Personality Disorders and Autism Spectrum Disorder: What Is Similar and What Is Different?

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1 Introduction

Personality disorders (PDs) are mental disorders characterized by enduring maladaptive patterns of cognition and behavior, causing difficulties in personal and social functioning (American Psychiatric Association 2013). The origins of PDs are poorly understood; recent pathogenic models are based on traumatic negative (early) childhood experiences and on genetic background (Ballard et al. 2015; Chanen and Kaess 2012). In contrast to personality, i.e., mental traits that characterize human beings, PDs are associated with significant dysfunctional experience and behavior, a number of comorbid conditions, and severe personal disadvantages. These endanger personal well-being, education, social and occupational integration, and relationships (Biskin 2015).

Autism spectrum disorder (ASD) is a heterogeneous group of pervasive disorders characterized by problems of social communication and interaction, restrictive interests, and repetitive behaviors (American Psychiatric Association 2013). Caused by genetic deficiencies that impair brain networking, ASD shows a broad range of severity of impaired intellectual functioning, inflexibility, and personal and social dysfunction. There are multiple comorbid conditions, e.g., concerning sensory integration, attention, neurology, anxiety, and PDs. ASD are not curable, but improvements of development and personal and social functioning may be attained through

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structured therapy for low-functioning children and through appropriate psychological and pharmacological therapy for higher-functioning children and adolescents. In this paper, we will focus on ASD patients with normal intellectual capacity; the relationships between low-functioning ASD patients and PDs have not yet been explored.

The relationships and overlapping psychopathology between ASD and PDs, although evident (especially for clusters A and C), are only poorly described and understood. These areas of overlap include pervasive impairment, abnormal development, stable patterns, long duration, onset in childhood to adolescence or early adulthood, and social impairment (Miller and Ozonoff 1997). However, a PD should be excluded "if the enduring pattern is not better accounted for as a manifestation or consequence of any other mental disorder" (American Psychiatric Association 2013). In this article, we will focus on these complex relationships, drawing upon available literature and aiming to contribute to the knowledge and (medical) handling of both disorders. We will especially focus on prevalence, phenomenology and clinical issues, common and uncommon features of PDs and ASD, and assessment and treatment.

2 Prevalence

PDs are among the most prevalent disorders in adulthood, reaching rates of 10 % in community samples (Coid et al. 2006) and up to 50 % in clinical samples (American Psychiatric Association 2000; Sevecke and Krischer 2008). Longitudinal studies suggest that prevalence rates of PDs are higher in adolescence and decline linearly up to the age of 27 (Chanen and Kaess 2012). Differences in prevalence rates may be explained by nonuniform interpretation of diagnostic criteria and the various classification systems.

The diagnosis of a PD is less stable than formerly expected (Biskin 2015; Skodol et al. 2005; Stepp 2012) and differs among the various types. PD traits such as "self-injurious behavior" are less stable than states such as "affective instability" and "impulsivity." Symptom stability is relatively stable over time for borderline (BPD), histrionic, and schizotypal PD and less stable for other categorical diagnoses.

3 Phenomenology, Comorbidity, and Clinical Issues

PD types are differently described by the various diagnostic systems: ICD-10 lists eight categories and a number of "others"; DSM-IV defined three clusters (A: odd, B: dramatic, C: anxious) and ten PDs in a separate diagnostic axis; and DSM-5 created a hybrid model, defining six categories of core impairments (antisocial, avoidant, borderline, narcissistic, obsessive-compulsive, and schizotypal PD, Criterion A) and five high-order traits (negative emotionality, detachment, antagonism, disinhibition, psychoticism, Criterion B). This new model reflects the complexity of PDs, improves discriminant validity and stability of the diagnosis, reduces comorbidity, and allows for assessing personal (identity and self-directedness) and

interpersonal (empathy and intimacy) functioning. Although personality traits may be present in early childhood, a definite PD diagnosis is now permitted in adolescence, respecting that early intervention and adequate psychological and pharmacological therapy may result in significant functional improvement and even cure. In so doing, therapeutic nihilism and the mischaracterization of PDs as lifelong conditions should be avoided.

Patients with an increased symptom level are more severely affected in their psychosocial functioning. A high level of cluster A symptoms has been related to lower educational levels and job performance (Cohen et al. 2005).

Diagnostic and statistical manual of mental disorders- Fifth edition (DSM-5) has also introduced revised specifications for ASD: ASD is now a spectrum disorder, with two main symptoms – (1) deficits in social communication and interaction and (2) restricted interests and repetitive behavior. The previous categories no longer exist. Moreover, DSM-5 has introduced specifiers, intellectual and language impairment, association with a known medical or genetic condition, and three severity levels.

PDs and ASD share many comorbid conditions that aggravate the severity of the disorders. Comorbidities of BPD mainly relate to attention deficit (ADHD), mood, anxiety, posttraumatic stress disorder (PTSD), and eating disorders (EDs). The close relationship between antisocial PD (ASPD) and ADHD remains stable into adulthood (Mannuzza et al. 1998; Nigg et al. 2002; Rösler et al. 2008; Sevecke and Krischer 2008). In addition, characteristics of ASPD, BPD, and early complex PTSD and problems of interaction may overlap (Brunner et al. 2001; Schmid et al. 2010), and there are close similarities between PTSD and BPD (Jucksch et al. 2009; Krischer and Sevecke 2008). Comorbidities of ASD mainly relate to ADHD and sensory integration problems and mood, tic, and anxiety disorders.

There are only a few studies relating PS and ASD. Generally, adolescents and adults with PDs and ASD face problems in social interaction, mentalizing abilities, and building and maintaining stable relationships. Cluster A and C PDs seem to be more prevalent in ASD patients (Lugnegård et al. 2012), whereas ASD psychopathology seems to protect patients from cluster B PDs. This could be related to early difficulties and less interest in social interaction in ASD, limiting the effects of poor parenting and inconsistent parent-child interaction on the developing personality. Subjectively experienced excessive emotional demands leading to temper tantrums could be a link between ASD and cluster B PDs, but the backgrounds seem to be different: children with ASD are overburdened because of their limited cognitive flexibility, whereas BPD patients are overburdened by their difficulties in expressing and regulating their emotions (Jarnecke et al. 2015). Moreover, although BPD and ASD patients exhibit social problem-solving deficits, the former have intense but unstable social relationships and strive to avoid abandonment, whereas ASD patients seem to be socially more independent and to display less interest in social interactions (King-Casas and Chiu 2012). There is also a sex difference: ASD patients are predominantly male, while PDs show a female predominance.

Tantam (1988) and Esterberg et al. (2008) have described more severe current and past autistic features, such as social impairment and unusual interests and

behavior in adolescents with schizotypal PD (SPD) compared to normal and other PD controls. Hurst et al. (2006), studying patients with Asperger's syndrome and SPD, found overlapping diagnostic criteria and correlations between social-interpersonal and communication-disorganized areas between the two disorders. Personality traits that are similar but less severe than in ASD are seen in the "broader autism phenotype" (BAP), comprising the traits "rigid," "impulsive," "aloof," "shy," "tactless," "reserved/schizoid," "irritable," "hypersensitive to criticism," "neurotic," "undemonstrative," and "anxious" (Sucksmith et al. 2011; Vannucchi et al. 2014).

Lugnegård et al. (2012), investigating 54 young adults with the clinical diagnosis of Asperger's syndrome (DSM-IV criteria, SKID-II interview), found marked PD symptoms in one-half of the autistic patients, two-thirds of the male, and one-third of the female population. The PD criteria were fulfilled for only four PDs, schizoid, schizotypal, avoidant, and obsessive-compulsive PD, and there were no cluster B diagnoses.

According to Hare and Neumann (2009), psychopathy as a specific PD subtype and ASD are both associated with strong deficits in empathy. Empathy deficits differed, however, in both groups of patients in genetic, cognitive, and neurologic aspects (Wallace et al. 2012). Specifically, individuals with psychopathic personality traits had problems in processing emotional stimuli, had reduced levels of anxiety, and had deficits in ethical reasoning, whereas patients with ASD had difficulties with the cognitive processing of emotional cues (Rogers et al. 2006). Roberz et al. (2013) discuss the differential diagnosis in regard to a case report about a 17-year-old boy. ASD patients perceive the suffering of others as aversive (Blair 1999; Sigman et al. 2003). ASD patients also have difficulties understanding the reasoning and feeling of others and seem to act insensitively. Yet when information is presented in such a way that enables them to emotionally understand the problems of others, they feel and care empathically normal (Jones et al. 2010).

Anckarsater (2006) reviewed brain imaging similarities between ASD and aggressive PD or psychopathy, especially in regard to hypoactivity and structural reduction of the prefrontal cortex and the limbic circuitry. Patients with ASPD show smaller prefrontal and cingulate cortices (Yang et al. 2009), while ASD patients show volume reductions in the temporal and parietal areas (Scheel et al. 2011; Wallace et al. 2010, 2012).

Diagnostic evaluation for PDs includes semi-structured instruments such as SCID-II (Structured Clinical Interview for DSM-IV (First et al. 1997)) and IPDE (International Personality Disorder Examination (Loranger 1994)). The diagnosis of ASD relies on ADOS 2 (Autism Diagnostic Observation Schedule (Poustka et al. 2015)) and ADI-R (Autism Diagnostic Interview – Revised (Bölte et al. 2006)), both of which assess autistic symptoms in a semi-structured way. Patients with PD or ASD are not always aware of their clinical problems, and this leads to interpersonal difficulties. As patients may not report their problems, it is essential to also rely on external sources of information, such as parents, relatives, and teachers. Furthermore, experienced clinicians will detect interpersonal dysfunctions by analyzing transference and countertransference phenomena.

4 Treatment

4.1 Nonmedical Interventions

A number of effective psychotherapeutic programs and manuals for treating (B)PDs are available for adults. For adolescents, these therapies – with the exception of DBT-A – have not yet been fully evaluated:

- Specific psychodynamic therapies: Transference-Focused Psychotherapy (TFP (Clarkin et al. 2004)) and Mentalization-Based Treatment (MbT (Bateman and Fonagy 2006)) and Adolescent Identity Treatment (AIT (Foelsch et al. 2013))
- Specific cognitive behavioral therapies: Dialectical Behavior Therapy (DBT and DBT-A (Linehan 1993)) and Schema Therapy (Young et al. 2003)

Ongoing clinical trials for the treatment of adolescents with PDs aim to respect the specific needs of this age group (see overviews: Krischer et al. 2006; Krischer and Sevecke 2010). Examples of modified adult treatment protocols are Adolescent Identity Treatment (AIT (Foelsch et al. 2008)), Transference-Focused Psychotherapy for Adolescents (TFP-A (Fleischhaker et al. 2011)), and Dialectic Behavioral Therapy for Adolescents (DBT-A (Miller et al. 1997)). These therapies focus more on identity diffusion than on identity crisis (Foelsch et al. 2010). Identity diffusion entails the lack of an integrative self-concept, similarly lacking in regard to significant persons. Identity diffusion is a prerequisite for developing adolescent PDs. The patient describes himself or herself and others in a highly chaotic way, unable to detect or integrate contradictions (Clarkin et al. 2004). Adolescent PD therapy aims at improving interpersonal relationships with friends, parents, and teachers. It further aims at defining lifetime goals, developing a sense of self-worth, and achieving a stable identity.

4.2 Pharmacological Interventions

Pharmacological interventions for ASD and PDs are only symptomatic and supportive. The main target symptoms are aggressive behavior/temper tantrums, depression, sleep problems, and ADHD; in ASD patients with seizures, anticonvulsive medication is indicated. A recent field study on psychopharmacological treatment in Germany (Bachmann et al. 2013) included 1,124 patients, 0.5 % having the diagnosis of ASD. One-third of the ASD patients received medication, with methylphenidate and risperidone the most frequently prescribed substances. Pharmacological studies on treating PDs mainly focus on BPD (Paris 2011), with low evidence (Klar and Siever 1984). Treatment guidelines, e.g., for BPD (Oldham et al. 2001), are poorly evidence supported and are no longer up to date.

Neuroleptics are widely used to decrease intrapersonal stress levels, enabling a distancing from environmental stressors and a reduction of impulsivity and reactive aggressive behavior. Although widely used, neuroleptic treatment shows poor

evidence of efficacy because large RCTs are lacking, especially for second-generation antipsychotics.

Anticonvulsants – aside from their use in epilepsy and bipolar disorder – are commonly used to reduce impulsivity and aggressive behavior. The evidence of such treatment is, however, poor (Huband et al. 2010).

Antidepressants are generally less effective in ASD (Williams et al. 2013) and PD (Paris 2011). This may be caused by the background and unchanging intrapersonal strain (due to flexibility problems) or by neuro-functional differences that are poorly understood.

The efficacy of ADHD medication, specifically methylphenidate and atomoxetine, has been proven in several prospective trials in ASD patients. Both medications seem to be less effective in ASD and PD patients than in normotypic and non-PD ADHD patients. However, methylphenidate (Simonoff et al. 2013) and atomoxetine (Handen et al. 2015) are effective in children with ASD, with atomoxetine showing a slightly better side effect profile. (B)PD patients with comorbid ADHD also benefit from these two medications (Newcorn et al. 2007). In addition, aggressive behavior (Blader et al. 2013) and the occurrence of SUD (Steinhausen and Bisgaard 2014) are reduced, and psychotherapeutic adherence improved with long-term medication (Prada et al. 2015).

5 Discussion and Future Directions

Clinical diagnosis and treatment of adolescents with PDs and ASD are sensitive, complex tasks that require specific knowledge and experience. The available diagnostic tools, mainly structured interviews and clinical observation, are relatively sensitive, specific, and reliable.

The findings to date and the abandoned age limitation for the assessment of PD (DSM-5) clearly justify the diagnosis of PDs in adolescence, but more work is necessary to better identify and understand age-specific processes and characteristics. Early, careful diagnosis of adolescents with PS, followed by specific treatment, is certainly indicated. It is noteworthy that large numbers of patients, when treated appropriately, are able to go into remission, rendering therapeutic nihilism as a consequence of presumed "incurability" unwarranted.

The new DSM-5 criteria for both groups of disorders provide a courageous approach, enhancing the quality of dimensional diagnosis. For PDs, abandoning the age limit of 18 and introducing a hybrid disorder model, keeping "old" categories and adding five high-order traits and 25 trait facets, will help characterizing the clinical picture more precisely. In ASD patients, emphasizing common problems, adding specifiers and severity levels, and abandoning categorization will likely increase diagnostic precision. Nevertheless, longitudinal studies for both groups of disorders are needed to better characterize the stability of the diagnostic criteria and the effects of specific therapies. For both groups of disorders, access to specific therapy and early, accurate diagnosis are essential to providing appropriate support.

As in all disorders, comorbidity is crucial for clinical severity, vocational success, and quality of life. The two disorders share multi-comorbid conditions, including ADHD and mood, anxiety, dysexecutive, and other disorders that further impair social functioning. Treatment must therefore consider the part of impairment caused by comorbid conditions.

The relationship between PDs and ASD is complex and not clearly defined. There are similarities, e.g., social ostracism, poor emotional control, comorbid conditions, unknown stability of symptoms, and therapy-related alleviation, as well as differences, e.g., age at onset, intellectual performance, social skills, and fears of abandonment. Some PDs and ASD and schizotypal PD show significant overlap. One of the main differences, besides age at onset, is etiology: ASD is primarily related to genetic problems of brain networks, with less parental impact, whereas PDs usually have a family and social background but are predominantly acquired disorders, related, for example, to poor parental functioning and repeated traumatization.

Nevertheless, PDs, especially those with schizotypal personality traits, and ASD share impaired social functioning due to emotional dysfunction. They appear to be of different etiology, however, which is apparent in their differing genetic, cognitive, and neuronal characteristics. The key point for differential diagnosis between PD and ASD thus appears to lie in the precise assessment of patients' empathy deficit.

Future work will likely focus on long-term epidemiology, etiology (genetics, inborn and acquired problems), and therapy. Precise diagnostics will influence specific therapies. The introduction of the dimensional-categorical hybrid model of the DSM-5, with its assessment of traits and trait facets, now allows for a better differentiation and characterization within personality pathology profiles. It may indeed also be helpful to create such profiles for patients with ASD.

Other questions concern social integration and specific vocational training as well as tailor-made and cost-effective psychotherapies for specific clinical entities. Similarly, the long-term effect of psychotherapies remains to be explored.

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