may be one of the factors that differentiate incarcerated psychopaths from "successful" ones in business, law, and other professions (Lilienfeld, Watts, & Francis-Smith, 2015; Spironelli et al., 2014). Intelligence may act as a protective factor during the onset of disorders by helping at-risk individuals compensate

for psychological dysfunctions, avoid maladaptive outcomes, or stop dangerous self-reinforcing feedback loops between symptoms before they develop into full-blown conditions. Likewise, cognitive skills may promote recovery once a disorder has developed (e.g., high IQ is the main predictor of eventual remission from autism; Helt et al., 2008; Seltzer et al., 2004). Finally, there is a small but reliable negative correlation between g and neuroticism (Chapter 3), and neuroticism has been associated with smaller brain volume and reduced white matter integrity (Bjørnebekk et al., 2013). Thus, low intelligence may indirectly increase the risk for defense activation disorders. To reinforce this effect, poor performance in laboratory tests of executive functions (which are robustly associated with g) predicts a higher frequency of stressors that depend at least in part on one's behavior, as for example divorce and job loss; in turn, those stressors lead to increased levels of anxiety and depression (Snyder & Hankin, 2016).

All of the preceding suggest that low cognitive ability should work as a generalized risk factor for many kinds of psychiatric conditions. This is precisely what the evidence shows (Caspi et al., 2014; Castellanos-Ryan et al., 2016; Koenen et al., 2009; Neumann et al., 2016; Urfer-Parnas et al., 2010). The association should be especially strong for disorders that involve dysfunctions and/or clearly maladaptive outcomes. A case in point is schizophrenia, which shows remarkably strong associations with low premorbid intelligence. On average, the early IQ of people who will develop schizophrenia is about 0.5 SD lower than that of healthy controls, and declining IQ through adolescence is a powerful risk factor for this disorder (Khandaker et al., 2011; Koenen et al., 2009; MacCabe et al., 2010; Meier, Caspi et al., 2014; Woodberry et al., 2008). Symmetrically, negative associations with intelligence should be attenuated—or even reversed—for conditions that reflect adaptive or potentially adaptive strategies. For example, OCPD and NPD are not associated with low IQ, and there is some evidence that people with NPD tend to have above-average intelligence (Coid, 1999; Hengartner et al., 2014). Manic symptoms correlate with higher IQ, and the risk for bipolar disorders has been found to increase with higher academic performance (Hagenaars et al., 2016; Koenen et al., 2009; MacCabe et al., 2010; Smith, Anderson et al., 2015). The picture for autism is more complicated; while mild forms of ASD grade into the territory of adaptive strategies, the more severe cases—especially those with intellectual disability—are unquestionably dysfunctional. The overall correlation between autistic symptoms and IQ in children is somewhat negative (around −.20); however, genetic risk scores for autism based on common alleles also predict *higher* intelligence and academic success (Clarke et al., 2016; Hagenaars et al., 2016; Hoekstra et al., 2010). Taken together, these findings suggest that rare and de novo mutations (which tend to lower cognitive ability and are not included in genetic risk scores in the studies I just cited) are largely responsible for the dysfunctional aspects of ASD (more on this in Chapter 10).

Intelligence shows a significant degree of assortative mating, with correlations between partners of about .40 (Plomin & Deary, 2015; Vinkhuyzen et al., 2012). Assortative mating for intelligence may contribute to explain findings of partner similarity for some mental disorders. Indeed, the highest levels of similarity

between partners have been documented in autism, schizophrenia, and ADHD (Nordsletten et al., 2016). As I discuss in Part III of the book, all these diagnostic categories include at least one subtype that is robustly associated with low IQ.

Intelligence and Socioeconomic Status

A final point to consider is the robust correlation that exists between g and measures of socioeconomic status. This association does not simply reflect the fact that better education and a stimulating family environment may increase performance on intelligence tests. On the contrary, there is strong evidence that intelligence plays an important *causal* role in determining an individual's eventual occupation and income and that the same genetic factors that raise g also tend to increase SES (see Gottfredson, 2011; Ericsson et al., 2017; Trzaskowski et al., 2014). The main implication for psychopathology is that some well-established associations between low SES and risk for psychiatric disorders are likely to be partly confounded by individual differences in intelligence. For example, the finding that autistic disorders with and without intellectual disability are differentially associated with socioeconomic factors (discussed in an earlier section) is consistent with the idea that genetic risk for intellectual disability is higher in low-SES families. The strong link between socioeconomic disadvantage and risk for ADHD should also be considered in this light, given that ADHD symptoms correlate with lower IQ owing to shared genetic factors (Clarke et al., 2016; Kuntsi et al., 2004).

Comparison with Transdiagnostic Models

The FSD taxonomy offers an evolutionary alternative to transdiagnostic models of the structure of mental disorders. Over the years, the original model based on the internalizing–externalizing distinction has evolved and become more complex. Most authors now include a thought disorder factor for SSDs and other psychotic conditions, and there have been initial attempts made to add a separate factor for the autism spectrum (e.g., Carragher et al., 2016; Kotov et al., 2011; Noordhof et al., 2015). The HiTOP model includes a detachment factor and splits the domain of externalizing symptoms into two overlapping spectra (disinhibition and antagonism; Kotov et al., 2017). Above and beyond these factors or spectra, it is possible to identify a "p factor" that reflects generalized risk for all kinds of mental disorders. The p factor is about 40% heritable, with a major influence of common genetic variants (Carragher et al., 2016; Caspi et al., 2014; Laceulle et al., 2015; Lahey et al., 2015; Neumann et al., 2016; Waldman et al., 2016).

While most conditions can be located within the model's structure with reasonable confidence, some disorders correlate strongly with more than one factor and are said to occupy "interstitial" positions: in particular, BPD and PTSD correlate with both the internalizing and externalizing factors, while BDs correlate with the internalizing and thought disorder factors. Also, OCD has been alternatively classified as an internalizing or thought disorder (Carragher et al., 2015; Caspi et al., 2014; Forbes et al., 2016; Kotov et al., 2017). Despite minor inconsistencies between

different versions of the model, its basic structure has been empirically supported in many large studies. In this section, I compare the FSD and transdiagnostic models in some detail and discuss how the existing data on the structure of psychopathology can be interpreted and explained from a life history perspective.

Internalizing and Externalizing Disorders

In earlier discussions of the life history framework, I argued that the internalizing spectrum is not a fully coherent dimension of psychopathology as it comprises a mixture of functionally divergent conditions (Del Giudice, 2014*a*, 2016*d*). This criticism was based on the original version of the framework, which lacked a separate category for defense activation disorders. With the inclusion of D-type conditions, the FSD model has recovered key aspects of the internalizing spectrum, and its structure has become more similar to that of the standard transdiagnostic model. At the same time, some important differences remain. In the transdiagnostic approach, disorders are assigned to the internalizing category if they show strong empirical correlations with the internalizing factor. In contrast, the classification of D-type conditions relies on the functional criterion of whether the symptoms of a disorder primarily reflect the activation of defensive mechanisms. Thus, eating disorders are classified as internalizing because of their comorbidity with anxiety and depression but do not fit the FSD criteria for D-type conditions. In fact, the inclusion of EDs in the internalizing spectrum is problematic: when symptoms are clustered based on the associated personality profiles, a subtype of EDs turns out to be strongly associated with externalizing behaviors, while another subtype shows very little comorbidity with either depression or anxiety (Chapter 13). Even though interstitial conditions such as BPD and BDs have marked depressive components, from a life history perspective they are better understood as F-type and S-type conditions rather than pure disorders of defense activation. In sum, the internalizing spectrum of transdiagnostic models does include a functionally coherent "core" of defense-related conditions, but instead is rendered heterogeneous by the inclusion of other disorders such as EDs, BDs, and BPD.

Similar considerations apply to the externalizing spectrum. Antisocial and conduct disorders such as ASPD, CD, and ODD clearly form a coherent cluster linked to the antagonistic/exploitative profile. However, the status of ADHD is much less straightforward, even if this condition is routinely classified as externalizing because of its robust associations with CD and ODD (e.g., Castellanos-Ryan et al., 2016; Kotov et al., 2017). As I discuss in Chapter 11, the fast spectrum subtype of ADHD overlaps not just with antisocial conditions but also with schizophrenia and psychosis, whereas the slow spectrum subtype is closer to autism. I also argue ADHD contains a subtype that is mainly linked to low intelligence and has no functional connections with life history strategy (O-ADHD). This complex pattern challenges the classification of ADHD as a simple externalizing disorder. Substance use disorders (SUDs) are also routinely included in the externalizing spectrum (externalizing-disinhibited in the HiTOP) but are not covered in the current version of the FSD model. While there is a strong link between fast-related traits and risk for SUDs (e.g., Quintelier et al., 2013; Richardson et al., 2014, 2016;

Vincke, 2016), the picture is likely more complex: for instance, some personality profiles associated with substance use may be closer to the slow spectrum or more characteristic of D-type disorders such as depression and social anxiety (e.g., Bulley et al., 2016; Del Giudice, 2014*b*; Yeo et al., 2014). More generally, the FSD and transdiagnostic model differ in the structural role assigned to externalizing disorders. According to the transdiagnostic approach, the externalizing spectrum is one of the fundamental dimensions of psychopathology. In the FSD model, externalizing conditions such as ASPD, CD, ODD, and F-ADHD are only one subset within the broader category of fast spectrum disorders, which also includes a number of internalizing and interstitial disorders (BPD, the fast spectrum subtypes of BDs and EDs), thought disorders (SSDs), and other conditions (NPD). Figure 6.5 illustrates the relations between the FSD taxonomy and the main categories of the transdiagnostic model.

With respect to the internalizing–externalizing distinction, an important advantage of the FSD model is that it makes "interstitial" classifications unnecessary. For example, defense activation conditions can occur in association with both S-type and F-type disorders; thus, the fact that BPD is strongly comorbid with depression and anxiety does not create a classification problem. Another advantage of the FSD model is its parsimony: with just two main dimensions of variation (a bipolar fast–slow continuum and a defense activation axis), the model provides an integrated structural account of internalizing and externalizing conditions but also autism, psychosis, and disorders such as NPD and OCPD that still have no clear placement in the transdiagnostic model. However, the real added value of the FSD model lies in the underlying theoretical framework, which brings coherence to the taxonomy and increases its heuristic power. For example, on a purely descriptive level, the D-type cluster of conditions is similar to the internalizing spectrum of the transdiagnostic model. Still, framing the same conditions as disorders of evolved defensive mechanisms affords unique insights into their developmental and comorbidity patterns, risk factors, and other important features.

The p Factor

In the most recent versions of the transdiagnostic model, the p factor captures a dimension of generalized risk that is shared by all kinds of psychopathology. The FSD model does not include such a general factor, although it recognizes that low cognitive ability tends to increase the risk for many (but not all) mental disorders. From the standpoint of the FSD model, the p factor is an artifact that reflects a mixture of three functionally distinct dimensions of variation: risk for fast spectrum psychopathology, risk for defense activation disorders, and low intelligence. These dimensions are not just conceptually distinct but also statistically separable, although they are expected to correlate to some degree—first because stressful conditions increase the risk for both D-type and F-type disorders, and second because low intelligence is weakly associated with both defense activation (neuroticism) and fast life history–related traits (Chapter 4). The p factor remains approximately valid at a descriptive level, as some people *are* at higher risk for a wide range of psychiatric conditions; however, such a general factor fails to capture the

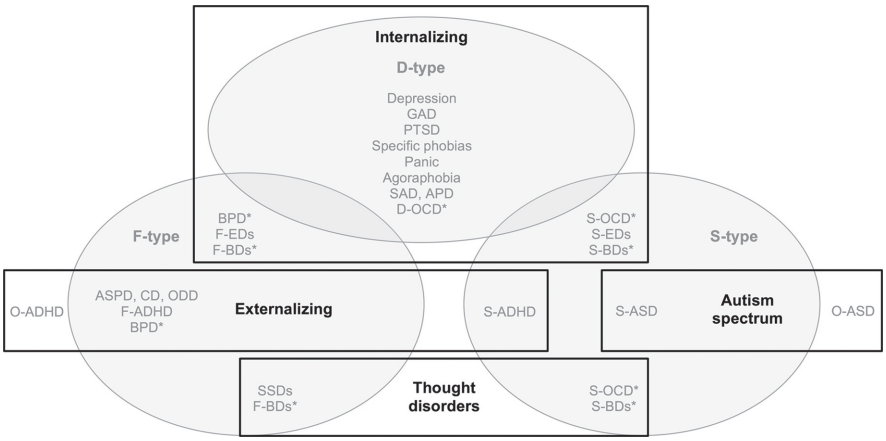

Figure 6.5. Relations between disorder categories in the FSD taxonomy and the main dimensions of transdiagnostic models. The internalizing spectrum includes defense activation (D-type) disorders as well as a number of F-type and S-type conditions, some of which are classified as "interstitial" (e.g., borderline personality disorder, bipolar disorders). The externalizing spectrum includes a number of F-type conditions (antisocial and conduct disorders; borderline personality disorder), in addition to attention-deficit/hyperactivity disorder (which has both F-type and S-type variants, and an O-type variant characterized by low intelligence and unrelated to life history variation). Thought disorders comprise schizophrenia spectrum disorders and bipolar disorders (which have both F-type and S-type variants); OCD is sometimes grouped with thought disorders, other times with internalizing disorders. In the FSD model, autism spectrum disorders include a slow spectrum subtype with normal/high intelligence, and an O-type variant with intellectual disability that is unrelated to life history variation. Asterisks denote conditions that are usually regarded as interstitial or have been classified in different spectra of the transdiagnostic model. ADHD = attention-deficit/hyperactivity disorder. APD = avoidant personality disorder. ASD = autism spectrum disorder. ASPD = antisocial personality disorder. BDs = bipolar disorders. BPD = borderline personality disorder. CD = conduct disorder. EDs = eating disorders. GAD = generalized anxiety disorder. NPD = narcissistic personality disorder. OCD = obsessive-compulsive disorder. OCPD = obsessive-compulsive personality disorder. ODD = oppositional-defiant disorder. PTSD = posttraumatic stress disorder. SAD = social anxiety disorder. SSDs = schizophrenia spectrum disorders.

specific risk for slow spectrum disorders beyond the component associated with low intelligence and/or elevated mutation load. More importantly, the p factor is not a coherent functional construct, emerging as it does from the combination of three separate sources of variation with different biological underpinnings.

This alternative interpretation of the p factor is supported by simulation results and consistent with the available empirical data. In a simulation study, I showed that the p factor, the internalizing factor, and the externalizing factor emerge reliably when standard data analysis methods are applied to virtual symptom scores generated from a continuum of risk for F-type versus S-type disorders (Del Giudice, 2016*d*). Adding a largely or completely unrelated dimension of variation in cognitive ability did not change the results. (Note that the simulation did

not include a separate defense activation category as it was based on a previous version of the framework; however, the simulated model is formally equivalent to one in which the risk for D-type disorders increases equally at both ends of the fast–slow continuum, and thus compatible with the updated FSD taxonomy.) These findings demonstrate that standard analytic approaches are going to recover the "internalizing–externalizing–p factor" triad even when the true underlying structure of psychopathology conforms to the FSD model and does not include a general p factor. This happens for three main reasons. First, standard diagnostic categories do not distinguish between F-type and S-type variants of heterogeneous conditions. Second, studies testing the transdiagnostic model typically do not include slow spectrum conditions such as OCPD and S-ASD. Third, the factor analysis techniques employed in those studies rely on linear correlations between symptoms, which fail to capture the nonlinear associations that arise when the risk for a disorder (e.g., depression) increases at both ends of the fast–slow continuum (for details, see Del Giudice, 2016*d*).

Simulation results are further corroborated by the empirical correlations between the p factor and a range of cognitive, personality, and environmental variables. The available data show that high scores on the p factor are associated with indices of low intelligence and reduced neural integrity, including low IQ scores, neurological abnormalities, and damage to the retinal vasculature; fast spectrum markers such as low conscientiousness, low agreeableness, impulsivity, high time discounting, and reduced self-control; defense activation markers such as high neuroticism and negative emotionality; and high levels of early stress and adversity, which should increase the risk of both F-type and D-type disorders (Carver et al., 2017; Caspi et al., 2014; Castellanos-Ryan et al., 2016; Neumann et al., 2016; Waldman et al., 2016). This pattern of correlations is consistent with the idea that the p factor is a functionally heterogeneous construct, in line with the alternative interpretation advanced here.

ANALYSIS OF COMMON MENTAL DISORDERS

In this chapter, I presented an overview of the FSD model and listed the psychiatric conditions included in the current version of the taxonomy. In Part III of the book (Chapters 7–20) I examine those conditions in more depth. For each disorder (or group of disorders), I begin with an overview of key symptoms and correlates and summarize the evidence on the condition's genetic structure, comorbidity, developmental features, and risk factors. I then review the main evolutionary models of the disorder, examine them in relation to the taxonomy presented in Chapter 5, and present a selection of relevant empirical findings. Finally, I analyze each diagnostic category from the standpoint of the FSD model and consider the possibility that it may comprise functionally distinct subtypes.