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## The co-occurrence of personality disorders and substance use disorders

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### Abstract

**Purpose of review:** Despite significant negative outcomes, the co-occurrence of personality disorders (PDs) and substance use disorders (SUDs) continues to be underrecognized, and the mechanisms contributing to this co-occurrence remain unclear. This review summarizes recent work on PD-SUD co-occurrence, with a focus on borderline and antisocial PDs, general substance use patterns among those with PDs, and the association of personality traits with SUDs.

**Recent findings:** The prevalence of co-occurring PD-SUD is generally high, with estimates ranging depending on the type of PD and SUD, the population assessed, and the sampling methods and measures used. Current theoretical explanations for co-occurrence include shared etiology and predisposition models, with research highlighting the importance of transactional processes. Potential underlying mechanisms include personality traits and transdiagnostic characteristics.

**Summary:** Recent research has increased focus on substances besides alcohol, dimensional models of personality pathology, and transactional explanations of co-occurrence, but more research is needed to disentangle the nuanced PD-SUD relationship.

### Keywords

personality disorders; substance use disorders; substance use; borderline personality disorder; antisocial personality disorder

### Introduction

People with personality disorders (PDs) often use substances, experience use-related problems, and meet criteria for substance use disorders (SUDs). In turn, people with SUDs often struggle with challenges related to personality dysfunction. This co-occurrence of PDs and SUDs leads to greater negative consequences, poorer treatment outcomes, and higher severity and chronicity of both disorders compared to either alone (1–3). Despite this, the co-occurrence of PDs and SUDs continues to be relatively underrecognized, and the mechanisms that contribute to this co-occurrence remain unclear. The current review summarizes recent work on PD-SUD co-occurrence. We focus on borderline and antisocial PDs, as most existing work has studied these two PDs. We then consider theoretical

explanations for PD-SUD co-occurrence and identify potential mechanisms for the overlap, concluding the review with implications and future directions.

### Setting the stage: Definitions

The *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) (4) groups ten PDs into three clusters: “odd or eccentric” Cluster A (paranoid, schizoid, schizotypal PDs); “dramatic, emotional, or erratic” Cluster B (antisocial, borderline, histrionic, narcissistic PDs); and “anxious or fearful” Cluster C (avoidant, dependent, obsessive-compulsive PDs). Borderline personality disorder (BPD) and antisocial personality disorder (ASPD) are among the most diagnosed and studied PDs and are prevalent in the general population (estimated 3.8% ASPD and 2.7–5.9% BPD) (5). BPD is characterized by impulsive and self-destructive behaviors, and instability of affect, identity, and interpersonal relationships. ASPD involves behavior patterns showing deceit, impulsivity, and a lack of regard for the rights of others. The difficulties with emotional and behavioral control involved in these disorders manifest in a variety of maladaptive behaviors, including problematic substance use.

SUDs span ten separate classes of drugs (e.g., alcohol, cannabis, opioids) and are characterized by cognitive, behavioral, and physiological symptoms resulting from ongoing substance use despite substance-related physiological, behavioral, and interpersonal problems (4). In the United States, past-year prevalence of SUD and alcohol use disorder (AUD) for those 12 and older was 16.5% and 10.6%, respectively (6), and lifetime AUD prevalence rates in adults approach 30% (4).

With ten PDs and ten SUDs, there are myriad possible combinations even before factoring in that many people meet for multiple PDs and use multiple substances. Perhaps it is no surprise, then, that there is heterogeneity in PD-SUD co-occurrence rates across studies, potentially reflecting different populations, sampling methods, and measures used. Co-occurrence also varies based on demographic factors like race, gender/sex, and age, among other characteristics. The existing literature has only scratched the surface in understanding the heterogeneity in PD-SUD co-occurrence.

### Prevalence part I: Substance use disorders in people with personality disorders

The prevalence of SUDs across PDs is generally high, with estimates ranging depending on PDs and SUDs assessed. In the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), a population-based study of 36,000 people, 41.9% of those with any PD had lifetime DSM-IV alcohol dependence, 18.7% drug dependence, and 48.2% nicotine dependence (5). More recent work finds similar rates. A 2018 review found that 38.9% of people with undifferentiated PDs or PDs other than BPD/ASPD had lifetime AUD (7). In an Asian cohort of 1,172 adults with PDs, AUD was the most prevalent SUD (43.2%), followed by methamphetamine use disorder (23.6%) and other SUDs (33.2%) (8). In a representative Brazilian sample of 2,942 adults, more than half of individuals with any PD used alcohol in the previous year, with 80.6% of those who drank being regular drinkers (9). Among regular

drinkers, 42.3%, 70%, and 46.1% of those with Cluster A, B, and C PDs, respectively, met for AUD. The presence of a Cluster B PD increased the odds of not only AUD, but also alcohol consumption and more severe AUD (9). In a treatment seeking sample, 69.8% of patients with a PD had a co-occurring SUD (66.4% AUD, 58.5% cocaine use disorder, 27.1% opioid use disorder [OUD], 43.5% cannabis use disorder, and 16.2% sedative use disorder) (10).

Specifically examining BPD, a recent review found approximately half of those with BPD have at least one SUD, with average co-occurrence rates of 45.5% for current and 75.3% for lifetime SUDs (11). Among those with BPD, AUD was the most common SUD, with average lifetime rates of 52.2%–59.5% (7,11), while 39.2% met criteria for current drug use disorder (DUD) (11). Men with BPD from a community sample were somewhat more likely to have comorbid AUD and SUD (53.1% and 59.3%) than women with BPD (36.9% and 44.3%) (12). Compared to the general population, youth with BPD show between four and nine times higher prevalence of alcohol dependence (20% prevalence), daily tobacco use (63% prevalence), and past-month use of illicit substances (44% cannabis and 17% amphetamine prevalence) (13). The prevalence of substance and polysubstance use, as well as levels of alcohol-related problems and risk, are also significantly higher among youth with BPD than age-matched controls (13–14).

Co-occurring SUDs are also prevalent in ASPD, with rates ranging from 55.3% for any past-year SUD (10.5% DUD, 31.2% AUD) to 76.7% for lifetime AUD (7,15). Among 427 Chilean prison inmates, 26% had comorbid ASPD-SUD, and 39.8% comorbid BPD-SUD (16). In adults 50 years and older in NESARC Waves I and III, ASPD was associated with a higher likelihood of several SUDs, including alcohol, marijuana, cocaine, and nicotine (17). In a representative English population ( $N=7,218$ ), hazardous alcohol use was most common in ASPD (30.3% prevalence), while alcohol dependence was most common in BPD (17.7% prevalence) (18).

## **Prevalence part II: Personality disorders in people with substance use disorders**

Prevalence estimates of co-occurring PDs in people with SUDs vary widely, with overall PD rates ranging from 50% to 92%, BPD rates from 12% to 53%, and ASPD rates from 9% to 23% (19). Presence of alcohol, opioid, sedative, stimulant, or heroin use disorder, as well as having multiple SUDs, was associated with increased risk of PD in NESARC (20). Almost a quarter (22.1%) of those with a current SUD met criteria for BPD (11), which was most associated with opioid, cocaine, and alcohol use disorders (21). Specifically examining alcohol, the prevalence of BPD ranges from 12–17% and ASPD from 19–22% (1). Compared to men, women with AUD in NESARC had a greater likelihood of BPD, and women with SUD had an increased likelihood of all PDs (22). Although receiving less attention, avoidant, histrionic, and paranoid PDs are also prevalent in AUD samples (19). Lower estimates of co-occurring AUD and ASPD/BPD (1.6–9.8%) have also been identified, which, together with high estimates of mixed/not specified PD (23), highlight the unreliability of prevalence estimates and diagnostic issues in categorical PDs. Additionally,

although many studies report general PD rates, PD type has been shown to account for most of the heterogeneity in lifetime AUD prevalence (7), emphasizing the potential value of differentiating personality dysfunction.

Recent research has linked PD diagnoses and PD traits to substances besides alcohol. PDs were highly prevalent among 577 males inpatients treated for methamphetamine use disorder in China, particularly ASPD (71.4%), BPD (20.2%), obsessive-compulsive (17.9%), avoidant (16.2%), and paranoid (14.3%) PDs (24). BPD features have been linked to heroin and methamphetamine use disorders in a small sample of Taiwanese adults (25), as well as to pre-incarceration alcohol, cannabis, cocaine, and opioid dependence among 510 people incarcerated in jail (26). Cannabis use in adolescence is associated with paranoid and schizotypal personality traits in young adulthood, consistent with other findings in linking early cannabis use to increased risk of psychotic symptoms and disorders (27). Conversely, schizotypal traits are also related to lower alcohol consumption, even among adolescents who are also elevated in borderline traits, potentially suggestive of less frequent engagement in social activities that may be associated with alcohol use (28). Different traits may be related to different substance use patterns, as shown in a study where paranoid PD traits were related only to cannabis use, whereas avoidant and dependent PD traits were more strongly linked to cannabis use disorder (29).

Given the impact of the ongoing opioid epidemic (30), it is important to examine the association of PDs and opioid use. Reviews find a 33.6% lifetime prevalence of ASPD and 18.2% BPD among those with OUD (31), indicating that a PD diagnosis may be a risk factor for OUD (32). Among 100 males in opioid treatment, PDs were among the most common comorbidities (27.6%), particularly ASPD (17.1%) and BPD (7.9%) (33). ASPD and BPD traits are also prevalent among people who use opioids, misuse prescription opioids, and meet criteria for OUD (34–36).

Considering that SUDs frequently co-occur and co-occurrence is associated with more severe problems (37), it is important to study this comorbidity in the context of PDs, which are associated with higher risk of polysubstance use (13,37). In NESARC, people with both alcohol and cannabis dependence had higher rates of PDs and a greater number of PDs than those with either diagnosis alone. Specifically, 77% of people with both SUDs had any lifetime PD, 41% were diagnosed with ASPD, 32.9% BPD, 29.1% obsessive-compulsive, 26.5% narcissistic, 24.4% paranoid, 21.2% schizotypal, 12.9% schizoid, 12.4% histrionic, 11.9% avoidant, and 4.4% dependent PDs (39). Co-occurring sedative use disorder and OUD have been linked to ASPD in both men and women, and schizotypal PD among women only (40). The co-occurrence and interaction of multiple PDs and SUDs likely results in distinctive clinical profiles warranting unique considerations. These relationships, although challenging to disentangle, are important to further study to improve our understanding and treatment of complex multimorbid disorders.

### **Prevalence part III: Dimensional models of personality disorders**

Existing work is largely based on categorical diagnoses, the limitations of which include arbitrary boundaries for diagnosis, heterogeneity of symptoms and presentations, and

artificially high comorbidity, particularly among PDs (41). PDs can alternatively be conceptualized as configurations of extreme variations of personality traits. SUDs have been linked to a number of personality traits and individual differences, including impulsivity, novelty seeking, harm avoidance, and negative thinking, with many of these related to the severity of drug and alcohol use (25,42–44).

### **Alternative model of personality disorders**

In contrast to the categorical PD classification, the DSM-5 Alternative Model of PD (AMPD) involves five broad personality domains: negative affectivity, detachment, antagonism, disinhibition, and psychoticism. For instance, both ASPD and BPD are characterized by disinhibition, with BPD additionally involving negative affectivity, and ASPD additionally involving antagonism (4). Research has started to explore relationships between AMPD personality facets and various SUD profiles, linking all five PD facets to drug and polydrug use (45–46). Cannabis use disorder has been linked to facets of schizotypal PD (i.e., suspiciousness, eccentricity, unusual perceptions), while patients with alcohol, heroin, and cocaine use disorders showed higher scores on pathological facets related to both schizotypal PD and BPD (i.e., emotional lability, separation insecurity, impulsivity) (45). A recent study found associations between AUD criteria and PD facets of risk taking, callousness, and irresponsibility, bridging externalizing personality traits with AUD criteria (47). These PD facets have also been linked to premature SUD treatment termination (48). As these facets are associated with BPD and ASPD, this may help explain why patients with these PDs have higher rates of relapse and treatment termination (47).

### **Externalizing spectrum**

The broad externalizing “superspectrum” of the Hierarchical Taxonomy of Psychopathology (HiTOP), which is composed of disinhibited and antagonistic spectra, encapsulates a variety of mental disorders associated with difficulties with impulsive control and self-insertion, including SUDs and Cluster B PDs (49). Several factor analytic studies have pointed to shared externalizing components underlying SUDs and ASPD, such as impulsivity, disinhibition, and role interference (50–52). BPD, although highly heterogeneous and traditionally placed at the externalizing-internalizing intersection, has similarly been linked to substance use via externalizing pathology (53,46). AUD is also heterogeneous, with some symptom clusters reflecting disorder-specific expressions while other symptom combinations differentially relate to internalizing and externalizing dimensions (54). Compared to traditional disorder classification systems, HiTOP provides improved characterization of SUDs (55), offering promising avenues for disentangling PD-SUD co-occurrence.

BPD and ASPD share key externalizing features, including deficits in emotional and behavioral control, and their co-occurrence may thus be particularly related with substance use and SUDs. Supporting this, the co-occurrence of BPD and ASPD was associated with a fivefold increase in SUD risk compared to those with only ASPD or BPD (15). A recent twin study found, in a bifactor model, that a general factor encompassing ASPD and BPD criteria was related to general substance use (56). Although bifactor models have important limitations (57), their findings may suggest that risk for substance use is not specific

to BPD or ASPD, but reflects a shared common pathway (56). These findings support transdiagnostic models of ASPD, BPD, substance use, and related behavioral disinhibition, in line with externalizing spectrum conceptualizations. Taken together with other findings on the structure of PD and SUD (e.g., (46,54)), research points to possible shared pathology as well as unique variance underlying the complexities of PD-SUD co-occurrence.

## Why do people with personality disorders use substances?

At the most basic level, SUD development requires substance use and, thus, it is important to understand what factors lead people with PDs to use substances and whether those factors differ in strength or form from people without PDs. Unfortunately, little research has examined how or why people with PDs use substances. Some research has explored the role of emotion dysregulation in substance use among people with BPD, who may be particularly likely to use substances to alleviate negative affect. Although several motives for use have been highlighted in the literature (e.g., enhancement, social, conformity), coping motives, in particular, have been linked to substance use among those with BPD features (58–60). These coping motives may mediate the association of BPD pathology with SUDs (61,62). For example, a cross-sectional study found that BPD symptoms were indirectly related to self-reported alcohol-related problems via coping and enhancement drinking motives (63).

A better understanding of how substance use leads to use disorder requires examining how and why people with PDs use substances in their everyday lives. Research using ecological momentary assessment (EMA) has found that drinking motives moderate momentary associations between affect and alcohol use in BPD in daily life (64). Specifically, the interaction of greater self-reported drinking to cope and negative affect was associated with subsequent alcohol use, and the interaction of greater enhancement motives and positive affect was associated with starting a drink episode, but did not significantly predict whether an individual continued drinking. Among people who inject drugs, BPD was similarly associated with greater momentary negative affect intensity and greater instability of both negative and positive affect (65).

## Theoretical models of co-occurrence

Comorbidity and co-occurrence have long been topics of importance in the field of psychopathology (e.g., (66)). Explanations range from pure chance to symptom overlap, to one disorder generating symptoms of the other as an epiphenomenon, to distinct disorders sharing the same underlying liability, to one disorder causing the other. Models of PD-SUD co-occurrence have largely focused on explanations involving shared liabilities and/or causation.

## Developmental processes

Before briefly reviewing models of co-occurrence, we want to explicitly highlight the importance of understanding PD-SUD co-occurrence from a developmental lens, which can offer insights into not only the emergence of these problems during adolescence/young adulthood, but also their dynamic and transactional interplay throughout the lifespan. Adolescence and emerging adulthood are marked by increased substance use and elevated

impulsivity, sensation-seeking, and neuroticism (1), thus being critical periods for the development of co-occurrence. Early traumatic or stressful experiences and other family factors may also play important roles in the development of both PDs and SUDs. Individuals with family history of drug use and SUDs may have increased exposure to early life adversity and increased antisocial tendencies (67,68), and conversely, those with family history of BPD and ASPD are at higher risk of SUDs and early adolescent onset drug use (69,70). Adulthood antisocial and impulsive traits may then further mediate the effects of childhood stressful events on adulthood SUDs (71). These complex mechanisms highlight the dynamic and transactional nature of PD-SUD co-occurrence, emphasizing that models described below are not mutually exclusive, and co-occurrence likely involves many different factors.

### Shared etiology models

Shared etiology models, such as the spectrum model, suggest that SUDs and PDs have a shared vulnerability rising from common etiological factors (1,72,73). For example, that trait impulsivity/disinhibition serves as a risk factor for both BPD/ASPD and SUD (1,72,73). As a core feature in both SUDs and many PDs, impulsivity may be higher in people with BPD-SUD comorbidity than with either disorder alone (73,74), although other studies have not found this (75,76). Personality pathology and substance use may represent different manifestations of shared underlying impulsivity and disinhibition (73). In addition to influencing substance use onset, impulsivity can contribute to its continued use and overuse, particularly in the context of more stress relief from substances for those with higher disinhibition (73,77). A longitudinal study found that changes in early adolescent impulsivity predicted late adolescent ASPD and AUD, mediated by changes in mid-adolescent alcohol use and conduct problems (78). These findings are consistent with the behavioral disinhibition model, which posits that, while early vulnerability toward impulsivity underlies the co-emergence of antisocial behavior and alcohol use in early/mid adolescence, the subsequent development of these problem behaviors, rather than impulsivity itself, predicts the emergence of ASPD and AUD in late adolescence (78).

Shared etiology models are supported by the literature on the externalizing spectrum, which highlights common externalizing mechanisms underlying PD-SUD co-occurrence. Several genetics studies have identified the same genes underpinning both personality pathology and substance use (79,80). SUDs are also among the most common psychiatric diagnoses in probands/relatives of those with BPD (81). Moreover, individual differences in BPD and ASPD traits appeared as the strongest phenotypic and genetic correlates of lifetime use and problematic use of cannabis (29). In addition to common traits, other mechanisms have been proposed to underpin both personality pathology and substance use. Meta-analytic findings show that dysfunctions in social processing and distress tolerance may be core transdiagnostic phenotypes of BPD and problematic substance use, including SUD (82,83).

### Causal models

Causal models posit that existing PDs lead to the onset of SUDs or vice versa (84). Similar to shared etiology models, these models may also highlight the role of traits, such as impulsivity, novelty-seeking, and neuroticism, that contribute to both disorders. However,

these models propose distinct disorders and that one disorder either causes or exacerbates the other.

The predisposition/vulnerability model proposes that existing personality pathology elicits environmental consequences (e.g., interpersonal conflict) that provide an impetus for substance use and SUD development (1). Presence of a PD is an established risk factor for substance use and SUDs in cross-sectional assessments, with some longitudinal studies supporting this association (5,10,13). For example, a quasi-longitudinal latent class analysis in a community-based twin sample showed that ASPD increased persistent polydrug use risk (38). Moreover, a longitudinal study with 1,354 juvenile offenders showed ASPD diagnosis and BPD symptom severity predicted increased drug use variety, with sensation-seeking mediating this relationship in BPD, and self-control and deviant peer association in ASPD (85,86). ASPD also increased risk for opioid use, with self-control and moral disengagement identified as important mechanisms (87). Those with higher levels of certain personality traits may also select into high-risk environments for substance use. For instance, the traits of low constraint and negative emotionality in adolescence may predict selection into negative social contexts during early adulthood, which can then reinforce those traits and associated AUD symptoms through young adulthood (88). Moreover, increased risk of violence exposure may help explain the effect of BPD symptoms on increased opioid use frequency (89).

With the emergence of personality pathology in childhood and onset of PDs in adolescence/early adulthood, causal theories have focused on PDs leading to substance use, rather than the reverse. However, a longitudinal study of 4,576 individuals suggests early drug use may be a precursor to delinquency and crime via impulsivity (90). Similarly, early use of alcohol, cigarettes, and cannabis in adolescence has been associated with higher risk of adult ASPD onset (91). In the complication model, an existing Axis I disorder is theorized to complicate or “scar” one’s personality (84). Indeed, the presence of a comorbid SUD tends to exacerbate existing PD pathology, and using substances can influence subsequent personality changes (1,72).

## Clinical outcomes and implications

### Prognosis, course, and treatment

PD-SUD co-occurrence tends to lead to poorer prognoses than either condition alone, with higher severity and chronicity of both disorders and unfavorable treatment outcomes (1–3). SUDs exacerbate PD symptomatology and contribute to its chronicity (11). The reverse is also true – ASPD and BPD are associated with persistence of alcohol dependence (92), and the severity of personality pathology is one of the main risk factors for concurrent SUDs in AUD (23). Despite this exacerbation, rates of SUD among BPD patients, similar to the general population, tend to decrease over time (93). Alcohol outcomes and psychosocial functioning can also improve for those with co-occurring AUD-ASPD who stay in treatment, although attrition is high (1). Specialized treatments for comorbid PD-SUD are thus critically needed. Several psychotherapies for PD-SUD co-occurrence have demonstrated some effectiveness, including Dialectical Behavior Therapy for SUD, Dynamic Deconstructive Psychotherapy, and Dual-Focus Schema Therapy, which all use



systematic and integrative approaches of treating PD simultaneously with SUD ((1,3,94) for detailed reviews). Nevertheless, further research is needed.

### **Self-harm and suicide**

The increased risk of self-harm and suicide are two particularly notable negative outcomes in PD-SUD co-occurrence and, thus, often a focus of clinical intervention. Those with co-occurring PD-SUD in Taiwan had a risk of suicide 74 times higher than the general population (8), while U.S. veterans with comorbid PD-SUD were 13.5 times more likely to have a lifetime suicide attempt compared to the healthy group, and a higher rate of lifetime suicide history than those with SUD alone (27% vs 5%) (95). ASPD is associated with suicide the Epidemiologic Catchment Area study (96) and in SUD treatment samples (97). BPD is itself strongly linked with self-harm and suicidal behavior (98), with recent evidence of compounded risk for those with SUD and BPD (99).

The interactive effects of substance use, self-harm, and PD are important to understand from a developmental lens. Recent engagement in both risky alcohol use and self-injurious behavior were longitudinal predictors of BPD symptoms in adolescence (100). Moreover, once adolescents engaged in excessive alcohol use, the risk for BPD symptoms increased regardless of later terminating use, suggesting symptoms may often shift from one impulsive behavior to another (e.g., alcohol use to self-harm). Transdiagnostic factors, particularly high impulsivity and low distress tolerance, have been proposed to explain the high incidence of suicide and self-harm among those with comorbid PD-SUD (83). More empirical and clinical focus is needed on interventions for suicide/self-harm in PD-SUD co-occurrence, with transdiagnostic mechanisms offering particularly important treatment targets.

### **Conclusions and Future Directions**

Prevalence estimates of PD-SUD co-occurrence are generally high, with significant heterogeneity across different types of PDs, SUDs, and demographic factors. Research should continue exploring personality traits and dimensional models of PD, substances besides alcohol, and multiple comorbid SUDs. Several models have been proposed for PD-SUD co-occurrence, with some (e.g., shared etiology and predisposition models) receiving more empirical attention and support than others. Nevertheless, the complexities underlying PD-SUD co-occurrence suggest that models are not mutually exclusive and multiple mechanisms are at play. Key temperamental vulnerabilities (e.g., impulsivity, emotion dysregulation) together with environmental factors (e.g., traumatic experiences) may predispose individuals to a higher risk of both PDs and SUDs (i.e., shared etiology model), and the earlier onset of PDs may further contribute to SUD development (i.e., predisposition/vulnerability model). For some individuals, substance use becomes problematic and develops into SUDs, which in turn can make PD symptoms more severe or chronic (i.e., complication model). The bidirectionality inherent in PD-SUD co-occurrence warrants research testing dynamic, reciprocal effects rather than unidirectional effects, which requires intensive longitudinal studies.

The assessment of mechanisms underlying PD-SUD co-occurrence has focused on broad/heterogeneous constructs (e.g., impulsivity, emotion regulation). Studying more nuanced variables is needed to improve our understanding of PD-SUD development and maintenance. More rigorous methodology (e.g., EMA) can further aid in these efforts. Research on PD-SUD co-occurrence has also largely focused on BPD and ASPD, yet more emphasis on other PDs and co-occurring PDs could help in further disentangling these mechanisms. Moreover, significant issues in the measurement and diagnosis of PDs and SUDs are important to consider. The ongoing shift towards dimensional conceptualization of psychopathology, including personality and substance use pathology, may aid in a more nuanced understanding of the PD-SUD relationship. As research has uncovered both common and distinct underlying components, it is important to balance broad transdiagnostic conceptualizations with retaining enough heterogeneity and specificity for targeted assessment and treatment approaches. Both empirical and clinical efforts warrant continued improvements in tailoring screening, prevention, and treatment for the complex and heterogeneous of PD-SUD co-occurrence.

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