

Cluster A Personality Disorders: Schizotypal, Schizoid and Paranoid Personality Disorders in Childhood and Adolescence

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Abstract Cluster A personality disorders (PD), including schizotypal personality disorder (SPD), paranoid personality disorder (PPD), and schizoid PD, are marked by odd and eccentric behaviors, and are grouped together because of common patterns in symptomatology as well as shared genetic and environmental risk factors. The DSM-IV-TR describes personality disorders as representing stable and enduring patterns of maladaptive traits, and much of what is understood about Cluster A personality disorders in particular stems from research with adult populations. Less is known about these disorders in children and adolescents, and controversy remains regarding diagnosis of personality disorders in general in youth. The current paper reviews the available research on Cluster A personality disorders in childhood and adolescence; specifically, we discuss differentiating between the three disorders and distinguishing them from other syndromes, measuring Cluster A disorders in youth, and the nature and course of these disorders throughout childhood and adolescence. We also present recent longitudinal data from a sample of adolescents diagnosed with Cluster A personality disorders from our research laboratory, and suggest directions for future research in this important but understudied area.

Keywords Schizotypal personality disorder · Schizoid personality disorder · Paranoid personality disorder · Personality disorders · Cluster A

Personality disorders are typically referred to as a class of personality types which deviate from contemporary societal expectations (Berrios 1993) and are characterized by relatively stable patterns of maladaptive traits and behaviors (Grilo et al. 2004). In general, individuals diagnosed with personality disorders are classified based on the traits that tend to cause them to feel and behave in socially dysfunctional ways, and these traits are often an extreme deviation from the way an average person in a particular culture perceives, thinks, feels, and relates to others. According to the American Psychiatric Association (APA), these presumably enduring patterns of behaviors, generally stable and pervasive across contexts, are often congruent with inner experiences, and are therefore subjectively perceived by the individual as appropriate or normative (APA 2000). While some of the ten personality disorders defined by the *Diagnostic and Statistical Manual, Fourth Edition, Text Revision* (DSM-IV-TR; APA 2000) have very little in common, those that appear to share similar characteristics are classified into one of three “clusters”. Those diagnosed with schizotypal, schizoid, and paranoid personality disorders are grouped together in Cluster A, and are classified by the DSM-IV-TR as representing “odd and eccentric behaviors” (APA 2000).

Considered as the more severe personality disorders (Leaf et al. 1992; Vaglum et al. 1990), Cluster A personality disorders are assumed by many to be resistant to treatment (Kosky and Thorne 2001). Furthermore, individuals diagnosed with these personality disorders have been noted to see the world as being ‘out of line’ (Derksen 1995), rather than themselves being out of ‘sync’ with the world around them. As a result, others generally consider these individuals to be overly self-centered, leading to significant difficulties in the initiation and maintenance of relationships (Hirschfeld 1993; Kosky and Thorne 2001). Due to the striking similarities between symptomatology of

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Cluster A personality disorders and Axis I diagnoses, particularly schizophrenia (Kalus et al. 1993; Rouff 2000), these disorders can also be difficult to both diagnose and manage (Tredget 2001). In the current paper, we describe and differentiate the three Cluster A personality disorders and provide a review of previous literature concerning: (1) common approaches to measuring Cluster A personality disorders throughout the lifespan; (2) what is known about the developmental trajectory of the three Cluster A personality disorders throughout childhood and adolescence; and (3) outcomes of such disorders in children and adolescents. Finally, we offer recent empirical longitudinal data from a sample of adolescents diagnosed with Cluster A personality disorders, and suggest avenues of future empirical research on Cluster A personality disorders in youth.

Cluster A Personality Disorders: Description and Differentiation

Prevalent in approximately 3–4% of the general population (APA 2000; Johnson et al. 2000b), *schizotypal personality disorder* (SPD) was first described in DSM-III, and criteria of the disorder were based on characteristics of first-degree relatives of patients with schizophrenia (Siever and Gunderson 1983; Spitzer et al. 1979). According to the most recent version of the DSM, SPD is characterized by nine signs and symptoms, including ideas of reference, odd beliefs (ideas that the individual can “know” what others are thinking, premonitions about when something bad is going to happen; Beck and Freeman 1990), unusual perceptual experiences, odd thinking and speech (vague, circumstantial, or tangential), paranoid ideation, inappropriate or constricted affect, odd or eccentric appearance or behavior, lack of close friends, and paranoia-associated social anxiety.

Raine (2006) hypothesizes that there are two forms of SPD: one form that represents a constellation of neurodevelopmental impairments that makes an individual vulnerable to developing schizophrenia, and a second form that is characterized by more psychosocial difficulties and greater symptom variability. Others have focused more on the dimensionality of SPD. Specifically, Raine et al. (1994) proposed three factors that underlie the DSM-IV-TR construct of SPD: cognitive-perceptual, interpersonal, and disorganization. Recent research has shown support for this three-factor model irrespective of age (Fossati et al. 2003; Mata et al. 2005) and sex (Reynolds et al. 2000). However, while the factor structure of the disorder seems to remain constant across populations, severity of SPD symptoms has been shown to vary depending on age and sex (Claridge et al. 1996; Fonseca-Pedrero et al. 2008; Páino-Piñeiro et al. 2008).

Paranoid personality disorder (PPD) has a prevalence rate of approximately 2–4% in the general population (Grant et al. 2004; Torgersen et al. 2001), and the DSM-IV-TR characterizes individuals with PPD as displaying pervasive and enduring suspiciousness and guardedness. This suspiciousness is hypothesized to be due to a perceived vulnerability to anticipated mistreatment and exploitation by others, who are seen as devious, deceptive, and manipulative (APA 2000). As a result, individuals who meet criteria for PPD generally experience feelings of anger over presumed abuse, anxiety over perceived threats, and a heightened sense of fear that is often perceived by others as argumentative, stubborn, defensive, and uncompromising (Beck and Freeman 1990; Ward 2004). Individuals diagnosed with PPD are hesitant to confide in others, hostile when feeling schemed against, excessively concerned about confidentiality, overly jealous about the faithfulness of partners, and have a tendency to blame others or have difficulty in considering alternative perspectives (Beck and Freeman 1990; Carrasco and Lecic-Tosevski 2000). Recent research has suggested that like SPD, PPD can also be characterized as being multidimensional rather than as a single “taxon” (Edens et al. 2009). Falkum et al. (2009), using exploratory and confirmatory factor analyses, provided evidence that PPD is best represented as two separate dimensions: suspiciousness and hostility.

Schizoid personality disorder (PD), estimated at occurring in less than 1% of the general population (Weissman 1993), is characterized by a lack of interpersonal relationships and lack of desire to seek such relationships. People who meet diagnostic criteria for schizoid PD tend to organize their lives in a manner that results in limited interaction with others, generally selecting occupations that require little social connectedness even if such positions fall below their level of ability (Beck and Freeman 1990). Thinking of themselves as observers rather than as participants in the world around them, individuals with schizoid PD manifest a tendency to sacrifice intimacy in order to preserve the autonomy that is required to maintain beliefs of self-sufficiency and independence (Beck and Freeman 1990; Kalus et al. 1993). Vague, impoverished, or concrete speech and cognition, as well as limited eye contact, gesturing, inflection, or tonal changes in speech further hampers communication (Beck and Freeman 1990; Carrasco and Lecic-Tosevski 2000). While some people with schizoid PD are drawn to conventional lifestyles, most are unable to respond appropriately to social stimuli (Carrasco and Lecic-Tosevski 2000). This can lead to a tendency to form emotional attachments to objects or animals, and in general, individuals with schizoid PD are often viewed as withdrawn, reclusive, isolated, and dull.

These three disorders are grouped together in Cluster A because they are characterized by traits that resemble either

the positive and negative features of psychotic disorders, or both (Kalus et al. 1993; Stone 1993). For example, the suspiciousness and social anhedonic symptoms of SPD parallel those of psychotic disorders, such as schizophrenia; stress from comorbid Axis I psychiatric disorders such as depression or anxiety can further complicate the diagnostic picture of SPD by resulting in even less opportunity for reality testing and an increased risk of deteriorating into a psychotic condition (Bornstein et al. 1988; Carrasco and Lecic-Tosevski 2000).

Similarly, individuals with PPD are at risk of experiencing brief psychotic episodes, as evidenced by delusional ideas or distorted perceptions that are manifested from extreme suspiciousness or paranoia. These can be difficult to differentiate from delusions and may result in misdiagnosis as a schizophrenia-spectrum disorder. For those diagnosed with schizoid PD, it has been suggested that excessive over- or under-stimulation may lead to a comorbid Axis I disorder, such as an anxiety disorder. The depersonalization experienced by individuals with schizoid PD, resulting from lack of contact with and emotional engagement with others, may engender preoccupations with fantasy and, for some, brief psychotic or manic episodes (Beck and Freeman 1990). Further support for the validity of the Cluster A construct has been provided by recent research that has shown that each of the three disorders share genetic and environmental risk factors (Kendler et al. 2006).

However, although the three disorders are all categorized by “odd and eccentric” behaviors, it is important to differentiate them from one another. For example, SPD represents a unique combination of *both* cognitive-perceptual (i.e., positive-like) experiences and social and interpersonal (i.e., negative-like) deficits, while PPD is characterized more by paranoia- and suspicious-related “positive” symptoms in the absence of negative-like symptoms and schizoid PD is characterized more by extreme social isolation caused from a lack of desire for interpersonal relationships in the absence of positive-like symptoms. Furthermore, while SPD and schizoid PD can both be characterized by these negative-like symptoms and interpersonal deficits, SPD can be distinguished from schizoid PD in that the social deficits and anxiety evidenced in those with SPD stem more from paranoid fears about others rather than the lack of desire for close relationships that is part of the clinical picture of schizoid PD (APA 2000).

It is also important to distinguish the Cluster A personality disorders from other disorders, including the presence of and risk for Axis I psychopathology. In particular, both SPD and schizoid PD have been shown to be phenomenologically similar to autistic-spectrum disorders, especially Asperger’s disorder (Gillberg 1989; Tantam 1988; Wing 1981). For example, both Asperger’s disorder and SPD involve social

deficits and odd behaviors, as well as difficulties with emotional functioning. Interestingly, there is evidence that adolescents who meet criteria for SPD also manifest an elevated rate of autistic-like behaviors (ALB), both currently and earlier in childhood (Esterberg et al. 2008). It has also been shown that schizotypal features are associated with features of Asperger’s disorder in non-clinical individuals (Hurst et al. 2007). Thus, while diagnostic confusion can be present, it is typically the presence of perceptual abnormalities, ideas of reference, magical thinking or odd beliefs, and suspiciousness that distinguish the SPD individual from an individual with Asperger’s disorder.

The phenomenology of Cluster A symptomatology also shows a striking resemblance to symptomatology and functioning that are characteristic of the schizophrenia prodrome, a term used to describe the time period directly preceding the onset of illness (Gennaro and Gould 1979). In an effort to adopt a preventative model for psychosis, characterizing the prodrome has been a focus for many schizophrenia researchers. More recently, eight research sites in North America pooled prospective data from nearly 300 individuals with an average age of 18 years (Cannon et al. 2008). These individuals were classified as being in a psychosis prodromal state based on the presence of three separate clinical conditions: (1) measurement using the Structured Interview for Prodromal Syndromes (SIPS; Miller et al. 2003; Miller et al. 2002), which emphasizes positive-like symptoms; (2) brief, intermittent psychotic symptoms beginning within 3 months of assessment; and (3) a diagnosis of SPD or a first-degree relative with a psychotic disorder.

Schizotypal personality disorder is considered to be a risk disorder for the development of schizophrenia, in that nearly 30% of adolescents with this personality disorder eventually go on to develop a psychotic disorder (Yung et al. 2004). Furthermore, SPD has been found at a higher prevalence rate in the family members of individuals diagnosed with schizophrenia. Over 30% of the Cannon et al. (2008) sample was diagnosed with SPD, and conversion to psychosis was 35% within a 2.5-year follow-up period; it was determined that the presence of a genetic risk for schizophrenia, unusual thought content, greater paranoia, severe social impairment, and substance abuse each contributed to predicting the onset of psychosis in this sample (Cannon et al. 2008). Thus, while SPD represents an important risk factor for psychosis, it can be difficult to distinguish from the prodromal phase of schizophrenia, given the strong phenomenological similarities between the two syndromes. Additionally, while several instruments have shown moderate discriminant validity in distinguishing SPD from the prodrome (Bedwell and Donnelly 2005), most researchers do not consider the prodrome to be fully characterized. Until this happens, diagnostic uncertainty will persist.

Measurement of Cluster A Personality Disorders

The current diagnostic system for personality disorders, which is dictated by the DSM-IV-TR in the United States, conceptualizes personality disorders as ten discrete disorders; therefore adopting a categorical approach to diagnosis (APA 2000). However, given the high rates of comorbidity among personality disorders and the heterogeneity within diagnostic categories (Blais and Norman 1997; Watson and Sinha 1998; Oldham et al. 1992; Pilkonis et al. 1995), some have asserted that categorical diagnostic systems are inappropriate, and that the process of “counting” symptoms imposes arbitrary and unreliable thresholds (Francis 1982; Widiger 1992; 1999). However, while some have argued for the implementation of a dimensional classification system, the categorical approach continues to dominate the fields of psychiatry and psychology; especially with respect to clinical practice. But regardless of whether personality disorders are studied dimensionally or categorically, an emerging body of longitudinal research indicates that there is moderate change in personality pathology over time (Johnson et al. 2000a, b; Lenzenweger 1999; Lenzenweger et al. 2004; Shea et al. 2002; Zanarini et al. 2005).

There are a host of instruments currently being utilized to study the presence of categorically diagnosed personality disorders. These include the *Diagnostic Interview for Personality Disorders* (Zanarini et al. 2005), which is a semi-structured interview containing over 250 questions that are guided by DSM personality disorder criteria. The *Structured Interview for DSM-IV Personality Disorders* (SIDP-IV; Pfohl et al. 1995) is a semi-structured interview that assesses DSM-IV Axis II criteria using questions about relationships, interests and activities, and emotions. This measure emphasizes trait functioning, states, moods, or behaviors induced by an external stimulus. Finally, the *Structured Clinical Interview for DSM-IV Axis II Personality Disorders* (SCID-II; Maffei et al. 1997) also has been widely used to diagnose the presence of the 10 personality disorders.

With respect to dimensional measurement of personality pathology, a number of researchers have utilized exploratory and confirmatory factor analyses to determine the construct validity of a unitary syndrome for each of the Cluster A personality disorders. Results from much of this research has indicated that these disorders are best represented as multidimensional constructs, and recent efforts have been aimed at empirically deriving clusters of symptomatology in each disorder within a variety of clinical and non-clinical samples. Of the three Cluster A disorders, SPD is the most common, and the bulk of this research has focused on the measurement of “schizotypal” signs or symptoms using self-report inventories that have been developed to measure a multidimensional “schizotypal” construct (Bolinsky et al. 2003; Claridge and Broks

1984; Nielsen and Petersen 1976; Rust 1987; 1988; Mason et al. 1995; Venables et al. 1990).

The *Schizotypal Personality Questionnaire* (SPQ; Raine 1991), which was developed to measure DSM-IV-TR symptom criteria for SPD, is one example of a self-report measure that assesses multidimensional schizotypal symptoms. Developed to measure all nine features of SPD, it has been widely studied and utilized. For example, several potential latent factor structures have been proposed for the SPQ (Compton et al. 2009; Kendler et al. 2003; Kline 2005; Raine et al. 1994; Siever and Gunderson 1983; Stefanis et al. 2004; Wuthrich and Bates 2006). In general, these factor-analytic studies yield either three (positive or cognitive-perceptual, negative or interpersonal, and disorganized) or four (positive, negative, disorganized, and paranoid) major factors or dimensions. Others are continuing to explore the validity of these factors in a variety of populations. For example, Fonseca-Pedrero et al. (2009) recently demonstrated that the factor scores derived from the Schizotypal Personality Questionnaire-Brief (SPQ-B; Raine and Benishay 1995) are useful in screening for adolescents in the general population.

Other measures have also been developed to identify those with elevated levels of schizotypy, including the *Wisconsin Schizotypy Scales*, which are more commonly known as the *Chapman scales* (Chapman et al. 1976, 1978; Chapman et al. 1984; Eckblad and Chapman 1983; Eckblad et al. 1982). Termed the *Perceptual Aberration Scale*, the *Magical Ideation Scale*, the *Physical Anhedonia Scale*, and the *Revised Social Anhedonia Scale*, these scales are true/false, self-report questionnaires that have been suggested to be reliable and valid measures for assessing risk for psychosis (Grove 1982; Lenzenweger 1994). They do not map directly onto DSM-IV-TR symptoms of SPD, but rather follow a characterization of schizotypy proposed by Paul Meehl (1964). Recent factor analyses of these scales showed a two-factor model that emphasizes both positive schizotypy and negative schizotypy (Kwapil et al. 2008).

In a recent survey-based study, Tackett et al. (2009) used the Dimensional Assessment of Personality Pathology—Basic Questionnaire (DAPP-BQ; Livesley and Jackson 2009), the SPQ, and the Chapman scales to examine Cluster A personality pathology in a sample of family members of patients with schizophrenia and bipolar disorder. Factor analyses of the survey results revealed a five-factor solution, four of which mapped onto the DAPP-BQ (*introversion/inhibition*, *antagonism/dissocial*, *emotional dysregulation*, and *compulsivity*), and a fifth factor (*peculiarity*) that mapped onto the SPQ and Chapman scales. Tackett et al. (2009) argue that this five-factor model represents a broader model of personality pathology that incorporates aspects of Cluster A personality disorders, especially cognitive and perceptual abnormalities.

Compared to the research on SPD, there is a relative dearth of literature available on the dimensional measurement of PPD and schizoid PD. However, paranoid and schizoid symptoms can also be measured by self-report inventories that are designed to measure the defining symptoms of a range of personality disorders. One example is the use of the paranoia scale of the *Personality Assessment Inventory* (PAI; Morey 1991), which assesses broader constructs of personality and psychopathology. Another example is the *Paranoia Scale* (Fenigstein and Vanable 1992), which is a 20-item, self-report questionnaire designed for non-clinical populations that was derived from items on the *Minnesota Multiphasic Personality Inventory* (MMPI; Butcher et al. 1989). Finally, the *Millon Clinical Multiaxial Inventory-II* (MCMI-II; Choca and Van Denburg 1997; Millon 1987) used to measure both paranoia-and schizoid-related symptoms, is a 175-item, true/false, self-report measure of symptoms that map onto DSM-III-R personality disorders.

In summary, there are several widely used instruments available for the categorical and dimensional measurement of Cluster A personality disorders, especially in the area of SPD. Continued efforts to derive more reliable and valid measurements of Cluster A personality disorders are critical to advancing understanding of both the genetic and environmental etiologies, given that some research has demonstrated that heritable genetic factors play a role in the etiology of Cluster A personality disorders, especially SPD (Parnas et al. 2005). More accurate measurement of these personality disorders and their associated symptomatology is also vital to understanding the nature and course of these disorders, especially in childhood and adolescence.

The Development of Personality and Diagnosing Personality Disorders in Youth

Temperament, along with experience, has been hypothesized to be the earliest formations of personality development in children (Rothbart 2007). Lemery et al. (1999) have provided evidence that infant and child behavior actually varies according to six temperament traits, including activity level, positive emotions, irritable distress, fearful distress, soothability, and attention span. Normal adult personality has been frequently assessed according to the Five-Factor Model (FFM) of personality (Digman 1990; Goldberg 1993), which provides a framework for conceptualizing personality functioning according to five overarching factors: agreeableness, conscientiousness, extraversion, openness to experience, and neuroticism. More recent research has shown support for a relationship between these temperament traits and adult personality structure (Graziano 2003).

However, this temperament-personality relationship is made even more complex given the changes in normal

personality development over time. A meta-analysis of the stability of personality traits across the lifespan revealed that test-retest correlations of personality at two points in time are relatively moderate, and that this stability improves as individuals age. Furthermore, as the time between personality assessments increases, stability in these traits decreases (Roberts and DelVecchio 2000). However, as Caspi et al. (2005) discuss in their review of this meta-analysis, what is most interesting is that while there is evidence for fluctuation over the lifespan, continuity of personality functioning in childhood is moderate and increases throughout adolescence and young adulthood.

With respect to personality pathology, stability and course have been more difficult to study and findings have been mixed. Findings from such groups as the *Collaborative Longitudinal Study of Personality Disorders* (CLPS) have shown that personality disorders tend to be less stable than what is predicted by the DSM-IV (Grilo et al. 2004; Shea et al. 2002). Furthermore, more recent research has suggested that symptoms of the various personality disorders become more correlated over time, suggesting an underlying vulnerability for general personality pathology (Sanislow et al. 2009). However, a dimensional model of personality pathology may show more stability over time; Lenzenweger (1999) showed only modest declines on a dimensional measure of personality disorders over a four-year follow-up period in a non-clinical population.

Understanding personality pathology in youth is even more multifaceted and complex. Research on the occurrence of personality disorders in childhood and adolescence has been controversial, primarily because most have assumed that personality is not fully formed until adulthood. The DSM-IV-TR defines personality disorders as having “an onset in adolescence or early adulthood,” and notes that the diagnosis of a personality disorder requires an “evaluation of the individual’s long-term patterns of functioning.” Furthermore, the DSM-IV-TR recognizes that while a personality disorder diagnosis may be applied to children and adolescents, it typically occurs in “unusual instances” in which the individual’s personality traits are “particularly maladaptive” and have been present for at least 1 year (APA 2000).

Thus, research in this area has been limited, partly due to the assumption that childhood and adolescent personality characteristics are unstable or labile, and do not persist into adulthood. As Cohen (2008) astutely notes, research in the area of adult personality functioning has also consistently shown longitudinal variation, although not to the degree that is seen in childhood and adolescent personality. Thus, there is some evidence that personality disorders are less stable in children than adults, and the above-cited research indicates that stability of normal personality traits in childhood is moderate at best. These and other findings

are providing the impetus for increased investigation into the developmental trajectory and assessment of personality disorders in youth. Furthermore, an increased focus on early prevention and intervention has spurred interest in examining the developmental pathways of personality disorders in youth.

While some researchers encourage caution in diagnosing personality disorders in youth, few deny that the origins of personality disorders are rooted in childhood and adolescence, and that maladaptive personality characteristics are present in younger populations (Bernstein et al. 1993a, b; Shiner 2005). For example, research has shown that rates of childhood maltreatment and trauma are higher in adults with personality disorders than in healthy controls, with approximately 73% of adult patients reporting various forms of childhood abuse (Battle et al. 2004). In a recent review of the literature on childhood risk factors for adult personality disorders, Tackett et al. (2009) conclude that factors such as parental conflict, low socioeconomic status, parental psychopathology, and maternal over-control are all associated with the development of personality disorders.

Not surprisingly, childhood Axis I psychopathology, as well as emotional and behavioral difficulties, have also been linked with later development of Axis II personality disorders (Bernstein et al. 1996; Kasen et al. 2001). For example, individuals diagnosed with childhood attention deficit/hyperactivity disorder (ADHD) have been shown to be at heightened risk for PPD in late adolescence (Miller et al. 2008). However, while informative, most of these longitudinal data are from retrospective studies of early childhood and adolescent antecedents of adult personality disorders. Thus, more research resulting from prospective studies of youth with personality disorders is essential.

Current theories assume that personality disorders arise from a complex combination of genetics and early life experiences (Caspi et al. 2005). However, we have limited knowledge of the trajectories of personality disorders that are diagnosed early in childhood or adolescence; much of what we do know concerns the incidence and stability of personality disorders from later adolescence to adulthood and on. For example, in a recent review, Shiner (2009) reports that approximately one in 10 adolescents are likely to be diagnosed with a personality disorder, with rates of occurrence for particular personality disorders being around 1–2%. Other studies, including the *Children in the Community Study* (CICS; Cohen et al. 2005), the *Longitudinal Study of Personality Disorders* (LSPD; Lenzenweger 2006), the *McLean Study of Adult Development* (MSAD; Zanarini et al. 2005) and the CPLS (Skodol et al. 2005), have studied diagnostic stability of personality disorders in adolescents. Taken together, the results show that the rate of personality disorder symptoms decline over time, such that many adolescents with personality disorder diagnoses

experienced stable remissions or considerable reductions in the rate and severity of symptoms as they progressed into adulthood (Skodol 2008).

Making this issue even more complex is the evidence for a great variation in normal personality across populations. This has been especially studied with respect to “psychotic-like” or schizotypal experiences. For example, Yung et al. (2009) found that infrequent psychotic-like experiences were common in a large, school-based sample from the general population in Australia. In the United States, the Epidemiologic Catchment Area study showed a lifetime prevalence of hallucinations of 10% for males and 15% for females (Tien 1991). Another study revealed that up to one-third of individuals from the general population experience paranoid thoughts on a regular basis (Freeman et al. 2005). Furthermore, findings have suggested that the expression of psychotic-like experiences, which overlap with schizotypal syndromes, is quite common in younger populations and tends to decline with age (Johns and van Os 2001; Myin-Germeys et al. 2003). Thus, even normal personality can vary greatly, which complicates the study of personality disorders.

However, some children and adolescents have severe symptoms of personality disorders that persist until adulthood, and those with comorbid Axis I psychopathology have significantly poorer prognoses (Crawford et al. 2008). Additionally, adolescent personality disorder diagnoses are predictive of adult Axis I disorders (Cohen et al. 2005). However, as Lilienfeld (2005) aptly summarizes, the concept of multifinality is especially important with respect to personality disorders, because while it is apparent they can be stable and have maladaptive outcomes, in many cases the outcomes are less negative than what was originally thought. Nonetheless, given the individual variability in stability, continued efforts at studying personality pathology in youth are vital.

Cluster A Personality Disorders in Childhood and Adolescence: Nature and Course

As mentioned earlier, of the Cluster A personality disorders in childhood and adolescence, the most is known about SPD. Raine (2006) conceptualized SPD as a neurodevelopmental disorder with genetic, prenatal, and early postnatal origins, and a resultant vulnerability that impacts biological processes and psychosocial functioning. Research findings on youth with SPD are consistent with this model. There is now a substantial body of literature showing that youth with SPD manifest many of the same functional impairments and biological abnormalities that have been observed in adults with SPD, as well as patients with schizophrenia. For example, when compared to

healthy youth of the same age, adolescents with SPD have been shown to have greater cognitive deficits (Diforio et al. 2000; Trotman et al. 2006), more movement abnormalities (Mittal et al. 2008), and heightened cortisol secretion (Mittal et al. 2007). Taken together, these findings provide further support for the notion that childhood and adult SPD have a common etiology. They also suggest the presence of a biological vulnerability, given the shared deficits with more severe disorders such as schizophrenia.

To date, there is only one report on the heritability of schizotypal traits in adolescents, and the results are consistent with those reported for schizotypal traits in adults (Lin et al. 2006, 2007). The study participants were 330 pairs of twins, ages 12–16, who completed the SPQ. Scores were derived for the three SPQ factors: cognitive-perceptual abnormalities, interpersonal deficits, and disorganization. The scores on these factors were substantially heritable, with heritability coefficients ranging from 41 to 49%. Further, the three schizotypy scores were significantly inter-correlated, and the pattern of findings indicated that common genetic factors influenced all three scores in these adolescents.

As is the case with most psychiatric disorders, there is evidence that psychosocial stress is associated with SPD in youth. When compared to healthy control subjects, adolescents with SPD have been shown to have increased exposure to stressful life events (Tessner et al. 2009) and a greater likelihood of early separation from mother (Anglin et al. 2008). In addition, recent findings indicate that low family socioeconomic status contributes independently to risk for SPD in adolescents, even when controlling for trauma history, recent stressful life events, intellectual capacity, poor parenting, and comorbid symptomatology (Cohen et al. 2008). It is possible that the heightened cortisol secretion observed in youth with SPD is a consequence of greater exposure to stressful events.

A number of studies have examined the comorbid behavioral problems observed in youth with SPD. Adolescents who meet criteria for SPD show an elevated rate of autistic features in childhood (Esterberg et al. 2008), and greater aggressive tendencies (Seah and Ang 2008). Studies using self-report measures of schizotypal signs, as opposed to categorical SPD diagnoses, have also revealed relationships with other symptom dimensions. For example, adolescents who have report psychotic-like experiences are also more likely to manifest difficulty concentrating, irritability, hypersensitivity to noise, sleep disturbances, suicidal ideation and attempts, and heightened anxiety (Nishida et al. 2008). Another recent investigation examined the relation of fantasy proneness, a common tendency in youth, with schizotypal symptom ratings (Sanchez-Bernardos and Avia 2006). The authors found that fantasy proneness correlated with the positive features of schizo-

typy, namely the magical ideation and the cognitive-perceptual dimensions, but not with the interpersonal symptoms, such as social anxiety. Another study showed that the positive features of schizotypy in adolescent patients were linked with more severe mood symptoms, including depression and anxiety, as well as self-monitoring dysfunctions (Deurell et al. 2008).

These findings suggest that adolescents with schizotypal features or SPD are characterized by a range of environmental risk exposures, as well as deficits in multiple functional domains that suggest the presence of brain dysfunction. Although we are not aware of any reports on brain function or structure in relation to diagnosed SPD in youth, a recent report on children and adolescents with 22q11.2 deletion syndrome did address the relation of schizotypal signs with brain structure using neuroimaging (Campbell et al. 2006). Individuals with 22q11.2 deletion syndrome (22qDS), a single deletion of chromosome 22q11.2, suffer from a variety of psychological disorders. In particular, many manifest schizophrenia-spectrum disorders, including SPD. The authors report positive correlations between ratings of schizotypy symptom severity in these youth and grey matter volume of the temporo-occipital regions and the striatum. Consistent with this, another study revealed that adolescents with higher scores on schizotypy, showed increased prefrontal gyrification (i.e., cortical folding; Stanfield et al. 2008). Although the functional significance of larger grey matter volume and increased gyrification is not yet known, these findings lend support to the assumption that schizotypal signs are linked with differences in brain structure.

Interestingly, much of what is known about the developmental trajectory of SPD comes from studies of the relationship between SPD and schizophrenia. As noted, all three Cluster A personality disorders have symptoms that are milder versions of those that define Axis I disorders, especially schizophrenia-spectrum disorders. In fact, SPD is often referred to as a disorder on the milder end of the schizophrenia spectrum of disorders, and has been described as the prototype of schizophrenia spectrum disorders (Siever et al. 2003). Furthermore, others have suggested that SPD may be a more common phenotypic expression of the underlying neural diathesis in the schizophrenia spectrum (Siever et al. 2003; Walker and Diforio 1997).

Support for a relation between childhood SPD and adult-onset psychosis comes from several lines of investigation. In a prospective study, Asarnow (2005) showed that childhood SPD is relatively stable over time and is associated with risk for more severe schizophrenia-spectrum disorders in adulthood. Specifically, Asarnow (2005) reports that in a small sample of clinically-referred children, ages 10 to 16 years, initially presenting with SPD, rates of schizophrenia-

spectrum disorders across the three follow-up years ranged from 75% to 92%. The most common clinical outcome for children with SPD was continuing SPD, supporting the hypothesis of continuity between childhood and later SPD. Moreover, 25% of the SPD sample developed more severe schizophrenia-spectrum disorders, which also supports the assumption that SPD can be a developmental precursor to schizophrenia.

Similarly, research focused on identifying ‘prodromal’ syndromes has revealed an elevated rate of adult-onset psychosis in youth with SPD. As previously mentioned, the prodrome is defined as the period of functional decline prior to the onset of the first psychotic episode. This period, which can last for months to several years, typically has its onset in adolescence and often entails the full syndrome of SPD. Based on studies of the prodrome, it is estimated that between 25–45% of those diagnosed with SPD in adolescence go on to develop schizophrenia (Miller et al. 2002; Yung et al. 2003). Thus, for many patients, the prodromal phase of Axis I psychotic disorders, such as schizophrenia, begins with SPD in adolescence. However, as indicated above, it is important to note that the stability of personality disorders in general is moderate, and many youth with SPD experience remissions or substantial reductions in the severity of their symptoms as they progress toward adulthood.

In contrast to SPD, relatively little is known about the course of PPD in children and adolescents. PPD is sometimes diagnosed in children and adolescents, and it has been shown that adult forms of this disorder have their foundations in childhood factors, such as abuse and neglect (Grant et al. 2004; Johnson et al. 1999). Further, Johnson et al. (2000a, b) found that adolescents with PPD show a heightened rate of externalizing disorders, such as violence and criminal acts, in adulthood. Similarly, Natsuaki et al. (2009) found that adolescent PPD symptoms were associated with childhood maltreatment, poor peer relations and bullying, and externalizing problems during childhood. There is evidence that PPD is a risk syndrome for the later development of schizophrenia-spectrum disorders, however, the relation is weaker than that established for SPD.

Even less is known about schizoid PD, in part because it has not been found to be as strongly associated with more severe psychopathology, relative to the other Cluster A personality disorders. However, the limited available evidence does suggest that features of schizoid PD, like SPD, are moderately stable in youth. Early studies by Wolff and colleagues (Wolff 1991a, 1991b; Wolff et al. 1991) described a group of predominately male school-age children with schizoid PD who demonstrated impaired empathy, mental rigidity, increased interpersonal sensitivity, odd styles of communication, and solitariness. Follow-up studies have shown these characteristics remain quite stable

into adulthood, and that many later met criteria for SPD as adults. Furthermore, two children went on to develop schizophrenia as adults (Wolff et al. 1991).

Recent Data on the Longitudinal Course of Cluster A Personality Disorders in Adolescence

Over the past 15 years, our research group has been conducting longitudinal studies of youth who meet criteria for SPD. Given the evidence that these youth are at heightened risk for the development of an Axis I psychotic disorder, a primary objective was to further enhance predictive power by characterizing the subgroup with the greatest likelihood of conversion to psychosis. Specifically, a range of psychological and biological factors was examined, with the primary goal of deriving a multi-factor index of psychosis risk. Recruitment focused on youth with signs of SPD, and was conducted through announcements directed at parents and clinical practitioners using descriptors of SPD DSM-IV-TR criteria.

We report here on the 36 youth (23 males), ranging in age from 12 to 18 years (mean age = 14.2 years), who met criteria for SPD at baseline in our cohort from 1995 to 1999. Exclusion criteria at baseline were current Axis I disorder, mental retardation, substance addiction (DSM-IV-TR criteria for a substance disorder), and neurological disorder. Of the 36 SPD youth assessed at baseline, 33 underwent assessment for Axis I and II disorders at one-year follow-up.

Assent and written consent was obtained from all participants and a parent, in accordance with guidelines of the Emory University Human Subjects Review Committee. The Structured Interview for DSM-IV Personality Disorder (SIDP-IV; Pfohl et al. 1995) was administered at baseline and follow-up. The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al. 2002) was administered to diagnose Axis I disorders at each assessment throughout the study. After completing a telephone-screening interview, adolescents and at least one parent/guardian underwent a videotaped baseline assessment conducted by a trained graduate-level examiner. Following training of interviewers, inter-rater reliability for SIDP symptom dimension ratings were high, ranging from $r=0.80$ to $r=0.94$, and Cohen's Kappa for diagnostic categories exceeded .80 for all pairs of raters. Final diagnoses were made by consensus of project staff, including an experienced psychologist and psychiatrist, after reviewing the videotaped interviews, medical histories, parent reports, and other materials. Symptoms and other behavioral data were carefully reviewed to document all personality disorders categories for which the subject met criteria. Follow-up assessments were conducted annually to determine Axis I and II diagnostic status.

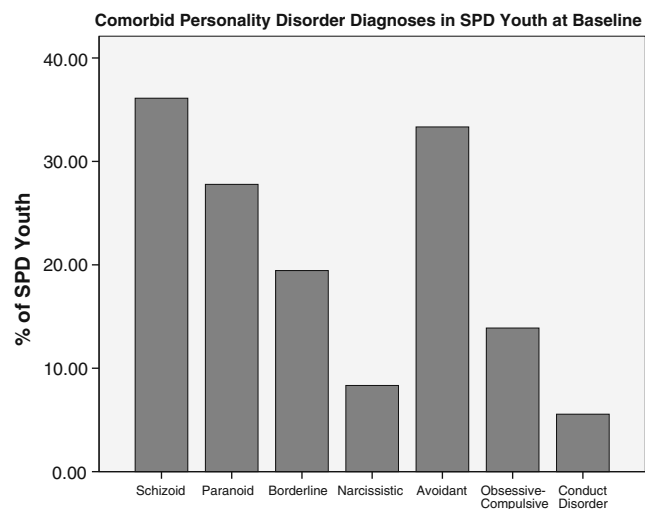


Fig. 1 Comorbid personality disorder diagnoses in SPD youth at baseline

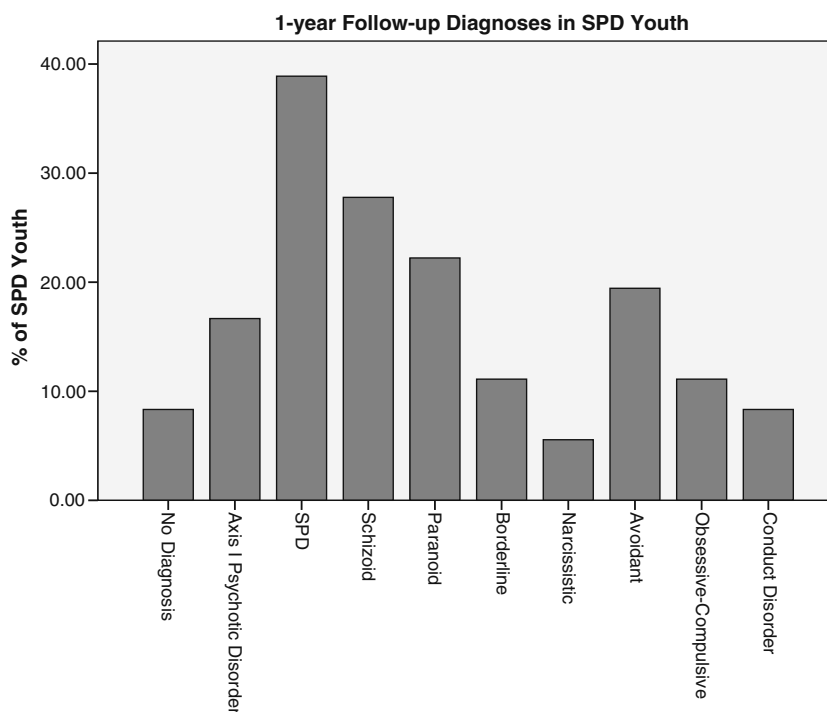
Of the 36 adolescents who met criteria for SPD at baseline, 26 also met criteria for another Axis II personality disorder or Conduct Disorder at baseline. Figure 1 shows the proportions of all comorbid disorders diagnosed in the SPD group. It should be noted that some met criteria for multiple disorders, so the proportions do not equal 100%. As illustrated in Fig. 1, other Cluster A disorders, especially schizoid, were the most common comorbid disorders; roughly 36% and 27% of SPD youth met criteria for

schizoid and/or paranoid personality disorders. Interestingly, about 34% also met criteria for avoidant personality disorder.

Diagnoses at one-year follow-up are shown in Fig. 2. At one-year follow-up, about 9% did not meet criteria for any Axis I or II disorder. However, a total of 6 (18%) met criteria for an Axis I psychotic disorder; schizophrenia ($n=3$), schizoaffective disorder ($n=2$), and bipolar I disorder with psychotic features ($n=1$). The diagnosis of SPD, with no Axis I diagnosis, remained consistent from baseline in 39% of the youth. Thus, more than half, about 57%, either remained in the SPD category or converted to psychosis. Of the remaining 43%, 9% no longer met criteria for any Axis I or II disorder, and 34% now met criteria for another personality disorder, predominantly Cluster A, schizoid or paranoid personality disorder. The proportion with sole or comorbid diagnoses of avoidant personality disorder was lower than at baseline, dropping to under 20%.

In summary, the findings from our study are consistent with previous reports that a substantial proportion (57%) of youth who meet criteria for SPD continue to do so, or become more severely disturbed, 1 year later. Of course, given the young age of the sample, it is not surprising that there is some change over time. Nonetheless, it is noteworthy that over 90% of the sample show persistent behavioral dysfunction that meets criteria for one or more DSM-IV-TR diagnoses over the course of 1 year, from the mean age of 14 to 15 years. Clearly, these findings highlight the importance of further research on the course

Fig. 2 1-year follow-up diagnoses in SPD youth



of Cluster A disorders in youth, especially SPD, and demonstrate concordance with prior research on the moderate stability of personality pathology in youth.

Summary and Conclusions

The literature on children and adolescents with Cluster A personality disorders is limited, which is influenced greatly by the controversy surrounding the diagnosis of personality disorders in youth. This controversy is primarily centered on the stability of personality pathology from childhood to adulthood, as well as the greater incidence of normal patterns of deviation in pathological experiences during childhood and adolescence. However, research has demonstrated that both normal personality and the personality pathology can be quite stable, with most studies demonstrating moderate correlations over time.

Given this moderate stability and empirical findings demonstrating relationships between childhood experiences and adult functioning with respect to personality disorders, there is little doubt that continued research in the area of personality functioning and pathology in youth is essential. In particular, the relationships between youth Cluster A personality disorders and other areas of functioning (i.e., cognitive deficits, aggressiveness, childhood maltreatment, and other externalizing disorders) make this an extremely relevant area of study, especially with respect to risk factors for functional impairment.

As seen from the above review, SPD has received much greater attention from researchers relative to PPD and schizoid PD. This is primarily due to the evidence base showing a strong link between SPD and Axis I schizophrenia-spectrum disorders; especially relevant for childhood-diagnosed SPD that later develops into schizophrenia. Further, with greater focus on the prodromal phase of psychotic disorders, the SPD syndrome will play an increasingly important role in the identification of clinical high-risk groups. Future research directions include focusing on the exploration of PPD and schizoid PD in youth samples, and differentiating them from other Axis I disorders, as well as aiming efforts at understanding their developmental trajectories.

Another important area with respect to Cluster A personality disorders in childhood and adolescent is intervention. Given evidence suggesting that antipsychotic medication may ameliorate SPD symptoms, we can expect more research on the psychopharmacologic treatment of SPD in youth (Deurell et al. 2008). This trend suggests a need for greater focus on indicators that differentiate those SPD youth at greatest risk for conversion to Axis I diagnoses versus those for whom the diagnosis of SPD reflects transitory adjustment problems that will resolve

without intervention (Correll et al. 2008). Thus, the feasibility of future screening of children for risk and the provision of preventive intervention will depend upon progress in developing indicators with strong positive predictive power (Laurens et al. 2007).

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