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Personality pathology in youth

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Introduction

Despite the acknowledgement that personality disorder onsets in adolescence (American Psychiatric Association, 1987, 1994, 2000, 2013), the assessment, diagnosis, and treatment of personality pathology in children and adolescents were regarded as highly controversial until very recently (Chanen, 2015; Sharp, 2016; Shiner & Tackett, 2014). Concerns over the clinical management of personality pathology in youth have focused on the belief that personality in youth is too unstable to justify the diagnosis of personality disorder; uncertainty whether the diagnosis of personality disorder in youth was endorsed by psychiatric nomenclature; beliefs about the normative nature of certain features of personality pathology (e.g. impulsivity, affective instability, or identity disturbances) specifically in adolescence; and worries regarding the demarcation between symptoms of personality pathology and symptoms of internalizing and externalizing disorders. Clinicians have also expressed significant worry over the possibility that labeling an adolescent with a personality disorder may be stigmatizing. Concerns over many of these beliefs have been laid to rest due to accumulating empirical evidence challenging these assumptions. Importantly, researchers and clinicians working in this area have noted that it is unlikely that a person wakes up on her or his 18th birthday with a personality disorder, and that turning a blind eye to personality pathology in youth was potentially perpetuating a stigma surrounding this type of pathology, which has been shown to be as treatable and "syndrome-like" as traditional axis I disorders (Chanen, Sharp, Hoffman, Global Alliance for, & Early Intervention for Borderline Personality, 2017; Zanarini, Frankenburg, Reich, & Fitzmaurice, 2012).

However, certain controversies remain. As may be observed throughout this handbook(see e.g. Chapter X, this volume), a longstanding tension in describing and

understanding personality pathology continues to be a focus of discussion also in the developmental psychopathology of personality (Tackett, Hertzhoff, Balsis, & Cooper, 2016) - that is, the tension between a categorical and dimensional approach to phenomenology. There are many reasons why a categorical approach like the DSM-system has served adult psychopathology reasonably well (Krueger & Markon, 2014); for instance, the fact that by the time that categorical diagnoses were introduced in the DSM-III, enough research had been conducted on these categories to minimally justify a categorical approach (Hudziak, Achenbach et al 2007). This is, however, not true for child and adolescent psychopathology, which has a much shorter empirical research history despite rich theoretical and clinical foundations (Hinshaw, 2017). Several unique features of child and adolescent pathology necessitate a dimensional approach (Hudziak, Achenbach, Althoff, & Pine, 2007), especially where personality pathology is concerned (De Clercq, Decuyper, & De Caluwé, 2014). First, psychopathology manifests differently across development, demonstrating either homotypic or heterotypic continuity. For instance, externalizing behavior in an 8-year old may include oppositional and aggressive behavior, but may morph into moodiness and substance use in adolescence. Second, behavior that is considered typical or adaptive in one developmental period may be considered atypical or maladaptive in another. A dramatic emotional melt-down may, for instance, be considered typical for a 2-year old, but indicative of underlying pathology in a 10-year old. Third, whereas assessment in adults is over-reliant on self-report, the assessment of psychopathology in children and adolescents, for obvious reasons, has to include multiple sources of information (parents, teachers, and children themselves; De Fruyt & De Clercq, 2014); however, research shows modest agreement between sources on problem behaviors (De Los Reyes, Thomas, Goodman, & Kundey, 2013). A child may therefore be considered above

clinical threshold from one perspective, but not another, thereby calling into question the usefulness of categorical approaches to assessment and diagnosis in children and adolescents. In short, then, due to multiple sources of variance in child and adolescent psychopathology, quantitative differences (rather than qualitative, categorical differences) may be more informative in youth.

Notwithstanding these obvious advantages, the reality in most clinical settings is that of a categorical approach to assessment, diagnosis, and treatment (Hudziak et al., 2007). Moreover, a categorical nosology is still very much in place for adult personality disorders (see DSM-5 Section II) (Herpertz et al., 2017), and a developmentally informed dimensional system of personality pathology (as well as most other disorders) in children and adolescents is yet to be developed for the DSM. It must be noted, however, that the alternative dimensional model of personality disorders in DSM-5 (AMPD) has abandoned the traditional age limit, thus leaving room for research on its validity in younger age groups. Indeed, in order to "dimensionalize" DSM personality disorders (i.e., facilitate the inclusion of a quantitative axis that can take developmentally specific sources of variance into account; Hudziak et al., 2007), the validity and reliability of well-researched DSM-based adult personality constructs must be evaluated in youth to determine their value in this population. In parallel, dimensional conceptualizations derived from trait-based approaches to personality pathology must be tested in youth. In this chapter, we review research on child and adolescent personality pathology that has emerged from both these perspectives with the goal of exploring whether the knowledge gained from these perspectives could provide complementary evidence in support of the idea of adolescence as a sensitive period for the development and manifestation of personality pathology.

A few limitations of our review should be mentioned at the outset. First, although a rich literature has developed on the phenomenology and course of maladaptive personality traits dimensionally defined, the translation into clinical utility has not yet taken place; thus, information on the prevalence, etiology, and treatment of personality pathology in youth, dimensionally defined, is lacking. We will therefore discuss the prevalence, etiology, and treatment of personality pathology in youth from categorically defined studies, whereas the phenomenology and course of youth personality disorder will include evidence from both the categorical and dimensional perspectives.

Second, for both practical and substantive reasons, the current chapter will mostly focus on borderline personality pathology (BPP), especially when discussing personality pathology from a categorical perspective. Whereas a dimensional perspective makes it easier to cover the full spectrum of personality pathology in one chapter, the coverage of 10 discrete personality disorders in one chapter is hard to achieve. Fortunately, most of the research on categorically-defined youth personality pathology has focused on borderline personality disorder (BPD; Sharp & Fonagy, 2015; Shiner & Tackett, 2014). Therefore, sections on categorically defined personality pathology will focus mostly on this disorder. We do not, however, consider this practical constraint catastrophic – which bring us to the substantive rationale for focusing on BPP. Although more research is obviously needed on other manifestations of personality pathology (that is, other PDs) in youth, recent factor analytic work at the level of both the disorder (Jahng et al., 2011; Nestadt et al., 1994; Nestadt et al., 2006) and the item/criterion (e.g. Sharp et al., 2015; Wright, Hopwood, Skodol, & Morey, 2016), have called into question the discrete nature of PDs, suggesting that the covariation between PDs and/or their symptoms is not explained by 10 underlying discrete disorders. This evidence has led to suggestions -- consistent

with early theories of personality pathology (Kernberg, 1967) -- that BPP may represent the common features shared by all personality pathology (Clark, Nuzum, & Ro, 2017; Sharp & Wall, 2017; Sharp et al., 2015). Clark, Nuzum, and Ro (2017) argue for BPP as a possible indicator of general personality impairment *severity*, such that PD severity is defined as a latent construct that can be modeled with four indicators: within-PD comorbidity, problematic course/prognosis of both PD and comorbid clinical syndromes, PD-associated psychosocial dysfunction, and features of DSM-5-II BPD. In contrast, but not mutually exclusive, Sharp and colleagues (Sharp, Vanwoerden, & Wall, in press; Sharp & Wall, 2017) argue for BPP as an indicator of *general maladaptation in self-other function;* i.e., of all the PD criteria, BPD criteria most closely capture problems in self-definition, self-reflection, identity, self-determination, and relatedness with others. In time, empirical research will clarify the subtle nuances within these distinctions; however, the point is that BPP appears to be indicative of general personality dysfunction and, as such, allows for the generalization of BPD research, at least to some extent, to personality pathology in general. We think this is especially justified for an understudied area such as youth personality pathology, where there seems to be some urgency in translating research findings into preliminary and useful guidance for clinicians who wish to interrupt further perpetuation of the stigma associated with PD.

Finally, we also wish to clarify another important point when considering the categorical-dimensional debate. For the purposes of this chapter, when we talk about PD categorically defined, we use the *constructs* developed by the categorically-informed DSM to talk about personality pathology – in this case BPD. However, within the boundaries of the BPD construct, we may also talk about BPD symptoms dimensionally assessed with, for instance, the help of self-report measures. In contrast, when we talk about PD dimensionally defined, we refer to the

dimensions that emerge empirically when personality pathology items are factor analyzed. This represents a more bottom-up approach to defining personality pathology because the DSM-based structure of 10 categorically defined PDs is not imposed top-down on covariation structures. Instead, underlying dimensions (or factors) that account for covariation among personality pathology items are allowed to emerge empirically.

Phenomenology, Assessment, and Construct Validity

Personality pathology in youth from a categorical perspective

With the above broader context in mind, we can now consider the definition of Section II BPD and evaluate the evidence in support of its construct validity from studies conducted in youth. The DSM defines BPD as characterized by affective instability, chronic feelings of emptiness, inappropriate or intense anger, stress-related paranoia or dissociative symptoms, fear of abandonment, unstable or intense interpersonal relationships, identity disturbance, impulsivity, and self-injurious behaviors. DSM-based BPP has been operationalized and assessed through both interview-based and self-report measures in youth. Both sources of evidence will be reviewed below.

Studies using interview-based measures. Whereas adult tools, most notably the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID; First, Spitzer, Gibbon, & Williams, 2002), have been used in youth samples (e.g. Chanen, Jovev, et al., 2008), there has been an attempt in recent years to develop more developmentally sensitive interview-based tools. These have included, for instance, the Childhood Interview for Borderline Personality Disorder (Zanarini, 2003) which has been evaluated for its psychometric properties in both clinical and community samples (Sharp, Ha, Michonski, Venta, & Carbone, 2012; Zanarini et al., 2011). Specifically, Sharp et al. (2012), used a confirmatory factor analytic

approach to examine the internal factor structure of the nine CI-BPD items in a sample of 254 inpatient adolescents and found support for a unidimensional factor structure, indicating that the DSM-IV criteria of BPD constitute *a coherent combination of symptoms* in adolescents, including adequate coefficients of internal consistency and high inter-rater agreement between self- and parental reports. CI-BPD diagnoses were further significantly related to clinician diagnosis and to two questionnaire-based measures of BPD, i.e., the PAI-BOR (Morey, 2007) and the BPFSC (Crick, Murray-Close, & Woods, 2005). The CI-BPD was also able to distinguish those who self-harmed and those who showed poor emotion regulation from those who did not, further underscoring its clinical utility.

In another study (Michonski, Sharp, Steinberg, & Zanarini, 2013b)– this time in a large, population-based sample (n = 6,339) of young adolescents from the United Kingdom (ages 11 to 12), item response theory (IRT) was used to investigate the extent to which each BPD criterion (as assessed in the CI-BPD) contributed to variability in the latent borderline trait. A *single underlying dimension* adequately accounted for covariation among the BPD criteria. Each criterion was found to be discriminating to a degree comparable to what has been reported in adult studies. BPD criteria were most informative within a range of severity of BPD pathology between +1 and +3 standard units, suggesting good discrimination at the more severe end of the latent trait. Five criteria were found to exhibit differential item functioning (DIF) between boys and girls. However, DIF balanced out for the total interview score, supporting the use of the total CI-BPD score to identify youth with possible personality pathology.

Additional interview-based measures of DSM-based BPD that have been validated for use in adolescents include the MSI-BPD (Zanarini et al., 2003), which was validated in at least two studies (Chanen, Jovev, et al., 2008; Noblin, Venta, & Sharp, 2013) and the Borderline

Personality Disorder Severity Index-IV-adolescent and parent versions (BPDSI-IV-ado/p; Schuppert, Bloo, Minderaa, Emmelkamp, & Nauta, 2012). Findings suggest that both the MSI-BPD and the BPDSI-IV-ado/p are valid and reliable instruments for the assessment of BPD symptom severity in adolescents.

Studies using self-report measures. DSM-based BPP has also been operationalized in several self-report measures for assessing BPD in youth – either as part of omnibus psychopathology measures or BPD-specific measures. Using standard criteria for measure evaluation (that is, the AERA, APA, NCME Standards for Educational and Psychological Testing (Association, 1999), studies have demonstrated strong psychometrics for these measures, further bolstering the notion that "adult-like" BPD can be reliably assessed and operationalized in youth. For instance, Morey (2007) adapted the adult Personality Assessment Inventory (PAI) borderline subscale (PAI-BOR) and provided norms for the assessment of BPP in adolescents. This subscale was recently evaluated in two high-risk samples, an inpatient sample of adolescents and justice-involved adolescents (Venta, Magyar, Hossein, & Sharp, in press). The purported four factor structure of the PAI-BOR did not hold, suggesting that covariation in BPD symptoms in youth is not accounted for by four underlying factors as suggested by earlier studies of the PAI-BOR, but by one general factor. However, the scale showed good internal consistency (*alpha*= .88, .82, respectively) and good diagnostic accuracy (AUC = 0.834) for predicting a BPD diagnosis (via structured interview).

In a further extension of DSM-defined BPD, Crick et al. (2005) adapted the PAI-BOR subscale for use in children and adolescents, resulting in the Borderline Personality Features Scale for Children (BPFSC). The BPFSC has shown excellent criterion validity (Chang, Sharp, & Ha, 2011), as well as concurrent validity (Sharp, Mosko, Chang, & Ha, 2010). A parent

version was adapted and also demonstrated good psychometric properties (Sharp et al., 2010). Recently, the original 24-item measure has been shortened through IRT to an 11-item version (BPFSC-11; (Sharp, Steinberg, Temple, & Newlin, 2014) to improve its item effectiveness. Factor analyses demonstrated a unidimensional factor structure and excellent criterion validity in the form of sensitivity and specificity in an independent clinical sample. In summary, using standard criteria for measure evaluation, adequate psychometrics for the BPFSC or BPFSC-11 have now been demonstrated for samples in Denmark (Bo et al., 2017), Italy (Fossati, Sharp, Borroni, & Somma, 2016), and Canada (Haltigan & Vaillancourt, 2016), with several studies in other countries underway (Mexico, Spain, China, Germany, France and Portugal). Other omnibus or more circumscribed self-report measures of Section II-defined BPD with demonstrated construct validity include the Minnesota borderline personality disorder scale (Bornovalova, Hicks, Patrick, Iacono, & McGue, 2011; Rojas et al., 2014), the Minnesota Multiphasic Personality Inventory– Adolescent version (Archer, Ball, & Hunter, 1985), and the Borderline Personality Questionnaire (Chanen, Jovev, et al., 2008).

Informant discrepancies. The use of multiple sources of information (i.e. self, informant, or clinician report) when assessing or diagnosing PDs is important given the longstanding view of PDs as relatively pervasive and persistent across contexts (American Psychiatric Association, 2013), as well as the deficits in self-reflective capacities inherent in the disorders (Association, 2013; Hopwood, Wright, Ansell, & Pincus, 2013). The importance of multi-informant designs in younger age groups can be additionally understood from the finding that younger people are more sensitive to response styles (Soto, John, Gosling, & Potter, 2008) when providing self-reports, or may provide less reliable answers due to immature meta-cognitive abilities or language skills (Achenbach, McConaughy, & Howell, 1987). Although

research is not extensive in this area, a couple of studies of youth personality pathology have been conducted, albeit using non-DSM-based tools of personality pathology (Tackett, 2011; Tromp & Koot, 2008). These studies have shown that interrater agreement varies across traits, potentially due to differences in observability, as reflected in higher agreement for more externalizing versus internalizing traits. From a DSM-perspective, Wall, Sharp, Ahmed, Goodman, and Zanarini (2017) found high diagnostic concordance for adolescent BPD between inpatient adolescents and their parents on the Revised Diagnostic Interview for Borderlines (DIB-R; Zanarini et al., 1989) and the CI-BPD (Zanarini, 2003). Sharp et al. (2010) also found significant but modest concordance in a community sample of parent and child reports on the Borderline Personality Features Scale (BPFSC). Given research that informant report discrepancies are often statistically and clinically significant if appropriately interpreted (De Los Reyes et al., 2013), latent class analyses were recently used to evaluate the clinical significance of parent- versus self-report concordance or divergence of DSM-based BPD symptoms in a large sample of inpatient adolescents (Wall, Ahmed, & Sharp, 2018). LCA identified 3 classes of parent-adolescent dyads: 2 convergent classes demonstrating BPFS-P and BPFS-C agreement at a moderate and high level and a divergent class consisting of dyads reporting clinically significant scores on the BPFS-P but clinically negligible BPFS-C scores. Both convergent classes evidenced higher rates of psychiatric severity and less access to internal resources to protect against the effects of psychopathology (i.e., emotion regulation and experiential acceptance).

Together, these studies suggest that personality pathology, as exemplified here with BPD studies, can be measured through either adolescent- or parent-report, and support the particular clinical utility of symptoms with high interrater agreement. However, these studies also point to

important sources of variability based on the source of the report and recommend the use of multiple sources in the assessment of youth personality pathology. Also, discrepancies across informants may represent meaningful content to discuss with parents (or teachers) and the child in order to evaluate to what extent these discrepancies actually represent context-specific or transient maladaptive manifestations rather that personality disturbances (De Clercq, 2017).

Comorbidity. Similar to adult BPD, adolescent BPD demonstrates high comorbidity with both internalizing and externalizing disorders, ranging from 50% in the Children in the Community study (Cohen, 2008) to 86% in a clinical sample (Speranza et al., 2011). Similarly, Chanen, Jovev, and Jackson (2007a) found significantly higher rates of comorbidity in adolescents with BPD, compared to adolescents with either no PD or no disorder, and Ha, Balderas, Zanarini, Oldham, and Sharp (2014) reported elevated rates of mood (70.6%), anxiety (67.3%), and externalizing (60.2%) disorders in adolescent inpatients with BPD relative to non-BPD psychiatric controls (39.2%, 45.5%, 34.4%, respectively). Adolescents with BPD also showed significantly higher scores on dimensional measures of internalizing and externalizing psychopathology than psychiatric controls, as well as significantly higher likelihood of meeting criteria for complex comorbidity (as defined by Zanarini et al. [1998] as any mood or anxiety disorder plus a disorder of impulsivity). Recent studies have also demonstrated in both adults (Eaton et al., 2011; James & Taylor, 2008) and adolescents (Sharp, Elhai, Kalpakci, Michonski, & Pavlidis, 2014) that whereas BPD appears to be a confluence of both internalizing and externalizing pathology (i.e. loaded onto both internalizing and externalizing latent factors; Roysamb et al., 2011), enough variance remains *uncaptured* by these latent factors to suggest that BPD cannot be fully explained by these pathologies. Taken together, this evidence suggests that although BPD is neither an internalizing disorder (Akiskal et al., 1985) nor a female

expression of antisocial PD (Paris, 1997), it likely represents a confluence of internalizing and externalizing problems; that is, the construct of BPD contains characteristics of both internalizing and externalizing disorders, while still retaining its independence as a separate disorder. Elsewhere, it has been argued that personality pathology constitutes a qualitatively different type of pathology on the severity continuum between internalizing/externalizing pathology on the one hand and psychotic disorders on the other (Sharp et al., in press; Sharp & Wall, 2017).

Summary: construct validity. A considerable amount of work (of which we presented only a representative sample) has been done to evaluate the construct validity of adult-like BPD in adolescence operationalized through interview-based and self-report measures. The framework suggested by the AERA, APA, and NCME (American Psychiatric Association, 1999) for organizing evidence to evaluate construct validity includes five categories of evidence, each varying in their importance according to how test scores are used. These include (1) evidence based on test content (i.e., themes, wording, and format of the items, questions, guidelines for administration and scoring, and the like), (2) evidence based on response processes (i.e., the fit between the latent constructs of the test and the detailed nature of performance by the examinee and conduct of the examiner), (3) evidence based on internal structure (i.e., the degree to which the relationships among the component parts of the test conform to the hypothesized constructs), (4) evidence based on relations to other (external) variables (i.e., the relationships between test scores and variables external to the test, including developmental variables and scores on other tests of similar and dissimilar constructs), and (5) evidence based on consequences of testing (i.e., the intended and unintended outcomes of the use or application of a test). In this section of the paper, we provided evidence in support of all of these categories, suggesting that BPP tools

capture something about adolescent function that is scientifically sound and clinically useful. As a field, we can name the construct captured by these measures whatever we want. What we cannot do, however, is ignore the fact that standard approaches to assessing the validity of tools support the downward extension of the DSM-based conceptualizations of BPD to adolescent populations. Overall, this evidence justifies the use of the BPP construct in younger age groups in a similar way as it has been conceived in adults, although phenomenologically speaking it should be mentioned that the more acute symptoms of BPD, such as self-harm and excessive risk taking behaviors, are often more explicitly seen in adolescents than adults (Kaess, Brunner, & Chanen, 2014). The developmental difference in the phenotypic manifestation of these symptoms may be understood in the context of reduced self-control in adolescence and the linear increase in impulse control from late adolescence to early adulthood, as will be outlined later in this chapter. This finding is important, as it points to the necessity of age-specific norms for the diagnosis of BPP in younger age groups.

Personality pathology in youth from a trait perspective

Conceptually, evidence on developmental manifestations of personality pathology from a trait approach has grown from two perspectives, including a general trait as well as a specific maladaptive trait perspective. Both viewpoints conceptualize antecedents of personality pathology as dimensional constructs, but differ in their focus on the trait continuum. The strength of such trait perspectives lies in their fundamental dimensional approach to PDs, enabling the description of young individuals on a set of trait vulnerabilities that are much more dynamic compared to a static and formal PD diagnosis (Clark, 2007; De Fruyt& De Clercq, 2014, Skodol et al., 2005). Conceptualizing a PD condition in terms of concrete and workable traits also reduces the stigma associated with a PD diagnosis and offers welcome leads to clinicians who

aim to effectuate change in daily functioning (De Clercq, 2017). Indeed, a trait profile is always generated from characteristic daily behavior, cognitions, and emotions, hence facilitating communication and appropriate therapeutic goal-setting. Moreover, the hierarchical conceptualization of most trait taxonomies enables the targeting of very specific trait vulnerabilities at the facet-level (Bach et al., 2015), which provides a clinically feasible way to fine-tune perspectives, especially in younger age groups. Finally, a dimensional trait perspective allows one to assess personality pathology in line with the fundamental nature of the pathology. The schizotypal PD, for example, is traditionally considered a unitary construct (APA, 2013), whereas multiple sources of evidence have outlined its multidimensional nature, including both positive and negative schizotypal traits already observable during adolescence (Verbeke, De Clercq, Van der Heijden, Hutsebaut, & Van Aken, 2015).

As the PD field is currently moving toward an increased familiarity with the DSM-5 alternative model for personality pathology (AMPD; Krueger et al., 2012), it may be informative to indicate that this section of the chapter is situated at the Criterion B trait-assessment of PDs and will review evidence on the validity of traits for conceptualizing personality pathology in younger age groups. According to the latest DSM-5 AMPD standards, however, the diagnostic process of a PD requires an additional assessment of self and interpersonal processes (Criterion A; American Psychiatric Association, 2013). These are traditionally not described as separate components in trait models (Tackett, 2016), although some debate exists as to the extent to which these self and interpersonal dysfunctionalities are already intertwined within the trait scores (Widiger et al., in press). As the answer to this question should result from continuing empirical exploration, this section will exclusively focus on the trait-level description of personality pathology in younger age groups.

From a general trait perspective, convincing evidence has shown that early individual differences in Emotional Stability, Extraversion, Imagination/Openness, Agreeableness, and Conscientiousness as represented by the Five-Factor Model (Costa & McCrae, 1992) are meaningfully related to each of the DSM-based PDs (De Clercq& De Fruyt, 2003; De Clercq, De Fruyt, & Van Leeuwen, 2004) in a largely similar way as has been demonstrated for adults (Saulsman & Page, 2004; Widiger et al., 2002). Beyond these trait associations at the level of the categorical PD scale, Tackett (2016) has recently provided an impressive review of the evidence in support of Five-Factor Model equivalents of the main developmental clinical features of PDs. This review indicates that for each of the ten DSM-based PDs, the most prominent phenotypic PD symptoms in youth can be translated into either higher- or lower-order level trait aspects. This evidence accentuates that a significant amount of the variability in personality pathology can be traced back to individual differences in the main building blocks of personality, implying that children at the extremes of these traits are at increased risk for developing a less adaptive or pathological personality. Whereas some traits reflect shared underlying dispositional components across different disorders, other traits are rather unique vulnerabilities for specific PD symptomatology.

For example, just like in adults, low emotional stability is a significant trait component for almost all DSM- based PDs assessed in youth, whereas high Openness to Experiences is a unique correlate for schizotypal personality pathology. It is important to understand these shared versus unique developmental trait correlates to gain insight into not only the trait-based nature of different manifestations of PD symptoms, but the nomological net of personality pathology in youth, as well as to increase our understanding of comorbidity and the overall dimensional nature of psychopathology.

Although the validity of this general trait perspective on personality pathology has been convincingly demonstrated across age, it has been argued that the extremes of general trait measures may not always assess the richness of personality pathology (Clark, 2007). From this perspective, it is exactly at these extremes that a more specific maladaptive trait perspective was elaborated by more narrowly defining a set of maladaptive traits considered to capture early manifestations of personality pathology in the most comprehensive way. Work in this area can be understood from top-down approaches, translating relevant adult PD traits into developmentally appropriate equivalents, such as the childhood borderline pathology construct (Crick et al., 2005; Chang, Sharp, & Ha, 2011), childhood psychopathy (Frick & Hare, 2001; Hare, 2003; Lynam, 1997) or the core trait of Narcissism (Thomaes, Stegge, Bushman, Olthof, &Denissen, 2008). Not surprisingly, this maladaptive trait perspective has been particularly elaborated for traits characteristic of Cluster B PDs. This can be explained by the fact that Cluster B pathology is socio-demographically seen more frequently in younger ages (Bernstein et al., 1993; Widiger & Costa, 2013), partly because of the heavy acting-out behavior that is easily observed by others and also because the seriously impairing character of cluster B pathology results in more frequent and quicker health-care seeking behavior or forced mental health care (Chanen, Jovev, & Jackson, 2007; Krabbendam et al., 2015; Winsper et al., 2015).

Beyond the work focusing on these more narrowly defined maladaptive traits, a subgroup of researchers has attempted to construct omnibus taxonomies of early personality pathology. Independent from each other, these researchers found an underlying maladaptive trait structure parallel to the well-established structure in adults (Widiger & Simonsen, 2005). From a likely similar top-down approach, the SNAP-Y (Linde, Stringer, Simms, & Clark, 2013) and the DAPP-BQ-A (Tromp &Koot, 2008) resulted from modifications of their adult counterparts for

use in younger age groups, and showed adequate psychometric properties, including construct and criterion validity. In addition, the SNAP-Y showed meaningful relations with the MMPI-A (Butcher et al., 1992), one of the most commonly used DSM-based measures of adolescent personality pathology (Archer & Newsom, 2000). Also, the recently released adult DSM-5 PID-5 measure (Krueger et al., 2012), including 25 trait facets along a similar five dimensional higher-order trait structure, can be reliably and validly used in younger age groups with both a non-referred (De Clercq et al., 2014) and referred (Somma et al., 2016: De Caluwé et al., 2018) status. Several authors have pointed to the potential of the latter instrument for a more official developmentally appropriate assessment of personality pathology at a young age (Shiner & Allen, 2013; Sevecke, Schmeck, &Krischer, 2014), as it is the first instrument integrated within DSM-5 that is built upon the well-established five major building blocks of personality that account for individual differences in trait characteristics from middle childhood onwards (Shiner & DeYoung, 2013).

From an age-specific bottom-up approach, De Clercq and colleagues (2006) constructed an omnibus measure (the Dimensional Personality Symptom Itempool; DIPSI) for early maladaptive traits, initially structured in the traits of Emotional Dysregulation, Introversion, Disagreeableness, and Compulsivity, and later amended with an item-set representing a fifth factor of Oddity (Verbeke& De Clercq, 2014; Verbeke, De Caluwé, & De Clercq, 2016). Interestingly, these bottom-up and top-down measures for youth were developed by independent research groups following different strategies, but proved to have significant and meaningful interrelationships (Kushner, Tackett & De Clercq, 2013), hence underscoring their construct validity.

Given the comprehensiveness of the content covered by omnibus measures, these taxonomies can be readily used to construct and examine age-specific PD constructs, such as the childhood borderline construct (De Clercq, Decuyper, & De Caluwé, 2014), childhood psychopathy (Decuyper, De Bolle, De Fruyt, & De Clercq, 2011), schizotypal pathology (Verbeke, et al., 2015), or PD-related constructs, such as the Dark Triad (De Clercq, Hofmans, Vergauwe, De Fruyt, & Sharp, 2017). The construction of such childhood PD trait constructs is interesting from a conceptual point of view, but their validity should not be assumed before extensive empirical exploration has underscored their value for understanding developmental antecedents of personality pathology. As outlined by Tackett (2016), these constructs should be the starting point, rather than the end, and their relevance should always be mirrored against the developmental principles and empirical evidence on the course of personality pathology from early age onwards. Although these developmental processes through which early trait vulnerabilities are shaped into consolidated patterns of personality pathology are complex and cannot be entirely defined by a set of principles or theoretical assumptions, they are important to consider, as they may unravel some of the density of childhood development and create guidelines for early assessment and intervention programs.

The developmental course of youth personality pathology

Longitudinal evidence has convincingly demonstrated that both stability and change characterize the developmental course of personality pathology (Tackett, 2016), which is underscored by both categorical as well as dimensional oriented (trait) studies. Overall, mean-level change can be understood from the *maturation principle*, reflecting the natural growth process toward adaptation in terms of more Emotional stability (or less Emotional Dysregulation), more Agreeableness (or less Antagonism), and more Conscientiousness (or less

Disinhibition) as children achieve more emotion regulation skills, impulse control, moral reasoning, and empathy with increasing age. The timing of this age-related decline, however, may vary across disorders. Core symptoms of borderline pathology, for example, have been demonstrated to increase in adolescence, peak in early adulthood, and then decline (Arens et al., 2013; Chanen & Kaess, 2012b), whereas core Disinhibited traits such as Impulsivity generally tend to start to decline from 10 years of age (Steinberg et al., 2008). Some studies also showed differences between traits in the pace of natural change over time (De Clercq, et al., 2009; Durbin et al., 2016; Van den Akker, Dekovic, Asscher, &Prinzie, 2014). Introversion, for instance, appears to show less mean-level change over time, suggesting the relative stability of Introverted- like traits throughout childhood compared to other basic maladaptive traits. In a related vein, the balance of evidence on borderline pathology traits suggests that whereas impulsive-type symptoms do reduce over time, affective-type symptoms, which include negative affect and feelings of emptiness, are more likely to persist (Meares, Gerull, Stevenson, & Korner, 2011).

Although the maturation principle is believed to be universal, some individuals do not experience an age-related decline in personality pathology symptoms. For instance, in the Children in the Community study, one-fifth of the sample of youth showed an increase in PD symptoms over the decade from mid-adolescence to early adulthood (Cohen, Crawford, Johnson, & Kasen, 2005). Moreover, remission from a categorical diagnosis of BPD does not imply that remitted patients are healthy (Wright et al., 2016). As with adults, poor functional outcomes persist for years in individuals who showed borderline features in adolescence, including increased risk for substance use and mood disorders, interpersonal problems, poorer quality of life, higher levels of general distress (Crawford et al., 2008; Winograd, Cohen, & Chen, 2008),

higher service utilization (Cailhol et al., 2013), and increased rates of pain, physical illness, and mortality over time (Chen et al., 2009). Krueger (2005) has suggested in this regard that categorically assessed remission often simply implies a shift towards a different disorder, because time and environmental context may change the phenotypic expression of an underlying trait vulnerability, whereas the trait itself remains rather stable. Indeed, the categorical stability of BPD is modest in both adolescents and adults (Chanen et al., 2004; Skodol et al., 2005; Zanarini et al., 2011), whereas the stability for dimensionally assessed BPD appears to be somewhat higher (Bornovalova, Hicks, Iacono, & McGue, 2009; A. M. Chanen et al., 2004; Cohen et al., 2008). Independent from age-related mean-level changes, convincing meta-analytical evidence has shown a high *rank-order stability* of traits (Roberts, DelVecchio, 2000), pointing at the stability of a child's trait position relative to her or his peer-group, and suggesting that vulnerable children remain vulnerable over time compared to others. Whereas rank-order stability for personality pathology has been shown to be moderate, it still appears to be more stable than common internalizing and externalizing psychopathology (Cohen et al., 2005; de Clercq, van Leeuwen, van den Noortgate, de Bolle, & de Fruyt, 2009). Together, these data point to the possibility that a subgroup of youngsters fail to follow the normative decline in maladaptive trait features, and may become "stuck" in adolescence. Presumably, many of these adolescents already manifested more explicit maladaptive behaviors compared to their peers in terms of frequency or intensity, although trajectories of a *steady increase* and thus a later onset of pathology have also been observed. We will return to the mechanisms that may account for these developmental delays or deviating pathways later.

Beyond the principles of maturation and rank-order stability, several studies have also underscored longitudinal measurement invariance of borderline symptomatology from

adolescence onwards (Haltigan & Vaillancourt, 2016; Vanwoerden, Garey, Ferguson, Temple, & Sharp, revise and resubmit; Wright, Zalewski, Hallquist, Hipwell, & Stepp, 2016). These studies are important against the background of other longitudinal studies on borderline pathology, which have shown that levels of borderline features increase until mid-adolescence and then level out through adulthood (Chanen & Kaess, 2012a). Put differently, the prospective course of borderline pathology reported in the literature appears to be reflective of true (mean level) changes and not due to reporting biases, thus pointing to possible *homotopic continuity* of DSM-based borderline symptoms at least throughout adolescence. Personality pathology appears to provide additional explanatory value above and beyond other traditional Axis I disorders as well as other PDs in predicting current psychosocial functioning (Chanen, Jovev, & Jackson, 2007b) and suicidal outcomes (Sharp, Green, Venta, Pettit, & Zanarini, 2012).

Prevalence

As a reminder to the reader, we will be focusing mostly on BPD (most often categorically defined) going forward, as coverage of all PDs is beyond the scope of this chapter and most empirical work in prevalence, etiology, and treatment has been carried out on narrowly defined BPD. In adults, BPD occurs in approximately 1–3% of the general population (Leichsenring, Leibing, Kruse, New, & Leweke, 2011; Lenzenweger, 2008). Whereas few population-based studies of BPD exist for children and adolescents, early studies reported high rates of BPD in community studies, with values ranging from 11% (Bernstein et al., 1993) to 26.7% (Chabrol et al., 2002)[1]. Two recent reports estimated prevalence using different scoring algorithms in a large

[1] We would like to note at this point that in some of the older studies, prevalence estimates may have been inflated due to the use of measures that were not validated for youth at that time, hence increasing the chance that normative turmoil was presented as BPD symptoms.

birth cohort of 6,330 British children 11 years of age (the Avon Longitudinal Study of Parents and Children; ALSPAC study) and found a prevalence of 3.27% (Zanarini et al., 2011) and of 0.006% (Michonski, Sharp, Steinberg, & Zanarini, 2013a), respectively. Other reports have estimated point prevalence for adolescents in the community at around 1% in the U.S. (Johnson, Cohen, Kasen, Skodol, & Oldham, 2008; Lewinsohn, Rohde, Seeley, & Klein, 1997) and 2% in China (Leung & Leung, 2009), and cumulative prevalence at 3% (Johnson et al., 2008). The picture in clinical populations is more concerning, with reported rates of 11% in outpatients (Chanen et al., 2004), 33% (Ha et al., 2014) and 43–49% in inpatients (Levy et al., 1999). The take-home message from these data is that, like phenomenology discussed above, prevalence rates for BPD appear to be comparable between adults and adolescents.

Etiology of youth personality pathology

Increasing etiological evidence suggests that various manifestations of psychopathology, including personality pathology, evolve from a more general genetically-based propensity to psychopathology (Caspi et al., 2014; Kotov et al., 2017; Lahey et al., 2012), which is partly reflected in a large common growth factor of early trait pathology (De Clercq, et al., 2017[a b]; Wright, Zalewski, Hallquist, Hipwell, & Stepp, 2016). Although there is some evidence for significant homotypic continuity of early internalizing versus externalizing tendencies toward either internalizing or externalizing trait outcomes (Luby, Si, Belden, Tandon, &Spitznagel, 2009; Mesman & Koot, 2001, Snyder, Young, & Hankin, 2016), adult personality pathology as currently structured in the DSM-5 does not result from phenotypically-similar trait antecedents at a more lower-level trait operationalization, at least not before adolescence. Indeed, from the principle of trait crystallization (Shiner, 1998), it was recently shown that the discriminatory power of youth maladaptive traits for conceptually related outcomes becomes significant only

from mid-adolescence onwards (De Clercq, et al., 2017). As outlined later in this chapter, this finding is important because it signifies that adolescence in particular can be considered a sensitive developmental period in which specific configurations of personality pathology are shaped. These specific manifestations of personality pathology may thus flow from either steady-high trajectories of childhood trait vulnerabilities, or result from increasing trajectories of maladaptive traits as a consequence of recurrent failures in achieving developmental milestones (De Clercq, et al., 2017[a b]).

Personality pathology does not, of course, evolve exclusively from child factors, as multiple environmental factors, as well as child x environmental processes, also contribute to PD outcomes. Two main developmental theories of BPD, for example, agree that an interaction of genetic predispositions and environmental stressors is likely at play (Gunderson & Lyons-Ruth, 2008; Sharp & Fonagy, 2015). Marsha Linehan's biosocial theory (Crowell, Beauchaine, & Linehan, 2009; Linehan, 1993) suggests that BPD results from biological predispositions manifesting themselves under stress from an invalidating environment. However, due to varying thresholds of sensitivity among children, even the most well-intentioned of families can create an environment that, to a sensitive child, is perceived as invalidating. This can lead to some instances of people with BPD reporting highly stressful or traumatic childhoods that may not agree with reports from other sources, such as parents or siblings. Growing up in an environment experienced as invalidating, a child will begin to believe that her or his feelings and thoughts do not matter, ultimately hindering the capacity to recognize and label emotions, both within the self and in others. Overall, Linehan's theory posits that BPD represents dysfunction in how an individual experiences and regulates emotions, with all symptoms stemming from this deficit. The emotional instability, anger, and self-destructive impulsivity observed in BPD are all

manifestations of the inability to effectively regulate unpleasant internal experiences. Similarly, Peter Fonagy's mentalization model of BPD (Fonagy & Bateman, 2008; Fonagy & Luyten, 2009, 2016) suggests that an interaction between a constitutional vulnerability to emotional distress and disruptions in attachment relationships may account for the development of the disorder. This combination of risk factors leads to hyper-responsiveness of attachment systems, resulting in deficits in mentalizing, or the ability to understand the internal experiences of others and the self, especially when experiencing stress or emotional arousal, and ultimately impeding the development of the self.

These developmental theories provide an important framework for studying the correlates and causes of personality pathology. Indeed, research in the genetics of personality pathology as well as its neurobiology in youth is emerging (see Goodman, Perez-Rodriguez, & Siever, 2014) for a review). For instance, Belsky et al. (2012) examined borderline-related features in 1,116 pairs of twins aged 12. The correlation for BPD traits between MZ twins were found to be 0.66 compared to .29 for DZ twins. Genetic factors were found to account for 66% of the variance in borderline traits, suggesting very similar heritability for adolescents compared to adults. Bornovalova et al. (2009) found that borderline traits were moderately heritable, with average heritability across age of approximately .3-.5. Developmentally, heritability appeared to increase from ages 14 and 18. Importantly, this study also showed that both stability and change of BPD traits were influenced profoundly by genetic factors, and modestly, but increasingly, by non-shared environmental factors, underscoring the etiological significance of young people progressively selecting their own environment. Developmentally-specific manifestations of biologically-based etiological factors are also apparent from volumetric and functional neuroimaging studies conducted in youth. Structural imaging research has demonstrated volume

reduction in the frontolimbic network in adolescents with BPD, including the orbitofrontal cortex (Brunner et al., 2010; Chanen, Velakoulis, et al., 2008b), the anterior cingulate cortex (Goodman et al., 2011), and the amygdala and hippocampus (Chanen, Velakoulis, et al., 2008a).

Of course, the most robust evaluation of the developmental models of personality pathology would be biology x environmental studies (Sharp & Kim, 2015). A nice example is provided by a study using a twin design (Bornovalova et al., 2013). Temperamental traits of behavioral disinhibition or externalizing (EXT; impulsivity and inability to inhibit undesirable actions) and negative emotionality or internalizing (INT; predisposition to experience depression, anger, and anxiety) were evaluated for their interaction with child abuse (CA) to predict borderline traits over time. Three causal models were tested: a direct causal model (CA → BPD); a diathesis stress model (INT/EXT x CA → BPD), and a genetic mediation model where the CA-BPD association was better accounted for by common genetic risk factors (i.e., INT, EXT, or additive INT and EXT psychopathology could account for genetic or environmental influences common to CA and BPD). The authors found the strongest support for a genetic mediation model where the association between exposure to traumatic events and BPD may be better accounted for by common genetic influences rather than the former causally influencing the latter.

Reconciling categorical and dimensional approaches of youth personality pathology: Shared perspectives on the sensitive period of adolescence

In the above sections, we have covered the research on phenomenology, comorbidity, course, prevalence, and etiology of personality pathology in youth, specifically borderline pathology. In reflecting on its content, certain conclusions can be drawn that point to adolescence as a sensitive period for the development of personality pathology, regardless of whether one

takes a categorical or dimensional perspective in defining personality pathology. First, adult-like personality pathology has its onset in adolescence. Second, rank order stability of personality pathology is moderately stable in children and adolescents and increases with age. Third, it appears that the discriminatory power of youth maladaptive traits for conceptually related outcomes becomes significant only from mid-adolescence onwards, suggesting the crystallization of early manifestations of personality pathology during adolescence. What accounts for this crystallization? Here and elsewhere (Sharp et al., in press; Sharp & Wall, 2017), it has been argued that, if internalizing and externalizing pathology is left untreated and in the context of biological vulnerability and stressful life events, we can observe the manifestation of personality pathology in its adult-like form because it is during adolescence that an agentic, self-determining author of the self emerges (McAdams & Olson, 2010). Although the development of self begins as early as infancy, identity formation has long been understood to be a key developmental achievement of adolescence (Erikson, 1950). In contrast to other related self-concepts, identity is defined as the way in which an individual makes sense of or meaning from her or his self-concept (McLean & Pratt, 2006). Thus, identity is often studied with autobiographical narratives in which people are evaluated in their ability to integrate their autobiographical past and imagined future in a coherent way (McAdams & McLean, 2013). The complex process of reflecting and integrating disparate pieces of information across multiple domains of functioning is a metacognitive capacity that does not emerge until adolescence (Sebastian, Burnett, & Blakemore, 2008; Shaw et al., 2008; Somerville et al., 2013). Adolescents expand their social lives to include peers and romantic partners, offering additional data points that need integration. Cognitively, they are able to handle perspective-taking, but the task of integrating multiple self-hypotheses is a complex process that can be easily disrupted (Harter,

1999). The developmental toll of these transformations in self-other relatedness (individuation) appears to impose a heavy burden on some youngsters and their families. Whereas most adolescents grow out of the normative inter- and intra-personal conflict, confusion, distress, and instability in self-representation, others do not (Sharp & Rossouw, in press). We suggest that it is this group of adolescents who do not show the normative decline in maladaptive traits, but whose internalizing and externalizing problems mature into a disturbance of identity, which may be conceptualized as part of the core of personality dysfunction (Hopwood et al., 2013; Sharp et al., in press) as represented in Criterion A of Section III of the DSM-5 (American Psychiatric Association, 2013).

Early Intervention, Treatment, and Prevention

The question then arises whether early manifestations of personality pathology can be effectively treated and whether full-blown personality pathology can be prevented. The well-established evidence on maturation effects, rank-order stability, and a-specificity of youth trait pathology is important in several ways for those who aim to translate empirical findings into good practice. First, maturational evidence should refrain us from diagnosing children at a very young age, as many of these early problematic behaviors are transient in nature and will likely turn into normative tendencies with increasing age. Relatedly, a specific personality disorder diagnosis cannot be justified throughout childhood given the a-specific predictive validity of early maladaptive traits. On the other hand, and based upon the same evidence, it is clear that overall trait vulnerabilities are traceable in younger age groups and that especially high-scoring children should be the focus of early intervention, given the differential continuity and that maturation effects are generally linear in nature. Because of the a-specificity of childhood trait vulnerability, however, early interventions should not be built around disorder-specific protocols,

but should explicitly target the traits that have proven to be the shared underlying liabilities for later PDs. From this perspective, youth with explicit manifestations of the BPD symptom cluster may be the target group par excellence, given that across PDs, BPD is the only disorder that comprehensively covers an overall pathology factor with meaningful correlates of impairment across various life domains.

However, central to the notion of early intervention and prevention, and consistent with a more dimensional approach to conceptualizing personality pathology, is the well-known fact that the threshold for distinguishing patients with and without PDs is arbitrary and there is no strict demarcation between 'cases' and 'noncases'(Clark, 2007; Herpertz et al., 2017); as such, there is also no distinct point of 'onset'(Chanen & Thompson, 2018). With no distinct point of onset, early intervention (defined as intervention at an early stage of disease progression) is justified either by preventing the onset of new cases (indicated prevention) or through case identification and early treatment, which involves formal diagnosis and intervention using DSM-based approaches. Chanen and Thompson (2018) identified several empirically informed principles when considering early intervention for personality pathology. First, due to issues of comorbidity (discussed earlier), as well as the fact that the relation between early manifestations of personality pathology and PD in adulthood are neither specific nor linear, personality pathology cannot be considered separate from other psychopathology. Rather, psychopathology must be viewed as a system, rather than a category. This allows for the consideration of phenotypic (e.g., trait-based; symptom-based), endophenotypic (e.g., neurobiology), and contextual factors to be considered over time in deciding which services to provide to a young person. This approach is represented in a "clinical staging" framework to assessment and diagnosis (Chanen, Berk, & Thompson, 2016), defined as a pragmatic, heuristic, and transdiagnostic integrative framework

to the assessment of individual patients, emphasizing identification of risk factors for persistence or deterioration of symptoms or problems, rather than just focusing on the initial onset of disorder. Interventions are selected that are proportionate to the phase and stage of disorder, such that they may be simpler and more benign during early stages of disorder, increasing in intensity with disorder progression, and adapted for co-occurring psychopathology as the severity and comorbidity begin to increase over the course of disorder progression (Chanen & Thompson, 2018).

At higher levels of severity, clinicians may turn to evidence-based approaches for treating personality pathology in adolescence. Again, we review here randomized-controlled trials (RCTs) conducted for BPD in adolescence, as we are not aware of RCTs conducted for other PDs in adolescents. The most compelling evidence in support of not only the mentalization-based model of adolescent BPD, but also the efficacy of a mentalization-based treatment approach, was derived from an RCT conducted by Rossouw and Fonagy (2012). In this study, 80 adolescents (85% female) consecutively presenting to mental health services with self-harm and comorbid depression were randomly allocated to either Mentalization-based Therapy – Adolescents (MBT-A) or Treatment as Usual (TAU). Adolescents were assessed for self-harm, risk-taking, and mood at baseline and at 3-month intervals until 12 months. Their attachment style, mentalization capacity, and BPP were also assessed at baseline and at the end of the 12-month treatment. Results indicated that MBT-A was more effective than TAU in reducing self-harm and depression. This superiority was explained by improved mentalization and reduced attachment avoidance and reflected improvement in emergent BPD symptoms and traits.

Several other evidence-based intervention programs have been evaluated for DSM-based BPD, including Cognitive Analytic Therapy (Chanen, Jackson, et al., 2009; Chanen &

McCutcheon, 2013; A. M. Chanen, McCutcheon, et al., 2009) and Dialectical Behavior Therapy (Mehlum et al., 2012). DBT synthesizes a change orientation from behavior therapy with an acceptance orientation from Zen philosophy to target the emotion dysregulation, distress tolerance, and interpersonal difficulties in BPD. DBT has been evaluated in adolescents with nonsuicidal self-injury (NSSI) and two BPD criteria in Norway (Mehlum et al., 2012), adolescents with a history of NSSI and suicide attempts in New Zealand (Cooney et al., 2012), and adolescents with bipolar disorder (Goldstein, Axelson, Birmaher, & Brent, 2007). Of the three studies, the Norwegian study was the most BPD-relevant and had the most rigorous study design and demonstrated a significant decrease in NSSI in DBT but not in Enhanced Usual Care condition. In addition, DBT resulted in greater improvements in BPD symptoms and depression.

Other approaches being used in adolescents, but for which RCTs have not yet been conducted, include Transference-Focused Therapy (Normandin, Ensink, Yeomans, & Kernberg, 2014), Systems Training for Emotional Predictability and Problem Solving (Harvey, Blum, Black, Burgess, & Henley-Cragg, 2014), and Emotion Regulation Individual Therapy for Adolescents (Bjureberg, Sahlin, Hellner, Hedman-Lagerlöf, Gratz, Bjärehed, Jokinen, Tull, & Ljótsson, 2017).

Summary and conclusion

In this chapter, we reviewed the evidence in support of the phenomenology, assessment, diagnosis, etiology, course, and treatment of personality pathology in youth – from both a categorical and dimensional perspective. Consistent with recent views on the categorical-dimensional debate (Sharp & Wright, 2018), this review highlights significant commonalities in conclusions drawn from the traditions underlying categorical versus dimensional approaches. Most pertinent in this regard is the evidence in support of adolescence as a unique developmental

period for the crystallization of personality pathology. Many questions remain unanswered, however. For instance, whereas theoretically-driven mechanisms have been suggested to account for developmental crystallization of personality pathology (e.g., narrative identity), research will have to demonstrate that early manifestations of personality pathology morph into adult-like personality through such mechanisms. Whether these mechanisms mirror Criterion A function beyond that of Criterion B function, and whether Criterion B function is sufficient to capture crystallization of maladaptive traits into a disorder that was not crystallized already in pre-adolescence should be clarified. Either way, consistent with the recent position statement by the Global Alliance for the Prevention and Early Intervention for Borderline Personality Disorder (Chanen et al., 2017), the studies reviewed in this paper form part of the proliferation of knowledge about personality pathology in adolescents and emerging adults ("youth") over the past two decades that provides a firm basis for establishing early diagnosis and treatment ("early intervention") for threshold and subthreshold personality pathology, and represent the continuation of research in this vibrant area.

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