Adolescent personality disorder

Developmental perspective

- Sharp, Kerr, and Chanen (2021)'s developmental model draws on research identifying a **distinct developmental period from 10-25 years,** which in economically developed nations has been argued to support the **acquisition of culturally embodied knowledge, skills, and self-regulatory capacities necessary in independent adult life** (Dahl, Allen, Wilbrecht, & Suleiman, 2018; Sawyer, Azzopardi, Wickremarathne, & Patton, 2018). Alongside the development of these capacities, youth also represents a **time of vulnerability and coincides with the peak period of clinical onset for major mental disorders** (Chanen & Thompson, 2019).
- A developmental lens allows for recognition of the impact of mental health problems on the transition to adulthood, and that personality pathology occurs in a wider mental health and developmental context. For example, the **Clinical High at Risk Mental State (CARMS)** aims to identify young people experiencing clinical distress due to subthreshold symptoms, and includes borderline personality disorder (McGorry, Hartmann, Spooner, & Nelson, 2018). This can help to identify the impact on functioning before reaching a threshold for adult mental health problems.

Addressing concerns about the diagnosis of adolescent personality disorder

- **Clinicians are hesitant to diagnose adolescents** with personality disorders, with less than 25% of a sample of British child and adolescent psychiatrists (Griffiths, 2011) and less than 10% of a group of Dutch psychologists reporting using the diagnosis in practice (Laurenssen, Hutsebaut, Feenstra, Busschbach, & Luyten, 2013).

Assessment and diagnosis of BPD in young people

- Addressing concerns from clinicians that personality disorder cannot be reliably diagnosed in young people, both the Borderline Personality Features Scale for Children (BPFS-C) (Crick, Murray–Close, & Woods, 2005) and the Personality Assessment Inventory – Borderline Subscale (PAI-BOR) adolescent version (Morey, 2007) have been adapted from the adult Personality Assessment Inventory **– Borderline Subscale (PAI-BOR) (Morey, 1991). The BPFS-C has also been adapted for parent report, and both have shown good va**lidity (Chang, Sharp, & Ha, 2011; Sharp, Mosko, Chang, & Ha, 2011; Venta, Magyar, Hossein, & Sharp, 2018).
- Looking at the factor structure of BPD in adolescence, a **unidimensional structure generally** seems to be supported, for example from studies of the PAI-BOR-A (Venta et al., 2018) and CI-BPD (Michonski, Sharp, Steinberg, & Zanarini, 2013; Sharp, Ha, Michonski, Venta, & Carbone, 2012), although a two factor model has been found in a study involving the BPFS-C (Haltigan & Vaillancourt, 2016).

Distinguishing BPD from typical adolescent development

- Another concern about diagnosing BPD in adolescents is related to the likelihood that observed BPD features actually reflect the typical 'storm and stress' of adolescence. One

assumption supporting this is that relevant developmental processes, including personality development and identity formation, are complete by age 18. However, as mentioned above, these processes are more accurately described as continuing into the mid-twenties and sometimes longer still (Newton-Howes, Clark, & Chanen, 2015).

- Numerous cross-sectional studies have concluded that the **extent and severity of BPD features in adolescents are non-normative** (Chanen, 2017), including when looking at **impulsivity** (Lawrence, Allen, & Chanen, 2010), **substance use** (Scalzo, Hulbert, Betts, Cotton, & Chanen, 2018), **sexual behaviour** (Penner et al., 2019) and **psychosocial** functioning (Kramer et al., 2017). There is also evidence that **identity disturbance** in adolescence resembles that in adults, with a clinically meaningful construct operating from adolescence to adulthood (Westen, Betan, & DeFife, 2011).
- Longitudinal studies suggest that BPD symptoms **emerge in early adolescence, peak** during **mid-adolescence** and **decline** during **early adulthood**, although The Children in the Community Study (Cohen, Crawford, Johnson, & Kasen, 2005) reported that 20% of their sample of 800 young people instead showed increased symptoms during **adulthood**. Considering whether these **young people** might meet the diagnostic threshold for BPD, a study comparing 104 adolescent and 290 adult inpatients with BPD found **similar rates and severity of symptom**s between the groups, suggesting that they were experiencing BPD above a diagnostic threshold rather than emerging symptoms (Zanarini et al., 2017). Of the 24 symptoms of BPD studied, **adolescents** with BPD only showed significantly **lower** levels of i) **quasi-psychotic thought**, ii) **dependency/**masochism, iii) **devaluation/manipulation/sadism** and iv) countertransference problems.

BPD and adolescent personality stability

- A further concern about diagnosing BPD in adolescents is that adolescent personalities are still developing so cannot show the stability of personality disorder symptoms seen in adults. However, evidence supporting relative personality stability from childhood to adulthood includes a longitudinal study assessing **2,450 girls at ages 5-8 years and 14-22 years** (Stepp & Lazarus, 2017). During the follow up, 12.2% of the sample saw conversion to BPD status and the authors found that temperament and symptoms reported by teachers and parents in childhood predicted BPD onset in adolescence and early adulthood, with emotionality as the strongest predictor. Participants with the **lowest scores on emotionality** from parents and teachers, hyperactivity/impulsivity and depression severity from parent ratings and inattention ratings from teachers had a **3.7%** predicted chance of developing BPD, whereas participants with the **highest scores** on these measures had a **54.2% predicted chanc**e, representing a **14-fold increase in risk.**
- BPD also seems to show moderate to strong rank-order stability from adolescence to adulthood. The Children in the Community Study mentioned above found coefficients from 0.4-0.7 (Cohen et al., 2005), which is similar to coefficients for other personality traits among children and adults. A longitudinal twin study had similar findings, with mean-level BPD traits declining from adolescence to adulthood, but high rank-order stability (coefficients 0.53-0.73) (Bornovalova, Hicks, Iacono, & McGue, 2009).

Associated clinical features and causal models for adolescent BPD

- Although there are concerns that causal models for adult BPD may not extend to adolescent BPD and they may have different associated clinical features, there is evidence linking risk

factors and etiological models from adult research to adolescent BPD. For example, similar genetic associations have been found in relation to the short allele of the serotonin transporter promoter gene (5-HTTLPR) (Hankin et al., 2011) and it seems that orbitofrontal alterations found in adults with BPD may already be present in adolescents (Brunner et al., 2010; Chanen, Velakoulis, et al., 2007). In terms of etiology, evidence has been most consistent with both Linehan's biosocial or stress-diathesis model (Crowell, Beauchaine, & Linehan, 2009; Crowell, Kaufman, & Beauchaine, 2014) and the mentalization-based developmental model (Fonagy & Bateman, 2008; Sharp & Fonagy, 2008). More recently, a study of **adolescents diagnosed with BPD compared with controls found higher levels of childhood neglect, lower levels of childhood competence and higher levels of trait neuroticism among the adolescents with BPD** (Zanarini et al., 2020).

- A series of papers using data from the **Avon Longitudinal Study of Parents and Children** (ALSPAC) have investigated the development of personality disorder symptoms:
    - From a sample of 6,050 mothers and their children born between April 1991 and December 1992, **at 11 years old 6.4%** of children had five or mor**e probable BPD symptoms and 0.9% had five or more definite BPD sympt**oms. The authors found that family adversity assessed during pregnancy had a direct impact on BPD symptoms at 11 years and also had indirect effects via suboptimal parenting, parental conflict, poorer cognitive functioning and DSM-IV diagnoses. The authors reported a dose-response effect, with increases in family adversity and maladaptive parenting leading to increased odds of BPD symptoms (Winsper, Zanarini, & Wolke, 2012).
    - From the same sample who completed the Childhood Interview for DSM-IV BPD at 11-12 years old, **persistent nightmares between 2.5 years and 6.8 years wa**s significantly associated with BPD symptoms at 11-12 years. This relationship was still significant after controlling for psychopathological and psychosocial (e.g. maladaptive parenting, family adversity) confounders, while sleep maintenance problems and persistent sleep onset problems were not significantly associated with BPD symptoms after controlling for confounders. This study also found that the relationship between early risk factors such as emotional temperament or maladaptive parenting and BPD symptoms was significantly mediated by persistent nightmares and that the association between persistent nightmares and BPD was significantly mediated by emotional and behavioural problems at 9.5 years (Lereya, Winsper, Tang, & Wolke, 2017).
    - Looking at relationships between adolescent BPD symptoms and subsequent depressive, psychotic and hypomanic symptoms, data from 1,758 ALSPAC participants indicated **that BPD symptoms at age 11-12 years increased the risk of psychotic symptoms and depression diagnosis at 18 years,** as well as clinically relevant lifetime hypomanic symptoms reported at 22-23 years. After controlling for confounders the direct relationship between BPD symptoms at 11-12 years and depression diagnosis at 18 years was no longer significant, but BPD was significantly indirectly associated with depression diagnosis at 18 years via depressive symptoms at 12 years (Winsper et al., 2020).
- Similarly to adults, adolescents with BPD also have high rates of comorbidity and severe psychosocial difficulties compared to adolescents with other mental health difficulties, with the most common comorbidities being mood disorders, eating disorders, dissociative and posttraumatic stress disorders, other personality disorders and substance use disorders (Chanen, Jovev, & Jackson, 2007; Ha, Balderas, Zanarini, Oldham, & Sharp, 2014; Kaess,

Brunner, & Chanen, 2014). However, there do seem to be differences in BPD symptomatology across age groups, which has been attributed to heterotypic continuity during adolescence and young adulthood. Specifically, **externalizing behaviour seems to be mostly expressed in younger age groups and more acute symptoms such as self-harm and excessive risk taking expressed in adolescence**. Longitudinal data from the Pittsburgh Girls Study has shown that higher **ADHD and ODD scores at age 8 uniquely predicted BPD symptoms at age 14** (Stepp, Burke, Hipwell, & Loeber, 2012). This study also found that the rate of growth of ODD scores from ages 8-10 and ADHD scores from 10-13 uniquely predicted higher BPD symptoms at age 14. The increase of internalizing symptoms and decrease of externalizing symptoms with age has been related to the developmental trajectory of impulsivity, which decreases from adolescence to young adulthood and then remains stable. **Among 2,488 participants from a combination of the Life-Events and Gene-Environment Interaction in Depression and Greifswald Family Study in Germany, there was a sharp decline in BPD prevalence between adolescents (5.4%) and young adults (0.9%) which then remained stable in the older age groups.** This study found the same course for impulsivity, whereas levels of depressivity increased from young, middle aged to older adults (Arens et al., 2013). **Affective instability also seems to decline with age** regardless of BPD severity or comorbidity (Santangelo et al., 2017), suggesting together that **BPD presents differently with ag**e, rather than that adolescent BPD is categorically different from adult BPD.

Distinguishing adolescent BPD from internalizing and externalizing disorders

- In general, factor analytic studies suggest that internalizing and externalizing disorders do not account fully for variability in BPD features (Eaton et al., 2011) and among adolescents, BPD has been shown to be a significant predictor above Axis I disorders and PDs for psychopathology, general functioning, peer relationships, self-care, and family and relationship functioning (Chanen, Jovev, et al., 2007). **Personality disorders appear to be more stable than internalizing and externalizing disorders during adolescence** (Cohen et al., 2005) and adolescent BPD symptoms have also **shown moderate to strong associations with psychosocial functioning, self-perception, social skills and sexual behaviour** (Wright, Zalewski, Hallquist, Hipwell, & Stepp, 2016).
- Instead, internalizing and externalizing disorders seem to precede BPD. For example, results from a large twin study have shown that **BPD characteristics at age 12 were associated with internalizing and externalizing disorders in earlier childhood** (Belsky et al., 2012), while there is **no evidence that BPD precedes** either of these disorder types (Bornovalova, Hicks, Iacono, & McGue, 2013; Lazarus, Beardslee, Pedersen, & Stepp, 2017).

Stigma associated with adolescent personality disorder

- A final concern about diagnosing adolescent personality disorders relates to the potential **stigma** of the diagnosis. Sharp argues that this partly relies on mistaken assumptions that personality disorders are **both chronic and untreatable**. Current systematic review evidence suggests that a range of specialized adult treatments are effective in reducing borderline features and related problems. From **33 trials, psychotherapies** have been found to be **moderately more effective than control** interventions for borderline-relevant outcomes, with dialectical behaviour therapy and psychodynamic approaches specifically being more effective than control conditions (compared with CBT and 'other' interventions which were

not significantly more effective) (Cristea et al., 2017). Additionally, there is evidence that **BPD features remit over time for the great majority of individuals**, with a 10 year follow up study of 242 18-35 year olds with BPD finding that overall 88% of the sample achieved remission (39.3% by two years, an additional 22.3% by four years, an additional 21.9% by six years, an additional 12.8% by eight years and an additional 3.7% by ten years). (Zanarini, Frankenburg, Hennen, Reich, & Silk, 2006).

- Adult treatments that have been adapted for adolescent BPD include dialectical behaviour therapy (DBT-A), mentalization-based therapy (MBT-A), cognitive analytic therapy (CAT) and emotion regulation training (ERT).
    - DBT-A includes family members in skills groups and adds a family therapy element to adult DBT. Compared to individual and group supportive therapy (IGST) during an RCT involving 173 adolescents with elevated suicide risk and who met three or more BPD criteria, DBT-A was more effective at post-treatment in reducing self-harm (no self-harm for 54.2% DBT-A vs 36.9% IGST, OR 0.33), suicide attempts (no suicide attempts for 90.3% DBT-A vs 78.9% IGST, OR 0.3) and non-suicidal self-injury (no self-injury for 56.9% DBT-A vs 40% IGST, OR 0.32) (McCauley et al., 2018).
    - Compared to adult MBT, MBT-A adds a family therapy component and focuses on impulsivity and affect regulation. Compared to controls, adolescents with recent self-harm who received MBT-A showed greater decreases in self-harm (self-harm in the last 3 months 56% MBT-A vs 83% TAU, NNT 3.66) and depressive symptoms (probable clinical depression 49% MBT-A vs 68% TAU, NNT 5.31), and were less likely to meet criteria for BPD (33% MBT-A vs 58% TAU) after 12 months (Rossouw & Fonagy, 2012). Group-based MBT-A has also shown clinically significant changes in BPD symptoms, mentalizing and trust among adolescents who had symptoms meeting four BPD criteria (Bo et al., 2017).
    - The Helping Young People Early (HYPE) programme is based on CAT and aims to intervene early with elements of psychoanalytic object relations theory and cognitive psychology (Chanen, McCutcheon, & Kerr, 2014; Chanen et al., 2009). In older adolescents with at least one childhood risk factor and at least three BPD criteria, CAT led to more rapid recovery compared with treatment as usual although differences between the groups were reduced at the 2 year follow up point (Chanen et al., 2008; Chanen et al., 2009).
    - ERT was adapted from Systems Training for Emotional Predictability and Problem Solving (STEPPS) and involves both cognitive-behavioural elements and dialectical behaviour skills training. An RCT of ERT reported decreased symptoms at 6 months, although not below the level of treatment as usual (individual pharmacotherapy and psychotherapy) (Schuppert et al., 2009; Schuppert et al., 2012).
- While the perception of personality disorders as chronic and untreatable may not be completely accurate, the stigma accompanying a BPD diagnosis remains. For example, a study with adolescents who had a range of diagnoses found that those with personality disorders experienced the greatest stigma, and that BPD was the strongest predictor of experiences of stigma (Catthoor, Feenstra, Hutsebaut, Schrijvers, & Sabbe, 2015).
- However, not diagnosing adolescent BPD excludes young people from treatment options and potentially perpetuates negative stereotypes and stigma. There is some evidence that a personality disorder diagnosis can be helpful to adolescents (Courtney & Makinen, 2016) and that a diagnosis may help to validate their experiences.

The Alternative DSM-5 Model for Personality Disorders (AMPD)

- Sharp et al. (2021) advocate that the AMPD allows for a more developmentally sensitive approach to understanding, preventing and intervening early with personality disorders through a Clinical Staging approach.
- Adolescence seems to be a sensitive period for the onset of BPD symptoms in Criterion A functioning (self, identity, intimacy and empathy), while Criterion B represents internalizing-externalizing spectra symptoms. This conceptualisation has empirical validity through the personality-psychopathology spectrum model (Shiner & Caspi, 2003; Tackett, 2006) and maladaptive self and interpersonal functioning (Criterion A) seems to build upon pre-adolescent maladaptive trait functioning (Criterion B).
- Maladaptive Criterion A functioning becomes noticeable during adolescence as this is the time that metacognitive capacities develop that enable the construction of a narrative identity (McAdams, 1995; McAdams et al., 2004) which allows connection of past, present and future and a coherent, organised identity. Supporting this, there is evidence that borderline features correlate with identity diffusion and lower levels of narrative coherence (even when identity components of the measure were removed) and that narrative coherence increases with age (Lind, Vanwoerden, Penner, & Sharp, 2020).

Clinical Staging approach to prevention and early intervention

- Clinical staging describes the progression of a disorder through five stages, from 'at risk' to a persistent personality disorder. These stages allow a more dynamic approach to assessment and treatment and a structure for any remittance or worsening of symptoms.
    - **Stage 0 – Increased risk for disorder but no current symptoms**: adolescents may show high levels of Criterion B maladaptive traits and community prevention interventions may be important to prevent progression to Stage 1.
    - **Stage 1a – Mild or nonspecific symptoms with mild functional change or decline**: adolescents may show symptoms of both internalizing and externalizing disorders and could have mild issues in social and school functioning. Community based interventions could be helpful at this level, such as mental health literacy interventions, family psychoeducation, parenting skills interventions or supportive counselling.
    - **Stage 1b – Ultra-high risk adolescents who show moderate but subthreshold symptoms**: adolescents with a confluence of internalizing and/or externalizing disorders and high scores on Criterion B traits. There may also be an escalation of social and educational challenges. At this stage, Stage 1a interventions would be helpful with additional time-limited evidence based interventions (such as CBT).
    - **Stage 2 – First episode of threshold disorder with moderate to severe symptoms and functional decline**: young people showing high levels of Criterion A dysfunction and a maintenance of high levels of Criterion B traits with moderate to severe problems in social, educational and/or work functioning. Interventions include those at Stage 1b with additional outpatient case management, educational or vocational interventions, family psychoeducation and support, time-limited psychotherapy and targeted psychopharmacotherapy.
    - **Stage 3a – Recurrence of subthreshold symptoms**: young people may experience a return of high levels of Criterion B traits and challenges in social and educational

settings. Interventions include those at Stage 2 with additional emphasis on maintenance medication and psychosocial strategies for full remission.

- **Stage 3b – First threshold relapse of personality disorder**: young people with the first threshold relapse of Criterion A dysfunction, high levels of Criterion B traits and moderate to severe problems in social, educational and/or work functioning. Additional relapse prevention strategies and outpatient or intensive outpatient services in addition to Stage 3a interventions.
- **Stage 3c – Multiple relapses of personality disorder**: Young people who have experienced multiple relapses of Criterion A function, high levels of Criterion B traits and near chronic levels of social, relationship, educational and/or work impairment. Additional intensive stabilisation in addition to Stage 3b interventions involving intensive outpatient services or inpatient treatment.
- **Stage 4 – Persistent, unremitting disorder**: Persistent and unremitting Criterion A dysfunction alongside high levels of Criterion B traits and no participation in social and professional life. Intensive outpatient services with additional intensive psychosocial interventions such as DBT or MBT and psychopharmacological intervention in addition to Stage 3c interventions.

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