Personality Disorders in Children and Adolescents

REBECCA L. SHINER
JENNIFER L. TACKETT

Children and adolescents differ strikingly in their emerging personalities. Already by childhood, youth vary in their typical positive and negative emotions; capacities for self-control and positive relationships with others; feelings of empathy and warmth versus hostility and alienation; and views of themselves, others, and their life experiences. For some youth, their typical personality patterns may begin to cause them difficulties in life; for example, their problematic personality patterns may lead them to experience high levels of distress or serious impairment in their daily lives, particularly in their relationships or self-development. These difficulties may become severe enough for some youth to be diagnosed with a personality disorder (PD); for others, the problems may not reach clinical significance, yet may still bear negative consequences. Both the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV; American Psychiatric Association [APA], 1994) and DSM-5 (APA, 2013) acknowledge that youth may experience PDs warranting treatment. The diagnostic manuals define PDs in terms of problematic cognition, affectivity, interpersonal functioning, and impulse control—all personality differences that vary in children and adolescents and that may become disturbed well before adulthood.

The present chapter surveys the existing state of knowledge about PDs in the first two decades of life. Although there is far less research on PDs in childhood and adolescence than on other early-emerging disor-

ders, the research that does exist has made it clear that personality pathology does occur in childhood and adolescence and poses significant risks for mental health problems and impairment both concurrently and later in life (Cohen, Crawford, Johnson, & Kasen, 2005; De Fruyt & De Clercq, 2012; Freeman & Reinecke, 2007; Hill, 2008; Johnson et al., 2012; Mervielde, De Clercq, De Fruyt, & van Leeuwen, 2005; Shiner, 2007, 2009; Tackett, 2010; Tackett, Balsis, Oltmanns, & Krueger, 2009; Westen & Chang, 2000). This is an exciting time for research on PDs because researchers are finally turning their attention to the early manifestations of personality pathology and to the antecedents of adult PDs (see, e.g., the recent special issues of *Clinical Psychology: Science and Practice*, DeFife & Ritschel, 2013; *Development and Psychopathology*, Cicchetti & Crick, 2009; *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, Biskin & Paris, 2013; *Journal of Personality Disorders*, Tackett & Sharp, 2014; *Journal of Psychopathology and Behavioral Assessment*, Tackett, 2010). Borderline PD (BPD) in youth and the childhood antecedents of antisocial PD (ASPD—e.g., conduct disorder and psychopathy) have received considerable attention, but researchers have begun to explore many of the other PDs and broader personality pathology domains in youth as well.

Throughout the chapter, we adopt a developmental psychopathology perspective on PD (Cicchetti, 1993, 2013). In particular, we draw on two especially impor-

tant tenets of developmental psychopathology. First, the study of normal development is critical for understanding pathological development. The same basic biological, psychological, and contextual processes underlie both normal and abnormal development, and therefore findings and theories from the study of normal development are relevant for explaining the development of psychological disorders. The converse is true as well (i.e., the study of pathological development has the potential to inform research on normal development), but at this point, far more is known about normal than about pathological personality development. Thus we draw on current research on personality development to explain patterns and fill gaps in the literature on PDs in youth.

Second, it is not possible to achieve a complete understanding of psychological disorders without charting the pathways both leading to and following from the development of those disorders (Cicchetti, 1993, 2013). These pathways are often complex (Cicchetti & Rogosch, 1996); different pathways and sets of processes may lead to similar outcomes (known as *equifinality*), and similar origins may yield a broad range of outcomes (known as *multifinality*). The developmental pathways leading to PD in adolescence and adulthood remain poorly understood. At present, there is only one large-scale longitudinal study that has examined the pathways leading to the full set of PDs included in DSM-IV and DSM-5—the Children in the Community study (Cohen, Crawford, et al., 2005). This study began with approximately 800 children ages 1–10 years living in upstate New York and has followed the participants at multiple time points, approximating ages 14, 16, 22, and 33; PDs were assessed at all four time points, as were a variety of other psychiatric disorders, risk factors, and outcomes. This study has provided a wealth of information about the prevalence, development, and course of PDs. Because other large-scale, longitudinal studies of all the PDs are lacking, we sometimes review findings from the literature on PDs in adults to supplement the relatively more scant developmental data.

This chapter proceeds in seven sections. The first section reviews the history of PDs in the DSM system, summarizes the nature of the PD diagnoses in DSM-IV and in DSM-5 Section II, and addresses the still-controversial status of PDs in youth. In its main section (Section II), DSM-5 retains the categorical PD diagnoses in exactly the same form as found in DSM-IV. The second section of the present chapter offers a conceptual framework for describing and explaining the nature of personality pathology in youth; this framework takes into account the ways that personality traits, mental representations, coping strategies, and life narratives may become disturbed in PDs earlier in life. The third section presents several dimensional personality models as diagnostic alternatives to the categorical model of PDs. This section also reviews the trait-based dimensional model for PD offered in Section III of DSM-5; this new section in DSM-5 addresses conditions requiring further research, including a proposed dimensional model of PD. The fourth section of this chapter provides a synopsis of recent research on the epidemiology of PDs, comorbidity among PDs, and links between PDs and other psychiatric disorders (previously called Axis I disorders). The fifth section charts what is known about the stability of early personality pathology and associated life outcomes. The sixth section surveys what is known about the etiology of PDs in general in the first two decades of life and addresses the etiology of specific PDs: Cluster A PDs (paranoid, schizoid, and schizotypal); BPD, ASPD, psychopathy, and narcissism; and the Cluster C PDs (avoidant, dependent, and obsessive–compulsive). The seventh section concludes the chapter with suggestions for future research on PDs in youth.

PDs IN YOUTH IN THE DSM SYSTEMS

A History of PDs from DSM-I to DSM-5

Although PDs have been present in every DSM from the beginning, their formulation has varied over time. The present section reviews the changing structure of PDs across all of the DSM systems, including the decision to retain the DSM-IV PD diagnoses in DSM-5, against the recommendation of the DSM-5 Personality and PDs Work Group. Millon (2012), Oldham (2005), and Widiger (2012) offer more complete reviews of the DSM history, and this history is drawn from their reviews.

DSM-I (APA, 1952) differentiated among three main types of disorders: psychoses, neuroses, and character disorders. The character disorders consisted of "personality disturbances," the name given in the first manual to PDs. Neuroses were seen as being milder and treatable through psychoanalysis, whereas personality disturbances were viewed as patterns that were essentially permanent by early adulthood, and thus difficult (if not impossible) to treat. The manual

recognized that these personality disturbances varied in severity, with some being highly impairing and others being only significantly impairing if patients faced high levels of stress. As for the causes of these disturbances, in DSM-I "personality disorders were generally viewed as deficit conditions reflecting partial developmental arrests or distortions in development secondary to inadequate or pathological early caretaking" (Oldham, 2005, p. 6). DSM-II (APA, 1968) attempted to shift from more theory-based diagnoses to diagnoses describing conditions that could be easily observed and measured; however, many of the specific PDs were retained, and they were still conceptualized as being enduring over time.

DSM-III (APA, 1980) involved a significant overhaul of the entire manual, and it was this manual that had the greatest impact on current conceptualizations of PDs. The first two manuals had presented narrative descriptions of the disorders, whereas DSM-III listed specific criteria to be met for each diagnosis; these criterion lists were added to increase the reliability of the diagnoses. The descriptions of the PDs thus included lists of specific symptoms for each disorder. In addition, DSM-III introduced a multiaxial system, with disorders seen as more episodic placed on Axis I and disorders seen as more enduring placed on Axis II. The Axis II disorders included mental retardation and the PDs. The manual itself suggested that the PDs were placed on Axis II for another reason—to ensure that "consideration is given to the possible presence of disorders that are frequently overlooked when attention is directed to the usually more florid Axis I disorder" (APA, 1980, p. 23).

DSM-III retained several PD diagnoses that had been present in some form in the previous two manuals: paranoid, schizoid, histrionic, passive–aggressive, compulsive, and antisocial. Two previous PD diagnoses were moved to Axis I: intermittent explosive disorder and cyclothymic disorder. In addition, several new PDs were added that are still present in the newest manual: BPD and schizotypal, narcissistic, avoidant, and dependent PDs. Millon (2012) describes the rationale for adding these new PDs in this way: "A major goal of the newly appointed *DSM-III* Task Force was to include as many clinically useful personality syndromes as could be justified. Despite objections from certain quarters, a decision was made to incorporate categories that had not been fully validated by systematic research but nevertheless had much to commend them in terms of their everyday clinical applicability" (p. 11). Another impor-

tant addition to the manual was the cluster system for the PDs, which has been retained in later manuals; this clustering is described more fully in the next section.

It is interesting to note that DSM-III included five childhood disorders that were seen as potential antecedents to adult PDs: avoidant disorder, schizoid disorder, identity disorder, oppositional disorder, and conduct disorder (Widiger, De Clercq, & De Fruyt, 2009). These were described as possible precursors to adult avoidant PD, schizoid PD, BPD, passive–aggressive PD, and ASPD, respectively. This explicit focus on possible childhood precursors of adult PDs was lost in later editions of the DSM because schizoid disorder and identity disorder in childhood were deleted; childhood avoidant disorder was merged with social phobia in DSM-IV; and the adult counterpart to oppositional disorder (passive–aggressive PD) was eliminated. Only ASPD continued to have an explicit childhood antecedent in the form of conduct disorder. Because conduct disorder and its related conditions (e.g., oppositional defiant disorder, childhood aggression) have been widely studied in the intervening years, much more is known about the developmental pathways leading to ASPD than the pathways leading to other PDs.

As hoped, the amount of research and clinical attention devoted to the PDs did increase significantly following the publication of DSM-III. DSM-III-R (APA, 1987) involved relatively few changes to the PDs. Likewise, DSM-IV (APA, 1994) retained almost all of the PD diagnoses and the cluster system of DSM-III; the continuity from DSM-III to DSM-IV was not surprising because DSM-IV was designed to take a conservative stance to making changes to the diagnoses (Frances & Widiger, 2012). Passive–aggressive PD was moved to Appendix B of DSM-IV, and a set of general diagnostic criteria for a PD was added to the chapter on PDs. DSM-IV-TR (APA, 2000) changed only the narrative text, not the diagnostic criteria, but even the changes to the narrative text for PDs were minimal.

The APA considered making major changes to the PD diagnoses for DSM-5. As more research was conducted on the PDs following the publication of DSM-III, DSM-III-R, and DSM-IV, it became clear that there were some significant flaws in the PD diagnostic system; these are described in more detail in this chapter's section on dimensional models. As a result of these concerns about the PD diagnoses, the APA opted to focus the first of a series of international conferences, held in 2004, on psychiatric classification on dimensional models of PDs (Widiger, Simonsen, Sirovatka, & Regi-

er, 2005). The Personality and PDs Work Group for DSM-5 thus undertook the task of revising the PD diagnoses, with an eye toward implementing a dimensional system for these diagnoses; this process is described in detail in Skodol (2012). It is interesting to note that although all of the DSM-5 Work Groups were initially encouraged to consider making substantial changes to the conceptualization and operationalization of the disorders, all of these groups except for the Personality and PDs Work Group eventually took a conservative stance toward revision and focused on making minor modifications instead of sweeping changes (Skodol, 2012; Widiger, 2013).

The Personality and PDs Work Group eventually submitted a final proposal that retained six of the PD diagnoses—ASPD, BPD, and avoidant, narcissistic, obsessive–compulsive, and schizotypal PDs—and proposed new diagnostic criteria for them (Skodol, 2012; Skodol, Bender, et al., 2011). These diagnoses were retained based on some combination of prevalence in community and clinical samples, associated psychosocial impairment, and evidence for the validity and clinical utility of the disorders. The proposal also included a new diagnosis of PD—Trait Specified, which was defined by the presence of significant impairment and specified by each individual's most prominent personality difficulties on a set pathological personality trait dimensions. This model is described more fully in the section on dimensional models.

Ultimately, the APA Board of Trustees rejected the proposal from the Personality and PDs Work Group (APA, 2012; Krueger, 2013). Instead, the board opted to retain the categorical PD classification system presented in DSM-IV and the 10 PD diagnoses in their exact form from DSM-IV. Thus, although the text has been updated in DSM-5, the PD diagnoses in Section II are identical to the ones presented in DSM-IV. The Board of Trustees also voted to eliminate the multiaxial system, so the PDs now appear in Section II, along with all of the other categorical psychiatric disorders. Two PDs are now cross-referenced in other chapters; in each case, the PD has close ties to non-PD disorders. Specifically, schizotypal PD is also listed in the chapter on schizophrenia spectrum and other psychotic disorders, and ASPD is also listed in the chapter on disruptive, impulse-control, and conduct disorders. The proposed pathological personality dimensional system is presented as the "alternative DSM-5 model for PDs" in Section III, "Emerging Measures and Models," which includes measures and diagnoses requiring further study before

potential inclusion in future diagnostic manuals (similar to, e.g., Appendix B in DSM-IV). At this point, the plan is to update DSM-5 as more research is conducted, with future updates being numbered in decimals (e.g., DSM-5.1, DSM-5.2), so it is possible that the alternative model will be moved into Section II if more research substantiates this model.

Several themes stand out in this history of the PDs in the DSM system. First, the PDs have been conceptualized consistently as long-lasting conditions that start at least by early adulthood. The presumed chronic nature of PDs has been part of their conceptualization from DSM-I onward, and it was this nature that was thought to set them apart from more episodic disorders. Second, the PD diagnoses included in the manuals were chosen for inclusion based on experts' clinical experience with "types" of personalities that tend to be accompanied by significant impairment, not based on empirical research on how best to define the nature of personality pathology. Third, some of the current PD diagnoses have been included in similar forms since the original 1952 manual (ASPD and paranoid, schizoid, histrionic, and obsessive–compulsive PDs), and others have been included since 1980 (BPD and schizotypal, narcissistic, avoidant, and dependent PDs). Thus all of the diagnoses have been in use for 30–60 years, and it is not surprising that there would be resistance to removing any of them, regardless of whether there is research supporting their validity. Taken together, it is striking how relatively consistent the PD framework has been throughout the DSM systems; yet, as we review elsewhere in this chapter, newer research has called into question some of the most basic assumptions about the PDs as defined in these systems.

PDs in DSM-IV and DSM-5 Section II

This section describes in more detail the nature of PD diagnoses in DSM-IV and DSM-5. (We focus here on the PD chapter in Section II of DSM-5; we review the Section III alternative DSM-5 model for PDs in this chapter's later section on alternative dimensional models of PDs.) These manuals provide an overarching framework for what constitutes a PD. According to this general framework, PDs consist of deviant patterns of inner experience and behavior in at least two of the following four areas: "(1) cognition (i.e., ways of perceiving and interpreting self, other people, and events); (2) affectivity (i.e., the range, intensity, lability, and appropriateness of emotional response); (3) interpersonal

functioning; (4) impulse control" (APA, 1994, p. 633; APA, 2013, p. 646).

Skodol (2005) has fleshed out what these four areas often include. Cognition typically manifests as disturbances in how patients view themselves and others—for example, overinflated self-views or unduly negative views of the self, profound mistrust or alienation toward others, or tendencies to idealize or devalue others. Cognition also includes deviant thinking about the world, such as expectations for perfectionism or odd, delusional beliefs. Affectivity involves a wide range of disturbances in patients' typical emotions, including disrupted mean levels of emotions (e.g., restricted emotional experience), as well as problems with emotion regulation (e.g., excessively intense and labile emotions). The emotions that are disturbed include the full gamut of human emotions: sadness, anxiety, anger and irritation, joy and pleasure, and love and affection. Difficulties in interpersonal functioning typically involve problems with one or both of the two main dimensions of interpersonal behavior: agency (ranging from dominance and self-assuredness to submission) and communion (ranging from affiliation and warmth to detachment and cold-heartedness) (Pincus & Hopwood, 2012). Finally, several PDs involve problems with impulse control—either deficits in self-control (poor planning, thinking without acting, poor self-regulation of behavior and emotions) or excessive levels of self-restraint and inhibition of healthy impulses.

These deviant personality patterns are further defined by DSM-IV and Section II of DSM-5 in several ways (APA, 1994, pp. 630–631; APA, 2013, pp. 646–647). Consistent with the definition of PDs in all of the DSM systems to date, the patterns must be enduring, inflexible, and pervasive across many contexts in the person's life. The patterns are expected to have started at least by adolescence or early adulthood. The personality patterns must be distressing to the person or must cause impairment in important arenas of daily life, such as social relationships, school, or work. Finally, the pattern must not be better accounted for as a consequence of another disorder, a medical condition, or the use of some substance.

The diagnostic manuals present the PDs as personality "types" made up of combinations of pathological personality tendencies. DSM-IV and DSM-5 outline diagnostic criteria for 10 specific PDs, which are grouped into three clusters: Cluster A, odd or eccentric (paranoid PD, schizoid PD, and schizotypal PD); Cluster B, dramatic, emotional, or erratic (ASPD, BPD, histrionic PD, and narcissistic PD); and Cluster C, anxious or fearful (avoidant PD, dependent PD, and obsessive–compulsive PD) (APA, 1994, pp. 629–630; APA, 2013, p. 646). The essential features of these 10 PD diagnoses are presented in Table 18.1. DSM-5 acknowledges: "It should be noted that this clustering system, although useful in some research and educational situations, has serious limitations and has not been consistently validated" (p. 646).

DSM-IV provided the option of diagnosing PD not otherwise specified (NOS), for those cases in which the general criteria for a PD are met and PD symptoms are present, but in which the person does not fulfill the criteria for any specific PD in the manual. However, DSM-5 has eliminated all NOS diagnoses. Instead, there are two options for Section II diagnoses for patients who exhibit a PD but don't meet criteria for a specific PD: other specified PD (when the clinician wants to note why the patient fails to meet criteria for a specific PD) and unspecified PD (when the clinician does not want to specify why the patient fails to meet such criteria). DSM-5 also offers the option of diagnosing personality change due to another medical condition, for instances in which a patient displays "a persistent personality disturbance that represents a change from the individual's previous characteristic personality pattern" (p. 682) as a result of a neurological or other medical condition.

PDs in Youth in the DSM Systems

Like DSM-IV, DSM-5 offers some directives that are specific to diagnosing PDs in children and adolescents under the age of 18. DSM-5 Section II cautions clinicians to be careful about diagnosing children and adolescents with a personality disorder, except in "those relatively unusual instances in which the individual's particular maladaptive personality traits appear to be pervasive, persistent, and unlikely to be limited to a particular developmental stage or another mental disorder" (APA, 2013, p. 647). For all of the PD diagnoses except for ASPD, the diagnostic criteria for children and adolescents are the same as those used for adults, but for youth under age 18, the patterns must have been present for at least a year. Youth under 18 may not be diagnosed with ASPD. Typically, youth with antisocial behavior are diagnosed with conduct disorder instead, and conduct disorder with onset before age 15 is required for an adult diagnosis of ASPD. Interestingly, the Section III alternative DSM-5 model for PDs does

TABLE 18.1. DSM-5 Personality Disorders (Section II)

- **Paranoid personality disorder** is a pattern of distrust and suspiciousness such that others' motives are interpreted as malevolent.
- **Schizoid personality disorder** is a pattern of detachment from social relationships and a restricted range of emotional expression.
- **Schizotypal personality disorder** is a pattern of acute discomfort in close relationships, cognitive or perceptual distortions, and eccentricities of behavior.
- **Antisocial personality disorder** is a pattern of disregard for, and violation of, the rights of others.
- **Borderline personality disorder** is a pattern of instability in interpersonal relationships, self-image, and affects, and marked impulsivity.
- **Histrionic personality disorder** is a pattern of excessive emotionality and attention seeking.
- **Narcissistic personality disorder** is a pattern of grandiosity, need for admiration, and lack of empathy.
- **Avoidant personality disorder** is a pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation.
- **Dependent personality disorder** is a pattern of submissive and clinging behavior related to an excessive need to be taken care of.
- **Obsessive–compulsive personality disorder** is a pattern of preoccupation with orderliness, perfectionism, and control.
- **Personality change due to another medical condition** is a persistent personality disturbance that is judged to be due to the direct physiological effects of a medical condition (e.g., frontal lobe lesion).
- **Other specified personality disorder and unspecified personality disorder** is a category provided for two situations: 1) the individual's personality pattern meets the general criteria for a personality disorder, and traits of several different personality disorders are present, but the criteria for any specific personality disorder are not met; or 2) the individual's personality pattern meets the general criteria for a personality disorder, but the individual is considered to have a personality disorder that is not included in the DSM-5 classification (e.g., passive–aggressive personality disorder).

Note. Reprinted with permission from *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (pp. 645–646). Copyright 2013 by the American Psychiatric Association.

not include any cautions about diagnosing PDs before the age of 18; this model also does not require that the symptoms have lasted for a specific period of time, but rather simply requires that they be "relatively stable across time, with onsets that can be traced back to at least adolescence or early adulthood" (APA, 2013, p. 761). The alternative system thus does not appear to discourage diagnosis of PDs in children and adolescence.

Unfortunately, some clinicians and researchers have misinterpreted the DSM-IV guidelines to mean that PDs may never be diagnosed in childhood or adolescence. A recent study of Dutch and Belgian psychologists found that one-quarter of the psychologists wrongly believed that diagnostic manuals do not permit PD diagnosis in adolescents (Laurenssen, Hutsebaut, Feenstra, Van Busschbach, & Luyten, 2013). This explicit hesitance to diagnose PDs in youth may arise from several sources (Chanen & McCutcheon, 2008; Freeman & Rigby, 2003; Miller, Muehlenkamp, & Jacobson, 2008; Shiner, 2007; Westen & Chang, 2000). First,

because PD has been conceptualized as long-lasting, difficult to treat, and severe, especially compared to many Axis I disorders, clinicians and researchers may have concerns about stigmatizing youth by giving them a PD diagnosis. Second, for centuries Western thinkers have suggested that adolescence is a tumultuous period characterized by erratic moods and impulsive behavior (Arnett, 1999), termed famously the "storm and stress" of adolescence (Hall, 1904). Perhaps a certain amount of personality "pathology" is seen as being normative during the adolescent period, and thus not worthy of clinical attention. Finally, youth's personalities are often viewed as being "under construction" during childhood and adolescence, and therefore too unstable to have lasting significance (Elliott, Tyrer, Horwood, & Fergusson, 2011). There is empirical evidence that all three of these reasons may prevent clinicians from making a PD diagnosis in adolescent patients (Laurenssen et al., 2013). This hesitance to diagnose PDs in youth has had a significant negative impact on researchers' interest in studying the development of PDs,

though fortunately this is changing as more work focuses on this topic.

The hesitance about diagnosing PDs may also lead clinicians to overlook the presence of personality pathology in their young patients. Westen, Shedler, Durrett, Glass, and Martens (2003) conducted a study in which they asked practicing psychologists and psychiatrists to report on a particular adolescent patient in their practices. Although only 28.4% of patients were assigned a PD diagnosis by their clinicians, 75.3% of the patients met criteria for a PD, based on their clinicians' reports of PD symptoms. Similarly, among a sample of practicing European psychologists, only 9% of clinicians reported diagnosing PDs in adolescence, and even fewer offered specialized treatments for adolescent PDs (Laurenssen et al., 2013). In short, misconceptions about the nature of PD in youth may prevent some clinicians from recognizing that their adolescent patients meet criteria for PDs. This is a serious problem, especially given the evidence reviewed in this chapter that PDs in youth are potentially serious and impairing, and certainly worthy of assessment and treatment.

THEORETICAL FRAMEWORK

As research on normal-range personality traits and their development in childhood and adolescence grows, the relevance of normal personality development for the emergence of personality pathology becomes ever more salient (Shiner, 2009; Tackett & Kushner, in press). Although the DSM PD system is largely nondevelopmental, it is possible to draw from the existing literature on normal personality development to provide a more truly developmental perspective on the development of personality pathology. As noted earlier, the developmental psychopathology perspective emphasizes the importance of normal-range and adaptive development for understanding the development of psychopathology (Cicchetti, 1993, 2013), providing a framework for integration of normal and abnormal phenomena. In this section, we review theory and research on normal personality constructs in youth, highlighting the relevance of this work for early-life personality pathology. Specifically, we use a very rich and comprehensive personality model developed by McAdams and colleagues (McAdams, 2013; McAdams & Olsen, 2010; McAdams & Pals, 2006). This model differentiates three levels of individual differences in personality. First, we discuss personality *traits*, which McAdams

and Pals call the "dispositional signature" of personality. Next, we discuss "characteristic adaptations"—"a wide range of motivational, social-cognitive, and developmental adaptations, contextualized in time, place, and/or social role" (McAdams & Pals, 2006, p. 208). We focus on two specific characteristic adaptations that hold particular relevance for youth PDs: attachment/social cognition and emotion regulation/coping (Shiner, 2009). Finally, we discuss the third level of "personal narratives"—stories that individuals begin to develop in adolescence to help them make sense of their identities over time. We believe that personality pathology in youth may involve disruptions at all of these levels of analysis.

Temperament and Personality Traits

A predominant theoretical and conceptual approach to personality across the lifespan focuses on personality traits as constructs that summarize characteristic patterns of thinking, feeling, and behaving that are pervasive across situations and stable across time. In particular, the "Big Five" model defines five broadly defined traits that capture salient features across persons: Extraversion (tendencies such as sociability, gregariousness, and experiencing positive emotions); Neuroticism (tendencies to experience negative emotions, such as sadness, anxiety, and distress); Conscientiousness (tendencies toward persistence, responsibility, and organization); Agreeableness (tendencies toward empathy and communion vs. hostility and aggression); and Openness to Experience/Intellect (tendencies toward intellectual engagement and exploration/enjoyment of stimulating experiences; John, Naumann, & Soto, 2008). These traits characterize the personalities of children as early as the preschool period (De Pauw, Mervielde, & Van Leeuwen, 2009), and they robustly characterize children's traits in later childhood and adolescence as well (Shiner & DeYoung, 2013). Table 18.2 illustrates the nature of the Big Five traits by presenting components of each trait; these components are taken from measures of temperament and personality traits in childhood and adolescence.

These traits are linked to early-emerging temperament traits, which have historically represented the primary constructs of interest for individual difference researchers focusing on infancy and early childhood (Rothbart & Bates, 2006; Shiner & Caspi, 2012). Early in life, children manifest individual differences in their experiences and expressions of positive and negative

TABLE 18.2. Child Temperament and Personality Facets Constituting the Big Five Higher-Order Traits in Childhood and Adolescence

Big Five higher-order domains	Child temperament facets	Child and adolescent personality facets
Neuroticism	Frustration[a] (CBQ/EATQ-R) Discomfort (CBQ) Fear (CBQ; EATQ-R) Sadness (CBQ)	Fearful/insecure (ICID) Anxiety (HiPIC) Negative affect (ICID) Self-confidence—rev. (HiPIC)
Extraversion	Activity level (CBQ; EATQ-R) Approach (CBQ) High-intensity pleasure (CBQ; EATQ-R) Shyness—rev. (CBQ; EATQ-R) Smiling and laughter (CBQ)	Positive emotions (ICID) Sociability (ICID); shyness—rev.[a] (HiPIC) Activity level (ICID); energy (HiPIC) Expressiveness (HiPIC) Optimism (HiPIC)
Agreeableness	Affiliation[a] (EATQ-R)	Antagonism—rev. (ICID); altruism (HiPIC) Strong-willed—rev. (ICID) Dominance—rev. (HiPIC) Egocentrism—rev. (HiPIC) Compliance (HiPIC); irritability—rev. (HiPIC)
Conscientiousness	Attention (CBQ; EATQ-R) Impulsivity—rev. (CBQ) Inhibitory control (CBQ; EATQ-R) Activation control (EATQ-R)	Organized (ICID); order (HiPIC) Achievement orientation (ICID); achievement motivation (HiPIC) Distractible (ICID); concentration (HiPIC) Perseverance (HiPIC)
Openness to Experience/Intellect	Low-intensity pleasure (CBQ) Pleasure sensitivity (EATQ-R) Perceptual sensitivity (CBQ; EATQ-R)	Intellect (ICID; HiPIC) Creativity (HiPIC) Curiosity (HiPIC)

Note. Rev., reversed (meaning that the facet loads negatively on the higher-order trait).

CBQ, Children's Behavior Questionnaire (Rothbart, Ahadi, Hershey, & Fisher, 2001); EATQ-R, Early Adolescent Temperament Questionnaire—Revised (Ellis & Rothbart, 2001); ICID, Inventory of Child Individual Differences (Halverson et al., 2003); HiPIC, Hierarchical Personality Inventory for Children (Mervielde & De Fruyt, 2002).

[a]Facets potentially loading on more than one higher-order Big Five domain.

emotions, as well as in their ability to regulate their emotions and behavior. Temperament trait models typically converge on three higher-order traits (rather than the five traits of the Big Five): Surgency or Positive Emotionality (akin to Extraversion); Negative Emotionality (akin to Neuroticism); and Effortful Control (most clearly linked with Conscientiousness, but with some association with Agreeableness as well). Recent efforts have focused on merging our understanding of three- and five-factor trait models, and have offered evidence of empirical links among these traits in both childhood and adulthood (Markon, Krueger, & Watson, 2005; Tackett et al., 2012). Thus temperament and personality traits are now linked both theoretically and empirically (De Pauw et al., 2009; Shiner, 2010; Shiner & DeYoung, 2013), and greater merging of these

literatures is expected to increase as the field moves forward.

Although advancing research in the domain of child personality traits has provided increasing evidence for connections with adult models such as the Big Five (e.g., Digman & Shmelyov, 1996; Goldberg, 2001), differences across development have emerged as well. For example, some studies have suggested that Neuroticism, compared to other traits, may be more difficult to measure in early life (Tackett, Krueger, Iacono, & McGue, 2008; Tackett et al., 2012). This challenge potentially reflects the restricted access that standard informants (e.g., parents, teachers) have to the type of internalized affect that defines trait Neuroticism (Grills & Ollendick, 2002; Tackett, 2011; Vazire, 2010). It is also unclear how distinct Agreeableness and Consci-

entiousness traits emerge across development from the broad Effortful Control trait defined in temperament models (Rothbart, Ahadi, & Evans, 2000; Tackett et al., 2012). The content of child personality traits is typically analogous, but not identical, to adult personality traits (De Pauw et al., 2009); researchers therefore need to maintain a developmentally sensitive perspective in such work and to guard against atheoretical top-down approaches, often seen in the application of adult personality theory and research to younger age groups.

Children's early personalities shape their experiences of the environment through a number of important processes (Caspi & Shiner, 2006; Shiner & Caspi, 2012): the ways that children are conditioned by their environments, the responses children evoke from the people in their lives, the ways that children interpret their experiences, the ways children evaluate themselves and form a sense of identity, the environments that children "select" themselves into, and the ways that children modify and manipulate their environments. The personalities of young people can help explain why children who are exposed to relatively similar environments do not have the same outcomes—an excellent example of the principle of multifinality. For example, a child who is intensely anxious and irritable, and who lacks good self-control, is going to have a very different experience of parental divorce than a child who is emotionally stable and behaviorally restrained; these differences in the experience of divorce could then lead to differing outcomes for the children.

Personality traits represent an important focus in understanding the emergence and development of personality disorder in youth because traits show similar levels of heritability (or genetic influence; Saudino & Wang, 2012) to PD constructs; they are salient and measurable from early life (Rothbart & Bates, 2006); and they reach moderate levels of stability by early childhood (Roberts & DelVecchio, 2000). We elaborate on these points later in this chapter. Work on adults suggests that PD symptom-level change *follows* change in normal personality traits (Warner et al., 2004), highlighting their importance as early core components of personality pathology; we return to this point as well later in the chapter. In addition, early efforts at utilizing personality traits as selection factors for indicated prevention efforts (i.e., prevention efforts delivered to a group defined as high-risk on the basis of some key vulnerability feature) have already shown great promise in reducing the emergence and severity of adolescent personality pathology (e.g., Chanen, Jovev, Djaja, et al., 2008).

Characteristic Adaptations: Attachment and Social Cognition

McAdams and Pals (2006) describe characteristic adaptations as those components of individual personality that are more closely tied to situations, contextual factors, and personal roles. One such aspect of individual functioning that holds great relevance for youth PDs is attachment to a primary caregiver and social-cognitive functioning more broadly. Attachment reflects a specific type of mental representation; mental representations are defined by children's perceptions of themselves, their experiences, their relationships, and their environments (Shiner, 2010). These perceptions hold predictive value in understanding later behavior and play an important role in shaping adaptive and maladaptive developmental trajectories. Attachment theory has played a central role in the conceptualization and theoretical underpinnings of a number of PDs, with empirical support for the importance of attachment in PD development (e.g., Crawford et al., 2006; Sroufe, Carlson, Levy, & Egeland, 1999; Weston & Riolo, 2007). Development from infancy to early childhood has been identified as a critical developmental period for PDs because of its relevance for adaptive attachment (Tackett et al., 2009), when patterns of security and insecurity form in response to the child's relationship with a primary caregiver (Mikulincer & Shaver, 2007). In this way, the central role played by attachment in PD conceptualization has anchored PD developmental origins to infancy and early childhood, underscoring the idea that PD emergence begins early in the lifespan (Paris, 2003).

The mental representation of this early relationship is thought to provide a context for the children's future relationships and responses to the world around them (Sroufe, 2005; Sroufe et al., 1999). Modern models of attachment define two key dimensions of attachment styles: the first reflecting the extent to which a person worries versus feels secure about the availability of a partner (or caregiver), and the second reflecting the extent to which a person prefers independence and detachment versus affiliation and intimacy (Fraley & Shaver, 2008). Disrupted and maladaptive attachment patterns have been a long-standing component of the theoretical background behind multiple PDs, but have played a particularly important role in the conceptualization of BPD (Levy, 2005). Empirical data indicate a higher prevalence of disrupted attachment styles (e.g., attachment styles characterized by fears of rejec-

tion and abandonment) among adolescents with BPD (Westen, Nakash, Thomas, & Bradley, 2006).

Although the transition from infancy to early childhood has been the key critical developmental period in attachment theory and is thus highly relevant for early PDs, two other critical developmental periods for PD development are also closely tied to interpersonal relationships (Tackett et al., 2009). Specifically, the transition from middle childhood to adolescence is marked by the increasing salience of the peer group, whereas the transition from late adolescence to adulthood is marked by the shift toward intimate partners as the primary relational context. Certainly, mental representations formed during the early years in the context of attachment to a primary caregiver may serve as risk or resiliency factors when youth are faced with these new relational tasks across development. Theory and research at these later stages has focused on broader definitions of social-cognitive factors that play a role in PD emergence. For example, children's sense of alienation from their peer group, perceptions of their self-competence, perceptions of the hostile intentions of other people, and beliefs about the malleability of their own behavior are all mental representations with implications for adjustment and maladjustment (Shiner, 2009; Tackett et al., 2009).

Three specific categories of youth's social cognition have been highlighted as especially relevant for personality pathology: emotion recognition, theory of mind (also called "mentalizing"), and trust (Sharp, 2012b). The relevance of emotion recognition for PD may emerge either via biases in emotion recognition, or via dampened/heightened emotion recognition. For example, BPD in adolescents has been associated with a negativity bias, as well as with potentially heightened recognition of one's own and others' emotions (Sharp, in press). Areas of social cognition show relevance across diverse forms of personality pathology, although sometimes in divergent directions. For example, hypermentalizing (i.e., overinterpreting the thoughts and behaviors of others) is associated with BPD, whereas hypomentalizing (i.e., impoverished interpretations of others' thoughts and behaviors) is associated with ASPD in youth (Sharp, 2012). Social-cognitive tendencies may also play a role in shaping adaptive versus maladaptive functioning. For example, "agentic" motives (meaning goals focused on achieving power, mastery, and assertion over others) differentiate children with narcissistic tendencies from children with adaptive high self-esteem, who are primarily motivated by communal motives (goals focused on achieving intimacy and affiliation; Thomaes, Stegge, Bushman, Olthof, & Denissen, 2008).

Characteristic Adaptations: Emotion Regulation and Coping

Another aspect of personality that is highly relevant for youth PDs and is best defined as a characteristic adaptation consists of emotion regulation and coping. The manner by which children learn to respond to and cope with stressors falls under the domain of characteristic adaptations, as this aspect of functioning is closely linked with those specific environments that an individual might encounter (Shiner, 2010). Coping strategies can be both adaptive and maladaptive, and have been closely linked to the development of personality pathology over time. Coping strategies have been broadly categorized into two domains: strategies reflecting engagement (or approach-motivated behaviors) and those reflecting disengagement (or avoidance-motivated behaviors; Skinner & Zimmer-Gembeck, 2007). In addition, coping strategies may include both conscious processes (e.g., active distraction from a negative stimuli) and unconscious processes (e.g., the use of defense mechanisms; Cramer, 2008).

Predominant coping strategies in childhood include problem solving, escape, distraction, and support seeking (Skinner & Zimmer-Gembeck, 2007). Adolescents develop a more complex repertoire of coping strategies, including adaptive strategies such as cognitive restructuring, as well as less adaptive strategies such as rumination and externalization of blame. Adolescence in particular may be viewed as a developmental stage of skill attainment and experimentation, as youth begin to discover new coping strategies and examine their effectiveness at goal attainment. Emotion regulation is an important aspect of coping, and it refers specifically to an individual's self-regulatory responses to emotions, rather than to the status or content of emotions themselves (Gratz et al., 2009). Deficits in emotion regulation include poor behavioral control in the context of emotional distress, as well as difficulties with the modulation of emotion arousal.

Youth PDs may be differentially associated with problems in emotional regulation and ineffective coping. Cluster B PDs seem likely to be associated with maladaptive emotion regulation strategies, whereas Cluster C PDs are likely to reflect maladaptive overreliance on disengagement coping approaches. Research-

ers may also differentiate these categories of personality pathology as defined by emotional underregulation (Cluster B) versus emotional overregulation (Cluster C), whereas Cluster A PDs are more likely to reflect general problems in the actual nature or quality of emotions (specifically, their absence). BPD in particular has been both theoretically and empirically associated with problematic emotion regulation approaches. A recent investigation by Gratz and colleagues (2009) highlights the nature of emotion regulation as a characteristic adaptation. Specifically, in this study the influence of a vulnerability trait (affective dysfunction) on child BPD symptoms was mediated by dysfunctional emotion regulation. In other words, this study supported the idea that an existing trait vulnerability may increase risk for later BPD, but showed that it did so (at least partly) through its impact on maladaptive emotion regulation processes. However, other aspects of maladaptive coping may cut across PDs and PD clusters. For example, experiential avoidance (a maladaptive coping technique defined by attempts to avoid internal distress) is present in BPD and has been historically associated with anxiety problems, and thus is probably connected to Cluster C PDs as well (Gratz, Tull, & Gunderson, 2008). Future research in this area should focus on core underlying components of maladaptive coping and emotion regulation, which are likely to be relevant for a variety of PD manifestations.

Narrative Identity

The final level in McAdams and Pals's (2006) model is that of personal narratives, or life stories. This level is of fundamental importance for youth PD, as a key function provided by personal narratives is identity development (McAdams & McLean, 2013; McLean & Pasupathi, 2012)—a process that may be disturbed in the development of certain types of personality pathology (Fonagy & Bateman, 2008). Thus considering this level is essential for a full understanding of how normal personality development influences the development and manifestation of PDs. Narrative identity development is a particularly important task for adolescence, when youth gain the cognitive and social skills to think about their lives in more coherent and complex ways (Habermas & de Silveira, 2008; Shiner, 2010).

The development of life narratives is firmly embedded in an individual's social context (McLean & Pasupathi, 2012; Shiner, 2009). Children begin co-constructing their narratives, primarily with their par-

ents, from an early age, and these experiences appear to influence narrative complexity (e.g., Fivush, Haden, & Reese, 2006). The social context of the peer group becomes an active part of narrative construction in adolescence. Thus the cross-cutting theme of interpersonal relationships for personality pathology in general highlights the potential relevance of life narratives for the development of personality disorder. Identity functioning is specifically embedded in the conceptualization of BPD, but it is likely to be relevant to many other PDs as well.

Shiner (2009) highlights two particularly problematic pathways in identity development with relevance for PD emergence. The first is problems with integrating negative experiences into the life narrative in constructive and adaptive ways, and the second is difficulties with progressive coherence of the life narrative. Regarding the first pathway, there are positive and adaptive ways of integrating negative experiences into a life narrative, such as utilizing positive explanatory frameworks and coping (Pals, 2006). A construct frequently studied by narrative psychologists is that of meaning making, or an individual's ability to develop positive meaning out of a potentially challenging or negative experience (McLean & Pasupathi, 2012). Meaning making is frequently associated with more adaptive functioning and life narratives. In contrast to narratives that construct positive meanings out of negative experiences, some life narratives contain a high number of "contamination sequences," in which descriptions of positive experiences are followed by descriptions of subsequent negative experience (McAdams, 2009); the negative experience spoils the rewards of the positive one. The presence of more frequent contamination sequences is associated with a variety of maladaptive psychological outcomes (McAdams, 2009). A second maladaptive pathway in identity development may involve problems in developing a coherent and integrated life narrative (Shiner, 2009). Specifically, some adolescents may struggle with committing to a specific pathway of identity development, and others may tend to recall few specific memories and instead focus only on diffuse or general memories; both of these problems with developing a coherent life story may result in negative or maladaptive consequences. Indeed, identity integration is a fundamental way in which personality-disordered youth differ from normal controls (Feenstra, Hutsebaut, Verheul, & van Limbeek, 2014). Furthermore, in this study by Feenstra and colleagues (2014), the majority of youth with a PD diagnosis showed in-

creasing levels of identity integration across the experience of inpatient psychotherapy, suggesting that this domain also represents an important target for treatment. As we describe later in this chapter, the alternative dimensional system for PDs in DSM-5 explicitly moves toward a more central role for problematic identity development and identity functioning in its definition of PDs.

Thus all three levels of normal personality development as described by McAdams and Pals (2006) are highly relevant for the development of PD in youth. Personality traits (Level 1) are likely to serve as both risk and resilience factors for the development of personality pathology. Characteristic adaptations (Level 2) show both general and specific connections to emerging PD, particularly via social-cognitive processes such as attachment and emotion regulation/coping strategies. The content and structure of adolescents' life narratives (Level 3) hold particular relevance for adaptive identity development and adjustment. We now turn from an examination of general constructs reflecting normal personality development to discussion of dimensional models of personality pathology.

ALTERNATIVE DIMENSIONAL MODELS OF PDs

In this section, we review dimensional alternatives to the categorical *DSM* PD system. First, we discuss the rationale for adopting a dimensional model for PDs. Second, we review research on a set of higher-order pathological personality traits obtained across studies of normal-range and pathological traits, in both youth and adults. Third, we present the alternative DSM-5 model for PDs, which includes a dimensional system for pathological personality traits. A move toward dimensional trait models of personality pathology is of great relevance for developmental research, as traits offer greater opportunity to investigate the development of these problems across the lifespan (Tackett et al., 2009). There is increasing evidence that some childhood conditions are best conceptualized as dimensions rather than categories (Coghill & Sonuga-Barke, 2012)—including attention-deficit/hyperactivity disorder (ADHD), posttraumatic stress disorder (PTSD), some forms of depression, and aggression—so dimensional models of personality pathology are worthy of further attention by researchers and clinicians working with children and adolescents.

The Rationale for Dimensional Models of PDs

A key issue in conceptualizing personality pathology is whether it is most validly described as categorical patterns or quantitative variations on dimensional traits. The model of PDs adopted in DSM-IV and now DSM-5 is a categorical one: The PDs are each seen as distinct patterns that differ qualitatively both from normal personality functioning and from each other. However, even within DSM-IV-TR, there is some recognition of the possibility of a dimensional approach: "An alternative to the categorical approach is the dimensional perspective that Personality Disorders represent maladaptive variants of personality traits that merge imperceptibly into normality and into one another" (APA, 2000, p. 689). As noted earlier, when the DSM-5 revision process first started, serious consideration was given to dimensional models of psychopathology across the diagnostic manual as a whole (Krueger, Watson, & Barlow, 2005; Rounsaville et al., 2002), but particularly within the PDs (Widiger et al., 2005).

The validity of the DSM PD categorical system has been challenged on a number of fronts (reviewed in Clark, 2007; Clark, Livesley, & Morey, 1997; Simonsen & Widiger, 2005; Trull & Durrett, 2005; Widiger & Trull, 2007). The PDs co-occur within patients at a rate that is much higher than would be expected if the disorders are truly distinct, categorical entities with distinct etiologies (Clark, 2007; Trull, Scheiderer, & Tomko, 2012); this is probably true for youth as well as adults, as we discuss later in the chapter. The cutoffs for the number of criteria needed for a PD diagnosis are arbitrary. The existing PD diagnoses include heterogeneous groups of patients within each category because of the polythetic criteria sets that are used. Despite the long list of PDs included in the DSMs, the existing PDs do not provide adequate coverage of the range of personality pathology that patients exhibit. As a result, PD-NOS has turned out to be the most common PD diagnosis used in actual practice with adults (Verheul & Widiger, 2004), and it is highly prevalent in psychotherapy outpatients (Verheul, Bartak, & Widiger, 2007) when the DSM-IV system is used. PD-NOS may also be the most prevalent DSM-IV PD in both adolescents and adults (Johnson, First, et al., 2005).

It seems, then, that personality pathology may be more validly conceptualized within a dimensional framework than via a number of discrete categories. In a dimensional taxonomy, it is recognized that psychopathology involves variation in underlying dimen-

sions of cognition, affect, and behavior. Implicit in such a model is the recognition that there is no clearcut boundary between normal and abnormal functioning; in other words, in a dimensional model, PDs differ from normal-range personality quantitatively rather than qualitatively. Dimensional models of personality pathology address the problems with the current categorical model. The high comorbidity of PDs makes sense if personality pathology is an expression of extreme standing on pathological trait dimensions because similar PD traits may be present across PD diagnoses. In addition, diagnostic heterogeneity within diagnoses probably results from a mixture of pathological traits in individuals within a PD category. Dimensional models should be able to describe the full range of individuals with PDs.

Evidence for a Set of Pathological Personality Traits

Research on dimensional approaches to PDs has relied on two key sources of evidence: research linking normal-range personality traits such as the Big Five traits with personality disorders, and research delineating the structure of pathological personality trait dimensions. In both lines of research, most of the focus has been on adult PDs, but the patterns observed for adult PD dimensions have been explored in youth as well.

The DSM-IV and DSM-5 PD diagnoses may be described in terms of variation of normal-range personality traits. In particular, extensive research has demonstrated that the Big Five traits described previously (Extraversion, Neuroticism, Conscientiousness, Agreeableness, and Openness to Experience/Intellect) may be used to characterize DSM-defined personality pathology (Widiger & Costa, 2013). Each of the broad, higher-order Big Five dimensions includes a number of more narrow, lower-order dimensions, or "facets" (e.g., Extraversion involves components such as activity level, gregariousness, and positive emotions). These facets are used to describe personality pathology in a more nuanced way than is possible with the Big Five traits alone. For example, BPD in adults can be characterized by specific facets of Neuroticism (emotionality lability, anxiousness, separation anxiety, hostility) and low Conscientiousness (impulsivity, risk taking) (Trull, 2012). As with adults, there is some evidence that PDs in adolescence can be described by using Big Five personality and temperament measures (De Clercq & De

Fruyt, 2003; De Clercq, De Fruyt, & Van Leeuwen, 2004; Decuyper, De Clercq, De Bolle, & De Fruyt, 2009; De Fruyt & De Clercq, 2013; Tackett & Kushner, in press). The findings of these studies with adolescents suggest that although patterns of links between Big Five facet scores and PD symptoms reasonably replicate patterns seen in adults, unexpected associations of personality traits and PD symptoms also occur, indicating possible developmental differences (De Fruyt & De Clercq, 2013). Another implication of this work is that some domains of PD in youth are not well captured by existing normative trait models (e.g., the role of identity disturbance in BPD; Tackett & Kushner, in press).

In addition to the work linking PDs with normal-range personality traits, many different pathological personality trait models have been proposed (Widiger & Simonsen, 2005). Several lines of research point to the evidence that personality pathology may be defined along four overarching dimensions (Clark, Simms, Wu, & Casillas, 2011; Livesley & Jackson, 2009; Markon et al., 2005; Trull & Durrett, 2005; Widiger & Mullins-Sweatt, 2005; Widiger & Simonsen, 2005). First, Extraversion versus Introversion/Detachment measures how outgoing, active, energetic, expressive, and emotionally positive a person is. At the pathological extremes, this dimension taps exhibitionism (high end) and detachment, social avoidance, and excessive shyness (low end). Second, Negative Affectivity versus Emotional Stability measures individual differences in the experience of negative emotions. At the pathological high end, this dimension taps anxiousness, insecure attachment, identity problems, affective lability, feelings of worthlessness, and poor coping with stress. It is not clear whether there is a pathological low end, but it is possible that it may involve an excessive lack of fear and anxiety (as in psychopathy). Third, Conscientiousness versus Disinhibition measures tendencies to be responsible, attentive, persistent, orderly, high-achieving, and planful versus irresponsible, unreliable, careless, and quitting easily. At the pathological extremes, this dimension taps compulsivity and workaholism (high end) and impulsiveness, irresponsibility, and excessive risk taking (low end). Fourth, Antagonism versus Agreeableness measures tendencies toward being hostile and cynical versus kind, modest, empathic, honest, and trusting. At the pathological high end, this dimension taps mistrust and alienation, aggression, entitlement, and callousness. Less often represented is a fifth factor reflecting Cluster A characteristics and sometimes labeled Psychoticism or Peculiarity versus

Lucidity (Harkness & McNulty, 1994; Tackett, Silberschmidt, Krueger, & Sponheim, 2008). Psychoticism, which is conceptualized as a pathological trait, reflects the tendency to experience cognitive or perceptual aberrations. Psychoticism is particularly notable for its relative lack of attention in the developmental literature and its absence from commonly used dimensional measures of personality pathology in youth (Tackett et al., 2009). Thus, although it is clearly relevant as a core component of personality pathology, much more work is needed to understand approaches to assessment of this trait and its utility in early life.

Although most of the research on pathological personality dimensions has focused on adults, there is newer evidence suggesting that the same pathological personality traits describe early PD manifestations in youth. PD trait questionnaire measures created for adults have been adapted for use with adolescents, and findings suggest that the same higher-order pathological traits validly represent the structure of personality pathology in adolescents (Linde, Stringer, Simms, & Clark, 2013; Ro, Stringer, & Clark, 2012; Tromp & Koot, 2008, 2010). In contrast to the "top-down" evidence from adult measures adapted for adolescents, "bottom-up" data on pathological personality traits in youth come from a questionnaire designed to measure maladaptive extreme variants of normal-range personality traits in youth (De Clercq, De Fruyt, Van Leeuwen, & Mervielde, 2006; De Clercq, De Fruyt, & Widiger, 2009; De Fruyt & De Clercq, 2013). This measure yields four higher-order traits comparable to those found in the adult research: Introversion, Disagreeableness, Compulsivity, and Emotional Instability. An attempt is currently being made to develop a measure of the Peculiarity dimension in youth (De Clercq & De Fruyt, 2012).

Despite similarities in the findings for the hierarchical structure of pathological personality traits in adults and youth, it is important to note that some differences are found in youth, much like the differences found for the structure of normal personality traits in youth (Kushner, Tackett, & De Clercq, 2013). For example, a robust pathological Introversion trait does not appear to be as salient in youth as in adults; this finding may be analogous to the difficulties in measuring "pure" Neuroticism in early life, when access to children's early experiences of sadness, anxiety, and anger may be more difficult to obtain. The use of dimensional measures of personality pathology in youth is becoming increasingly feasible with the advent of such measures, but it

will be important to remain sensitive to potential developmental differences in early personality pathology. For example, Westen and colleagues have obtained evidence for a larger number of PD-relevant dimensions in their work on clinician assessment of adolescent PD traits (Westen et al., 2003; Westen, Dutra, & Shedler, 2005). More work will be needed to identify early maladaptive personality traits in a developmentally sensitive manner.

Proposed Alternative DSM-5 Model for Personality Disorders

DSM-5 acknowledges the importance of dimensional models of PDs by its inclusion of the alternative DSM-5 model for PDs in Section III of the manual. This system is based on the research on the higher-order domains of pathological personality traits described in the preceding section. DSM-5 states the rationale for including both the categorical PD diagnoses and the alternative dimensional model thus: "The inclusion of both models in DSM-5 reflects the decision of the APA Board of Trustees to preserve continuity with current clinical practice, while also introducing a new approach that aims to address numerous shortcomings of the current approach to personality disorders" (APA, 2013, p. 761). In other words, the alternative model is designed to address the previously described limitations of the categorical PD approach.

Like the categorical formulation of PDs in Section II, the alternative model for PDs in Section III presents a set of general criteria for PDs (APA, 2013, p. 761). There are two key features to PDs in this new formulation: (1) impairment and (2) pathological personality traits. (See Table 18.3 for a general overview of these proposed diagnostic criteria for impairment in the elements of personality functioning and the presence of pathological personality traits.) As in the Section II PD diagnoses, the PD condition must be impairing; however, here "impairment" is defined in terms of moderate or greater impairment in self and interpersonal functioning. The person must also display one or more pathological personality traits. These two main features—impairment and pathological personality traits—are qualified in a number of ways: They must be relatively stable over time, present since adolescence or early adulthood, not better explained by another mental disorder, not merely the result of a substance or medical condition, and not normative for the person's age or sociocultural environment (APA, 2013, p. 761). This

TABLE 18.3. DSM-5 Proposed Diagnostic Criteria for Personality Disorder—Trait Specified (Alternative DSM-5 Model for Personality Disorders)

A. Moderate or greater impairment in personality functioning, manifested by difficulties in two or more of the following four areas:

  1. *Identity*
  2. *Self-direction*
  3. *Empathy*
  4. *Intimacy*

B. One or more pathological personality trait domains OR specific trait facets within domains, considering ALL of the following domains:

  1. **Negative Affectivity** (vs. Emotional Stability): Frequent and intense experiences of high levels of a wide range of negative emotions (e.g., anxiety, depression, guilt/shame, worry, anger), and their behavioral (e.g., self-harm) and interpersonal (e.g., dependency) manifestations.
  2. **Detachment** (vs. Extraversion): Avoidance of socioemotional experience, including both withdrawal from interpersonal interactions, ranging from casual, daily interactions to friendships to intimate relationships, as well as restricted affective experience and expression, particularly limited hedonic capacity.
  3. **Antagonism** (vs. Agreeableness): Behaviors that put the individual at odds with other people, including an exaggerated sense of self-importance and a concomitant expectation of special treatment, as well as a callous antipathy toward others, encompassing both unawareness of others' needs and feelings, and a readiness to use others in the service of self-enhancement.
  4. **Disinhibition** (vs. Conscientiousness): Orientation toward immediate gratification, leading to impulsive behavior driven by current thoughts, feelings, and external stimuli, without regard for past learning or consideration of future consequences.
  5. **Psychoticism** (vs. Lucidity): Exhibiting a wide range of culturally incongruent odd, eccentric, or unusual behaviors and cognitions, including both process (e.g., perception, dissociation) and content (e.g., beliefs).

Note. Reprinted with permission from *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (p. 770). Copyright 2013 by the American Psychiatric Association.

new framework acknowledges that PDs may be only relatively stable (unlike the Section II model, which describes PDs as enduring and inflexible), consistent with research described later in this chapter indicating that PDs may change over time. The dimensional framework does not specify exactly how long the condition needs to have persisted, also unlike the Section II framework. As noted earlier, the new formulation also does not suggest that PDs are relatively rare in childhood and adolescence, but instead asks the clinician or researcher to determine whether a patient is displaying traits and impairment that are not normative for the youth's developmental phase of life.

The first of the two key components required for a PD diagnosis in the dimensional system is impairment in the areas of self (including the elements of identity and self-direction) and interpersonal functioning (including the elements of empathy and intimacy). The DSM-5 definitions of these four elements are provided in Table 18.4. These elements are viewed as existing on a continuum and should be rated by researchers or clinicians on a scale with these four levels (APA, 2013, pp. 775–778): 0, little or no impairment; 1, some impairment; 2, moderate impairment; 3, severe impairment; and 4, extreme impairment. DSM-5 defines each scale point for each of the four elements of potential impairment. For example, a person displaying Level 3 (severe impairment) in the element of self-direction would exhibit this pattern: "Has difficulty establishing and/or achieving personal goals. Internal standards for behavior are unclear or contradictory. Life is experienced as meaningless or dangerous. Has significantly compromised ability to reflect on and understand own mental processes" (p. 777). A person must manifest Level 2 (moderate or greater impairment) in two or more elements to receive a PD diagnosis.

The DSM-5 Personality and PDs Work Group decided to include impairment as a key feature of PDs

TABLE 18.4. DSM-5 Proposed Elements of Personality Functioning

Self:

1. *Identity:* Experience of oneself as unique, with clear boundaries between self and others; stability of self-esteem and accuracy of self-appraisal; capacity for, and ability to regulate, a range of emotional experience.

2. *Self-direction:* Pursuit of coherent and meaningful short-term and life goals; utilization of constructive and prosocial internal standards of behavior; ability to self-reflect productively.

Interpersonal:

1. *Empathy:* Comprehension and appreciation of others' experiences and motivations; tolerance of differing perspectives; understanding the effects of one's own behavior on others.

2. *Intimacy:* Depth and duration of connection with others; desire and capacity for closeness; mutuality of regard reflected in interpersonal behavior.

Note. Reprinted with permission from *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (p. 762). Copyright 2013 by the American Psychiatric Association.

because substantial theoretical and empirical literatures point to the importance of problematic self and interpersonal functioning as a manifestation of personality pathology, separate from deviant personality traits (Bender, Morey, & Skodol, 2011; Livesley, 2007; Skodol, 2012; Skodol, Clark, et al., 2011; Tackett et al., 2009). Severity of dysfunction in self and interpersonal domains predicts PD outcomes in both adults (Hopwood et al., 2011) and adolescents (DeFife, Goldberg, & Westen, in press), and there is some preliminary support for the structure of the levels of personality functioning (Morey et al., 2011).

The second of the two key components required for a PD diagnosis is the presence of one or more pathological personality traits. These pathological traits are organized into five domains, and these five broad domains include between three and nine specific, narrow-band facets. The five personality trait domains and their specific trait facets are as follows (see APA, 2013, pp. 779–781):

1. Negative Affectivity (vs. Emotional Stability): emotional lability, anxiousness, separation insecurity, submissiveness, hostility, perseveration, depressiv-

ity, suspiciousness, and restricted affectivity (lack of).

2. Detachment (vs. Extraversion): withdrawal, intimacy avoidance, anhedonia, depressivity, restricted affectivity, suspiciousness.

3. Antagonism (vs. Agreeableness): manipulativeness, deceitfulness, grandiosity, attention seeking, callousness, hostility.

4. Disinhibition (vs. Conscientiousness): irresponsibility, impulsivity, distractibility, risk taking, and rigid perfectionism (lack of).

5. Psychoticism (vs. Lucidity): unusual beliefs and experiences, eccentricity, and cognitive and perceptual dysregulation.

The DSM-5 definitions for the five pathological personality trait domains are presented in Part B of Table 18.3. Some facets are included for more than one domain because, empirically, they are components of multiple domains.

The choice of these five PD trait domains was based on the research on pathological personality traits described in the previous section (Krueger, Eaton, Clark, et al., 2011; Skodol, 2012), and the domains clearly overlap with much previous work on normal and abnormal trait structure (Markon et al., 2005; Widiger, 2013). The primary domain names all focus on the more negative end of the trait dimension, however. The DSM-5 Personality and PDs Work Group opted to frame the domains in terms of pathological traits, rather than in terms of the normal-range personality models like the Big Five, in part because normal-range personality models do not adequately capture the full range of personality pathology (Krueger, Eaton, Clark, et al., 2011). A particular challenge that faced the work group was that although there is general consensus among researchers about the nature of the five domains, research has not clearly specified the facets that make up those domains (Clark, 2007; Krueger, Eaton, Clark, et al., 2011). The work group generated a set of facets based on the clinical relevance of the facets and based on current research on the ways that personality trait pathology may manifest itself, and then refined the list of facets based on research using a new questionnaire inventory of the DSM-5 facets—the Personality Inventory for DSM-5 (PID-5; Krueger, Derringer, Markon, Watson, & Skodol, 2011; Krueger, Eaton, Derringer, et al., 2011). Preliminary research on the PID-5 in adult samples suggests that it is structured in terms of the

five broad trait domains (Wright, Thomas, et al., 2012) and recovers much of the information obtained through use of the PD symptom lists in DSM-IV (Hopwood, Thomas, Markon, Wright, & Krueger, 2012).

Once a clinician or researcher has determined that a patient meets the general criteria for impairment and pathological personality traits, there are two possible routes to specifying the nature of the PD: (1) providing a specific PD diagnosis, or (2) providing a diagnosis of personality disorder—trait specified. To address the first route, there are six specific PD diagnoses retained from *DSM-IV*—ASPD, BPD, and avoidant, narcissistic, obsessive–compulsive, and schizotypal PDs—but the diagnoses are defined by new diagnostic criteria framed in terms of specific impairments and pathological personality traits, consistent with the general Section III framework. The typical features of these six diagnoses are presented in Table 18.5. As noted earlier, these diagnoses were retained based on some combination of prevalence in community and clinical samples, associated psychosocial impairment, and evidence for the validity and clinical utility of the disorders (Skodol, 2012; Skodol, Bender, et al., 2011). To address the second route to PD diagnosis, for people who do not display a pattern of impairment and pathological traits consistent with one of these six diagnoses, the diagnosis of personality disorder—trait specified is used instead; the nature of the diagnosis is made clear by noting the specific aspects of impairment and pathological personality exhibited by a particular patient. This new diagnosis is designed to provide more detail and nuance for what may have previously been a diagnosis of PD-NOS.

At this point, it is not clear to what extent this Section III model for the PDs will be used in both clinical and research settings. Obviously, the model was not unanimously well received by the APA Board; otherwise, the DSM-IV PD diagnoses would not have been retained in DSM-5's Section II. The model has been criticized for lacking adequate empirical support and breaking away too radically from the previous model (Frances & Widiger, 2012; Leising & Zimmerman, 2011). Other criticisms have been leveled against it as well: It deletes numerous PD diagnoses that have been useful for decades; it is unduly complex; and it does not adequately cover the full range of normal personality traits (Widiger, 2011). However, the model has much to commend it, especially in light of the research described in this section of the chapter, and future research will help refine it further.

TABLE 18.5. DSM-5 Proposed Specific Personality Disorders

Section III [of DSM-5, Emerging Measures and Models,] includes diagnostic criteria for antisocial, avoidant, borderline, narcissistic, obsessive–compulsive, and schizotypal personality disorders. Each personality disorder is defined by typical impairments in personality functioning (Criterion A) and characteristic pathological personality traits (Criterion B):

- Typical features of **antisocial personality disorder** are a failure to conform to lawful and ethical behavior, and an egocentric, callous lack of concern for others, accompanied by deceitfulness, irresponsibility, manipulativeness, and/or risk taking.

- Typical features of **avoidant personality disorder** are avoidance of social situations and inhibition in interpersonal relationships related to feelings of ineptitude and inadequacy, anxious preoccupation with negative evaluation and rejection, and fears of ridicule or embarrassment.

- Typical features of **borderline personality disorder** are instability of self-image, personal goals, interpersonal relationships, and affects, accompanied by impulsivity, risk taking, and/or hostility.

- Typical features of **narcissistic personality disorder** are variable and vulnerable self-esteem, with attempts at regulation through attention and approval seeking, and either overt or covert grandiosity.

- Typical features of **obsessive–compulsive personality disorder** are difficulties in establishing and sustaining close relationships, associated with rigid perfectionism, inflexibility, and restricted emotional expression.

- Typical features of **schizotypal personality disorder** are impairments in the capacity for social and close relationships, and eccentricities in cognition, perception, and behavior that are associated with distorted self-image and incoherent personal goals and accompanied by suspiciousness and restricted emotional expression.

Note. Reprinted with permission from *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (pp. 763–764). Copyright 2013 by the American Psychiatric Association.

EPIDEMIOLOGY AND COMORBIDITY

Epidemiology

It is important to estimate the prevalence of PDs across the lifespan, in order to obtain a clearer developmental perspective on the emergence and course of PDs. At this point, far more prevalence studies have been conducted in adult samples than in samples of youth.

In community samples of adults assessed with a structured or semistructured clinical interview, the average current prevalence of any PD is between 10.5 and 12% (Lenzenweger, 2008; Torgersen, 2012), and the current prevalence for any specific PD is around 1–2% (Torgersen, 2012). The most common PDs in adult samples appear to be avoidant PD and obsessive–compulsive PD; consistent with this finding, the Cluster C PDs appear to be more prevalent than the Cluster A and B PDs (Torgersen, 2012). The prevalence studies in adults are limited in several ways (relatively small, mostly urban, and mostly American samples), but the consistency of the findings across studies lends support to the idea that approximately 1 in 10 adults has at least one PD at any one point in time. The lifetime prevalence rates are of course higher, with estimates of at least 30% for any PD and 3–4% for specific PDs (Torgersen, 2012). Clinical samples of adults display high current rates of PDs, with an estimate range of 46–81%, and with estimates as high as 51–88% when PD-NOS is included (Torgersen, 2012).

The data on prevalence in youth are more limited, but they point to the likelihood that PDs may be slightly more prevalent earlier in life than in adulthood. Several studies with youth suggest that the rates of PDs may be higher in early and middle adolescence (Bernstein et al., 1993; Johnson, Cohen, Dohrenwend, Link, & Brook, 1999; Zaider, Johnson, & Cockell, 2000) than is typical in later adolescence and adulthood, although one unusual study found very low rates of PDs in a community sample of adolescents ages 14–18 (Lewinsohn, Rohde, Seeley, & Klein, 1997). The Children in the Community study has particularly helpful data on this issue because it has tracked the prevalence of PDs assessed by interviews in the same sample across time (Johnson, Cohen, Kasen, Skodol, & Oldham, 2008). The study obtained the following point prevalence rates: age 14, 14.6%; age 16, 12.7%; age 22, 13.9%; and age 33, 12.7%. The finding of slightly higher PD prevalence rates earlier in adolescence in several samples is consistent with findings that pathological personality traits are at highest levels during adolescence, as described later in this chapter. In adulthood, Cluster B PDs are more prevalent earlier in adulthood than later in adulthood (Torgersen, 2012). Interestingly, the Children in the Community study found that the Cluster B PDs were the most common PDs in adolescence (Johnson, Cohen, Kasen, Skodol, et al., 2000); this suggests that the Cluster B PDs may be most prevalent earlier in life, particularly adolescence. Two studies of PDs in clinical samples of adolescents indicate that, as in adult samples, rates of PDs are high, with estimates falling between 41 and 64% (Feenstra, Busschbach, Verheul, & Hutsebaut, 2011; Grilo et al., 1998). Taken together, the more limited data on youth indicate that (1) PDs are at least as common in adolescence as in adulthood; (2) the Cluster B PDs may be more prevalent in adolescence than in adulthood; (3) the Cluster C PDs may be more prevalent in adulthood than in adolescence; and (4) PDs are extremely common in clinical samples of youth.

As with general prevalence rates for PDs, much more is known about gender differences in PD rates in adults than in adolescents. Although the overall prevalence rates for PDs appear to be roughly equal for adult males and females, some specific PDs may be more prevalent in one gender or the other (Oltmanns & Powers, 2012; Paris, 2007; Torgersen, 2012). In adult community samples, ASPD is much more common in men (Torgersen, 2012), with rates five times as high in men than in women (Magnavita, Powers, Barber, & Oltmanns, 2013; Oltmanns & Powers, 2012). Dependent PD is more common in women (Torgersen, 2012). Other differences are less certain: Narcissistic PD and obsessive–compulsive PD may be more common in men, and histrionic PD and avoidant PD may be more common in women (Torgersen, 2012), but most gender differences in prevalence are nonexistent, small, or inconsistent across studies (Oltmanns & Powers, 2012). It is especially notable that rates of BPD do not appear to differ consistently by gender. The few cases where there are gender differences appear to reflect gender differences in related personality traits (Oltmanns & Powers, 2012; Paris, 2007); on average, men tend to be higher in assertiveness and excitement seeking, whereas women tend to be higher on facets of the higher-order factors Neuroticism and Agreeableness. In short, gender differences in adult PDs are not as common or as large in community samples of adults as often assumed.

The limited available information on community samples of PDs and PD traits in youth suggests that gender differences in prevalence rates or levels of symptoms are likewise small or nonexistent (Bernstein et al., 1993; see the review for BPD traits in Belsky et al., 2012), other than the consistent finding that conduct problems are more prevalent in samples of males (Moffitt, Caspi, Rutter, & Silva, 2001). Although gender differences in prevalence rates are typically small in adults and potentially small in youth, gender still seems to have an important impact on the manifestations of

specific PDs in adults (Oltmanns & Powers, 2012), and the same is likely to be true for youth as well. For example, although adolescent girls with BPD show correlates similar to those of adults with BPD, boys with BPD tend to be more disruptive and antisocial (Bradley, Conklin, & Westen, 2005). Clearly, this is an issue requiring more research in samples of youth.

Unfortunately, even less is known about variability in prevalence rates of PDs by ethnicity, race, or culture in adults and youth (Magnavita et al., 2013; Mulder, 2012). A recent meta-analysis compared rates of adult PDs across racial groups and found slightly lower rates among black than white populations, but no differences among white, Asian, and Hispanic populations (McGilloway, Hall, Lee, & Ghui, 2010); however, the studies included in the meta-analysis had significant limitations. There are no epidemiological studies of prevalence rates for PDs across cultures (Mulder, 2012). However, the existing evidence suggests that ASPD is found in all cultures studied, though the prevalence rates vary (Mulder, 2012); other PDs have been identified in most cultures, but again, prevalence rates vary. More work is needed to understand the validity of the use of PD diagnoses across cultures and to determine prevalence rates, if PDs are valid diagnostic categories in those cultures.

Comorbidity among PDs and between PDs and Other Psychiatric Disorders

Comorbidity appears to be the rule rather than the exception among PDs in both adolescents and adults. There tends to be a high level of comorbidity among PDs in epidemiological samples of adults (Skodol, 2005; Trull et al., 2012); in fact, it is relatively uncommon for an adult to have only one PD, and this is even rarer in clinical samples (Trull et al., 2012). In adult samples, BPD, paranoid PD, and dependent PD show the highest rates of co-morbidity with other PDs, and ASPD and obsessive–compulsive PD show the lowest rates (Trull et al., 2012). In contrast to the substantial literature on comorbidity among PDs in adults, there are surprisingly few studies of such comorbidity in youth. The Children in the Community study has not reported specific rates of PD comorbidity, but Cohen, Crawford, and colleagues (2005) noted that in this sample, "There is relatively high comorbidity and correlation among the criteria counts for the PDs" (p. 470). Becker, Grilo, Edell, and McGlashan (2000) reported that a sample of hospitalized adolescents with BPD showed unusually high rates of comorbidity with Cluster A and Cluster C PDs, compared to a comparison sample of adults. Similarly, De Clercq and colleagues (2004) found unusually high rates of overlap among PD symptoms in their adolescent sample. Future work should address the question of whether comorbidity among PDs is especially high in youth.

There is also a high rate of concurrent comorbidity between PDs and other psychiatric disorders in both adults (Links, Ansari, Fazalullash, & Shah, 2012) and adolescents (Cohen, Crawford, et al., 2005; Feenstra et al., 2011; Grilo et al., 1998). All three clusters of PDs in adolescence show high rates of comorbidity with other psychiatric disorders, including depressive, anxiety, and disruptive behavior disorders (Cohen, Crawford, et al., 2005), and PDs are associated with substance use problems as well (Serman, Johnson, Geller, Kanost, & Zacharapoulou, 2002). Adolescent PD-NOS also shows high comorbidity with non-PD conditions (Johnson et al., 2005). Furthermore, earlier disorders, including anxiety, depression, and disruptive behavior disorders, predict heightened risk for later emergence and continuation of PDs into adulthood (Cohen, Crawford, et al., 2005; Goodwin, Brook, & Cohen, 2005; Lewinsohn et al., 1997). The reverse is true as well: Earlier PDs predict greater risk for other early adult psychiatric disorders, including depressive, anxiety, and substance use disorders (Cohen, Chen, Crawford, Brook, & Gordon, 2007; Cohen, Crawford, et al., 2005; Daley et al., 1999; Levy et al., 1999), sometimes even after the presence of earlier PDs and other disorders is taken into account. In addition, when PDs co-occur with other psychiatric disorders in adolescence, the likelihood of the PDs' continuing into adulthood is increased (Cohen, Crawford, et al., 2005). It appears that there is often a transaction between PDs and other disorders across the years from adolescence to adulthood, with other psychiatric disorders contributing to the expression of PDs and vice versa.

Some patterns of associations between PDs and other disorders seem to be especially common for particular clusters of PDs. First, not surprisingly, Cluster A PDs seem especially associated with psychotic disorders, but they are associated with other disorders as well. Adolescents who exhibit schizotypal PD and who meet prodromal criteria for psychotic disorders show higher rates of transition to disorders with psychotic features (Correll et al., 2008); this finding is consistent with the idea that genetic risk for schizophrenia predisposes individuals to develop schizotypal PD (Fanous et

al., 2007). We discuss research supporting the idea that shared genetic factors underlie vulnerability to Cluster A PDs and psychotic disorders in the section on the etiology of the Cluster A PDs. Adolescent disruptive behavior disorders predict heightened risk of schizoid PD, and adolescent anxiety disorders predict risk of paranoid PD (Kasen et al., 2001). The persistence of Cluster A PDs from adolescence to adulthood is much greater in the presence of anxiety disorders than the persistence of Cluster B and C PDs' co-occurring with anxiety (Cohen, Crawford, et al., 2005).

Second, several lines of evidence indicate that Cluster B PDs show especially strong links with disruptive behavior, substance abuse, and depression. Cluster B PDs are substantially more stable when they co-occur in adolescence with disruptive behavior disorders or depression (Kasen, Cohen, Skodol, Johnson, & Brook, 1999); adolescent Cluster B PDs predict higher risks of substance abuse in adulthood (Cohen et al., 2007); and disruptive behavior disorders predict increased risks for Cluster B disorders (Cohen, Crawford, et al., 2005). Young adolescents with high levels of BPD traits also display heightened rates of other disorders—particularly depression, but also conduct disorder, psychosis, and anxiety disorders (Belsky et al., 2012). Childhood ADHD and oppositional defiant disorder predict heightened risks for BPD symptoms in early adulthood (Burke & Stepp, 2012; Stepp, Olino, Klein, Seeley, & Lewinsohn, 2013), and conduct disorder and anxiety disorders sometimes do as well (Stepp et al., 2013). A recent longitudinal twin study of adolescents found that although shared/familywide environmental influences accounted for an association between BPD and substance use at age 14, the association was accounted for by shared genetic factors at age 18 (Bornovalova, Hicks, Iacono, & McGue, 2013). Taken together, the findings across these studies suggest that the Cluster B disorders, particularly BPD, show strong links with externalizing and internalizing disorders both concurrently and across time.

Third, the more limited research on the Cluster C PDs suggests that they seem to show fewer specific links with other psychiatric disorders, but rather exhibit various associations with disruptive behavior disorders, depressive disorders, and anxiety disorders over time (Cohen, Crawford, et al., 2005). Major depression in adolescence predicts adult dependent PD (Kasen et al., 2001). Although the Cluster C PDs show strong co-occurrence with anxiety disorders in adolescence, they do not predict later anxiety disorders after controls

for earlier ones, although they do predict later disruptive behavior disorders (Johnson, Cohen, Skodol, et al., 1999).

Numerous researchers have suggested that the high rates of comorbidity among the PDs, and between the PDs and other disorders, indicate that genetic factors and personality traits are likely to underlie these co-occurrences (Clark, 2005, 2007; De Fruyt & De Clercq, 2012; Krueger, 2005; Krueger & Markon, 2008). Other psychiatric disorders probably include a strong component of personality functioning; these disorders would be better understood by considering their associations with personality functioning. There is evidence that symptoms of other psychiatric disorders are linked with PD traits in childhood (Mervielde et al., 2005): Antagonism and Disinhibition with externalizing symptoms, and Negative Affectivity and Detachment with internalizing symptoms. As we have discussed in the section on the trait models of PDs, disorders may co-occur because they arise from shared genetic sources and personality traits. For example, an adult twin study found evidence for a common genetic liability influencing the co-occurrence of major depression and dimensional representations of paranoid PD, BPD, and avoidant PD (Reichborn-Kjennerud et al., 2010); it seems possible that the genes influencing all of these conditions do so by shaping propensities toward Negative Affectivity.

The high rates of overlap between PDs and other psychiatric disorders suggest that the two types of disorders are not nearly as distinct as originally conceived. Empirical research on this topic almost certainly played some part in the decision to remove Axis II from DSM-5 and to put the categorical PDs in Section II with the rest of the disorders. Although PDs and other disorders show significant overlap in many respects, it is important to recognize that they may still differ somewhat, with PD traits being more stable and the symptoms of other disorders being more episodic. Improvement in PDs is typically more likely to lead to improvement in other conditions than the reverse (Clark, 2005). An adult twin study of the genetic and environmental structure of PDs and other psychiatric disorders in DSM-IV provided further evidence for the distinction between these two groups of disorders (Kendler et al., 2011). The results indicated four genetic factors that accounted for the observed covariation among disorders: Axis I internalizing (somatoform disorder, panic disorder, major depression, agoraphobia, specific phobia, generalized anxiety disorder, eating disorders); Axis

II internalizing (dysthymia, schizoid PD, schizotypal PD, avoidant PD, social phobia); Axis I externalizing (ASPD, drug abuse/dependence, conduct disorder, alcohol abuse/dependence); and Axis II externalizing (histrionic PD, narcissistic PD, obsessive–compulsive PD). Paranoid PD and dependent PD were related to the genetic factors for both internalizing and externalizing Axis II, and BPD was related to the genetic factors underlying Axis I and II externalizing disorders and an environmental factor underlying Axis I internalizing disorders. These results suggest that different genetic factors may underlie many of the PDs versus the other psychiatric disorders. The relationships among PDs, other psychiatric disorders, and personality traits in childhood and adolescence will be an especially exciting direction for future research.

COURSE: STABILITY AND LIFE OUTCOMES

Stability of PD Diagnoses/Traits and Pathological Personality Traits

Embedded in the DSM-IV and DSM-5 Section II PD diagnoses are some explicit claims about the stability and course of PDs. Specifically, in these diagnostic models, the PDs are described as *enduring* patterns that start by adolescence or early adulthood, and the patterns need to have existed for at least a year to warrant diagnosis in youth under age 18. These older views of PD have been challenged by a number of longitudinal studies that have examined the stability and course of PD diagnoses and symptoms in both youth and adults. These more recent studies have demonstrated that although PD symptoms show moderate rank-order stability by adolescence, PD diagnoses themselves are less stable than previously assumed. The findings for PD diagnoses and symptoms can be understood in light of recent research on the stability of normal-range personality traits over time. The newer view of PDs is reflected in the DSM-5 Section III requirement that PDs be only *relatively* stable over time.

Rank-Order Stability

Personality stability is itself a complex notion because there are many different kinds of continuity and change (Caspi & Shiner, 2006). First, "rank-order stability" refers to the degree to which the relative ordering of individuals on a given trait is maintained over time. Rank-order stability is high if people in a group maintain their position on a trait relative to each other over time, even if the group as a whole increases or decreases on that trait over time. It is typically indexed by correlations between scores on the same trait measured across two points in time (i.e., test–retest correlations). PD symptoms in adolescents and young adults display moderate to strong levels of rank-order stability across time, often in the range of .40–.65 (Bornovalova et al., 2013; Cohen, Crawford, et al., 2005; Crawford et al., 2005; Daley et al., 1999; Ferguson, 2010; Frick & White, 2008; Johnson, Cohen, Kasen, et al., 2000; Winograd, Cohen, & Chen, 2008); these are similar to the levels of PD symptom stability observed in adulthood (Clark, 2007, 2009; Ferguson, 2010; Grilo & McGlashan, 2005). Less is known about the rank-order stability of PD symptoms in childhood, but two studies suggest that PD symptoms and pathological traits may show similar levels of moderate to strong rank-order stability over periods of 1 and 2 years in childhood (Crick, Murray-Close, & Woods, 2005; De Clercq, Van Leeuwen, Van Den Noortgate, De Bolle, & De Fruyt, 2009). The De Clercq, Van Leeuwen, and colleagues (2009) study of pathological traits also found high within-person stability, meaning that the absolute levels of PD traits of each individual in the study tended to remain high.

The results for the rank-order stability of PD symptoms in youth parallel those found for normal-range personality traits. Personality traits are already moderately stable by childhood (Roberts & DelVecchio, 2000), but become increasingly stable from childhood through adolescence (Ferguson, 2010; Shiner, 2014). A recent meta-analysis demonstrated that the same is true for normal and pathological personality traits in adulthood, in that both kinds of traits show high levels of stability (Ferguson, 2010). The findings for rank-order stability of PD symptoms, pathological traits, and normal-range traits converge on a shared conclusion: There is nothing transformative about the age of 18 with regard to stability of PDs measured dimensionally. Moderate to strong stability is already apparent by adolescence and may already be in place by late childhood and early adolescence.

Mean-Level Stability

Second, "mean-level change" refers to increases or decreases in the average trait level of a population as a whole. In other words, investigations of mean-level

change address the question of whether people, on average, tend to increase or decrease on particular trait or symptom measures during different periods of life. In terms of mean-level change, findings from the Children in the Community study suggest that levels of PD symptoms may peak in adolescence and then decline across the years of later adolescence and early adulthood (Cohen, Crawford, et al., 2005; Johnson, Cohen, Kasen, et al. 2000). Narcissistic symptoms showed the greatest decline from adolescence to adulthood (Cohen, Crawford, et al., 2005; see also Carlson & Gjerde, 2009), whereas obsessive–compulsive symptoms did not decline at all (Cohen, Crawford, et al., 2005). A short-term study of pathological personality traits in childhood found slight mean-level decreases in such traits (except for Introversion) across 1 and 2 years in later childhood (De Clercq, Van Leeuwen, et al., 2009). BPD traits have been found to decline modestly from ages 14 to 18 (Bornovalova et al., 2013). Findings from longitudinal studies of adults suggest that PD symptom levels and pathological trait levels continue to decline in adulthood as well (Clark, 2007). Recent research in older adults, however, has called this finding somewhat into question (Cooper, Balsis, & Oltmanns, 2014). Specifically, Cooper and colleagues found that the pattern of declining PD symptoms over time only held when self-reports were examined; informant reports of PD symptoms actually showed slight increases over time. This study raises interesting questions about potentially confounding measurement factors in such studies, and challenges the general notion that PD symptoms decline across adulthood.

These findings for mean-level change are generally consistent with results for mean-level change of normal personality traits from childhood through adulthood, and these mean-level changes in normal-range traits may help to explain changes in the prevalence rates of PDs over time. In fact, a recent study demonstrated that mean-level changes in aspects of the Big Five traits from adolescence through later adulthood could explain parallel mean-level changes in psychopathy (a construct discussed later in this chapter) and its prevalence in forensic samples (Vachon et al., 2013). The studies on mean-level trait changes in childhood and early adolescence are not entirely consistent, but there is some evidence that although children develop better emotional self-regulation and greater Conscientiousness and Agreeableness across the childhood years (Shiner, in press), youth may show mean-level decreases in these positive traits in the transition from childhood to adolescence, followed by increases in those traits later in adolescence (Shiner, 2014; see, e.g., Soto, John, Gosling, & Potter, 2011). Across the late adolescent and early adult years, there is a movement toward greater personality maturity on average. Neuroticism decreases in young adulthood, and Agreeableness and Conscientiousness increase in young adulthood and middle age (Roberts, Walton, & Viechtbauer, 2006). Given that many PDs are characterized by high Neuroticism and low Agreeableness and Conscientiousness, it is not surprising that on average, PD symptoms may peak in early or mid-adolescence and later decline.

The positive growth in personality traits from late adolescence through adulthood is accounted for in part by young adults' greater investment in socially important roles as spouses or partners, workers, and parents (Lodi-Smith & Roberts, 2007). However, it is important to recognize that not all people benefit from increased personality maturity as they enter adulthood (Roberts, Wood, & Caspi, 2008). Rather, some people show changes in their personality traits in more negative directions. People who lack normative experiences with adult roles may be particularly vulnerable to such negative changes in personality (Roberts et al., 2008). Given that PDs in adolescence put youth at risk for problems with developmental tasks in the transition to adulthood, it is likely that youth struggling with personality pathology may sometimes miss out on the beneficial effects of adopting more adult roles. This is consistent with evidence that the transition from late adolescence to early adulthood represents a critical developmental period for PDs, and a time when individuals with PD diagnoses grow increasingly deviant from their peer group (Clark, 2005; Tackett et al., 2009).

Stability of PD Diagnoses

Finally, the stability of PD diagnoses over time is important for understanding the nature of PDs. If a person meets criteria for a particular PD, is it likely that the person will still warrant that diagnosis over time? Contrary to what might be expected from the classic view of PDs represented in all of the previous DSMs, the stability of particular PD diagnoses appears to be relatively modest in samples of adolescents (Bernstein et al., 1993; Chanen et al., 2004; Cohen, Crawford, et al., 2005; Daley et al., 1999; Mattanah, Becker, Levy, Edell, & McGlashan, 1995) and adults (Clark, 2007, 2009; Grilo & McGlashan, 2005; Skodol et al., 2005; Zanarini, Frankenburg, Hennen, Reich, & Silk, 2005).

This relatively modest stability is probably due to several causes. First, it may result in part from the categorical system used for diagnosis; patients can switch from having a PD to not having one, simply because they exhibit one or two symptoms fewer for a particular PD. Second, the instability in diagnoses also reflects the mean-level changes in PD symptoms and traits; as mean levels of PD symptoms and traits decline, these mean-level changes lead to changes in PD diagnoses over time as well (Clark, 2009). Third, the surprising remission rates also reflect the nature of PDs, in that there are more and less stable aspects to PDs (Clark, 2007; Skodol et al., 2005; Zanarini et al., 2005). The less stable aspects typically involve more acute behaviors, such as odd behavior, self-harm, or avoidance of particular situations; in contrast, the more stable aspects involve personality traits underlying the condition, such as the paranoid ideation seen in schizotypal PD or the feelings of inadequacy and social ineptness in avoidant PD (McGlashan et al., 2005). BPD similarly includes more acute aspects (substance abuse, chaotic relationships) and more temperamental, chronic aspects (anger and odd thinking) (Hopwood, Donnellan, & Zanarini, 2010). As the more acute aspects of PDs resolve over time, people may no longer qualify for PD diagnoses, even if the more chronic aspects remain in place.

It is important to note that despite the general improvements that typically occur in personality functioning, there may be some individuals whose PD symptoms worsen in adolescence and adulthood and become a more persistent pattern. In the Children in the Community study, adolescents with PD diagnoses frequently continued to display high PD traits in early adulthood (Johnson, Cohen, Kasen, et al., 2000), and a fifth of the youth showed an increase in PD symptoms over the decade from midadolescence through early adulthood (Cohen, Crawford, et al., 2005). In the short-term longitudinal study of older children's pathological personality traits described previously, the children who started with the highest levels of pathological traits exhibited less pronounced declines in those traits than the rest of the sample (De Clercq, Van Leeuwen, et al., 2009). And although rates of continuity may be low for specific PD diagnoses, there is some evidence that adolescent patients with a PD diagnosis may still be at higher risk of having *any* PD diagnosis over time (Chanen et al., 2004; Cohen, Crawford, et al., 2005). These youth with non-normative development in PD symptoms may be the ones who especially need research and clinical attention.

Life Outcomes Associated with PDs

Although data on some aspects of PDs in youth are sparse, there is a convincing literature about the negative life outcomes predicted by PDs earlier in life. In a previous section, we have described research indicating that an adolescent PD heightens the chances that a youth will develop a non-PD condition in adulthood. Youth PDs increase vulnerability for the development of a wide variety of other harmful and potentially risky behaviors as well. PDs from Clusters A and B in adolescence predict risks for adolescent and adult violence—including acts such as "arson, assault, breaking and entering, initiating physical fights, robbery, and threats to injure others" (Johnson, Cohen, Smailes, et al., 2000, p. 1406)—and for violence against romantic partners (Ehrensaft, Cohen, & Johnson, 2006), even when possible confounding variables are taken into account. Paranoid and narcissistic symptoms in particular are associated with later violence and criminality, perhaps because they fuel the suspiciousness and entitlement that often precipitate aggression (Cohen, Crawford, et al., 2005). Adolescents with PDs are also at heightened risk for having high numbers of sexual partners and for high-risk sexual behaviors more generally (Lavan & Johnson, 2002). Adolescent PDs from all three clusters are predictive of heightened risk of suicidal ideation or attempts in early adulthood (Brent, Johnson, Perper, & Connolly 1994; Johnson, Cohen, Skodol, et al., 1999). Nonsuicidal self-injury (NSSI) may also be present in youths with PDs; NSSI may take the form of cutting, burning, or punching oneself (Nock, 2010; see Cha & Nock, Chapter 7, this volume). A study of adolescent inpatients found that two-thirds of the patients who had engaged in NSSI prior to admission met criteria for a PD (Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006). Suicide and NSSI seem to be particularly associated with BPD, in that adolescent suicide attempts and NSSI are associated with the number of borderline symptoms (Jacobson, Muehlenkamp, Miller, & Turner, 2008), and adolescent inpatients with BPD are more likely to have experienced suicidal ideation earlier in life and with more frequency than psychiatric controls (Venta, Ross, Schatte, & Sharp, 2012). A study of adult patients with BPD found that, among the patients who had engaged in self-mutilation, approximately one-third reported having started harming themselves as children, and another third reported having started as adolescents (Zanarini et al., 2006). Taken together, the evidence suggests that particular adolescent PDs pose

risks in terms of violence, criminality, high-risk sexual behaviors, suicide attempts, and NSSI.

Beyond the effects of PDs on symptomatology and risky behaviors, there is evidence that adolescent PDs are associated with risks for problems with adaptation, both concurrently and later in adulthood. Adolescent PDs put youth at risk for later overall impairment in adulthood (Skodol, Johnson, Cohen, Sneed, & Crawford, 2007) and are associated with high health care costs and reduced quality of life among patients, especially when accompanied by a non-PD condition (Feenstra et al., 2012). Some PDs in both adolescents and adults seem to be associated with higher risks of impairment (e.g., BPD and schizotypal PD), whereas others seem to be associated with relatively little overall impairment (e.g., histrionic PD, narcissistic PD, and obsessive–compulsive PD) (Chen et al., 2006; Torgersen, 2012). Adolescent PDs and traits pose heightened risks for later conflicts with family members (Johnson, Chen, & Cohen, 2004); difficulties with child rearing in middle adulthood (Johnson, Cohen, Kasen, & Brook, 2008); and problems with romantic relationships, including stressful relationships, conflicts, and low partner satisfaction (Chen et al., 2004; Daley, Hammen, Davila, & Burge, 1998; Johnson et al., 2005; Winograd et al., 2008). Adolescents with PDs also have heightened rates of problems in other domains of life, including difficulties in friendships, few social activities, poor educational achievement, and work difficulties (Bernstein et al., 1993; Johnson et al., 2005; Winograd et al., 2008).

The Children in the Community study has identified several patterns of outcomes for the three clusters of PDs. The adolescents with high levels of Cluster A symptoms displayed the greatest degree of impairment in the transition from adolescence to adulthood (Cohen, Chen, et al., 2005). This is probably attributable to the fact that Cluster A symptoms may reflect vulnerability to symptoms of schizophrenia for some people. The participants in the Children in the Community study were asked to provide life narratives describing themselves in various roles and social settings, and these narratives revealed worse trajectories in terms of education and achievement. In addition, Cluster A symptoms in adolescence predicted a greater likelihood of teenage parenting (Cohen, Chen, et al., 2005) and higher levels of partner conflict through age 23 (Chen et al., 2004). Fortunately, some of the adolescents with high Cluster A symptoms did better in terms of life adaptation in the transition to adulthood, and this then predicted a decline in Cluster A symptoms over time (Cohen, Crawford, et al., 2005). Cluster B symptoms showed particular relevance for romantic relationships because of their links with identity disturbance (Crawford, Cohen, Johnson, & Sneed, 2004); specifically, adolescent Cluster B symptoms were associated with lower well-being and intimacy in relationships in adolescence, and the negative association with intimacy became stronger in adulthood. Cluster A symptoms in adolescence predicted heightened partner conflict over the next decade (Chen et al., 2004). In contrast, although youth with high levels of Cluster C symptoms were less likely to develop romantic relationships, those in romantic relationships showed higher levels of conflict until age 23 only, and then later showed even lower levels of conflict than was typical (Cohen, Crawford, et al., 2005). Thus, although most PDs are associated with some degree of impairment, the patterns of problematic adaptation may vary according to the symptoms a youth displays.

All of the findings for PDs and adaptation are consistent with research on personality in childhood and adolescence more generally; youth's personalities are predictive of many important life outcomes, including peer relationships, formation of romantic relationships, academic attainment, effectiveness at work, and health (Caspi & Shiner, 2006; Zentner & Shiner, 2012). The effects of PDs on the critical developmental tasks of adolescence and young adulthood—developing friendships and romantic relationships, and developing skills for education and work—may be one of the most negative outcomes of PDs in youth. Impairment may be quite stable, even when PD symptoms change (Clark, 2007, 2009). The risks for later impairment well into adulthood are as high for PDs as for other psychiatric disorders in adolescence (Crawford et al., 2008); the combination of PDs and non-PD conditions in adolescence is even more problematic for adult outcomes. The more persistent PDs are in adolescence, the greater the adaptive impairment in adulthood is likely to be (Skodol, Johnson, et al., 2007).

Despite the seemingly gloomy picture for adolescent PDs, it is important to recognize that not all youth with PDs suffer clear-cut impairment (Cohen, Crawford, et al., 2005; Johnson et al., 2005). Fortunately, some youth with PDs improve in their functioning as they age (Cohen, Crawford, et al., 2005). There appear to be transactions between youth's PD symptoms and their adaptation. Positive adaptation in school and in relationships can lead to improvements in some PD

symptoms over time (Skodol, Bender, et al., 2007). As PD symptoms improve, youth's sense of well-being may correspondingly improve as well (Crawford et al., 2004). Conversely, problems with adaptation are likely to cause and perpetuate PD symptoms. Poor school achievement, being suspended from school, and repeating a grade all predict later adolescent PD symptoms (Cohen, Crawford, et al., 2005). Similarly, young adults who perpetrate partner violence are less likely to experience the positive declines in PD symptoms that occur normatively during this span of life (Ehrensaft et al., 2006). The interaction between personality pathology and impairment is likely to be complex.

ETIOLOGY

Genetic, Family, and Broader Contextual Influences

As is true for many other topics in the study of PDs in youth, empirical research on the developmental pathways leading to the development of PDs is more limited than would be desirable. Much of the early clinical interest in PDs in the 20th century arose from rich, complex psychodynamic theories about the origins of these conditions. Most of these etiological theories were based on clinicians' discussions with their patients about their early histories. Although these theories spurred interest in PDs and provided a basis for interventions, relatively little is known empirically about the developmental pathways leading to many of the PDs. Nonetheless, there are several promising leads for potential causes of PDs. Because we have provided an overview of the personality characteristics likely to be involved in the development of PDs earlier in this chapter (see our description of the research on temperament and personality traits, mental representations [including attachment and social-cognitive processes], emotion regulation and coping, and life narratives), we focus here instead on three other potential contributors: genetic influences, experiences within the family, and broader contextual factors (peers, schools, socioeconomic resources, and cultural influences). We focus in this section on a discussion of the etiology of PDs in youth in general. Following this section, we turn to a review of research on the etiology of Cluster A PDs, BPD, ASPD/psychopathy/narcissism, and Cluster C PDs.

There has been some research on the neurobiological correlates of many of the PDs in adults, as well as on the neurobiological basis of many of the dimen-sions associated with the different clusters of PDs (e.g., psychotic-like perceptual distortions in Cluster A and affective instability in Cluster B; Roussos & Siever, 2012). Relatively few studies have examined the neurobiological basis of most of the PDs in youth; however, we review briefly the existing neuroscience research on schizotypal PD, BPD, and psychopathy in the relevant sections.

Genetic Influences

Most people who experience adversity do not go on to develop PDs. This simple finding suggests that there are almost certainly genetic factors that shape vulnerability to developing personality pathology in the face of adverse experiences. Thus far, three twin studies have been conducted to examine the genetic and environmental contributions to individual differences in PD symptom counts for all 10 PDs listed in DSM-IV (South, Reichborn-Kjennerud, Eaton, & Krueger, 2012). One of these examined parent reports of PD symptoms in children (Coolidge, Thede, & Jang, 2001) and obtained heritability estimates ranging from .50 to .81, with no shared/familywide environmental effects and with moderate effects of the nonshared/child-specific environment on PD symptoms. The other two studies examined PD symptoms in adult twin samples (Kendler et al., 2006; Reichborn-Kjennerud et al., 2007; Torgersen et al., 2000, 2008). The average heritability of PD symptoms obtained across these three studies was .4–.5, indicating moderate heritability, and the studies have been consistent in finding only limited shared or familywide environmental effects (South et al., 2012). Estimates of heritability for PD traits in adults are roughly similar in magnitude to those for PD symptoms (Cloninger, 2005; Livesley, 2005). These behavior genetic findings for PD symptoms and traits are consistent with findings for temperament and personality traits in childhood (Saudino & Wang, 2012) and personality traits in adulthood (Krueger & Johnson, 2008; South et al., 2012). Although more research is needed before firm conclusions can be drawn, the existing data suggest that genetic influences on PD symptoms, PD traits, and normal-range personality traits are moderate in size, and that environmental differences account for a substantial portion of the variation as well. What environmental experiences tend to do, however, is to create differences in PD outcomes between children growing up in the same family, rather than to make siblings more alike.

An adult multivariate twin study by Kendler and colleagues (2008) examined the genetic and environmental influences on the co-occurrence of symptoms of the 10 DSM-IV PDs. Three genetic risk factors were identified: first, one accounting for the general risk for PDs (interpreted by the authors as most likely to be a propensity for Negative Affectivity); second, one influencing BPD and ASPD (interpreted as reflecting high Disinhibition and Antagonism); and, third, one influencing schizoid and avoidant PDs (interpreted as reflecting high Detachment). These three genetic risk factors appear likely to be linked with four of the five domain-level pathological personality traits in the DSM-5 Section III PD diagnoses. In addition, three nonshared/person-specific environmental factors accounted for the associations among the disorders within each of the three clusters of PDs (Clusters A, B, and C). In other words, similar nonshared/person-specific environmental factors influenced all of the disorders within each cluster. Finally, multiple genetic and nonshared/person-specific environmental factors contributed to each of the PDs. This study suggests that genetic factors do not contribute to the co-occurrence of PDs within clusters, but environmental experiences that shape PDs within clusters may do so. These results point to three important areas for future investigation: the developmental influences on the basic pathological personality dimensions; the environmental factors that shape disorders within the three clusters; and the specific genetic and environmental sources of variation in more narrowly defined aspects of personality pathology.

Finally, it is important to note that molecular genetic techniques have been used in an attempt to identify some of the specific genes responsible for genetic influences on PDs and normal-range personality traits. At this point, the results of molecular genetic research on these topics has been disappointing, in that replicable molecular genetic influences have not been identified, or only trivial amounts of variance in outcomes have been accounted for (South et al., 2012).

It is not clear yet which individual differences are the mediators through which genes influence the development of PDs. The personality differences described previously in this chapter may be one such mediator. Some other individual differences have been identified as risk factors for the development of PDs, including "low IQ, poor achievement, having been suspended or expelled from school, having repeated at least one grade, and not being goal directed" (Cohen, Crawford, et al., 2005, p. 471). These other individual differences, which may reflect different aspects of cognitive and executive functioning, are other individual differences beyond personality worthy of investigation as vulnerability factors for the development of PDs.

Family Influences

The behavior genetic research points to the importance of environmental experiences in the development of PD symptoms and traits. Among the most likely sources of environmental influence on PDs are youth's experiences within their families. Although there have been many theories about the ways that families influence the development of PDs, there were few data on this topic until the last 15 years. Many of the studies have focused on the role of the family in the development of particular PDs, and we address that research in the following sections. However, some studies have looked at the family effects across all of the PDs.

Maladaptive parenting generally poses risks for the development of PDs in early adulthood; such maladaptive parenting includes low parental affection or nurturing and aversive parental behavior (such as harsh punishment) (Johnson, Cohen, Chen, Kasen, & Brook, 2006). The greater the number of negative parental behaviors, the higher the risk for young adult PDs (Johnson et al., 2006). Other family risks for PD development include single parenthood, parental conflict, and parental psychiatric disorders (Cohen, Crawford, et al., 2005); separation from parents, particularly before the age of 5 (Lahti et al., 2012); and parental suicide attempts or completion, parental history of being jailed, and history of a battered mother (Afifi et al., 2011).

There is now longitudinal evidence that childhood abuse (including sexual, physical, and verbal abuse) and neglect predict heightened risk for the later development of PDs (Johnson, Cohen, Brown, Smailes, & Bernstein, 1999; Johnson et al., 2001; Johnson, Smailes, Cohen, Brown, & Bernstein, 2000). Retrospective reports also suggest that adults with PDs reported having been maltreated at higher rates than adults without PDs (see, e.g., Afifi et al., 2011; Battle et al., 2004). A recent study of a nationally representative sample of adults found that childhood adversity, defined broadly as childhood maltreatment and household dysfunction, was particularly associated with schizotypal PD and most of the Cluster B PDs (Afifi et al., 2011). Many of these analyses linking adverse family experiences with adolescent or young adult PDs have controlled for

a variety of potential confounds, which strengthens the evidence for a potential causal role for family adversity in the development of PDs.

Negative experiences in the family may shape youth's emerging personality pathology through a number of processes. Children facing these adverse experiences lack the socialization experiences that normally help children learn how to follow societal rules, inhibit impulses, and regulate emotions and behavior (Bradley et al., 2011; Kim, Cicchetti, Rogosch, & Manly, 2009). Maltreatment may also undermine the development of healthy, realistic, and positive views of the self, others, and the self in relationship to others (Bradley et al., 2011; Feiring, Cleland, & Simon, 2010). Recent research has shown that parenting predicts changes in children's emerging personality traits. When parents fail to provide an environment that helps children manage negative emotions—specifically, when parents create an insensitive, punitive, chaotic, and hostile environment—children's negative emotionality tends to increase over time (Bates, Schemerhorn, & Petersen, 2012; Lengua & Wachs, 2012; Shiner, 2014). In addition, youth with poorer self-control are particularly negatively affected by adverse family environments (e.g., low maternal responsiveness, high parental punitiveness, single parenting) (Shiner, 2014). Thus family adversity may tend to promote a number of negative personality outcomes, including high Negative Affectivity and Disinhibition, troubled attachment styles, and more negative social-cognitive functioning.

Given that the behavior genetic research conducted thus far indicates a role for person-specific environmental influences on PD but not familywide environmental effects, it is important to note that the family experiences likely to be most relevant to the development of PDs are those that are unique to each youth in a family. Person-specific experiences within the family could include family events that are encountered by only one child in the family (e.g., separation from parents at a specific time, a specific parent–child relationship) or family events that are experienced uniquely by each child (e.g., parental psychopathology or marital conflict that is experienced uniquely by each sibling). In most of the studies looking at family predictors of PD, family factors are measured in a child-specific way (e.g., maltreatment of a specific child, affection toward a specific child). Other family factors are measured as familywide variables that are not specific to each child (e.g., parental suicide, socioeconomic status [SES]). It is possible that some of the familywide variables, such

as parental psychopathology, may predict the later development of youth PD not because the family factors are causing youth PD, but rather because the predictors (i.e., the familywide variables) and the outcomes (i.e., youth PD) are both the result of a third variable (e.g., genes shared between parents and offspring). As we note in the conclusion of this chapter, it will be important for future research to use sophisticated behavior genetic designs to tease apart these possibilities (the behavior genetic study by Belsky et al., 2012, described in the section on the etiology of BPD, provides an excellent example of such a study).

In addition, although family adversity poses significant risks for the development of personality pathology, it is crucial to recognize that early trauma and abuse are not present in the histories of all youths with PDs. In fact, in the Children in the Community Study, early trauma or abuse "do not account for all, or even most cases of PD observed in our longitudinal cohort" (Cohen, Crawford, et al., 2005, p. 482). Furthermore, even in cases of maltreatment, different children will be affected differently. In a recent study of adult PD, most of the participants who retrospectively reported a history of childhood maltreatment did not meet criteria for a PD (Afifi et al., 2011). These findings point to the importance of equifinality and multifinality in the links between family adversity and later PDs; we return to this topic in our final suggestions for future research on PDs.

Broader Contextual Influences

Beyond the family environment, there are also likely to be broader contextual factors influencing the development of PDs. First, peer relationships are an understudied potential contributor to the development of PDs in youth. Given that PDs involve difficulties in relationships, problematic peer relationships seem to be a likely influence on the emergence of PD symptoms. Peer relationships have been studied extensively in relation to the development of other disorders in childhood and adolescence (e.g., ADHD, conduct disorder, depression) (Deater-Deckard, 2013); aspects of peer relationships relevant to developmental psychopathology include social rejection/exclusion, lack of high-quality or the presence of poor-quality friendships, victimization/bullying, aggression, social withdrawal, peer contagion (adopting problematic behaviors from peers), and weaknesses in social skills. PD symptoms in early adulthood are predicted by a history of earlier social

isolation and low social competence (Cohen, Crawford, et al., 2005), and adolescent PDs are concurrently associated with shorter friendships, less enjoyment of others, lack of a confidant, and few social activities (Bernstein, Cohen, Skodol, Bezirganian, & Brook, 1996). Second, aspects of the school environment are likely to be relevant for the emergence and continuation of PD symptoms in childhood and adolescence. For example, students on average show declines in Cluster B PD symptoms in schools with a strong focus on learning (Kasen, Cohen, Chen, Johnson, & Crawford, 2009).

Third, the broader socioeconomic context (including family SES and poverty) seems likely to predict the development of PDs in youth. Adolescent PDs are associated with lesser parental education and lower occupational status and family income, even after researchers control for various potential confounds (Johnson, Cohen, Dohrenwend, et al., 1999), and adult PDs are linked with lower SES as well (Torgersen, 2012). Neighborhood-level characteristics may also influence PD symptoms (Hart & Marmorstein, 2009). There is considerable evidence linking poverty and low SES with difficulties in personality development and emotional and behavioral regulation more generally (Conger & Donnellan, 2007; Evans & Kim, 2013). Low SES, poverty, and risky neighborhoods are associated with declines in self-control in youth (Shiner, 2014). Fourth and finally, broader social forces (e.g., cultural values, customs, and mores accepted across societies or within societal subgroups) may be relevant to the development of PDs. For example, personality pathology characterized by poor constraint may be fostered in social contexts that do not provide structure or firm limits on the expression of impulsivity (Paris, 2005) or that offer lower levels of social cohesion (Millon, 2010). The very limited data on prevalence rates for Cluster B PDs indicate that ASPD and BPD may be more common in Western cultures, suggesting that there may indeed be significant cultural influences on these conditions (Mulder, 2012). Although there are good reasons to think that broader social contexts influence the development of PDs in youth, these potential contextual influences have received little attention in the literature on PDs and constitute an important direction for future research.

Etiology of Cluster A Disorders

The three Cluster A PDs—paranoid PD, schizoid PD, and schizotypal PD—are described in DSM-IV and DSM-5 as the "odd and eccentric" PDs. All three of these PDs involve a tendency to maintain distance in interpersonal relationships, although for different reasons in each case—distrust of and suspiciousness toward others in paranoid PD, emotional detachment from others in Schizoid PD, and discomfort with others in schizotypal PD (see Table 18.1 for more information). Although these three disorders do tend to co-occur frequently (Esterberg, Goulding, & Walker, 2010; Links et al., 2012; South et al., 2012), they also are frequently comorbid with avoidant PD in adolescents and adults (Esterberg et al., 2010; South et al., 2012); this is not surprising, given that avoidant PD is characterized by social inhibition and concerns about others' evaluations. Schizotypal PD and avoidant PD share genetic influences (Kendler et al., 2008). Thus avoidant PD is perhaps more appropriately studied in relation to the Cluster A PDs than in relation to the Cluster C PDs. At this point, there is far more research on schizotypal PD in both youth and adults than on the other two Cluster A PDs. Paranoid and schizoid PD are not included in the list of categorical PDs in DSM-5 Section III.

There is substantial evidence suggesting that the Cluster A PDs are schizophrenia spectrum disorders, meaning that they stem in part from the same genetic liabilities that predispose people to the development of psychotic disorders, including schizophrenia (South et al., 2012; see, e.g., Kendler et al., 2006). Schizotypal PD is the most closely and consistently linked with psychotic disorders, with paranoid PD and schizoid PD showing weaker and less consistent associations; schizotypal PD is even listed in the DSM-5 chapter on schizophrenia spectrum and other psychotic disorders, to indicate its close connection with this family of disorders. Schizotypal PD includes both positive symptoms (cognitive and perceptual abnormalities) and negative symptoms (social withdrawal, restricted emotions, lack of goal-directed behavior) seen in schizophrenia. Adolescent schizotypal PD that is accompanied by prodromal symptoms of schizophrenia heightens the risk of the later development of schizophrenia, schizoaffective disorder, or psychotic bipolar disorder (Correll et al., 2008), with one large-scale study indicating that approximately one-third of a sample of late adolescents with schizotypal PD developed schizophrenia within 2.5 years (Cannon et al., 2008). A small study of adolescents meeting criteria for schizotypal PD found that only about 40% of youth still met criteria for that disorder after a year; of those no longer meeting criteria for schizotypal PD, a third met criteria

for another PD, mostly paranoid or schizoid PD (Esterberg et al., 2010). This finding probably reflects the fact that the categorical diagnoses are unstable, but that the shared symptoms among the Cluster A disorders are more stable (Widiger, 2010). Taken together, the research on Cluster A disorders (especially schizotypal PD) and schizophrenia spectrum disorders suggests that these disorders have genetic influences and symptoms in common, but that numerous individuals who exhibit Cluster A PDs do not go on to develop clear-cut psychotic disorders.

Schizotypal PD in both adolescence and adulthood shares many of the cognitive, perceptual, and motor abnormalities seen in schizophrenia (Esterberg et al., 2010; Links et al., 2012). Schizotypal PD and schizotypy in adults are associated with a number of neurodevelopmental risk factors (Kwapil & Barrantes-Vidal, 2012): prenatal exposure to infection and malnutrition, obstetric complications, signs of prenatal androgen/estrogen disruptions (specifically, higher asymmetry in dermatoglyphic finger ridge counts), minor physical anomalies, and neurological "soft signs." Several neurodevelopmental risks have been identified in adolescents with schizotypal PD as well, including minor physical anomalies (Hans et al., 2009), neurological soft signs (Weinstein, Deforio, Schiffman, Walker, & Bonsall, 1999), and diminished gestural communication (Mittal et al., 2006). One large-scale prospective study found that signs of malnutrition at age 3 predicted lower performance IQ at age 11, which in turn predicted a heightened risk of schizotypal symptoms at age 23 (Venables & Raine, 2012). Although there have not yet been studies of brain anatomy and function in adolescents with schizotypal PD (to the best of our knowledge), research with adults points to several structural and functioning brain differences in adult schizotypal PD. Adults with schizotypal PD have been found to have structural abnormalities in the superior temporal gyrus, the posterior region of the fusiform gyrus, and the parahippocampus, whereas they seem to show fewer structural abnormalities than patients with schizophrenia in the frontal lobes and medial temporal lobes (Kwapil & Barrantes-Vidal, 2012). Adults with schizotypal PD likewise show diminished activation in the temporal lobes but more typical activation in the frontal lobes, perhaps accounting for the milder symptoms seen in schizotypal PD than in schizophrenia (Kwapil & Barrantes-Vidal, 2012). These findings have yet to be replicated in adolescents with schizotypal PD.

Several studies have examined a variety of nongenetic, experience-based contributors to schizotypal PD. Consistent with research linking early cannabis use with the development of schizophrenia, early cannabis use also predicts the development of schizotypal PD (Anglin et al., 2012). Early family predictors of schizotypal PD symptoms in adolescence and adulthood have also been identified; these include maternal separation in the first 2 years of life (Anglin, Cohen, & Chen, 2008) and high levels of family adversity, including abuse, neglect, and general household dysfunction (Afifi et al., 2011). Negative family experiences may potentially fuel the dissociation and interpersonal skill deficits observed in schizotypal PD. Low SES also predicts maintenance of schizotypal PD symptoms from adolescence through adulthood, in part through its effects on trauma, high stress, problematic parenting, and lower IQ (Cohen et al., 2008). Cluster A symptoms appear to decline more in schools that promote autonomy and minimize conflict and excessive informality among students and teachers (Kasen et al., 2009), and positive academic and social experiences in childhood or adolescence predict declines specifically in schizotypal symptoms (Skodol, Bender, et al., 2007). Thus, in addition to genetic influences on schizotypal PD, experiences that promote cognitive dysfunction (malnutrition and marijuana use) and that diminish positive social connections serve as risk factors for the development of schizotypal PD.

As noted, very little is known about the biological and contextual risk factors for paranoid and schizoid PDs, other than that the genetic and family risk factors for all PDs are relevant for these disorders as well. A prospective study examined childhood predictors of paranoid PD symptoms at age 15 (Natsuaki, Cicchetti, & Rogosch, 2009). Adolescent paranoid PD symptoms were predicted by an earlier history of maltreatment; by earlier increases in externalizing symptoms and in the youth's own bullying of other children (but not being bullied themselves); and by peer ratings of being less cooperative, less likely to be leaders, and more likely to start fights. These results are interesting, in that they suggest that early precursors of adolescent paranoid PD symptoms are expressions of interpersonal hostility and alienation, and the findings are consistent with previously described results indicating that adolescent paranoid PD predicts later violence and criminality. Schizoid PD may be related to experiences undermining the biologically based affiliative system that pro-

motes social interaction in most people (Lenzenweger, 2010), but there are not yet data testing this idea in youth. Because paranoid PD and schizoid PD have been dropped from the categorical diagnoses in DSM-5 Section III, they may not receive much research attention in the future. However, the alienation expressed in these conditions is important for understanding PDs more generally, so it should continue to be a focus of research.

Etiology of BPD

Within the limited research on the emergence and early development of most PDs, the predictors and processes underlying BPD have received significantly more attention. Several researchers have called for greater recognition of BPD in youth, in part because it is potentially associated with significant levels of impairment (Chanen, Jovev, McCutcheon, Jackson, & McGorry, 2008; Miller et al., 2008; Stepp, 2012).

Researchers have increasingly refined a trait-based conceptualization of BPD in youth, identifying several major dimensions: identity disturbance, affective instability, relationship difficulties, and impulsivity (Miller et al., 2008). These core dimensions map onto personality dimensions identified in child personality trait models, with best coverage for the impulsivity domain, followed by the dimensions of affective instability and relationship difficulties, and with the least coverage for the identity disturbance domain (Tackett & Kushner, in press). In other words, certain aspects of core youth BPD functioning are likely to be assessed with existing normal-range personality trait measures, whereas other aspects of the disorder (e.g., identity disturbance) are likely to call for supplemental assessment tools.

A number of the general risk factors described previously as predictors of PDs in youth have also been found specifically as risks for BPD, including genetics, family adversity, negative peer relationships, and problems with emotion regulation. There is evidence for a genetic basis for BPD symptoms; a recent twin study of 12-year-olds obtained a heritability of .66 for BPD characteristics (Belsky et al., 2012). Lower levels of executive functioning, IQ, and theory of mind at age 5 predict later BPD characteristics at age 12 (Belsky et al., 2012). Family risks include physical and sexual abuse, problematic parenting styles, and parental psychopathology (e.g., Cohen, Crawford, et al., 2005; Guzder, Paris, Zelkowitz, & Marchessault, 1996; Levy, 2005).

Adolescent BPD symptoms are associated with maternal disrupted communication patterns and disrupted attachment as well (Levy, 2005; Ludolph, Westen, Misle, & Jackson, 1990). The experience of bullying in childhood predicts an increased risk of BPD symptoms by age 11 (Wolke, Schreier, Zanarini, & Winsper, 2012). Emotion dysregulation and social cognitive deficits are also linked with youth BPD (Reich & Zanarini, 2001; Sharp, in press). Research has identified ragefulness and overwhelming emotions as characteristics of adolescent BPD in particular, which may account for the previously described links between BPD and self-harm behaviors (Crowell et al., 2005; Reich & Zanarini, 2001). Taken together, there is good evidence for both genetic and environmental contributors to the development of BPD symptoms and personality processes in childhood and adolescence.

Personality traits are highly relevant for understanding the etiology of disorder, with multiple theoretical links proposed between these two domains (Nigg, 2006; Tackett, 2006). Personality traits may represent risk or vulnerability factors for disorder, or they may reflect common underlying causal factors influencing both personality and psychopathology. Although direct tests of such associations have been infrequent, modern research may support both types of associations between personality and youth BPD. For example, the biosocial development model that has emerged from work by Crowell, Beauchaine, and Linehan (2009) highlights potential transactional influences between youth BPD traits (such as negative affectivity and impulsivity) and environmental risk. Specifically, their theory suggests that early traits may represent true risk factors (to the extent that, e.g., high levels of Negative Affectivity promote the experience of environmental risks such as negative peer group responses), in addition to sharing underlying common causes across personality traits and youth BPD constructs.

Evidence for common causes—both biological and psychosocial—also emerges from a comparison of the literature on normal personality development and youth BPD. For example, dysfunction in the dopamine system has been identified as a biological vulnerability for youth BPD (Crowell et al., 2009) and has also been linked to the personality traits of Extraversion and Conscientiousness (Noble et al., 1998). Similarly, dysfunction in the serotonin system has also been identified as a biological vulnerability for youth BPD (Crowell et al., 2009) and has been connected to Neuroticism

and Disagreeableness (Greenberg et al., 2000; Hamer, Greenberg, Shabol, & Murphy, 1999). Such findings point to potential biological pathways resulting in the phenotypic correlations observed between youth BPD and these personality traits, in that BPD symptoms are typically associated with high Neuroticism, low Agreeableness, low Conscientiousness, and low Extraversion (Tackett & Kushner, in press). Similarly, research also points to potential shared psychosocial factors between youth BPD and normal personality. Early life experiences such as problematic attachment and maltreatment appear both to increase risk for youth BPD (e.g., Carlson, Egeland, & Sroufe, 2010; Gratz, Latzman, Tull, Reynolds, & Lejuez, 2011; Paris, Zweig-Frank, & Gudzer, 1994) and to alter the development of normal personality traits (e.g., Fabes, Poulin, Eisenberg, & Madden-Derdich, 2002; Rogosch & Cicchetti, 2004), again highlighting potential common pathways to normal and abnormal personality development.

Spectrum associations between personality–psychopathology constructs emphasize the potentially dimensional relationships between traits and disorders (Tackett, 2006). A spectrum association is consistent with a common-cause model, but can also be investigated by examining evidence for potentially quantitative (rather than qualitative) relationships at the phenotypic level. One recent study examined evidence for a spectrum association between youth BPD traits and more typical externalizing constructs in youth (aggression and rule breaking; Tackett, Herzhoff, Reardon, De Clercq, & Sharp, in press). This study found evidence that the antagonism traits at the core of youth BPD showed very high correlations with a general externalizing factor, supporting the argument that core aspects of youth BPD may be linked to both normal personality traits and DSM-IV Axis I psychopathology; this finding is consistent with the previously described research linking Cluster B PDs with numerous externalizing disorders (e.g., oppositional defiant disorder, substance abuse). This work points to a further need to examine underlying core components of normal personality, abnormal personality, and Axis I psychopathology in joint, multivariate investigations. As noted previously, BPD is also associated with internalizing psychopathology in adults (see also Eaton et al., 2011). This is corroborated by work in youth, which finds primary associations for antagonistic traits (more closely reflecting externalizing behaviors) as well as secondary associations for emotional instability (which typically reflects internalizing behaviors; Tackett et al., in press)

relevant for the externalizing spectrum in youth. Thus BPD probably represents a more complex condition reflecting elements of both internalizing and externalizing problems across the lifespan.

Finally, in regard to brain differences in BPD, research with adults with BPD has pointed to several abnormalities in terms of brain structure, function, and neurochemistry (Hooley, Cole, & Gironde, 2012; Paris, 2012). In terms of structural differences, a meta-analysis of seven studies concluded that there are reductions in hippocampal and amygdalar volume in adults with BPD (Nunes et al., 2009); the hippocampus and amygdala are both part of the limbic system, which is involved in emotion processing and memory. Significant reductions in size have also been observed in the orbitofrontal cortex and anterior cingulate cortex, and alterations in the corpus callosum have been observed as well (Hooley et al., 2012); these are all areas that may be involved in the impulsivity and poor regulation seen in BPD. People with BPD also display reduced prefrontal regulation (Silbersweig et al., 2007) and dysregulation of the hypothalamic–pituitary–adrenocortical (HPA) system, which is an important component of stress response (Hooley et al., 2012). Hooley and colleagues (2012) have suggested, "It is reasonable to believe that BPD reflects stress-induced compromises in neural circuits that underlie regulatory processes" (p. 428), in light of the fact that the brain differences observed across studies of people with BPD point to problems with emotion regulation, stress reactivity, and behavioral control. There have been some recent attempts to examine structural differences in adolescents with BPD. These studies have found abnormalities in the orbitofrontal cortex (Chanen, Velakoulis, et al., 2008), but not in the hippocampus or amygdala (Chanen, Velakoulis, et al., 2008) or the corpus callosum (Walterfang et al., 2010). These preliminary studies serve as a good reminder that the biological abnormalities present in adult BPD may not be present in adolescent BPD.

Etiology of ASPD, Psychopathy, and Narcissism

As noted earlier in this chapter, the early development of ASPD has the largest existing evidence base from early life, likely because of the DSM-IV requirement for a conduct disorder diagnosis before age 15 in order to make a diagnosis of ASPD in adults. That is, the DSM-IV and DSM-5 approach to conceptualizing conduct disorder as the core early life feature of

ASPD would suggest that the entire body of literature on conduct disorder has implications for the etiology of ASPD. Conduct disorder diagnoses are assigned to a heterogeneous group of youth, however. Researchers have argued for distinctions based on age of onset and behavioral type, suggesting that earlier age of onset and physically aggressive behaviors may represent a more severe variant of the phenomenon (Burt, 2012; Moffitt, Caspi, Harrington, & Milne, 2002; Tackett, Krueger, Iacono, & McGue, 2005)—and one that potentially indicates greater prediction of later diagnoses such as ASPD. Distinguishing between child- and adolescent-onset conduct disorder is supported by the empirical literature (Moffitt et al., 2008), although some evidence suggests that the advantage of this distinction is better accounted for by differentiation of behavioral subtypes (Burt, Donnellan, Iacono, & McGue, 2011). Indeed, early violent behaviors indexing conduct disorder do increase risk for a later ASPD diagnosis (Gelhorn, Sakai, Price, & Crowley, 2007), although many children with conduct disorder will not go on to develop ASPD (Moffitt et al., 2008).

Extensive work has been conducted in recent years on extending the concept of psychopathy downward to childhood and adolescence (e.g., Frick, Bodin & Barry, 2000). Psychopathy includes a number of tendencies: risk taking and impulsivity, grandiosity, manipulativeness, lack of empathy and remorse, and shallow relationships (Lynam, 1997; Lynam & Gudonis, 2005). Psychopathy predicts a number of important associated features and outcomes, including more severe and stable conduct problems (Kotler & McMahon, 2005). DSM-5 has added a specifier to conduct disorder based on research on psychopathy, although the manual terms this specifier "with limited prosocial emotions" (APA, 2013, p. 470), presumably because "psychopathy" sounds too negative or stigmatizing. The limited prosocial emotions may be displayed in four ways: "lack of remorse or guilt," "callous—lack of empathy," "unconcerned about performance," and "shallow or deficient affect" (APA, 2013, pp. 470–471). As in adult populations, psychopathy in youth is associated with high rates of instrumental aggression (Blair, Peschardt, Budhani, Mitchell, & Pine, 2006). Psychopathy can be reliably measured in childhood and remains moderately stable across adolescence (Lynam et al., 2009), and psychopathy symptoms in youth predict later antisocial behavior (Salekin, Rosenbaum, & Lee, 2008).

In terms of the causes of psychopathy, childhood psychopathy appears to be at least partially heritable,

and these inherited characteristics are likely to result in impaired socialization across development (Blair et al., 2006). In addition, the stability in psychopathy symptoms across adolescence is primarily influenced by genetic factors (Forsman, Lichtenstein, Andershed, & Larsson, 2008). There is also evidence for functional brain differences in adolescents with high level of psychopathic traits relative to normal controls, in that they show reduced amygdala activity to negative stimuli (especially fearful faces), which may reduce their capacity for learning from punishment (Blair, 2010; Hyde, Shaw, & Hariri, 2013). In addition, a number of other brain regions in adolescent studies have shown functional abnormalities, including prefrontal regions, insula, anterior cingulate cortex, and caudate (Hyde et al., 2013); these regions may all be implicated in the abnormalities in reward processing, learning, and decision making that are observed in more severely psychopathic youth. Studies of structural brain differences in youth point to abnormalities in many of the same brain regions identified in the functional neuroimaging studies, including, for example, amygdala, prefrontal areas, and insula (Blair, 2010; Hyde et al., 2013). Many of the structural neuroimaging studies conflict in the direction of their findings, however (Hyde et al., 2013), so more work is needed to understand how the functional and structural differences in psychopathy relate to each other.

In addition, a number of contextual contributors to psychopathy have been identified. One study found the highest levels of stability in psychopathy symptoms from adolescence to adulthood in those youth who were exposed to psychosocial stressors, such as corporal punishment, low SES, and exposure to delinquent peers (Lynam, Loeber, & Stouthamer-Loeber, 2008). Early childhood predictors of later psychopathy—including earlier psychopathy characteristics, SES, parenting risk, and youth antisocial behavior—are generalizable across race and adult criminal status as well, speaking to their robustness and stability (Vachon, Lynam, Loeber, & Stouthamer-Loeber, 2012).

Callous–unemotional traits (i.e., lack of empathy and remorse, shallow emotions and relationships) are often thought to reflect a more narrowly defined core of the psychopathy construct in childhood (Frick & Viding, 2009; see Kimonis, Frick, & McMahon, Chapter 3, this volume); the literature on these traits overlaps substantially with research on youth psychopathy. The presence of callous–unemotional traits appears to be a particularly useful way of distinguishing children diag-

nosed with conduct disorder who are most likely to go on to an adult diagnosis of ASPD (Moffitt et al., 2008); the research on these traits heavily informed the decision to frame the conduct disorder specifier in terms of "limited prosocial emotions." Even among those youth exhibiting high levels of callous–unemotional traits, recent evidence supports heterogeneity on dimensions such as anxiety (high vs. low; Kimonis, Frick, Cauffman, Goldweber, & Skeem, 2012). Thus a better understanding of the phenomenology and utility of callous–unemotional traits continues to be a primary focus for future research.

Multiple studies have identified normal personality trait correlates of youth psychopathy, which are similar to those found in adult samples: low Agreeableness, low Conscientiousness, and high Neuroticism (e.g., Lynam et al., 2005; Salekin, Leistico, Trobst, Schrum, & Lochman, 2005). Youth callous–unemotional traits can also be characterized within a broader personality/temperament framework, and generally relate to high levels of Disinhibition, high levels of Negative Affectivity (particularly reflecting alienation and antagonism), and low levels of Positive Emotionality (Decuyper, De Bolle, De Fruyt, & De Clercq, 2011; Latzman, Lilienfeld, Latzman, & Clark, 2013; Roose et al., 2012; Salekin, Debus, & Barker, 2010). At the higher-order personality trait level, then, correlates for youth psychopathy and youth BPD are largely overlapping. Differentiation between these disorders is probably best reflected in the magnitude of associations at the domain level (e.g., youth BPD should show stronger associations with trait Neuroticism than should youth psychopathy), as well as in differentiation of associations at the lower-order trait, or facet, level.

Social-cognitive processing deficits have also been identified in youth psychopathy, such as the overattribution of conflict in friendship interactions (Munoz, Kerr, & Besic, 2008). A growing literature highlights problems with emotional recognition and social exchange behavior in youth with psychopathic traits (e.g., White, Brislin, Meffert, Sinclair, & Blair, 2013), and a recent meta-analysis suggests that emotion recognition deficits in youth psychopathy are broad and pervasive across emotions (Dawel, O'Kearney, McKone, & Palermo, 2012). In addition, one recent study found that specific components of psychopathy differentially predicted social cognitive processing in a sample of inpatient youth (Sharp, 2012). Specifically, this study found the affective component of psychopathy was related to hypermentalization (or overattribution of others'

intent), whereas the interpersonal component was related to hypomentalization (or underattribution). Thus numerous aspects of social cognition and interpersonal processing appear to be relevant for the development of psychopathy in early life.

A related trait that has been studied in conjunction with conduct disorder and psychopathy in youth is narcissism. Very little research has examined the origins of narcissistic PD, but there is increasing interest in the dimensional trait of narcissism, which "refers to a sense of grandiosity, coupled with a strong need to obtain attention and admiration from others" (Thomaes, Brummelman, Reijntjes, & Bushman, 2013, p. 22). Individual differences in this trait are measurable by at least late childhood (Barry, Frick, & Killian, 2003; Thomaes et al., 2008). Narcissism in youth tends to be associated with a more manipulative and less empathic stance toward others, difficulties with regulating self-esteem, and a preoccupation with others' evaluations (Thomaes et al., 2013; Weise & Tuber, 2004). These aspects of narcissism manifest themselves in the ways that narcissistic youth interact with peers. Specifically, narcissism is associated cross-sectionally with physical, verbal, and relational aggression, both in person and on the Internet, and with antisocial and delinquent behavior; these problems with aggression are made worse when youth's self-views are threatened (Thomaes et al., 2013). Among young adolescents who are aggressive, there is greater stability of aggression when the adolescents are also narcissistic (Bukowski, Schwartzman, Santo, Bagwell, & Adams, 2009). Thus narcissism in childhood and adolescence is associated with a number of troubling outcomes, particularly in the domain of peer relationships.

Relatively little is known about the pathways leading to narcissism because few longitudinal studies have examined precursors to later narcissism. Narcissism in adolescence and early adulthood is predicted by preschool measures of interpersonal antagonism, inadequate impulse control, histrionic tendencies, high activity level, and desire to be the center of attention (Carlson & Gjerde, 2009); these results suggest that there are a number of theoretically predicted early markers of later narcissism. One prospective study of adult narcissism found that both authoritarian and indulgent maternal parenting predicted adult narcissistic traits (Cramer, 2011). An interesting theory (Thomaes, Bushman, Orobio de Castro, & Stegge, 2009) ties together these findings by suggesting that children who are higher in approach tendencies will be more reinforced by re-

wards, such as praise from others; if there are problems with socialization, such as parental overvaluing of their children's characteristics, approach temperament could shape the development of narcissistic tendencies over time. Given the links between narcissism and youth's social behavior, it will be important to begin exploring the pathways leading to narcissistic tendencies.

Etiology of Cluster C Disorders

The three Cluster C PDs—avoidant PD, dependent PD, and obsessive–compulsive PD—are described in DSM-IV and DSM-5 as the "anxious or fearful" PDs (see Table 18.1 for the primary characteristics of each one). As a group, these PDs have received the least attention in the literature on PDs in youth, and very few longitudinal studies have been conducted exploring their development over time. However, despite this lack of research on the etiology of the Cluster C PDs, both avoidant PD and obsessive–compulsive PD have been retained in the list of DSM-5 Section 3 categorical disorders. Before we discuss the possible precursors to the Cluster C PDs, it is important to note that obsessive–compulsive PD seems to be less closely related to the other two Cluster C PDs than they are to each other. Obsessive–compulsive PD is associated with relatively low levels of impairment in adolescence and adulthood (Cohen, Crawford, et al., 2005; Torgersen, 2012), whereas both avoidant and dependent PDs are associated with significant impairment in adolescence and adulthood (Bornstein, 2012b; Cohen, Crawford, et al., 2005; Torgersen, 2012). Obsessive–compulsive PD has different genetic and environmental influences from the other two PDs (Kendler et al., 2011; Reichborn-Kjennerud et al., 2007), and it has the highest disorder-specific genetic influences of all the PDs (Kendler et al., 2008). Thus, its causes are likely to be different from those of avoidant and dependent PD. The relationship between obsessive–compulsive PD and obsessive–compulsive disorder (OCD) is complex, in that although the two are sometimes comorbid, obsessive–compulsive PD does not seem to be simply a milder version of OCD (Samuels & Costa, 2012). Rather, obsessive–compulsive PD co-occurs with a wide variety of anxiety, mood, and eating disorders.

Several predisposing factors seem likely to be relevant to the development of avoidant and dependent PDs. First, the same temperament and personality traits that predispose youth and adults to develop internalizing disorders may be relevant to the development of the Cluster C PDs, given the previously described research linking the Cluster C PDs with depression and anxiety. High Negative Affectivity predicts the development of all the internalizing disorders in both youth and adults, and poor self-control, including poor attentional control, is often implicated as well (Klein, Dyson, Kujawa, & Kotov, 2012). Consistent with this research, a study found that both high anger and low levels of attentional control were observed in children manifesting trajectories indicating higher levels of social withdrawal (Eggum et al., 2009). Behavioral inhibition, the tendency to respond to novel situations with fear and withdrawal, is also associated with the development of some anxiety disorders in youth (Klein et al., 2012) and seems likely to be involved in the development of avoidant and dependent PDs. Second, many of the family factors described previously predict the emergence of the Cluster C PDs in adolescence and adulthood. Third, peer relationships are likely to be disturbed. A retrospective study found that adult avoidant PD was associated with recollections of weaker athletic performance, less involvement in hobbies, and less peer popularity earlier in life (Rettew et al., 2003). Improvements in avoidant PD symptoms from adolescence to adulthood are predicted by positive achievement and interpersonal experiences in childhood and adolescence (Skodol, Bender, et al., 2007). Trait dependency is likewise associated with unpopularity and negative perceptions by peers in childhood, and with loneliness and peer rejection in adolescence (Bornstein, 2012a). Finally, although the origins of obsessive–compulsive PD are poorly understood, the pathological trait of compulsivity (the negative extreme end of high Conscientiousness) is especially associated with obsessive–compulsive PD in adolescence and adulthood (Aelterman, Decuyper, & De Fruyt, 2010); more research on this trait in youth should help facilitate a better understanding of obsessive–compulsive PD in adulthood.

CONCLUSIONS AND RECOMMENDATIONS FOR FUTURE RESEARCH

Research over the last two decades has made it clear that PDs exist in youth and are worthy of both research and clinical attention. PDs are prevalent by early adolescence, with at least 10% of adolescents meeting criteria for at least one PD. Although PD diagnoses are changeable in youth, PD symptoms and traits are modestly to strongly stable by adolescence and not substan-

tially less stable than in adulthood. PDs in youth pose considerable risks for development, including potential high-risk behaviors, emergence of other psychiatric disorders, and impairment in important life domains (e.g., academic achievement, relationships, work). When the diagnosis of PDs is discouraged in people under the age of 18, youth with personality pathology may receive incorrect treatment or may not receive the treatment they need (Shiner, 2007).

Although considerable progress has been made in research on PDs in youth over the last two decades, much remains to be learned about the nature and course of PDs. These conditions remain understudied, relative to other psychiatric conditions in childhood and adolescence. In the following sections, we offer suggestions for future research, focusing on two general areas: the measurement and manifestations of PDs in youth and the development of PDs over time.

Measurement and Manifestations of PDs in Childhood and Adolescence

With some notable exceptions (e.g., ASPD), the DSM systems have given little consideration to the childhood antecedents of later-emerging adult PDs, and this situation has led to a relative paucity of research on the pathways leading to PDs. In addition, contradictory views that PDs are rare in adolescence but that PD symptoms may be normative in adolescence have resulted in few attempts in the DSM systems to consider how childhood and adolescent PDs relate to other childhood disorders that involve relatively enduring patterns of behavior, cognition, and emotion (Ashton, 2007; De Fruyt & De Clercq, 2012). For example, oppositional defiant disorder involves a consistent pattern of hostile, defiant, and negativistic behavior; this sustained pattern describes a troubling pattern that could be considered an expression of pathological personality. Similarly, childhood anxiety disorders, especially social anxiety disorder (social phobia), may overlap considerably with avoidant PD symptoms in childhood and adolescence. Furthermore, in direct contrast to the normative hypothesis—often put forth to discourage research on early PDs—recent work suggests that youth personality pathology may show the strongest connections to psychopathology during developmental periods of greatest prevalence (Tackett et al., in press). In other words, diverting clinical and empirical attention from "normative" periods may be limiting attention to those periods most deserving of close scrutiny. The relation-

ship between childhood and adolescent PDs and other disorders in youth (e.g., ADHD, autism spectrum disorder) awaits further study.

Beyond the categorical definitions of PD diagnoses, the new alternative model for diagnosing PD is an important target for future research, and one that is highly amenable to developmental research with children and adolescents. The model will require much more empirical research to examine whether it is reliable, valid, and clinically useful; this is particularly true for its use with populations of children and adolescents, given that the published empirical work on the model has focused on adult samples. From a developmental perspective, however, the model seems potentially promising. The model incorporates the literatures described in this chapter showing that personality traits, attachment, social-cognitive mechanisms, coping styles, and identity may be disturbed in youth with PDs, and that these same processes may play a causal role in the development of adult PDs. The definition of impairment specifically takes into account disturbances in attachment, other mental representations, and identity, and the requirement of pathological personality traits builds nicely on the research on such traits in youth. Future work will help to clarify the usefulness of this model for diagnosing PDs in youth. Both the pathological trait domains and the domains of impairment will require intensive investigation in youth.

Developmental Pathways Leading to Disordered Personality

We currently lack information from multiple studies about the developmental pathways leading to the emergence of personality pathology in the first two decades of life. Prospective longitudinal studies that trace the developmental pathways leading to PD are sorely needed. The one prospective, longitudinal study of all the PDs—the Children in the Community study—has made impressive contributions to extant knowledge about the development of PDs and is the source of many of the findings reviewed in this chapter. New longitudinal work on PD development can build on the findings of this study by considering what is known about normal personality development, assessing a wide range of personality differences, and measuring multiple aspects of the environment. Studies using behavior genetic or molecular genetic methods would be particularly useful for clarifying the causes of individual differences in PD. It would also be extremely

informative to begin such studies earlier in childhood to pinpoint the earliest manifestations of and influences on PD development. Most of the research reviewed in this chapter has focused on personality pathology in adolescence, leaving PDs in childhood poorly understood. Further, although adolescence seems to represent a critical juncture in the emergence of persistent personality pathology, the origins of PD cannot be understood without beginning a study well before adolescence. Studies with more frequent assessments could better identify transactions between youth and their environments over time. Well-designed studies could also address fundamental epidemiological questions about PD in youth, including changing prevalence rates over time; gender differences; differences across socioeconomic groups, ethnicities, races, and cultures; and rates of comorbidity among PDs.

In future work, it will be especially important to examine the environmental contributions to the development of personality pathology. For personality traits (Shiner, 2014), characteristic adaptations (Pomerantz & Thompson, 2008), and personal narratives (McAdams, 2008), we already know a considerable amount about how the environment contributes to personality development. The insights from this research can be incorporated into new longitudinal research examining contextual contributors to personality pathology. Extreme adversity (including significant poverty) may have negative effects on personality development, including children's emerging capacity for self-regulation (Hart, Atkins, & Matsuba, 2008). Although there is some work investigating personality development in the context of real-life contexts, other important social, cultural, and global changes in children's lives have received relatively little attention, including immigration, war, violence, illness, and abuse (Belfer, 2008). These large-scale societal challenges are likely to play a critical role in both healthy and unhealthy personality development.

Both equifinality and multifinality are likely to be evident in the developmental pathways to PDs in youth. When applied to PDs, the principle of equifinality highlights the importance of exploring whether different processes may lead to similar patterns of personality pathology. As noted earlier, although early family adversity poses significant risks for the development of personality pathology, early trauma and abuse are unlikely to be present in the histories of all youth with PDs. In contrast, some youth may struggle with such extreme traits from early in life that those traits overwhelm the effects of a generally "good enough" environment (e.g., Zanarini & Frankenburg, 2007). In short, it is important to recognize that temperament may play a more central role in some pathways, whereas trauma or adversity may be more central in other ones (Nigg, Silk, Stavro, & Miller, 2005).

Likewise, youth with similar outcomes may vary in the time course over which their personality difficulties develop. For some youth, the pathway may be more continuous and linear. For example, a child who is temperamentally prone toward hostility and impulsivity may gradually become increasingly angry and poorly regulated over time, as that child encounters more and more experiences that contribute to the development of these negative traits. In contrast, other youth may show a course that is more abrupt and nonlinear. In this kind of pathway, vulnerable youth may encounter life experiences that lead to abrupt changes in their personality functioning. In future work, it will be important to recognize the possibilities of these diverse processes leading to PDs.

The progress made in understanding PD in youth has begun to accelerate in recent years; our hope is that the upsurge in new knowledge about PD in children and adolescents will have an increasingly positive impact on clinical practice for youth.

REFERENCES

Aelterman, N., Decuyper, M., & De Fruyt, F. (2010). Understanding obsessive–compulsive personality disorder in adolescence: A dimensional personality perspective. *Journal of Psychopathology and Behavioral Assessment, 32*, 467–478.

Afifi, T. O., Mathew, A., Boman, J., Fleisher, W., Enns, M. W., MacMillan, H., et al. (2011). Childhood adversity and personality disorders: Results from a nationally representative population-based study. *Journal of Psychiatry Research, 45*, 814–822.

American Psychiatric Association (APA). (1952). *Diagnostic and statistical manual of mental disorders.* Washington, DC: Author.

American Psychiatric Association (APA). (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.

American Psychiatric Association (APA). (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association (APA). (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association (APA). (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association (APA). (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

American Psychiatric Association (APA). (2012). American Psychiatric Association Board of Trustees approves DSM-5 diagnostic manual, passes major milestone before May 2013 publication [Press release]. Retrieved from *www.psychiatry.org/advocacy—newsroom/newsroom/current-news*

American Psychiatric Association (APA). (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.

Anglin, D. M., Cohen, P. R., & Chen, H. (2008). Duration of early maternal separation and prediction of schizotypal symptoms from early adolescence to midlife. *Schizophrenia Research, 103*, 143–150.

Anglin, D. M., Corcoran, C. M., Brown, A. S., Chen, H., Lighty, Q., Brook, J. S., et al. (2012). Early cannabis use and schizotypal personality disorder symptoms from adolescence to middle adulthood. *Schizophrenia Research, 137*, 45–49.

Arnett, J. J. (1999). Adolescent storm and stress, reconsidered. *American Psychologist, 54*, 317–326.

Ashton, M. C. (2007). *Individual differences and personality.* Burlington, MA: Elsevier Academic Press.

Barry, C. T., Frick, P. J., & Killian, A. L. (2003). The relation of narcissism and self-esteem to conduct problems in children: A preliminary investigation. *Journal of Clinical Child and Adolescent Psychology, 32*, 139–152.

Bates, J. E., Schermerhorn, A. C., & Petersen, I. T. (2012), Temperament and parenting in developmental perspective. In M. Zentner & R. L. Shiner (Eds.), *Handbook of temperament* (pp. 425–441). New York: Guilford Press.

Battle, C. L., Shea, T., Johnson, D. M., Yen, S., Zlotnick, C., Zanarini, M. C., et al. (2004). Childhood maltreatment associated with adult personality disorders: Findings from the Collaborative Longitudinal Personality Disorders Study. *Journal of Personality Disorders, 18*, 193–211.

Becker, D. F., Grilo, C. M., Edell, W. S., & McGlashan, T. H. (2000). Comorbidity of borderline personality disorder with other personality disorders in hospitalized adolescents and adults. *American Journal of Psychiatry, 157*, 2011–2016.

Belfer, M. L. (2008). Child and adolescent mental disorders: The magnitude of the problem across the globe. *Journal of Child Psychology and Psychiatry, 49*, 226–236.

Belsky, D. W., Caspi, A., Arseneault, L., Bleidorn, W., Fonagy, P., Goodman, M., et al. (2012). Etiological features of borderline personality related characteristics in a birth cohort of 12-year-old children. *Development and Psychopathology, 24*, 251–265.

Bender, D. S., Morey, L. C., & Skodol, A. E. (2011). Toward a model for assessing level of personality functioning in DSM-5: Part I. A review of theory and methods. *Journal of Personality Assessment, 93*, 332–346.

Bernstein, D. P., Cohen, P., Skodol, A., Bezirganian, S., & Brook, J. S. (1996). Childhood antecedents of adolescent personality disorders. *American Journal of Psychiatry, 153*, 907–913.

Bernstein, D. P., Cohen, P., Velez, C. N., Schwab-Stone, M., Siever, L. J., & Shinsato, L. (1993). Prevalence and stability of the DSM-III-R personality disorders in a community-based survey of adolescents. *American Journal of Psychiatry, 150*, 1237–1243.

Biskin, R. S., & Paris, J. (Eds.). (2013). Personality disorders in adolescence. [Special issue]. *Journal of the Canadian Academy of Child and Adolescent Psychiatry, 22*(3).

Blair, R. J. R. (2010). A cognitive neuroscience perspective on child and adolescent psychopathy. In R. T. Salekin & D. R. Lynam (Eds.), *Handbook of child and adolescent psychopathy* (pp. 156–178). New York: Guilford Press.

Blair, R. J. R., Peschardt, K. S., Budhani, S., Mitchell, D. G. V., & Pine, D. S. (2006). The development of psychopathy. *Journal of Child Psychology and Psychiatry, 47*, 262–275.

Bornovalova, M. A., Hicks, B. M., Iacono, W. G., & McGue, M. (2013). Longitudinal twin study of borderline personality disorder traits and substance use in adolescence: Developmental change, reciprocal effects, and genetic and environmental influences. *Personality Disorders: Theory, Research, and Treatment, 4*, 23–32.

Bornstein, R. F. (2012a). From dysfunction to adaptation: An interactionist model of dependency. *Annual Review of Clinical Psychology, 8*, 291–316.

Bornstein, R. F. (2012b). Illuminating a neglected clinical issue: Societal costs of interpersonal dependency and dependent personality disorder. *Journal of Clinical Psychology, 68*, 766–781.

Bradley, R., Conklin, C. Z., & Westen, D. (2005). The borderline personality disorder diagnosis in adolescence: Gender differences and subtypes. *Journal of Child Psychology and Psychiatry, 46*, 1006–1019.

Bradley, B., Westen, D., Mercer, K. B., Binder, E. B., Jovanovic, T., Crain, D., et al. (2011). Association between childhood maltreatment and adult emotional dysregulation in a low-income, urban, African American sample: Moderation by oxytocin receptor gene. *Development and Psychopathology, 23*, 439–452.

Brent, D. A., Johnson, B. A., Perper, J., & Connolly, J. (1994). Personality disorder, personality traits, impulsive violence, and completed suicide in adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 33*, 1080–1086.

Bukowski, W. M., Schwartzman, A., Santo, J., Bagwell, C., & Adams, R. (2009). Reactivity and distortions in the self: Narcissism, types of aggression, and the functioning of the hypothalamic–pituitary–adrenal axis during early adolescence. *Development and Psychopathology, 21*, 1249–1262.

Burke, J. D., & Stepp, S. D. (2012). Adolescent disruptive behavior and borderline personality disorder symptoms in young adult men. *Journal of Abnormal Child Psychology, 40*, 35–44.

Burt, S. A. (2012). How do we optimally conceptualize the heterogeneity within antisocial behavior?: An argument for aggressive versus non-aggressive behavioral dimensions. *Clinical Psychology Review, 32*, 263–279.

Burt, S. A., Donnellan, M. B., Iacono, W. G., & McGue, M. (2011). Age-of-onset or behavioral sub-types?: A prospective comparison of two approaches to characterizing the heterogeneity within antisocial behavior. *Journal of Abnormal Child Psychology, 39*, 633–644.

Cannon, T. D., Cadenhead, K., Cornblatt, B., Woods, S. W., Addington, J., Walker, E., et al. (2008). Prediction of psychosis in youth at high clinical risk: A multisite longitudinal study in North America. *Archives of General Psychiatry, 65*, 28–37.

Carlson, E. A., Egeland, B., & Sroufe, L. A. (2009). A prospective investigation of the development of borderline personality symptoms. *Development and Psychopathology, 21*, 1311–1334.

Carlson, K. S., & Gjerde, P. F. (2009). Preschool personality antecedents of narcissism in adolescence and young adulthood: A 20-year longitudinal study. *Journal of Research in Personality, 43*, 570–578.

Caspi, A., & Shiner, R. L. (2006). Personality development. In W. Damon & R. Lerner (Series Eds.) & N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 300–365). Hoboken, NJ: Wiley.

Chanen, A. M., Jackson, H. J., McGorry, P. D., Allot, K. A., Clarkson, V., & Yuen, H. P. (2004). Two-year stability of personality disorder in older adolescent outpatients. *Journal of Personality Disorders, 18*, 526–541.

Chanen, A. M., Jovev, M., Djaja, D., McDougall, E., Yuen, H. P., Rawlings, D., et al. (2008). Screening for borderline personality disorder in outpatient youth. *Journal of Personality Disorders, 22*, 353–364.

Chanen, A. M., Jovev, M., McCutcheon, L. K., Jackson, H. J., & McGorry, P. D. (2008). Borderline personality disorder in young people and the prospects for prevention and early intervention. *Current Psychiatry Reviews, 4*, 48–57.

Chanen, A. M., & McCutcheon, L. K. (2008). Complex case. Personality disorder in adolescence: The diagnosis that dare not speak its name. *Personality and Mental Health, 2*, 35–51.

Chanen, A. M., Velakoulis, D., Carison, K., Gaunson, K., Wood, S. J. Yuen, H. P., et al. (2008). Orbitofrontal, amygdala, and hippocampal volumes in teenagers with first-presentation borderline personality disorder. *Psychiatry Research: Neuroimaging, 163*, 116–125.

Chen, H., Cohen, P., Crawford, T. N., Kasen, S., Johnson, J. G., & Berenson, K. (2006). Relative impact of young adult personality disorders on subsequent quality of life: Findings of a community-based longitudinal study. *Journal of Personality Disorders, 20*, 510–523.

Chen, H., Cohen, P., Johnson, J. G., Kasen, S., Sneed, J. R., & Crawford, T. N. (2004). Adolescent personality disorders and conflict with romantic partners during the transition to adulthood. *Journal of Personality Disorders, 18*, 507–525.

Cicchetti, D. (1993). Developmental psychopathology: Reactions, reflections, projections. *Developmental Review, 13*, 471–502.

Cicchetti, D. (2013). An overview of developmental psychopathology. In P. Zelazo (Ed.), *Oxford handbook of developmental psychology* (pp. 455–480). New York: Oxford University Press.

Cicchetti, D., & Crick, N. R. (2009). Precursors and diverse pathways to personality disorder in children and adolescents. *Development and Psychopathology, 21*, 683–685.

Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in development. *Development and Psychopathology, 8*, 597–600.

Clark, L. A. (2005). Temperament as a unifying basis for personality and psychopathology. *Journal of Abnormal Psychology, 114*, 505–521.

Clark, L. A. (2007). Assessment and diagnosis of personality disorder: Perennial issues and emerging conceptualization. *Annual Review of Psychology, 58*, 227–257.

Clark, L. A. (2009). Stability and change in personality disorder. *Current Directions in Psychological Science, 18*, 27–31.

Clark, L. A., Livesley, W. J., & Morey, L. (1997). Personality disorder assessment: The challenge of construct validity. *Journal of Personality Disorders, 11*, 205–231.

Clark, L. A., Simms, L. J., Wu, K. D., & Casillas, A. (2011). *Manual for the Schedule for Nonadaptive and Adaptive Personality–2 (SNAP-2)*. Minneapolis: University of Minnesota Press.

Cloninger, C. R. (2005). Genetics. In J. M. Oldham, A. E. Skodol, & D. S. Bender (Eds.), *The American Psychiatric Publishing textbook of personality disorders* (pp. 143–154). Washington, DC: American Psychiatric Publishing.

Coghill, D., & Sonuga-Barke, J. S. (2012). Categories versus dimensions in the classification and conceptualization of child and adolescent mental disorders—implications of recent empirical study. *Journal of Child Psychology and Psychiatry, 53*, 469–489.

Cohen, P., Chen, H., Crawford, T. N., Brook, J. S., & Gordon, K. (2007). Personality disorders in early adolescence and the development of later substance use disorders in the general population. *Drug and Alcohol Dependence, 88S*, S71–S84.

Cohen, P., Chen, H., Gordon, K., Johnson, J. G., Brook, J., & Kasen, S. (2008). Socioeconomic background and the developmental course of schizotypal and borderline personality disorder symptoms. *Development and Psychopathology, 20*, 633–650.

Cohen, P., Chen, H., Kasen, S., Johnson, J. G., Crawford, T.,

& Gordon, K. (2005). Adolescent Cluster A personality disorder symptoms, role assumption in the transition to adulthood, and resolution or persistence of symptoms. *Development and Psychopathology, 17*, 549–568.

Cohen, P., Crawford, T. N., Johnson, J. G., & Kasen, S. (2005). The Children in the Community study of developmental course of personality disorder. *Journal of Personality Disorders, 19*, 466–486.

Conger, R. D., & Donnellan, M. B. (2007). An interactionist perspective on the socioeconomic context of human development. *Annual Review of Psychology, 58*, 175–199.

Coolidge, F. L., Thede, L. L., & Jang, K. L. (2001). Heritability of personality disorders in childhood: A preliminary investigation. *Journal of Personality Disorders, 15*, 33–40.

Cooper, L. D., Balsis, S., & Oltmanns, T. F. (2014). A longitudinal analysis of personality disorder dimensions and personality traits in a community sample of older adults: Perspectives from selves and informants. *Journal of Personality Disorders, 28*, 151–165.

Correll, C., Smith, C., Auther, A., McLaughlin, D., Shah, M., Foley, C., et al. (2008). Predictors of remission, schizophrenia, and bipolar disorder in adolescents with brief psychotic disorder or psychotic disorder not otherwise specified considered at very high risk for schizophrenia. *Journal of Child and Adolescent Psychopharmacology, 18*, 475–490.

Cramer, P. (2008). Seven pillars of defense mechanism theory. *Social and Personality Psychology Compass, 2*, 1963–1981.

Cramer, P. (2011). Young adult narcissism: A 20-year longitudinal study of the contribution of parenting styles, preschool precursors of narcissism, and denial. *Journal of Research in Personality, 45*, 19–28.

Crawford, T. N., Cohen, P., First, M. B., Skodol, A. E., Johnson, J. G., & Kasen, S. (2008). Comorbid Axis I and Axis II disorders in early adolescence: Outcomes 20 years later. *Archives in General Psychiatry, 65*, 641–648.

Crawford, T. N., Cohen, P., Johnson, J. G., Kasen, S., First, M. B., Gordon, K., et al. (2005). Self-reported personality disorder in the Children in the Community sample: Convergent and prospective validity in late adolescence and adulthood. *Journal of Personality Disorders, 19*, 30–52.

Crawford, T. N., Cohen, P., Johnson, J. G., & Sneed, J. R. (2004). The course and psychosocial correlates of personality disorder symptoms in adolescence: Erikson's developmental theory revisited. *Journal of Youth and Adolescence, 33*, 373–387.

Crawford, T. N., Shaver, P. R., Cohen, P., Pilkonis, P. A., Gillath, O., & Kasen, S. (2006). Self-reported attachment, interpersonal aggression, and personality disorder in a prospective community sample of adolescents and adults. *Journal of Personality Disorders, 20*, 331–351.

Crick, N. R., Murray-Close, D., & Woods, K. (2005). Bor-

derline personality features in childhood: A short-term longitudinal study. *Development and Psychopathology, 17*, 1051–1070.

Crowell, S. E., Beauchaine, T. P., & Linehan, M. M. (2009). A biosocial developmental model of borderline personality: Elaborating and extending Linehan's theory. *Psychological Bulletin, 135*, 495–510.

Crowell, S. E., Beauchaine, T. P., McCauley, E., Smith, C. J., Stevens, A. L., & Sylvers, P. (2005). Psychological, autonomic, and serotonergic correlates of parasuicide among adolescent girls. *Development and Psychopathology, 17*, 1105–1127.

Daley, S. E., Hammen, C., Burge, D., Davila, J., Paley, B., Lindberg, N., et al. (1999). Depression and Axis II symptomatology in an adolescent community sample: Concurrent and longitudinal associations. *Journal of Personality Disorders, 13*, 47–59.

Daley, S. E., Hammen, C., Davila, J., & Burge, D. (1998). Axis II symptomatology, depression, and life stress during the transition from adolescence to adulthood. *Journal of Consulting and Clinical Psychology, 66*, 595–603.

Dawel, A., O'Kearney, R., McKone, E., & Palermo, R. (2012). Not just fear and sadness: Meta-analytic evidence of pervasive emotion recognition deficits for facial and vocal expressions in psychopathy. *Neuroscience and Biobehavioral Reviews, 36*, 2288–2304.

Deater-Deckard, K. (2013). The social environment and the development of psychopathology. In P. Zelazo (Ed.), *Oxford handbook of developmental psychology* (pp. 527–548). New York: Oxford University Press.

De Clercq, B., & De Fruyt, F. (2003). Personality disorder symptoms in adolescence: A five-factor model perspective. *Journal of Personality Disorders, 17*, 269–292.

De Clercq, B., & De Fruyt, F. (2012). A five-factor model framework for understanding childhood personality disorder antecedents. *Journal of Personality, 80*, 1533–1563.

De Clercq, B., De Fruyt, F., & Van Leeuwen, K. (2004). A "Little Five" lexically based perspective on personality disorder symptoms in adolescence. *Journal of Personality Disorders, 18*, 479–499.

De Clercq, B., De Fruyt, F., Van Leeuwen, K., & Mervielde, I. (2006). The structure of maladaptive personality traits in childhood: A step toward an integrative developmental perspective for DSM-V. *Journal of Abnormal Psychology, 115*, 639–657.

De Clercq, B., De Fruyt, F., & Widiger, T. A. (2009). Integrating a developmental perspective in dimensional models of personality disorders. *Clinical Psychology Review*, 29, 154–162.

De Clercq, B., Van Leeuwen, K., Van Den Noortgate, W., De Bolle, M., & De Fruyt, F. (2009). Childhood personality pathology: Dimensional stability and change. *Development and Psychopathology, 21*, 853–869.

Decuyper, M., De Bolle, M., De Fruyt, F., & De Clercq, B.

(2011). General and maladaptive personality dimensions and the assessment of callous–unemotional traits in adolescence. *Journal of Personality Disorders, 25*, 681–701.

Decuyper, M., De Clercq, B., De Bolle, M., & De Fruyt, F. (2009). Validation of FFM PD counts for screening personality pathology and psychopathology in adolescence. *Journal of Personality Disorders, 23*, 587–605.

DeFife, J. A., Goldberg, M. G., & Westen, D. (in press). Dimensional assessment of self and interpersonal functioning in adolescents: Implications for DSM-5's general definition of PD. *Journal of Personality Disorders*.

DeFife, J. A., & Ritschel, L. A. (Eds.). (2013). Special series on personality disorders in adolescence. [Special issue]. *Clinical Psychology: Science and Practice, 20*(4).

De Fruyt, F., & De Clercq, B. (2012). Childhood antecedents of personality disorders. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 166–185). New York: Oxford University Press.

De Fruyt, F., & De Clercq, B. (2013). Childhood antecedents of personality disorder: A five-factor model perspective. In T. A. Widiger & P. T. Costa Jr. (Eds.), *Personality disorders and the five-factor model of personality* (3rd ed., pp. 43–60). Washington, DC: American Psychological Association.

De Pauw, S. S. W., Mervielde, I., & Van Leeuwen, K. G. (2009). How are traits related to problem behavior in preschool children?: Similarities and contrasts between temperament and personality. *Journal of Abnormal Child Psychology, 37*, 309–325.

Digman, J. M., & Shmelyov, A. G. (1996). The structure of temperament and personality in Russian children. *Journal of Personality and Social Psychology, 71*, 341–351.

Eaton, N. R., Krueger, R. F., Keyes, K. M., Skodol, A. E., Markon, K. E., Grant, B. F., et al. (2011). Borderline personality disorder comorbidity: Relationship to the internalizing–externalizing structure of common mental disorders. *Psychological Medicine, 41*, 1041–1050.

Ehrensaft, M. K., Cohen, P., & Johnson, J. G. (2006). Development of personality disorder symptoms and the risk for partner violence. *Journal of Abnormal Psychology, 115*, 474–483.

Eggum, N. D., Eisenberg, N., Spinrad, T. L., Valiente, C., Edwards, A., Kupfer, A. S., et al. (2009). Predictors of withdrawal: Possible precursors of avoidant personality disorder. *Development and Psychopathology, 21*, 815–838.

Elliott, T., Tyrer, P., Horwood, J., & Fergusson, D. (2011). Assessment of abnormal personality in childhood: A Delphi survey of questionnaire data. *Journal of Personality Disorders, 25*, 89–100.

Ellis, L. K., & Rothbart, M. K. (2001, April). *Revision of the Early Adolescent Temperament Questionnaire*. Poster presented at the biennial meeting of the Society for Research in Child Development, Minneapolis, MN.

Esterberg, M. L., Goulding, S. M., & Walker, E. F. (2010).

Cluster A personality disorders: Schizotypal, schizoid, and paranoid personality disorders in childhood and adolescence. *Journal of Psychopathology and Behavioral Assessment, 32*, 515–528.

Evans, G. W., & Kim, P. (2013). Childhood poverty, chronic stress, self-regulation, and coping. *Child Development Perspectives, 7*, 43–48.

Fabes, R., Poulin, R., Eisenberg, N., & Madden-Derdich, D. (2002). The Coping with Children's Negative Emotions Scale (CCNES): Psychometric properties and relations with children's emotional competence. *Marriage and Family Review, 34*, 285–310.

Fanous, A., Neale, M., Gardner, C., Webb, B., Straub, R., O'Neill, F., et al. (2007). Significant correlation in linkage signals from genome-wide scans of schizophrenia and schizotypy. *Molecular Psychiatry, 12*, 958–965.

Feenstra, D. J., Busschbach, J. J. V., Verheul, R., & Hutsebaut, J. (2011). Prevalence and cormorbidity of Axis I and Axis II disorders among treatment refractory adolescents admitted for specialized psychotherapy. *Journal of Personality Disorders, 25*, 842–850.

Feenstra, D. J., Hutsebaut, J., Laurenssen, E. M. P., Verheul, R., Busschbach, J. J. V., & Soeteman, D. I. (2012). The burden of disease among adolescents with personality pathology: Quality of life and costs. *Journal of Personality Disorders, 26*, 593–604.

Feenstra, D. J., Hutsebaut, J., Verheul, R., & van Limbeek, J. (2014). Changes in the identity integration of adolescents in treatment for personality disorders. *Journal of Personality Disorders, 28*, 101–112.

Feiring, C., Cleland, C. M., & Simon, V. A. (2010). Abuse-specific self-schemas and self-functioning: A prospective study of sexually abused youth. *Journal of Clinical Child and Adolescent Psychology, 39*, 35–50.

Ferguson, C. J. (2010). A meta-analysis of normal and disordered personality across the life span. *Journal of Personality and Social Psychology, 98*, 659–667.

Fivush, R., Haden, C. A., & Reese, E. (2006). Elaborating on elaborations: Role of maternal reminiscing style in cognitive and socioemotional development. *Child Development, 77*, 1568–1588.

Fonagy, P., & Bateman, A. (2008). The development of borderline personality disorder: A mentalizing model. *Journal of Personality Disorders, 22*, 4–21.

Forsman, M., Lichtenstein, P., Andershed, H., & Larsson, H. (2008). Genetic effects explain the stability of psychopathic personality from mid- to late adolescence. *Journal of Abnormal Psychology, 117*, 606–617.

Fraley, R. C., & Shaver, P. R. (2008). Attachment theory and its place in contemporary personality theory and research. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (3rd ed., pp. 518–541). New York: Guilford Press.

Frances, A. J., & Widiger, T. (2012). Psychiatric diagnosis:

Lessons from the *DSM-IV* past and cautions for the *DSM-5* future. *Annual Review of Clinical Psychology, 8*, 109–130.

Freeman, A., & Reinecke, M. A. (2007). *Personality disorders in childhood and adolescence.* Hoboken, NJ: Wiley.

Freeman, A., & Rigby, A. (2003). Personality disorders among children and adolescents: Is it an unlikely diagnosis? In M. A. Reinecke, F. M. Dattilio, & A. Freeman (Eds.), *Cognitive therapy with children and adolescents: A casebook for clinical practice* (2nd ed., pp. 434–464). New York: Guilford Press.

Frick, P. J., Bodin, S. D., & Barry, C. T. (2000). Psychopathic traits and conduct problems in community and clinic-referred samples of children: Further development of the Psychopathy Screening Device. *Psychological Assessment, 12*, 382–393.

Frick, P. J., & Viding, E. (2009). Antisocial behavior from a developmental psychopathology perspective. *Development and Psychopathology, 21*, 1111–1131.

Frick, P. J., & White, S. F. (2008). The importance of callous–unemotional traits for developmental models of aggressive and antisocial behavior. *Journal of Child Psychology and Psychiatry, 49*, 359–375.

Gelhorn, H. L., Sakai, J. T., Price, R. K., & Crowley, T. J. (2007). DSM-IV conduct disorder criteria as predictors of antisocial personality disorder. *Comprehensive Psychiatry, 48*, 529–538.

Goldberg, L. R. (2001). Analyses of Digman's child-personality data: Derivation of Big-Five factor scores from each of six samples. *Journal of Personality, 69*, 709–743.

Goodwin, R. D., Brook, J. S., & Cohen, P. (2005). Panic attacks and the risk of personality disorder. *Psychological Medicine, 35*, 227–235.

Gratz, K. L., Latzman, R. D., Tull, M. T., Reynolds, E. K., & Lejuez, C. W. (2011). Exploring the association between emotional abuse and childhood borderline personality features: The moderating role of personality traits. *Behavior Therapy, 42*, 493–508.

Gratz, K. L., Tull, M. T., & Gunderson, J. G. (2008). Preliminary data on the relationship between anxiety sensitivity and borderline personality disorder: The role of experiential avoidance. *Journal of Psychiatric Research, 42*, 550–559.

Gratz, K. L., Tull, M. T., Reynolds, E. K., Bagge, C. L., Latzman, R. D., Daughters, S. B., et al. (2009). Extending extant models of the pathogenesis of borderline personality disorder to childhood borderline personality symptoms: The roles of affective dysfunction, disinhibition, and self- and emotion-regulation deficits. *Development and Psychopathology, 21*, 1263–1291.

Greenberg, B. D., Li, Q., Lucas, F. R., Hu, S., Sirota, L. Q., Benjamin, J., et al. (2000). Association between the serotonin transporter promoter polymorphism and personality traits in a primarily female population sample. *American Journal of Medical Genetics, 96*, 202–216.

Grills, A. E., & Ollendick, T. H. (2002). Issues in parent–child agreement: The case of structured diagnostic interviews. *Clinical Child and Family Psychology Review, 5*, 57–83.

Grilo, C. M., & McGlashan, T. H. (2005). Course and outcome of personality disorders. In J. M. Oldham, A. E. Skodol, & D. S. Bender (Eds.), *The American Psychiatric Publishing textbook of personality disorders* (pp. 103–115). Washington, DC: American Psychiatric Publishing.

Grilo, C. M., McGlashan, T. H., Quinlan, D. M., Walker, M. L., Greenfeld, D., & Edell, W. S. (1998). Frequency of personality disorders in two age cohorts of psychiatric inpatients. *American Journal of Psychiatry, 155*, 140–142.

Guzder, J., Paris, J., Zelkowitz, P., & Marchessault, K. (1996). Risk factors for borderline pathology in children. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*, 26–33.

Habermas, T., & de Silveira, C. (2008). The development of global coherence in life narratives across adolescence: Temporal, causal, and thematic aspects. *Developmental Psychology, 44*, 707–721.

Hall, G. S. (1904). *Adolescence: Its psychology and its relation to physiology, anthropology, sociology, sex, crime, religion, and education* (2 vols.). Englewood Cliffs, NJ: Prentice-Hall.

Halverson, C. F., Havill, V. L., Deal, J., Baker, S. R., Victor, J. B., Pavlopoulos, V., et al. (2003). Personality structure as derived from parental ratings of free descriptions of children: The Inventory of Child Individual Differences. *Journal of Personality, 71*, 995–1026.

Hamer, D. H., Greenberg, B. D., Shabol, S. Z., & Murphy, D. L. (1999). Role of the serotonin transporter gene in temperament and character. *Journal of Personality Disorders, 13*, 312–328.

Hans, S. L., Auerbach, J. G., Neuchterlein, K. H., Asarnow, R. F., Asarnow, J., Styr, B., et alJ. (2009). Neurodevelopmental factors associated with schizotypal symptoms among adolescents at risk for schizophrenia. *Development and Psychopathology, 21*, 1195–1210.

Harkness, A. R., & McNulty, J. L. (1994). The Personality Psychopathology Five (PSY-5): Issue from the pages of a diagnostic manual instead of a dictionary. In S. Strack & M. Lorr (Eds.), *Differentiating normal and abnormal personality* (pp. 291–315). New York: Springer.

Hart, D, Atkins, R., & Matsuba, M. K. (2008). The association of neighborhood poverty with personality change in childhood. *Journal of Personality and Social Psychology, 94*, 1048–1061.

Hart, D., & Marmorstein, N. R. (2009). Neighborhoods and genes and everything in between: Understanding adolescent aggression in social and biological contexts. *Development and Psychopathology, 21*, 961–973.

Hill, J. (2008). Disorders of personality. In M. Rutter, D. Bishop, D. Pine, S. Scott, J. Stevenson, E. Taylor, et al. (Eds.), *Rutter's child and adolescent psychiatry* (5th ed., pp. 841–851). London: Blackwell.

Hooley, J. M., Cole, S. H., & Gironde, S. (2012). Borderline personality disorder. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 409–436). New York: Oxford University Press.

Hopwood, C. J., Donnellan, M. B., & Zanarini, M. C. (2010). Temperamental and acute symptoms of borderline personality disorder: Associations with normal personality traits and dynamic relations over time. *Psychological Medicine, 40*, 1871–1878.

Hopwood, C. J., Malone, J. C., Ansell, E. B., Sanislow, C. A., Grilo, C. M., McGlashan, T. H., et al. (2011). Personality assessment in *DSM-5*: Empirical support for rating severity, style, and traits. *Journal of Personality Disorders, 25*, 305–320.

Hopwood, C. J., Thomas, K. M., Markon, K. E., Wright, A. G. C., & Krueger, R. F. (2012). *DSM-5* personality traits and *DSM-IV* personality disorders. *Journal of Abnormal Psychology, 121*, 424–432.

Hyde, L. W., Shaw, D. S., & Hariri, A. R. (2013). Understanding youth antisocial behavior using neuroscience through a developmental psychopathology lens: Review, integration, and directions for research. *Developmental Review, 33*, 168–223.

Jacobson, C. M., Muehlenkamp, J. J., Miller, A. L., & Turner, J. B. (2008). Psychiatric impairment among adolescents engaging in different types of deliberate self-harm. *Journal of Clinical Child and Adolescent Psychology, 37*, 373–375.

John, O. P., Naumann, L. P., & Soto, C. J. (2008). Paradigm shift to the integrative Big Five trait taxonomy. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (3rd ed., pp. 114–158). New York: Guilford Press.

Johnson, J. G., Chen, H., & Cohen, P. (2004). Personality disorder traits during adolescence and relationships with family members during the transition to adulthood. *Journal of Consulting and Clinical Psychology, 72*, 923–932.

Johnson, J. G., Cohen, P., Brown, J., Smailes, E., & Bernstein, D. P. (1999). Childhood maltreatment increases risk for personality disorders during early adulthood. *Archives of General Psychiatry, 56*, 600–606.

Johnson, J. G., Cohen, P., Chen, H., Kasen, S., & Brook, J. S. (2006). Parenting behaviors associated with risk for offspring personality disorder during adulthood. *Archives of General Psychiatry, 63*, 579–587.

Johnson, J. G., Cohen, P., Dohrenwend, B. P., Link, B. G., & Brook, J. S. (1999). A longitudinal investigation of social causation and social selection processes involved in the association between socioeconomic status and psychiatric disorders. *Journal of Abnormal Psychology, 108*, 490–499.

Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S. (2008). Psychiatric disorders in adolescence and early adulthood and risk for child-rearing difficulties during middle childhood. *Journal of Family Issues, 29*, 210–233.

Johnson, J. G., Cohen, P., Kasen, S., Skodol, A. E., Hamagami, F., & Brook, J. S. (2000). Age-related change in personality disorder trait levels between early adolescence and adulthood: A community-based longitudinal investigation. *Acta Psychiatrica Scandinavica, 102*, 265–275.

Johnson, J. G., Cohen, P., Kasen, S., Skodol, A. E., & Oldham, J. M. (2008). Cumulative prevalence of personality disorders between adolescence and adulthood. *Acta Psychiatrica Scandinavia, 118*, 410–413.

Johnson, J. G., Cohen, P., Skodol, A. E., Oldham, J. M., Kasen, S., & Brook, J. S. (1999). Personality disorders in adolescence and risk of major mental disorders and suicidality during adulthood. *Archives of General Psychiatry, 56*, 805–811.

Johnson, J. G., Cohen, P., Smailes, E., Kasen, S., Oldham, J. M., & Skodol, A. E. (2000). Adolescent personality disorders associated with violence and criminal behavior during adolescence and early adulthood. *American Journal of Psychiatry, 157*, 1406–1412.

Johnson, J. G., Cohen, P. Smailes, E. M., Skodol, A. E., Brown, J., & Oldham, J. M. (2001). Childhood verbal abuse and risk for personality disorders during adolescence and early adulthood. *Comprehensive Psychiatry, 42*, 16–23.

Johnson, J. G., First, M. B., Cohen, P., Skodol, A. E., Kasen, S., & Brook, J. S. (2005). Adverse outcomes associated with personality disorder not otherwise specified in a community sample. *American Journal of Psychiatry, 162*, 1926–1932.

Johnson, J. G., Smailes, E. M., Cohen, P., Brown, J., & Bernstein, D. P. (2000). Associations between four types of childhood neglect and personality disorder symptoms during adolescence and early adulthood: Findings of a community-based longitudinal study. *Journal of Personality Disorders, 14*, 171–187.

Johnson, J. G., Tackett, J. L., Baugh, C. S., McCullough, C., Sneed, J. S., Lawson, H., et al. (2012). Emergent personality disorders among adolescents with anxiety disorders. In D. McKay & E. A. Storch (Eds.), *Handbook of anxiety disorders in children and adolescents* (pp. 187–201). New York: Springer.

Kasen, S., Cohen, P., Chen, H., Johnson, J. G., & Crawford, T. (2009). School climate and continuity of adolescent personality disorder symptoms. *Journal of Child Psychology and Psychiatry, 50*, 1504–1512.

Kasen, S., Cohen, P., Skodol, A. E., Johnson, J. G., & Brook, J. S. (1999). Influence of child and adolescent psychiatric disorders on young adult personality disorder. *American Journal of Psychiatry, 156*, 1529–1535.

Kasen, S., Cohen, P., Skodol, A. E., Johnson, J. G., Smailes, E., & Brooke, J. S. (2001). Childhood depression and adult personality disorder. *Archives of General Psychiatry, 58*, 231–236.

Kendler, K. S., Aggen, S. H., Knudsen, G. P., Roysamb, E., Neale, M. C., & Reichborn-Kjennerud, T. (2011). The structure of genetic and environmental risk factors for

syndromal and subsyndromal common *DSM-IV* Axis I and all Axis II disorders. *American Journal of Psychiatry, 168*, 29–39.

Kendler, K. S., Aggen, S. H., Czajkowski, N., Røysamb, E., Tambs, K., Torgersen, S., et al. (2008). The structure of genetic and environmental risk factors for *DSM-IV* personality disorders: A multivariate twin study. *Archives of General Psychiatry, 65*, 1438–1446.

Kendler, K. S., Czajkowski, N., Tambs, K., Torgersen, S., Aggen, S. H., Neal, M. S., et al. (2006). Dimensional representation of *DSM-IV* Cluster A personality disorders in a population-based sample of Norwegian twins: A multivariate study. *Psychological Medicine, 36*, 1583–1591.

Kim, J., Cicchetti, D., Rogosch, F. A., & Manly, J. T. (2009). Child maltreatment and trajectories of personality and behavioral functioning: Implications for the development of personality disorders. *Development and Psychopathology, 21*, 889–912.

Kimonis, E. R., Frick, P. J., Cauffman, E., Goldweber, A., & Skeem, J. (2012). Primary and secondary variants of juvenile psychopathy differ in emotional processing. *Development and Psychopathology, 24*, 1091–1103.

Klein, D. N., Dyson, M. W., Kujawa, A. J., & Kotov, R. (2012). Temperament and internalizing disorders. In M. Zentner & R. L. Shiner (Eds.), *Handbook of temperament* (pp. 541–561). New York: Guilford Press.

Kotler, J. S., & McMahon, R. J. (2005). Child psychopathy: Theories, measurement, and relations with the development and persistence of conduct problems. *Clinical Child and Family Psychology Review, 8*, 291–325.

Krueger, R. F. (2005). Continuity of Axes I and II: Toward a unified model of personality, personality disorders, and clinical disorders. *Journal of Personality Disorders, 19*, 233–261.

Krueger, R. F. (2013). Personality disorders are the vanguard of the post-DSM-5.0 era. *Personality Disorders: Theory, Research, and Treatment, 4*, 355–362.

Krueger, R. F., Derringer, J., Markon, K. E., Watson, D., & Skodol, A. E. (2012). Initial construction of a maladaptive personality trait model and inventory for *DSM-5. Psychological Medicine, 42*, 1879–1890.

Krueger, R. F., Eaton, N. R., Clark, L. A., Watson, D., Markon, K. E., Derringer, J., et al. (2011). Deriving an empirical structure of personality pathology for *DSM-5. Journal of Personality Disorders, 25*, 170–191.

Krueger, R. F., Eaton, N. R., Derringer, J., Markon, K. E., Watson, D., & Skodol, A. E. (2011). Personality in *DSM-5*: Helping delineate personality disorder content and framing the metastructure. *Journal of Personality Assessment, 93*, 325–331.

Krueger, R. F., & Johnson, W. (2008). Behavior genetics and personality: A new look at the integration of nature and nurture. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (3rd ed., pp. 287–310). New York: Guilford Press.

Krueger, R. F., & Markon, K. E. (2008). Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. *Annual Review of Clinical Psychology, 2*, 111–133.

Krueger, R. F., Watson, D., & Barlow, D. H. (2005). Introduction to the special section: Toward a dimensionally based taxonomy of psychopathology. *Journal of Abnormal Psychology, 114*, 491–493.

Kushner, S. C., Tackett, J. L., & De Clercq, B. (2013). The joint hierarchical structure of adolescent personality pathology: Converging evidence from two measures. *Journal of the Canadian Academy of Child and Adolescent Psychiatry, 22*, 199–205.

Kwapil, T. R., & Barrantes-Vidal, N. (2012). Schizotypal personality disorder: An integrative review. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 437–477). New York: Oxford University Press.

Lahti, M., Pesonen, A., Raikkonnen, K., Heinonen, K., Wahlbeck, K., Kajantie, E., et al. (2012). Temporary separation from parents in early childhood and serious personality disorders in adult life. *Journal of Personality Disorders, 26*, 751–762.

Latzman, R. D., Lilienfeld, S. O., Latzman, N. E., & Clark, L. A. (2013). Exploring callous unemotional traits in youth via general personality traits: An eye toward *DSM-5. Personality Disorders: Theory, Research, and Treatment, 4*, 191–202.

Laurenssen, E. M. P., Hutsebaut, J., Feenstra, D. J., Van Busschbach, J. J., & Luyten, P. (2013). Diagnosis of personality disorder in adolescents: A study among psychologists. *Child and Adolescent Psychiatry and Mental Health, 7*, 3.

Lavan, H., & Johnson, J. G. (2002). The association between Axis I and II psychiatric symptoms and high-risk sexual behavior during adolescence. *Journal of Personality Disorders, 16*, 73–94.

Leising, D., & Zimmermann, J. (2011). An integrative conceptual framework for assessing personality and personality pathology. *Review of General Psychiatry, 15*, 317–330.

Lengua, L. J., & Wachs, T. D. (2012). Temperament and risk: Resilient and vulnerable responses to adversity. In M. Zentner & R. L. Shiner (Eds.), *Handbook of temperament* (pp. 519–540). New York: Guilford Press.

Lenzenweger, M. F. (2008). Epidemiology of personality disorders. *Psychiatric Clinics of North America, 31*, 395–403.

Lenzenweger, M. F. (2010). A source, a cascade, a schizoid: A heuristic proposal from the Longitudinal Study of Personality Disorders. *Development and Psychopathology, 22*, 867–881.

Levy, K. N. (2005). The implications of attachment theory and research for understanding borderline personality disorder. *Development and Psychopathology, 17*, 959–986.

Levy, K. N., Becker, D. F., Grilo, C. M., Mattanah, J. J. F., Barnet, K. E., Quinlan, D. M., et al. (1999). Concurrent and predictive validity of the personality disorder diagno-

sis in adolescent patients. *American Journal of Psychiatry, 156*, 1522–1528.

Lewinsohn, P. M., Rohde, P., Seeley, J. R., & Klein, D. N. (1997). Axis II psychopathology as a function of Axis I disorders in childhood and adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 1752–1759.

Linde, J. A., Stringer, D., M., Simms, L. J., & Clark, L. A. (2013). The Schedule for Nonadaptive and Adaptive Personality for Youth (SNAP-Y): A new measure for assessing adolescent personality and personality pathology. *Assessment, 20*, 387–404.

Links, P. S., Ansari, J. Y., Fazalullash, F., & Shah, R. (2012). The relationship of personality disorders and Axis I clinical disorders. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 237–259). New York: Oxford University Press.

Livesley, W. J. (2005). Behavioral and molecular genetic contributions to a dimensional classification of personality disorder. *Journal of Personality Disorders, 19*, 131–155.

Livesley, W. J. (2007). A framework for integrating dimensional and categorical classifications of personality disorder. *Journal of Personality Disorders, 21*, 199–224.

Livesley, W. J., & Jackson, D. N. (2009). *Dimensional Assessment of Personality Pathology—Basic Questionnaire (DAPP-BQ): Technical manual*. Port Huron, MI: Sigma Assessment Systems.

Lodi-Smith, J. L., & Roberts, B. W. (2007). Social investment and personality: A meta-analytic analysis of the relationship of personality traits to investment in work, family, religion, and volunteerism. *Personality and Social Psychology Review, 11*, 68–86.

Ludolph, P. S., Westen, D., Misle, B., & Jackson, A. (1990). The borderline diagnosis in adolescents: Symptoms and developmental history. *American Journal of Psychiatry, 147*, 470–476.

Lynam, D. R. (1997). Pursuing the psychopath: Capturing the fledgling psychopath in a nomological net. *Journal of Abnormal Psychology, 106*, 425–438.

Lynam, D. R., Caspi, A., Moffitt, T. E., Raine, A., Loeber, R., & Stouthamer-Loeber, M. (2005). Adolescent psychopathy and the Big Five: Results from two samples. *Journal of Abnormal Child Psychology, 33*, 431–443.

Lynam, D. R., Charnigo, R., Moffitt, T. E., Raine, A., Loeber, R., & Stouthamer-Loeber, M. (2009). The stability of psychopathy across adolescence. *Development and Psychopathology, 21*, 1133–1153.

Lynam, D. R., & Gudonis, L. (2005). The development of psychopathy. *Annual Review of Clinical Psychology, 1*, 381–407.

Lynam, D. R., Loeber, R., & Stouthamer-Loeber, M. (2008). The stability of psychopathy from adolescence into adulthood: The search for moderators. *Criminal Justice and Behavior, 35*, 228–243.

Magnavita, J. J., Powers, A. D., Barber, J. P., & Oltmanns, T.

F. (2013). Personality disorders. In L. G. Castonguay & T. F. Oltmanns (Eds.), *Psychopathology: From science to clinical practice* (pp. 275–318). New York: Guilford Press.

Markon, K. E., Krueger, R. F., & Watson, D. (2005). Delineating the structure of normal and abnormal personality: An integrative hierarchical approach. *Journal of Personality and Social Psychology, 88*, 139–157.

Mattanah, J. J. F., Becker, D. F., Levy, K. N., Edell, W. W., & McGlashan, T. H. (1995). Diagnostic stability in adolescents followed up 2 years after hospitalization. *American Journal of Psychiatry, 152*, 889–894.

McAdams, D. P. (2008). Personal narratives and the life story. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (3rd ed., pp. 242–262). New York: Guilford Press.

McAdams, D. P. (2009). *The person: An introduction to the science of personality psychology* (5th ed.). Hoboken, NJ: Wiley.

McAdams, D. P. (2013). The psychological self as actor, agent, and author. *Perspectives on Psychological Science, 8*, 272–295.

McAdams, D. P., & McLean, K. C. (2013). Narrative identity. *Current Directions in Psychological Science, 22*, 233–238.

McAdams, D. P., & Olson, B. D. (2010). Personality development: Continuity and change over the life course. *Annual Review of Psychology, 61*, 517–542.

McAdams, D. P., & Pals, J. L. (2006). A new Big Five: Fundamental principles for an integrative science of personality. *American Psychologist, 61*, 204–217.

McGilloway, A., Hall, R. E., Lee, T., & Bhui, K. S. (2010). A systematic review of personality disorder, race and ethnicity: Prevalence, aetiology and treatment. *BMC Psychiatry, 10*, 33.

McGlashan, T. H., Grilo, C. M., Sanislow, C. A., Ralevski, E., Morey, L. C., Gunderson, J. G., et al. (2005). Two-year prevalence and stability of individual DSM-IV criteria for schizotypal, borderline, avoidant, and obsessive–compulsive personality disorders: Toward a hybrid model of Axis II disorders. *American Journal of Psychiatry, 162*, 883–889.

McLean, K. C., & Pasupathi, M. (2012). Processes of identity development: Where I am and how I got there. *Identity: An International Journal of Theory and Research, 12*, 8–28.

Mervielde, I., De Clercq, B., De Fruyt, F., & van Leeuwen, K. (2005). Temperament, personality, and developmental psychopathology as childhood antecedents of personality disorders. *Journal of Personality Disorders, 19*, 171–201.

Mervielde, I., & De Fruyt, F. (2002). Assessing children's traits with the Hierarchical Personality Inventory for Children. In B. De Raad & M. Perugini (Eds.), *Big Five assessment* (pp. 129-142). Ashland, OH: Hogrefe & Huber.

Mikulincer, M., & Shaver, P. R. (2007). *Attachment in adulthood: Structure, dynamics, and change*. New York: Guilford Press.

Miller, A. L., Muehlenkamp, J. J., & Jacobson, C. M. (2008). Fact or fiction: Diagnosing borderline personality disorder in adolescents. *Clinical Psychology Review, 28*, 969–981.

Millon, T. (2010). A sociocultural conception of the borderline personality disorder epidemic. In R. F. Krueger & E. Simonsen (Eds.), *Contemporary directions in psychopathology: Scientific foundations of the DSM-V and ICD-11* (pp. 111–123). New York: Guilford Press.

Millon, T. (2012). On the history and future study of personality and its disorders. *Annual Review of Clinical Psychology, 8*, 1–19.

Mittal, V. A., Tessner, K. D., McMillan, A. L., Delawalla, Z., Trotman, H. D., & Walker, E. F. (2006). Gesture behavior in unmedicated schizotypal adolescents. *Journal of Abnormal Psychology, 115*, 351–358.

Moffitt, T. E., Arseneault, L. Jaffee, S. R., Kim-Cohen, J. Koenen, K. C., Odgers, C. L. et al. (2008). Research review: DSM-V conduct disorder: Research needs for an evidence base. *Journal of Child Psychology and Psychiatry, 49*, 3–33.

Moffitt, T. E., Caspi, A., Harrington, H., & Milne, B. J. (2002). Males on the life-course-persistent and adolescence-limited antisocial pathways: Follow-up at age 26 years. *Development and Psychopathology, 14*, 179–207.

Moffitt, T. E., Caspi, A., Rutter, M., & Silva, P. A. (2001). *Sex differences in antisocial behaviour: Conduct disorder, delinquency, and violence in the Dunedin longitudinal study.* New York: Cambridge University Press.

Morey, L. C., Berghuis, H., Gender, D. S., Verheul, R., Krueger, R. F., & Skodol, A. E. (2011). Toward a model for assessing level of personality functioning in DSM-5, Part II: Empirical articulation of a core dimension of personality pathology. *Journal of Personality Assessment, 93*, 347–353.

Mulder, R. T. (2012). Cultural aspects of personality disorder. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 260–274). New York: Oxford University Press.

Muñoz, L. C., Kerr, M., & Besic, N. (2008). The peer relationships of youths with psychopathic personality traits. *Criminal Justice and Behavior, 35*, 212–227.

Natsuaki, M. N., Cicchetti, D., & Rogosch, F. A. (2009). Examining the developmental history of child maltreatment, peer relations, and externalizing problems among adolescents with symptoms of paranoid PD. *Development and Psychopathology, 21*, 1181–1193.

Nigg, J. T. (2006). Temperament and developmental psychopathology. *Journal of Child Psychology and Psychiatry, 47*, 395–422.

Nigg, J. T., Silk, K. R., Stavro, G., & Miller, T. (2005). Disinhibition and borderline personality disorder. *Development and Psychopathology, 17*, 1129–1149.

Noble, E. P., Ozkaragoz, T. Z., Ritchie, T. L., Zhang, X., Belin, T. R., & Sparkes, R. S. (1998). D2 and D4 dopamine receptor polymorphisms and personality. *American Journal of Medical Genetics, 81*, 257–267.

Nock, M. K. (2010). Self-injury. *Annual Review of Clinical Psychology, 6*, 339–363.

Nock, M. K., Joiner, T. E., Gordon, K. H., Lloyd-Richardson, E., & Prinstein, M. (2006). Non-suicidal self-injury among adolescents: Diagnostic correlates and relation to suicide attempts. *Psychiatry Research, 144*, 65–72.

Nunes, P. M., Wenzel, A., Borges, K. T., Porto, C. R., Caminha, R. M., & Reis, D. O. (2009). Volumes of the hippocampus and amygdala in patients with borderline personality disorder: A meta-analysis. *Journal of Personality Disorders, 23*, 333–345.

Oldham, J. M. (2005). Personality disorders: Recent history and future directions. In J. M. Oldham, A. E. Skodol, & D. S. Bender (Eds.), *The American Psychiatric Publishing textbook of personality disorders* (pp. 3–16). Washington, DC: American Psychiatric Publishing.

Oltmanns, T. F., & Powers, A. D. (2012). Gender and personality disorders. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 206–218). New York: Oxford University Press.

Pals, J. L. (2006). Authoring a second chance in life: Emotion and transformational processing within narrative identity. *Research in Human Development, 3*, 101–120.

Paris, J. (2003). Personality disorders over time: Precursors, course, and outcome. *Journal of Personality Disorders, 17*, 479–488.

Paris, J. (2005). A current integrative perspective on personality disorders. In J. M. Oldham, A. E. Skodol, & D. S. Bender (Eds.), *The American Psychiatric Publishing textbook of personality disorders* (pp. 119–128). Washington, DC: American Psychiatric Publishing.

Paris, J. (2007). An overview on gender, personality, and mental health. *Personality and Mental Health, 1*, 14–20.

Paris, J. (2012). Pathology of personality disorder: An integrative conceptualization. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 399–406). New York: Oxford University Press.

Paris, J., Zweig-Frank, H., & Guzder, J. (1994). Risk factors for borderline personality in male outpatients. *Journal of Nervous and Mental Disease, 182*, 375–380.

Pincus, A. L., & Hopwood, C. J. (2012). A contemporary interpersonal model of personality pathology and personality disorder. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 372–398). New York: Oxford University Press.

Pomerantz, E. M., & Thompson, R. A. (2008). Parents' role in children's personality development: The psychological resource principle. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (3rd ed., pp. 351–374). New York: Guilford Press.

Reich, D. B., & Zanarini, M. C. (2001). Developmental aspects of borderline personality disorder. *Harvard Review of Psychiatry, 9*, 294–301.

Reichborn-Kjennerud, T., Czajkowski, N., Neale, M. S., Orstavik, R. E., Torgersen, S., Tambs, K., et al. (2007). Genetic and environmental influences on dimensional representations of *DSM-IV* Cluster C personality disorders: A population-based multivariate twin study. *Psychological Medicine, 37*, 645–653.

Reichborn-Kjennerud, T., Czajkowski, N., Roysamb, E., Orstavik, R. E., Neale, M. C., Torgersen, S., et al. (2010). Major depression and dimensional representations of *DSM-IV* personality disorders: A population-based twin study. *Psychological Medicine, 40*, 1475–1484.

Rettrew, D. C., Zanarini, M. C., Yen, S., Grilo, C. M., Skodol, A. E., Shea, T., et al. (2003). Childhood antecedents of avoidant personality disorder: A retrospective study. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*, 1122–1130.

Ro, E., Stringer, D., & Clark, L. A. (2012). The Schedule for Nonadaptive and Adaptive Personality: A useful tool for diagnosis and classification of personality disorder. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 58–81). New York: Oxford University Press.

Roberts, B. W., & DelVecchio, W. F. (2000). The rank-order consistency of personality traits from childhood to old age: A quantitative review of longitudinal studies. *Psychological Bulletin, 126*, 3–25.

Roberts, B. W., Walton, K. E., & Viechtbauer, W. (2006). Patterns of mean-level change in personality traits across the life course: A meta-analysis of longitudinal studies. *Psychological Bulletin, 132*, 1–25.

Roberts, B. W., Wood, D., & Caspi, A. (2008). The development of personality traits in adulthood. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (3rd ed., pp. 375–398). New York: Guilford Press.

Rogosch, F. A., & Cicchetti, D. (2004). Child maltreatment and emergent personality organization: Perspectives from the five-factor model. *Journal of Abnormal Child Psychology, 32*, 123–145.

Roose, A., Bijttebier, P., Claes, L., Lilienfeld, S. O., De Fruyt, F., & Decuyper, M. (2012). Psychopathic traits in adolescence and the five-factor model of personality. *Journal of Psychopathology and Behavioral Assessment, 34*, 84–93.

Rothbart, M. K., Ahadi, S. A., & Evans, D. E. (2000). Temperament and personality: Origins and outcomes. *Journal of Personality and Social Psychology, 78*, 122–135.

Rothbart, M. K., Ahadi, S. A., Hershey, K. L., & Fisher, P. (2001). Investigation of temperament at three to seven years: The Children's Behavior Questionnaire. *Child Development, 72*, 1394–1408.

Rothbart, M. K., & Bates, J. E. (2006). Temperament. In W. Damon & R. Lerner (Series Eds.) & N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 99–166). Hoboken, NJ: Wiley.

Rounsaville, B. J., Alarcón, R. D., Andrews, G., Jackson, J. S., Kendell, R. E., & Kendler, K. (2002). Basic nomenclature issues for DSM-V. In D. J. Kupfer, M. B. First, & D. A. Regier (Eds.), *A research agenda for DSM-V* (pp. 1–29). Washington, DC: American Psychiatric Publishing.

Roussos, P., & Siever, L. J. (2012). Neurobiological contributions. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 299–324). New York: Oxford University Press.

Salekin, R. T., Debus, S. A., & Barker, E. D. (2010). Adolescent psychopathy and the five factor model: Domain and facet analysis. *Journal of Psychopathology and Behavioral Assessment, 32*, 501–514.

Salekin, R. T., Leistico, A. R., Trobst, K. K., Schrum, C. L., & Lochman, J. E. (2005). Adolescent psychopathy and personality theory—the interpersonal circumplex: Expanding evidence of a nomological net. *Journal of Abnormal Child Psychology, 33*, 445–460.

Salekin, R. T., Rosenbaum, J., & Lee, Z. (2008). Child and adolescent psychopathy: Stability and change. *Psychiatry, Psychology and Law, 15*, 224–236.

Samuels, J., & Costa, P. T. (2012). Obsessive–compulsive personality disorder. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 566–581). New York: Oxford University Press.

Saudino, K. J., & Wang, M. (2012). Quantitative and molecular genetic studies of temperament. In M. Zentner & R. L. Shiner (Eds.), *Handbook of temperament* (pp. 315–346). New York: Guilford Press.

Serman, N., Johnson, J. G., Geller, P. A., Kanost, R. E., & Zacharapoulou, H. (2002). Personality disorders associated with substance use among American and Greek adolescents. *Adolescence, 37*, 841–854.

Sharp, C. (2012). *The developmental building blocks of psychopathic traits: Revisiting the role for theory of mind.* Paper presented at the 1st World Conference on Personality, Stellenbosch, South Africa.

Sharp, C. (in press). The social-cognitive basis of BPD: Toward a theory of hypermentalizing. In C. Sharp & J. L. Tackett (Eds.), *Handbook of borderline personality disorder in children and adolescents.* New York: Springer.

Shiner, R. L. (2007). Personality disorders. In E. J. Mash & R. A. Barkley (Eds.), *Assessment of childhood disorders* (4th ed., pp. 781–816). New York: Guilford Press.

Shiner, R. L. (2009). The development of personality disorders: Perspectives from normal personality development in childhood and adolescence. *Development and Psychopathology, 21*, 715–734.

Shiner, R. L. (2010). Mapping the landscape of personality in childhood and adolescence. *Social and Personality Psychology Compass, 4*, 1084–1097.

Shiner, R. L. (2014). The development of temperament and personality traits in childhood and adolescence. In M. Mikulincer & P. Shaver (Eds.) & M. L. Cooper & R. Larsen (Assoc. Eds.), *APA handbook of personality and social psychology: Vol. 3. Personality processes and individual*

differences (pp. 85–105). Washington, DC: American Psychological Association.

Shiner, R. L., & Caspi, A. (2012). Temperament and the development of personality traits, adaptations, and narratives. In M. Zentner & R. L. Shiner (Eds.), *Handbook of temperament* (pp. 497–516). New York: Guilford Press.

Shiner, R. L., & DeYoung, C. G. (2013). The structure of temperament and personality traits: A developmental perspective. In P. Zelazo (Ed.), *Oxford handbook of developmental psychology* (pp. 113–141). New York: Oxford University Press.

Silbersweig, D., Clarkin, J. F., Goldstein, M., Kernberg, O. F., Tuescher, O., Levy, K. N., et al. (2007). Failure of fronto-limbic inhibitory function in the context of negative emotion in borderline personality disorder. *American Journal of Psychiatry, 164*, 1832–1841.

Simonsen, E., & Widiger, T. A. (2005). Introduction. In T. A. Widiger, E. Simonsen, P. J. Sirovatka, & D. A. Regier (Eds.), *Dimensional models of personality disorders: Refining the research agenda for DSM-V* (pp. xxv–xxxiii). Washington, DC: American Psychiatric Association.

Skinner, E. A., & Zimmer-Gembeck, M. J. (2007). The development of coping. *Annual Review of Psychology, 58*, 119–144.

Skodol, A. E. (2005). Manifestations, clinical diagnosis, and comorbidity. In J. M. Oldham, A. E. Skodol, & D. S. Bender (Eds.), *The American Psychiatric Publishing textbook of personality disorders* (pp. 57–87). Washington, DC: American Psychiatric Publishing.

Skodol, A. E. (2012). Personality disorders in *DSM-5*. *Annual Review of Clinical Psychology, 8*, 317–334.

Skodol, A. E., Bender, D. S., Morey, L. C., Clark, L. A., Oldham, J. M., Alarcon, R. D., et al. (2011). Personality disorder types proposed for DSM-5. *Journal of Personality Disorders, 25*, 136–169.

Skodol, A. E., Bender, D. S., Pagano, M. E., Shea, M. T., Yen, S., Sanislow, C. A., et al. (2007). Positive childhood experiences: Resilience and recovery from personality disorder in early adulthood. *Journal of Clinical Psychiatry, 68*, 1102–1108.

Skodol, A. E., Clark, L. A., Bender, D. S., Krueger, R. F., Morey, L. C., Verheul, R., et al. (2011). Proposed changes in personality and personality disorder assessment and diagnosis for *DSM-5:* Part I. Description and rationale. *Personality Disorders: Theory, Research, and Treatment, 2*, 4–22.

Skodol, A. E., Gunderson, J. G., Shea, M. T., McGlashan, T. H., Morey, L. C., Sanislow, C. A., . et al. (2005). The Collaborative Longitudinal Personality Disorders Study (CLPS): Overview and implications. *Journal of Personality Disorders, 19*, 487–504.

Skodol, A. W., Johnson, J. G., Cohen, P., Sneed, J. R., & Crawford, T. N. (2007). Personality disorder and impaired functioning from adolescence to adulthood. *British Journal of Psychiatry, 190*, 415–420.

Soto, C. J., John, O. P., Gosling, S. M., & Potter, J. (2011). Age differences in personality traits from 10 to 65: Big Five domains and facets in a large cross-sectional sample. *Journal of Personality and Social Psychology, 100*, 330–348.

South, S. C., Reichborn-Jennerud, T., Eaton, N. R, & Krueger, R. F. (2012). Behavior and molecular genetics of personality disorders. In T. A. Widiger (Ed.), *The Oxford handbook of personality disorders* (pp. 143–165). New York: Oxford University Press.

Sroufe, L. A. (2005). Attachment and development: A prospective, longitudinal study from birth to adulthood. *Attachment and Human Development, 7*, 349–367.

Sroufe, L. A., Carlson, E. A., Levy, A. K., & Egeland, B. (1999). Implications of attachment theory for developmental psychopathology. *Development and Psychopathology, 11.* 1–13.

Stepp, S. D. (2012). Development of borderline personality disorder in adolescence and young adulthood: Introduction to the special section. *Journal of Abnormal Child Psychology, 40*, 1–5.

Stepp, S. D., Olino, T. M., Klein, D. N., Seeley, J. R., & Lewinsohn, P. M. (2013). Unique influences of adolescent antecedents on adult borderline personality disorder features. *Personality Disorders: Theory, Research, and Treatment, 4*, 223–229.

Tackett, J. L. (2006). Evaluating models of the personality–psychopathology relationship in children and adolescents. *Clinical Psychology Review, 26*, 584–599.

Tackett, J. L. (2010). Measurement and assessment of child and adolescent personality pathology: An introduction to the special issue. *Journal of Psychopathology and Behavioral Assessment, 32*, 463–466.

Tackett, J. L. (2011). Parent informants for child personality: Agreement, discrepancies, and clinical utility. *Journal of Personality Assessment, 93*, 539–544.

Tackett, J. L., Balsis, S., Oltmanns, T. F., & Krueger, R. F. (2009). A unifying perspective on personality pathology across the life span: Developmental considerations for the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders. Development and Psychopathology, 21*, 687–713.

Tackett, J. L., Herzhoff, K., Reardon, K. W., De Clercq, B., & Sharp, C. (in press). The externalizing spectrum in youth: Incorporating personality pathology. *Journal of Adolescence.*

Tackett, J. L., Krueger, R. F., Iacono, W. G., & McGue, M. (2005). Symptom-based subfactors of DSM-defined conduct disorder: Evidence for etiologic distinctions. *Journal of Abnormal Psychology, 114*, 483–487.

Tackett, J. L., & Kushner, S. K. (in press). Conceptualizing youth BPD within a normative personality framework. In C. Sharp & J. L. Tackett (Eds.), *Handbook of borderline personality disorder in children and adolescents.* New York: Springer.

Tackett, J. L., & Sharp, C. (Eds.). (2014). A developmental

psychopathology perspective on personality disorder: Introduction to the special issue. *Journal of Personality Disorders, 28*(1), 1–4.

Tackett, J. L., Silberschmidt, A. L., Krueger, R. F., & Sponheim, S. R. (2008). A dimensional model of personality disorder: Incorporating DSM Cluster A characteristics. *Journal of Abnormal Psychology, 117*, 454–459.

Tackett, J. L., Slobodskaya, H., Mar, R. A., Deal, J., Halverson, Jr., C. F., Baker, S. R., et al. (2012). The hierarchical structure of childhood personality in five countries: Continuity from early childhood to early adolescence. *Journal of Personality, 80*, 1–33.

Thomaes, S., Brummelman, E., Reijntjes, A., & Bushman, B. J. (2013). When Narcissus was a boy: Origins, nature, and consequences of childhood narcissism. *Child Development Perspectives, 7*, 22–26.

Thomaes, S., Bushman, B. J., Orobio de Castro, B., & Stegge, H. (2009). What makes narcissists bloom?: A framework for research on the etiology and development of narcissism. *Development and Psychopathology, 21*, 1233–1247.

Thomaes, S., Stegge, H., Bushman, B. J., Olthof, T., & Denissen, J. (2008). Development and validation of the Childhood Narcissism Scale. *Journal of Personality Assessment, 90*, 382–391.

Torgersen, S. (2012). Epidemiology. In T. A. Widiger (Ed.), *The Oxford handbook of personality disorders* (pp. 186–205). New York: Oxford University Press.

Torgersen, S., Czajkowski, N., Jacobson, K., Reichborn-Kjennerud, T., Roysamb, E., Neale, M. S., et al. (2008). Dimensional representations of *DSM-IV* Cluster B personality disorders in a population-based sample of Norwegian twins: A multivariate study. *Psychological Medicine, 38*, 1617–1625.

Torgersen, S., Lygren, S., Oien, P. A., Skre, I., Onstad, S., Edvardsen, J., . et al. (2000). A twin study of personality disorders. *Comprehensive Psychiatry, 41*, 416–425.

Tromp, N. B., & Koot, H. M. (2008). Dimensions of personality pathology in adolescents: Psychometric properties of the DAPP-BQ-A. *Journal of Personality Disorders, 22*, 623–638.

Tromp, N. B., & Koot, H. M. (2010). Dimensions of normal and abnormal personality: Elucidating DSM-IV personality disorder symptoms in adolescents. *Journal of Personality, 78*, 839–864.

Trull, T. J. (2012). The five-factor model of personality disorder and DSM-5. *Journal of Personality, 80*, 1697–1720.

Trull, T. J., & Durrett, C. A. (2005). Categorical and dimensional models of personality disorder. *Annual Review of Clinical Psychology, 1*, 355–380.

Trull, T. J., Scheiderer, E. M., & Tomko, R. L. (2012). Axis II comorbidity. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 219–236). New York: Oxford University Press.

Vachon, D. D., Lynam, D. R., Loeber, R., & Stouthamer-Loeber, M. (2012). Generalizing the nomological network

of psychopathy across populations differing on race and conviction status. *Journal of Abnormal Psychology, 121*, 263–269.

Vachon, D. D., Lynam, D. R., Widiger, T. A., Miller, J. D., McCrae, R. R., & Costa, P. T. (2013). Basic traits predict the prevalence of personality disorder across the life span: The example of psychopathy. *Psychological Science, 24*, 698–705.

Vazire, S. (2010). Who knows what about a person?: The self–other knowledge asymmetry (SOKA) model. *Journal of Personality and Social Psychology, 98*, 281–300.

Venables, P. H., & Raine, A. (2012). Poor nutrition at age 3 and schizotypal personality at age 23: The mediating role of age 11 cognitive functioning. *American Journal of Psychiatry, 169*, 822–830.

Venta, A., Ross, E., Schatte, D., & Sharp, C. (2012). Suicide ideation and attempts among inpatient adolescents with borderline personality disorder: Frequency, intensity, and age of onset. *Personality and Mental Health, 6*, 340–351.

Verheul, R., Bartak, A., & Widiger, T. (2007). Prevalence and construct validity of personality disorder not otherwise specified (PD-NOS). *Journal of Personality Disorders, 21*, 359–370.

Verheul, R., & Widiger, T. A. (2004). A meta-analysis of the prevalence and usage of the personality disorder not otherwise specified (PD-NOS) diagnosis. *Journal of Personality Disorders, 18*, 309–319.

Walterfang, M., Chanen, A. M., Barton, S., Wood, A. G., Jones, J., Reutens, D. C., et al. (2010). Corpus callosum morphology and relationship to orbitofrontal and lateral ventricular volume in teenagers with first-presentation borderline personality disorder. *Psychiatry Research: Neuroimaging, 183*, 30–37.

Warner, M. B., Morey, L. C., Finch, J. F., Gunderson, J. G., Skodol, A. E., et al. (2004). The longitudinal relationship of personality traits and disorders. *Journal of Abnormal Psychology, 113*, 217–227.

Weinstein, D. D., Diforio, D., Schiffman, J., Walker, E., & Bonsall, R. (1999). Minor physical anomalies, dermatoglyphic asymmetries, and cortisol levels in adolescents with schizotypal personality disorder. *American Journal of Psychiatry, 156*, 617–623.

Weise, K. L., & Tuber, S. (2004). The self and object representations of narcissistically disturbed children: An empirical investigation. *Psychoanalytic Psychology, 21*, 244–258.

Westen, D., & Chang, C. (2000). Personality pathology in adolescence: A review. In A. H. Esman, L. T. Flaherty, & H. A. Horowitz (Eds.), *Adolescent psychiatry: Developmental and clinical studies* (Vol. 25, pp. 61–100). Mahwah, NJ: Analytic Press.

Westen, D., Dutra, L., & Shedler, J. (2005). Assessing adolescent personality pathology. *British Journal of Psychiatry, 186*, 227–238.

Westen, D., Nakash, O., Thomas, C., & Bradley, R. (2006).

Clinical assessment of attachment patterns and personality disorder in adolescents and adults. *Journal of Consulting and Clinical Psychology, 74*, 1065–1085.

Westen, D., Shedler, J., Durrett, C., Glass, S., & Martens, A. (2003). Personality diagnoses in adolescence: DSM-IV Axis II diagnoses and an empirically derived alternative. *American Journal of Psychiatry, 160*, 952–966.

Weston, C. G. & Riolo, S. A. (2007). Childhood and adolescent precursors to adult personality disorders. *Psychiatric Annals, 37*, 114–120.

White, S. F., Brislin, S. J., Meffert, H., Sinclair, S., & Blair, R. J. R. (2013). Callous–unemotional traits modulate the neural response associated with punishing another individual during social exchange: A preliminary investigation. *Journal of Personality Disorders, 27*, 99–112.

Widiger, T. A. (2010). Cluster A personality symptomatology in youth. *Journal of Psychopathology and Behavioral Assessment, 32*, 551–556.

Widiger, T. A. (2011). A shaky future for personality disorders. *Personality Disorders: Theory, Research, and Treatment, 2*, 54–67.

Widiger, T. A. (2012). Historical developments and current issues. In T. A. Widiger (Ed.), *Oxford handbook of personality disorders* (pp. 13–34). New York: Oxford University Press.

Widiger, T. A. (2013). A postmortem and future look at the personality disorders in DSM-5. *Personality Disorders: Theory, Research, and Treatment, 4*, 382–387.

Widiger, T. A., & Costa, P. T., Jr. (Eds.). (2013). *Personality disorders and the Five-Factor model of personality* (3rd ed.).Washington, DC: American Psychological Association.

Widiger, T. A., De Clercq, B., & De Fruyt, F. (2009). Childhood antecedents of personality disorder: An alternative perspective. *Development and Psychopathology, 21*, 771–791.

Widiger, T. A., & Mullins-Sweatt, S. N. (2005). Categorical and dimensional models of personality disorders. In J. M. Oldham, A. E. Skodol, & D. S. Bender (Eds.), *The American Psychiatric Publishing textbook of personality disorders* (pp. 35–53). Washington, DC: American Psychiatric Publishing.

Widiger, T. A., & Simonsen, E. (2005). Alternative dimensional models of personality disorder: Finding a common ground. *Journal of Personality Disorders, 19*, 110–130.

Widiger, T. A., Simonsen, E., Sirovatka, P. J., & Regier, D. A. (Eds.). (2005). *Dimensional models of personality disorders: Refining the research agenda for DSM-V.* Washington DC: American Psychiatric Association.

Widiger, T. A., & Trull, T. J. (2007). Plate tectonics in the classification of personality disorder. *American Psychologist, 62*, 71–83.

Winograd, G., Cohen, P., & Chen, H. (2008). Adolescent borderline symptoms in the community: Prognosis for functioning over 20 years. *Journal of Child Psychology and Psychiatry, 49*, 933–941.

Wolke, D., Schreier, A., Zanarini, M. C., & Winsper, C. (2012). Bullied by peers in childhood and borderline personality symptoms at 11 years of age: A prospective study. *Journal of Child Psychology and Psychiatry, 53*, 846–855.

Wright, A. G., Thomas, K. M., Hopwood, C. J., Markon, K. E., Pincus, A. L., & Krueger, R. F. (2012). The hierarchical structure of DSM-5 pathological personality traits. *Journal of Abnormal Psychology, 121*, 951–957.

Zaider, T. I., Johnson, J. G., & Cockell, S. J. (2000). Psychiatric comorbidity associated with eating disorder symptomatology among adolescents in the community. *International Journal of Eating Disorders, 28*, 58–67.

Zanarini, M. C., & Frankenburg, F. R. (2007). The essential nature of borderline psychopathology. *Journal of Personality Disorders, 21*, 518–535.

Zanarini, M. C., Frankenburg, F. R., Hennen, J., Reich, B., & Silk, K. R. (2005). The McLean Study of Adult Development (MSAD): Overview and implications of the first six years of prospective follow-up. *Journal of Personality Disorders, 19*, 505–523.

Zanarini, M. C., Frankenburg, F. R., Ridolfi, M. E., Jager-Hyman, S., Hennen, J., & Gunderson, J. G. (2006). Reported childhood onset of self-mutilation among borderline patients. *Journal of Personality Disorders, 20*, 9–15.

Zentner, M., & Shiner, R. L. (Eds.). (2012). *Handbook of temperament.* New York: Guilford Press.