# Antisocial Personality Disorder and Substance Use Disorders: A Continuing Education Manual for Psychologists on Comorbidity, Clinical Characterization, and Treatment Implications

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## Title Page

**Antisocial Personality Disorder and Substance Use Disorders: A Continuing Education Manual for Psychologists on Comorbidity, Clinical Characterization, and Treatment Implications**

A Continuing Education Reading Manual for Licensed Psychologists and Graduate-Level Clinicians

Prepared for Continuing Education in Clinical, Counseling, and Health Psychology

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## Abstract

Antisocial personality disorder (ASPD) and substance use disorders (SUDs) constitute one of the most consequential comorbidity dyads encountered in contemporary clinical practice. Their frequent co-occurrence has been documented across epidemiological surveys, treatment-seeking populations, forensic samples, and inpatient psychiatric services, yet the clinical, mechanistic, and phenomenological texture of the ASPD–SUD relationship remains incompletely characterized. This continuing education manual integrates contemporary empirical evidence, most notably the large-scale Yale-Penn analysis reported by Low and colleagues (2024) and the cross-sectional clinical study of adults with alcohol dependence syndrome comorbid with ASPD reported by Jarcuskova and colleagues (2024), to provide licensed psychologists and graduate-level clinicians with an authoritative, publication-quality reference on ASPD–SUD comorbidity. The manual synthesizes findings across substance

classes, examining associations between ASPD and diagnoses, severity thresholds, and individual diagnostic criteria of alcohol, cannabis, cocaine, opioid, and tobacco use disorders. It explores shared etiological pathways involving behavioral disinhibition, impulsivity, aggressivity, and dysregulated reward processing, and considers the developmental scaffolding of these conditions in adverse childhood experiences, family psychopathology, and neurobiological vulnerability. Cognitive correlates, sleep disturbance, affective dysregulation, and traumatization patterns among individuals with dual diagnoses are examined in depth. The manual also addresses methodological considerations, the limits of generalizability inherent in ascertainment-enriched samples, and the clinical implications of subtyping ASPD–SUD presentations for personalized intervention. Throughout, the emphasis is on cultivating conceptual sophistication, diagnostic precision, and therapeutic humility when working with a population marked by high burden, treatment complexity, and persistent stigma.

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## 1. Introduction

Antisocial personality disorder occupies a distinctive and, in many respects, uncomfortable position within the taxonomy of psychiatric conditions. Defined by the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5) as a pervasive pattern of disregard for and violation of the rights of others—manifesting through deceitfulness, impulsivity, irritability, aggressiveness, reckless disregard for the safety of self or others, consistent irresponsibility, and a lack of remorse—ASPD sits at the intersection of clinical psychopathology, forensic concern, and public health burden (American Psychiatric Association, 2013, as cited in Low et al., 2024; Jarcuskova et al., 2024). Its diagnostic architecture is unusual within the personality disorder taxonomy: unlike other personality disorders, it requires evidence of conduct disorder with onset before the age of 15 years, effectively situating ASPD as a developmentally continuous disturbance whose adult presentation is inseparable from its childhood antecedents (Jarcuskova et al., 2024).

Alongside this developmental grounding, ASPD is remarkable for the density of its comorbidities. Substance use disorders are, by a considerable margin, its most consistent psychiatric companions. Whereas nationally representative surveys estimate the prevalence of ASPD in the general adult population at approximately 3.6%, samples ascertained through

substance use treatment settings report prevalence figures ranging from 23.5% to 81.4%, depending on the substance in question and the population sampled (Low et al., 2024). Jarcuskova and colleagues (2024) observed a 35% prevalence of comorbid ASPD within a consecutive series of adults hospitalized with alcohol dependence syndrome (ADS), a figure that reinforces earlier findings from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), in which individuals with any personality disorder demonstrated a 42% rate of concurrent alcohol dependence and those with ASPD in particular were seven to eight times more likely than the general population to meet criteria for alcohol dependence.

For licensed psychologists, these figures are not merely epidemiological curiosities. They speak to the near-certainty that clinicians working in substance use treatment, forensic evaluation, community mental health, and integrated primary care will regularly encounter individuals whose antisocial features shape the presentation, trajectory, and treatment response of their substance use pathology—and vice versa. The clinical stakes are correspondingly high: comorbid ASPD–SUD presentations are associated with earlier onset of substance use, greater severity, more frequent hospitalizations, poorer treatment adherence, higher rates of relapse and drop-out, greater interpersonal and occupational disruption, and heightened risk of violence, incarceration, and premature mortality (Jarcuskova et al., 2024; Low et al., 2024).

Historically, however, the field has treated ASPD and SUD as somewhat parallel domains—linked by comorbidity data, certainly, but rarely examined at the granular level of diagnostic criteria, severity thresholds, or substance-specific patterns of association. The recent work of Low and colleagues (2024) in the Yale-Penn cohort represents a substantive advance in this regard, offering the first systematic examination of ASPD associations across DSM-5 SUD diagnoses, severity levels, and individual criteria for alcohol, cannabis, cocaine, opioid, and tobacco use disorders. Complementing this molecular-level analysis, Jarcuskova and colleagues (2024) provide a rich clinical portrait of the dual-diagnosis presentation, integrating cognitive assessment, affective symptomatology, sleep quality, traumatization history, and neuroimaging findings in a hospitalized alcohol-dependent population.

This continuing education manual integrates these two contemporary evidence streams into a unified scholarly narrative intended for practicing psychologists. Its aims are threefold. First, it seeks to deepen conceptual understanding of the mechanisms that bind ASPD to substance pathology, moving beyond the observation of comorbidity to the interrogation of shared etiological, developmental, and neurobiological substrates. Second, it aims to sharpen clinical

discrimination by examining how ASPD associates differently with different substances and different diagnostic criteria, a level of specificity with direct implications for assessment and formulation. Third, it invites critical reflection on the methodological, conceptual, and therapeutic limitations that shape our current understanding of ASPD–SUD comorbidity, and considers the pathways forward for research and practice.

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## 2. Historical and Theoretical Foundations

The conceptual lineage of ASPD extends through more than a century of psychiatric thought, from early notions of "moral insanity" and "psychopathic personality" through the mid-twentieth century construct of sociopathy and into the operationalized diagnostic criteria of the DSM series. Each iteration has reflected the intellectual currents of its time: the moralism of Victorian-era psychiatry, the emerging behavioral and social psychology of the postwar period, and the criterion-based operationalism of the DSM-III and its successors. What has remained constant, across these shifting frameworks, is the recognition that a subset of individuals exhibit a persistent, developmentally rooted pattern of behavior characterized by disregard for social norms, insensitivity to the rights and welfare of others, and a resistance to the socializing pressures that ordinarily shape prosocial conduct.

The linkage between antisociality and substance misuse is equally longstanding. Nineteenth-century clinical observers noted the tendency of "habitual drunkards" to display features that would today be recognized as antisocial, and mid-twentieth-century typological work on alcoholism—most notably Cloninger's distinction between Type I and Type II alcoholism—explicitly incorporated antisocial features as defining characteristics of one alcoholism subtype (Cloninger, 1987, as cited in Low et al., 2024). Cloninger's framework proposed that Type II alcoholism, characterized by early onset, male predominance, and antisocial behavior, was associated with a personality profile marked by high novelty seeking, low harm avoidance, and low reward dependence—a constellation that maps closely onto contemporary descriptions of ASPD. This early typological work anticipated the more recent recognition that ASPD and SUDs share a common personality substrate, one that Low and colleagues (2024) describe as encompassing "behavioral disinhibition," loosely composed of sensation seeking, impulsivity, and aggressivity.

Theoretical understanding advanced substantially with the introduction of the externalizing spectrum framework, most prominently articulated by Krueger and colleagues (Krueger, 1999; Krueger et al., 2007, as cited in Low et al., 2024). This framework proposes that ASPD, SUDs, conduct disorder, and adult antisocial behavior are not independent conditions but rather share a latent externalizing dimension that reflects a common vulnerability to disinhibited, outward-directed behavioral dysregulation. Empirical support for this model has been robust: factor-analytic studies consistently identify a latent externalizing factor onto which antisocial and substance-related pathology loads, and behavioral-genetic research has documented substantial shared heritability across these conditions (Krueger et al., 2007, as cited in Low et al., 2024; Witkiewitz et al., 2013, as cited in Low et al., 2024).

The externalizing framework carries important theoretical implications. It suggests that ASPD and SUDs are not simply co-occurring by chance or through unidirectional causation, but rather represent alternative phenotypic expressions of a shared underlying diathesis. This reframing invites clinicians to think in terms of transdiagnostic vulnerabilities—disinhibition, low harm avoidance, aggressivity, and reward-system dysregulation—rather than in terms of discrete disease entities. It also foreshadows contemporary dimensional approaches to psychopathology, including the Hierarchical Taxonomy of Psychopathology (HiTOP), which situate externalizing pathology within a broader nomological network of behavioral and personality dimensions.

Complementing the externalizing framework, developmental psychopathology traditions have emphasized the temporal continuity of antisocial features across the lifespan. The requirement that ASPD diagnosis be preceded by conduct disorder with onset before age 15 reflects the field's recognition that adult antisocial behavior rarely arises de novo. Rather, it typically emerges from a developmental cascade in which early temperamental vulnerabilities—difficult temperament, callous-unemotional traits, executive dysfunction—interact with adverse environmental conditions to produce a trajectory of escalating antisocial behavior and, frequently, early substance experimentation and dependence (Fairchild et al., 2019, as cited in Low et al., 2024; Jarcuskova et al., 2024).

The Slovak clinical context in which Jarcuskova and colleagues (2024) conducted their investigation offers a useful reminder that cultural factors shape both the expression and the treatment of these conditions. In Slovakia, alcohol use is socially normalized and even actively promoted, with 75% of adults reporting occasional or regular consumption. Alcoholism is the most common psychiatric diagnosis in the country, accounting for 26.5% of all psychiatric

hospitalizations. The cultural embeddedness of alcohol use in this and many other societies means that the pathways into alcohol dependence—and, by extension, into the ASPD–ADS dual diagnosis—are shaped by normative pressures that clinicians must consider when formulating cases and designing interventions.

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## 3. Conceptual Models and Mechanisms

Several complementary conceptual models help clinicians understand how ASPD and SUDs come to co-occur so frequently, and why their comorbidity carries such significant clinical implications. These models are not mutually exclusive; rather, each illuminates a distinct facet of a multiply determined relationship.

### *3.1 The Shared Vulnerability Model*

Perhaps the most influential contemporary model posits that ASPD and SUDs share underlying etiological factors, both genetic and environmental. Low and colleagues (2024) argue that the strong diagnosis-level associations they observed between ASPD and multiple SUDs, together with the fact that these associations were statistically stronger than associations with SUD severity, suggest that "ASPD–SUD comorbidity may be more related to the shared mechanisms between these pathologic conditions rather than ASPD association with increased risk symptoms across the substance misuse spectrum." In other words, the two conditions may co-occur not primarily because ASPD amplifies the severity of substance pathology but because both conditions arise from a common vulnerability that predisposes affected individuals to both antisocial behavior and substance misuse.

The shared vulnerability model draws support from behavioral-genetic research indicating substantial shared heritability between antisocial behavior and substance use pathology. Rhee and Waldman's (2002) meta-analysis, cited by Jarcuskova and colleagues (2024), documented additive and non-additive genetic contributions to antisocial behavior accounting for 32% and 9% of variance, respectively, with 16% attributable to shared environment and 43% to individual-specific environmental influences. More recent molecular-genetic work has begun to identify specific loci implicated in shared risk. Li and colleagues (2023), as cited by Jarcuskova and colleagues (2024), reported a genome-wide association study identifying a

novel signal on chromosome 15q26.1 near *SLCO3A1* that may contribute to genetic risk for both ADS and ASPD.

### *3.2 The Behavioral Disinhibition Model*

Closely related to the shared vulnerability model, the behavioral disinhibition model specifies the phenotypic pathway through which shared vulnerability is expressed. Gorenstein and Newman's (1980) foundational work on disinhibitory psychopathology, as cited by Low and colleagues (2024), proposed that a range of externalizing conditions—including antisocial behavior and substance abuse—reflect a common failure of behavioral inhibition, particularly in the face of appetitive stimuli. Subsequent research has refined this construct, identifying sensation seeking, impulsivity, and aggressivity as its principal components (Hawkins et al., 1992; Tarter et al., 2003, as cited in Low et al., 2024).

Behavioral disinhibition, so conceived, functions as a transdiagnostic mechanism that predisposes individuals to a wide range of externalizing outcomes. In the context of substance use, it may manifest as an inability to inhibit the pursuit of immediate substance-related reward despite adverse consequences—a description that maps closely onto DSM-5 SUD criteria such as continued use despite problems, use in hazardous situations, and failure to fulfill role obligations. Low and colleagues (2024) note that AUD in particular has been linked to low harm avoidance, high novelty seeking, and low reward dependence—characteristics shared with ASPD—and they hypothesize that these traits may extend across substances, accounting for the transdiagnostic association of ASPD with multiple SUDs.

### *3.3 Bidirectional Causal Pathways*

A third class of models focuses on causal pathways that operate in both directions between ASPD and SUDs. Jarcuskova and colleagues (2024) articulate several such pathways. On one hand, an existing personality disorder may elicit environmental responses—interpersonal conflict, occupational instability, social marginalization—that increase the risk of substance use. ASPD may also directly compromise the individual's capacity to inhibit habitual substance use, incapacitating "one's ability to ignore the immediate reward offered by alcohol" and jeopardizing the capacity to abstain despite significant psychological and physical costs. On the other hand, substance use itself may induce neuroadaptive changes—increased impulsivity, heightened negative emotionality, altered reward processing—that intensify

antisocial features. Early adolescent substance use may generate aggression and antisocial behavior that consolidates over time into a full ASPD presentation, particularly when reinforced by peer group dynamics, family dysfunction, and educational failure.

This bidirectional framing helps explain the vicious-cycle quality that many clinicians observe in dual-diagnosis presentations. Antisocial features drive substance use; substance use exacerbates antisocial features; the resulting behavioral pattern generates environmental adversity, which in turn reinforces both dimensions of the pathology.

### *3.4 Neurobiological Substrates*

Contemporary neuroscience has begun to specify the neural substrates through which shared vulnerability and reciprocal causation may operate. Dugré and Potvin's (2021) meta-analysis of resting-state functional connectivity studies in individuals with antisocial behaviors, cited by Jarcuskova and colleagues (2024), identified alterations across regions implicated in ventral and dorsal attention networks, including the anterior midcingulate cortex, pre-supplementary motor area, superior parietal lobule, premotor cortex, and cuneus. Connectivity deficits have also been documented in the amygdala, middle cingulate cortex, ventral posterior cingulate cortex-precuneus, ventromedial and dorsomedial prefrontal cortex, and superior parietal lobule.

Murray, Waller, and Hyde (2018), as cited by Jarcuskova and colleagues (2024), documented altered neural reactivity in ASPD during reward and loss processing, with impulsive-antisocial traits specifically associated with hypersensitivity in the ventral striatum and prefrontal cortex during reward anticipation. These findings converge with reward-processing accounts of addiction, suggesting a shared neurobiological locus for the reward-system dysregulation that characterizes both conditions. Additional evidence implicates serotonergic and endocannabinoid signaling systems, as well as weaker cortico-striatal connectivity associated with risk-taking and violence proclivity (Kolla et al., 2023, as cited in Jarcuskova et al., 2024).

### *3.5 Developmental and Environmental Models*

Finally, developmental models emphasize the role of adverse childhood environments in shaping the trajectory toward comorbid ASPD and SUD. Jarcuskova and colleagues (2024) document elevated rates of adverse childhood experiences (ACEs)—including physical,

emotional, and sexual abuse; parental substance use; witnessing domestic violence; parental incarceration; and emotional and physical neglect—among individuals with comorbid ADS and ASPD. The prenatal, perinatal, and postnatal risk factors identified in this literature include maternal smoking and substance use during pregnancy, obstetric complications, parental psychopathology, childhood maltreatment, and bullying. Together, these environmental exposures interact with genetic vulnerability to produce the developmental cascade that culminates in dual pathology.

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# 4. Empirical Findings Across Studies

The empirical evidence base on ASPD–SUD comorbidity spans epidemiological surveys, clinical samples, and, most recently, deeply phenotyped research cohorts that permit granular analysis of association patterns. The following synthesis integrates findings from these diverse sources, with particular attention to the contributions of Low and colleagues (2024) and Jarcuskova and colleagues (2024).

## *4.1 Prevalence Estimates Across Settings*

Prevalence estimates vary substantially by setting, reflecting the different populations sampled. In general population surveys, ASPD prevalence hovers near 3.6%, with substantially elevated rates in substance-using populations. Grant and colleagues (2004), analyzing the 2001–2002 wave of NESARC (N = 43,093), found that individuals with current AUD were 4.8 times more likely to meet ASPD criteria, and those with current drug use disorders were 11.8 times more likely. Gonzalez and colleagues (2019), studying patients seeking treatment at mental health or addiction centers in Spain, documented lifetime ASPD prevalence rates of 76.5% among individuals with AUD, 81.4% among those with cocaine use disorder, 45.1% for opiates, 67.6% for cannabis, and 23.5% for sedatives (as cited in Low et al., 2024).

Jarcuskova and colleagues (2024) observed a 35% rate of comorbid ASPD in their sample of 80 hospitalized adults with ADS in Slovakia—a figure lower than some Western European estimates but consistent with the broader literature documenting substantial ASPD burden in alcohol-dependent populations. Windle (1999), examining 740 inpatients admitted for

alcoholism treatment, likewise reported elevated rates of ASPD and familial alcoholism in dual-diagnosis cases (as cited in Jarcuskova et al., 2024).

### *4.2 ASPD Associations Across Substance Classes*

The Yale-Penn analysis by Low and colleagues (2024) provides the most detailed contemporary examination of how ASPD associates with different substance classes. Working with 1,660 ASPD cases matched to 6,640 controls on sex, age, and racial/ethnic background, the authors examined associations between ASPD and DSM-5 diagnoses of AUD, cannabis use disorder (CanUD), cocaine use disorder (CocUD), opioid use disorder (OUD), and tobacco use disorder (TUD). In multivariable analyses, ASPD was significantly associated—after Bonferroni correction—with AUD (OR = 1.89), CanUD (OR = 2.13), and TUD (OR = 1.50). Associations with CocUD and OUD were positive but did not survive multiple-testing correction, a finding that the authors attribute to the lower population prevalence of these conditions relative to AUD, CanUD, and TUD.

**Table 1** synthesizes the diagnosis-level associations between ASPD and each SUD as reported by Low and colleagues (2024).

| Substance Use Disorder | Odds Ratio (Diagnosis) | Odds Ratio (Severity) | Survived Multiple-Testing Correction |
|---|---|---|---|
| Alcohol Use Disorder | 1.89 | 1.25 | Yes |
| Cannabis Use Disorder | 2.13 | 1.32 | Yes |
| Cocaine Use Disorder | Positive, not significant | Positive, not significant | No |
| Opioid Use Disorder | Positive, not significant | Positive, not significant | No |
| Tobacco Use Disorder | 1.50 | 1.21 | Yes |

Notably, diagnosis-level associations were consistently stronger than severity-level associations, a pattern that led Low and colleagues (2024) to conclude that "ASPD–SUD comorbidity may be more related to the shared mechanisms between these pathologic conditions rather than ASPD association with increased risk symptoms across the substance misuse spectrum."

### *4.3 Diagnostic Criteria-Level Associations*

A particular strength of the Low et al. (2024) analysis was its examination of individual DSM-5 diagnostic criteria. Across all five SUDs examined, the "hazardous use" criterion showed the strongest and most consistent association with ASPD, with odds ratios ranging from 1.37 (CanUD) to 1.88 (TUD). Additional criterion-level associations included "social problems" for AUD and CanUD, "neglected roles" for CanUD and CocUD, "physical problems" for TUD, and, notably, an inverse association between "attempts to quit" and ASPD for both AUD (OR = 0.76) and CocUD (OR = 0.57). The latter finding suggests that individuals with ASPD may be less likely to attempt to reduce or cease their substance use—a pattern with obvious clinical significance for treatment engagement.

When criteria were entered into a multi-SUD omnibus model to account for polysubstance comorbidity, only three associations survived multiple-testing correction: CanUD severity, CocUD "attempts to quit" (inversely), and TUD "hazardous use." After further controlling for sex-interaction effects, only the inverse association between CocUD "attempts to quit" and ASPD remained significant (OR = 0.57, 95% CI = 0.36–0.89). This finding underscores the robustness of the observation that individuals with ASPD are less likely to make attempts to quit cocaine use, even after accounting for a range of covariates and interaction terms.

### *4.4 Clinical Characteristics in the Dual-Diagnosis Presentation*

Jarcuskova and colleagues (2024) provide a complementary clinical portrait of the ASPD–ADS dual-diagnosis presentation. In their sample, individuals with comorbid ASPD were significantly younger, began drinking at earlier ages, had more prior hospitalizations, and scored higher on the Alcohol Use Disorders Identification Test (AUDIT). They also demonstrated lower educational attainment, higher unemployment rates, and lower marriage rates. Family histories were more likely to include alcohol dependence, incarceration, and mental illness, and rates of traumatic childhood experiences were markedly elevated.

Depression, anxiety, and stress, as measured by the Depression Anxiety Stress Scales–42 (DASS-42), were all significantly correlated with ASPD in bivariate analyses, though these associations attenuated in multivariable models—likely reflecting the modest sample size. Sleep quality, assessed by the Pittsburgh Sleep Quality Index (PSQI), showed a trend-level association ($p = 0.058$) with ASPD status. On the Montreal Cognitive Assessment (MoCA),

participants with comorbid ASPD scored significantly lower and demonstrated particular difficulty on attention- and language-related subtests.

**Table 2** summarizes the demographic, clinical, and psychometric profile of individuals with comorbid ASPD relative to those with ADS alone, as reported by Jarcuskova and colleagues (2024).

| Characteristic | ADS Only | ADS + ASPD | Direction of Difference |
|---|---|---|---|
| Age (median) | 53.5 years | 45 years | Younger in ASPD |
| Age at first hospitalization | 46 years | 38.5 years | Earlier in ASPD |
| Number of hospitalizations | 2.5 | 6 | Higher in ASPD |
| Educational attainment | Higher | Lower | Lower in ASPD |
| Employment | Higher | Lower | Lower in ASPD |
| DASS-42 Stress score | 10.5 | 20.5 | Higher in ASPD |
| ACE-IQ Trauma score | 3.5 | 6 | Higher in ASPD |
| MoCA score | 23 | 19 | Lower in ASPD |
| Addicted family member | 26.25% | 31.25% | Higher in ASPD |
| Mental illness in household | 11.25% | 17.5% | Higher in ASPD |
| Incarcerated household member | 5% | 12.5% | Higher in ASPD |

### *4.5 Convergence and Divergence Across Studies*

The two contemporary studies converge on several key findings: ASPD is strongly and consistently associated with SUD diagnoses across substance classes; the comorbid presentation is characterized by earlier onset, greater severity, and higher hospitalization rates; and shared vulnerability factors—including family psychopathology, adverse childhood experiences, and behavioral disinhibition—likely underlie the observed association patterns. Divergences reflect the different aims and populations of the two studies: Low and colleagues

(2024) focused on molecular-level analysis of association patterns in a large, ascertainment-enriched sample, while Jarcuskova and colleagues (2024) provided rich clinical phenotyping in a smaller hospitalized sample. Together, they offer a multidimensional view of ASPD–SUD comorbidity that neither could provide alone.

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## 5. Psychological Pathways and Stress Responses

Understanding the psychological pathways that connect ASPD to substance use pathology requires attention to the transactional dynamics through which personality traits, environmental stressors, and coping strategies interact over time.

### *5.1 Impulsivity, Sensation Seeking, and the Externalizing Phenotype*

At the core of the ASPD–SUD relationship lies a shared personality substrate characterized by high impulsivity, high novelty and sensation seeking, low harm avoidance, and low reward dependence (Cloninger, 1987, as cited in Low et al., 2024; Ruiz et al., 2008, as cited in Jarcuskova et al., 2024). This constellation predisposes affected individuals to seek out reinforcing but risky experiences, including substance use, and to persist in these behaviors despite escalating consequences. Impulsivity, in particular, has been extensively documented as a predictor of both antisocial behavior and problematic substance use across the lifespan (Sargeant et al., 2012, as cited in Low et al., 2024).

Developmental research suggests that individual differences in the trajectory of impulsivity across adolescence and young adulthood are meaningfully related to substance use trajectories. Individuals whose impulsivity declines steeply across this developmental window tend to show corresponding declines in problematic alcohol use, whereas those whose impulsivity remains high—or increases—show stable or escalating alcohol use patterns (Jarcuskova et al., 2024). This developmental patterning helps to explain why the ASPD–SUD dyad, once established, tends to be chronic and difficult to treat.

### *5.2 Traumatization and the Stress Response*

Adverse childhood experiences constitute a second critical psychological pathway. Jarcuskova and colleagues (2024) documented substantially elevated rates of adverse childhood experiences among individuals with comorbid ASPD and ADS: 75% reported emotional abuse, 89% reported alcohol or drug abuse in the household, 75% reported witnessing violence between family members, 43% reported physical abuse, 43% reported physical neglect, and 32% reported bullying. These figures underscore the extent to which comorbid ASPD–SUD presentations emerge from environments marked by chronic adversity.

The clinical implications of this traumatic patterning are substantial. As DeLisi and colleagues (2019), cited by Jarcuskova and colleagues (2024), have argued, trauma provides a template that shapes both the perpetration and the reception of antisocial behavior across generations. Individuals who experience severe childhood trauma may develop insensitivity to stressful situations, presenting with blunted emotional reactivity alongside impulsive aggression. This numbed affect—alongside a compromised hypothalamic-pituitary-adrenal stress response system—may reduce the deterrent value of the aversive consequences that ordinarily inhibit antisocial behavior in healthier individuals.

The correlation between trauma exposure and ASPD traits appears to be robust, with exposure to high-betrayal trauma (i.e., trauma perpetrated by trusted others) especially predictive of adult antisocial features (Yalch et al., 2021, as cited in Jarcuskova et al., 2024). Fergusson and colleagues (2008), as cited by Jarcuskova and colleagues (2024), reported that individuals who had experienced childhood sexual abuse were two to four times more likely to meet criteria for ASPD in emerging adulthood, with similar patterns for physical abuse.

### *5.3 Affect Dysregulation and Self-Medication*

A third pathway involves the use of substances as a strategy for regulating affect. Although ASPD has traditionally been characterized by low neuroticism relative to other personality disorders and to SUDs, the co-occurrence of ASPD with mood and anxiety pathology is nonetheless common. Jarcuskova and colleagues (2024) observed that 68% of ASPD participants reported depression, 64% reported stress, and 79% reported anxiety—rates substantially higher than those observed among ADS participants without ASPD. Goldstein and colleagues (2017), cited by Jarcuskova and colleagues (2024), similarly documented that individuals with ASPD are four times more likely to experience a mood disorder and that up to

half may experience an anxiety disorder over the lifespan.

These affective symptoms may motivate substance use as a form of self-medication, a pathway that appears especially prominent in the context of anxiety-provoking events. The reinforcement value of alcohol as an anxiolytic, combined with the disinhibitory and impulsivity-enhancing effects of intoxication, creates a self-perpetuating cycle in which affective distress fuels substance use and substance use in turn exacerbates affective dysregulation.

### *5.4 Sleep Disturbance and Behavioral Regulation*

Sleep disturbance emerges as a further psychological pathway meriting clinical attention. Van Veen and colleagues (2017), as cited by Jarcuskova and colleagues (2024), documented poor sleep quality in 53.6% of patients with antisocial or borderline personality disorders, with 22.3% meeting criteria for severe chronic insomnia. In the Jarcuskova et al. (2024) sample, PSQI scores were elevated among individuals with comorbid ASPD, though this association reached only trend-level significance.

Sleep disturbance both reflects and exacerbates behavioral dysregulation. Individuals with poorer sleep quality report greater difficulty focusing, controlling their thoughts, and regulating impulsive behavior—symptoms that compound the disinhibitory features already characteristic of ASPD. In dual-diagnosis presentations, alcohol use itself further disrupts sleep architecture, generating a bidirectional feedback loop in which poor sleep drives substance use for its sedating effects and substance use in turn perpetuates sleep pathology.

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## 6. Mental Health Outcomes and Severity Spectrum

Comorbid ASPD substantially alters the presentation, trajectory, and severity of substance use pathology, generating a distinct clinical phenotype that warrants specific attention in assessment and treatment planning.

### *6.1 Earlier Onset and Accelerated Progression*

Individuals with comorbid ASPD begin substance use earlier in life and progress more rapidly through the stages of use, misuse, and dependence. Jarcuskova and colleagues (2024) documented significantly younger ages at first hospitalization among ASPD participants (38.5 years vs. 46 years in the ADS-only group), consistent with a broader literature demonstrating earlier onset of substance-related problems in individuals with antisocial features. This earlier onset is clinically significant not only because it reflects a more severe underlying phenotype but also because early-onset substance use is itself a robust predictor of long-term severity, treatment resistance, and mortality.

### *6.2 Severity and Treatment Utilization*

Beyond earlier onset, comorbid ASPD is associated with greater severity of substance-related pathology across multiple metrics. AUDIT scores were substantially higher in ASPD participants (median 33 vs. 22 in ADS-only), and the number of prior hospitalizations was more than doubled (median 6 vs. 2.5) (Jarcuskova et al., 2024). This pattern of elevated treatment utilization reflects both the greater severity of the underlying pathology and the poorer treatment response typical of dual-diagnosis cases.

Relapse rates in individuals with comorbid ASPD are notably elevated. More than half of individuals with alcohol dependence relapse within a year of treatment, with relapse risk particularly high among those with comorbid personality pathology. Multiple factors contribute to this elevated risk: marital instability, chronic traumatization, psychological distress, comorbid affective and anxiety pathology, and alterations in dopaminergic reward systems all combine to undermine sustained abstinence. Compounding these factors, individuals with ASPD frequently exhibit poor treatment adherence—reflecting the disorder's characteristic disregard for rules, authority, and long-term consequences—which further increases the likelihood of drop-out and relapse (Jarcuskova et al., 2024).

### *6.3 Cognitive Functioning*

Cognitive impairment represents an underappreciated dimension of the comorbid presentation. In the Jarcuskova et al. (2024) sample, 75% of ASPD participants demonstrated cognitive impairment on the MoCA, with particular difficulty on attention- and language-related subtests. Multivariable analyses confirmed that cognitive impairment was independently associated with ASPD status, with odds ratios ranging from approximately 3.6 to 6.2 across

models controlling for depression, family history, and other covariates.

These cognitive findings align with a broader literature documenting attentional, executive, decision-making, and verbal deficits in individuals with antisocial features. Chamberlain and colleagues (2016), cited by Jarcuskova and colleagues (2024), identified deficits in cognitive control, attentional processing, flexible responding, and planning among young adults with ASPD features. Verbal deficits and delayed language development are particularly well documented, with young offenders typically scoring approximately half a standard deviation lower on verbal than on performance-related domains of intelligence (Wallinius et al., 2019, as cited in Jarcuskova et al., 2024).

The neuroanatomical correlates of these cognitive deficits map onto regions belonging to ventral and dorsal attention networks, including the anterior midcingulate cortex, pre-supplementary motor area, superior parietal lobule, and premotor cortex (Dugré & Potvin, 2021, as cited in Jarcuskova et al., 2024). Alterations in these networks likely contribute to the attentional and executive difficulties observed in ASPD, though the causal direction—whether these alterations predispose to ASPD or arise from chronic substance use—remains incompletely resolved.

### *6.4 The Severity Spectrum: Mild, Moderate, and Severe*

Low and colleagues (2024) systematically examined the association between ASPD and DSM-5 SUD severity classifications (mild, moderate, and severe). Across AUD, CanUD, and TUD, ASPD was associated with greater severity, with odds ratios in the range of 1.21 to 1.32. However, as noted earlier, these severity-based associations were consistently weaker than the corresponding diagnosis-based associations, leading the authors to hypothesize that ASPD confers vulnerability to the categorical presence of substance pathology more than to its severity per se. This distinction carries important theoretical implications, suggesting that the shared vulnerability underlying ASPD–SUD comorbidity operates at the level of susceptibility to disorder rather than at the level of dose-response scaling.

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## 7. System-Level and Contextual Psychological Effects

The comorbid ASPD–SUD presentation does not exist in isolation; it is embedded within family systems, social networks, and broader cultural contexts that shape both its expression and its treatment.

## *7.1 Family Systems and Intergenerational Transmission*

Family psychopathology emerged as a robust correlate of ASPD in both contemporary studies. Jarcuskova and colleagues (2024) found that 89% of ASPD participants reported alcohol or drug abuse in the household of origin, compared with substantially lower rates in the ADS-only group. Rates of family incarceration and mental illness were similarly elevated. Multivariable analyses confirmed that the presence of an addicted family member and an incarcerated household member each independently predicted ASPD status, with odds ratios of 5.76 and 14.26, respectively.

These findings underscore the intergenerational transmission of externalizing pathology. Genetic vulnerability, modeled behavior, disrupted parenting, and chronic exposure to family conflict combine to produce a developmental context in which antisocial behavior and substance use are both normalized and reinforced. The presence of an incarcerated family member, in particular, may reflect a family environment in which extreme antisocial behavior is normative, providing a template for the developing child's own behavioral repertoire.

## *7.2 Socioeconomic and Occupational Consequences*

The socioeconomic footprint of comorbid ASPD–SUD is substantial. In the Jarcuskova et al. (2024) sample, participants with comorbid ASPD had significantly lower educational attainment and higher unemployment rates than those with ADS alone. These findings align with epidemiological data from NESARC-III (Goldstein et al., 2017, as cited in Jarcuskova et al., 2024) documenting that individuals with ASPD are disproportionately likely to have less than a college degree, lower family income, and unstable marital status.

The mechanisms underlying these socioeconomic consequences are multiple. Behavioral disinhibition and impulsivity interfere with academic and occupational functioning from an early age. School failure, in particular, is overrepresented in offenders and is closely linked to the development of aggressive antisocial behaviors. Occupational instability, in turn, generates chronic financial stress, exposure to criminogenic environments, and reduced

access to healthcare—all of which perpetuate both the antisocial and the substance-related pathology.

### *7.3 Sex Differences and Gender Considerations*

Sex differences shape the epidemiology, phenomenology, and clinical presentation of both ASPD and SUDs. ASPD is diagnosed substantially more frequently in men than in women, and the Yale-Penn sample analyzed by Low and colleagues (2024) reflected this pattern, with over 75% of participants being male. Despite careful matching on sex between ASPD cases and controls, Low and colleagues (2024) nonetheless observed that sex remained a significant covariate in multi-SUD criterion-level analyses, suggesting that symptom-level sex differences in SUD criteria exceed those observable at the diagnostic or severity level. The inclusion of sex-interaction terms in their models attenuated most sex effects but did not eliminate them, indicating that sex-specific pathways deserve further attention in future research and clinical practice.

Female presentations of comorbid ASPD–SUD may differ importantly from male presentations, with greater rates of internalizing comorbidity, distinct trauma histories, and different patterns of interpersonal disruption. Yalch and colleagues (2021), cited by Jarcuskova and colleagues (2024), reported that exposure to trauma at all levels of betrayal predicts ASPD traits for women, whereas only exposure to high-betrayal trauma predicts ASPD traits for men—a finding with implications for trauma-informed assessment in women presenting with dual pathology.

### *7.4 Cultural Context*

Cultural context shapes both the prevalence and the presentation of comorbid ASPD–SUD. The Slovak context studied by Jarcuskova and colleagues (2024) is characterized by high rates of alcohol consumption—75% of adults report occasional or regular use—and by social promotion of alcohol use as normative. In such contexts, the pathway from social drinking to alcohol dependence may be shortened and normalized, and the boundaries between culturally sanctioned use and clinically significant misuse may be blurred. Clinicians working across cultural contexts must remain attentive to these normative variations when formulating cases and designing interventions.

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## 8. Clinical Implications for Psychologists

The empirical evidence synthesized above carries substantial implications for clinical practice with individuals presenting with comorbid ASPD and SUDs.

### *8.1 Assessment and Case Formulation*

The high prevalence of ASPD in substance-using populations argues for routine screening for antisocial features in all clients presenting with SUDs, particularly those presenting with alcohol, cannabis, or tobacco use disorders. Structured diagnostic interviews such as the Structured Clinical Interview for DSM (SCID-II or its DSM-5 successor) offer the most rigorous approach, though briefer screening measures may be appropriate for initial assessment. Careful attention to childhood history is essential, given the requirement that conduct disorder features be documented as preceding age 15 for a valid ASPD diagnosis.

Assessment should extend beyond categorical diagnosis to include the specific SUD criteria most strongly associated with ASPD. The "hazardous use" criterion, which showed the most robust association with ASPD across substance classes in the Low et al. (2024) analysis, warrants particular clinical attention as a marker of externalizing vulnerability. The inverse association between ASPD and the "attempts to quit" criterion, particularly for cocaine use, has direct implications for treatment engagement: clinicians should anticipate that clients with ASPD may present with fewer prior quit attempts and less spontaneous motivation to change.

Assessment of adverse childhood experiences, using measures such as the Adverse Childhood Experiences International Questionnaire (ACE-IQ) employed by Jarcuskova and colleagues (2024), provides critical formulation-level information. Cognitive assessment, using instruments such as the MoCA, may identify attentional and language-based deficits that can shape treatment planning. Sleep quality assessment, using the PSQI or similar measures, addresses a frequently overlooked dimension of the comorbid presentation.

### *8.2 Treatment Considerations*

The treatment of comorbid ASPD–SUD presents substantial challenges. A recent meta-analysis of 24,801 subjects, cited by Jarcuskova and colleagues (2024), did not identify sufficient evidence to establish the efficacy of medication for ASPD itself. Psychological interventions have received similarly limited empirical support: a meta-analysis of 605 adults, cited by Jarcuskova and colleagues (2024), suggested that schema therapy, contingency management, and dialectical behavior therapy may be more effective than other approaches, but the certainty of the evidence remained low or very low.

In the absence of definitive treatment protocols, clinical practice must be guided by careful attention to the shared mechanisms underlying ASPD and SUDs. Interventions that target behavioral disinhibition, impulsivity, and reward-system dysregulation—including contingency management, cognitive-behavioral interventions for impulse control, and mindfulness-based approaches—may offer benefit across both dimensions of the pathology. Trauma-informed approaches are essential given the high rates of adverse childhood experiences documented in this population. Attention to sleep hygiene, affective regulation, and cognitive rehabilitation may address dimensions of the presentation that are often overlooked in traditional substance use treatment.

Treatment engagement presents particular challenges. Individuals with ASPD are, by disorder definition, prone to violate rules, deceive treaters, and disengage from treatment when its demands become aversive. Clinicians should anticipate these dynamics, structure the therapeutic frame accordingly, and avoid the countertransferential pull toward either punitive rigidity or naïve permissiveness. Motivational interviewing techniques may prove particularly valuable given the reduced spontaneous motivation to change documented in ASPD populations.

### *8.3 Managing Countertransference*

Working with individuals with ASPD and comorbid substance use pathology places distinctive demands on the clinician. Deceptive behavior, manipulation, hostility, and rule violation may provoke countertransferential responses that undermine the therapeutic alliance. Awareness of these dynamics—including one's own tendencies toward moralistic judgment, punitive impulses, or defensive withdrawal—is essential. Consultation, supervision, and peer support all play important roles in sustaining the clinician's therapeutic stance across the extended timeframes typically required for meaningful change in this population.

### *8.4 Attending to Comorbid Affective and Anxiety Pathology*

The elevated rates of depression, anxiety, and stress documented in comorbid ASPD–ADS presentations argue against the historical view that ASPD is characterized by uniformly low neuroticism and minimal affective distress. Clinicians should routinely assess for and treat comorbid mood and anxiety pathology, recognizing that these conditions may drive substance use through self-medication pathways and may also respond to targeted intervention. The comorbid presence of PTSD, in particular, deserves close attention given the high rates of childhood trauma documented in this population.

### *8.5 Personalized Interventions and Mechanism-Based Subtyping*

Low and colleagues (2024) argue for personalized interventions targeting mechanism-based subtyping in relation to ASPD–SUD comorbidities. This vision aligns with broader movements toward precision psychiatry, in which treatment selection is guided by individual-level phenotypic and mechanistic characteristics rather than by diagnostic category alone. Although the tools required to implement such subtyping in routine practice remain under development, the underlying principle—that clinical attention should be directed to the specific mechanisms driving each individual's presentation—is immediately applicable. For one client, the primary target may be trauma-related dysregulation; for another, impulsivity and reward-system dysfunction; for a third, cognitive deficits impeding engagement with standard interventions. Careful formulation, informed by the empirical literature synthesized here, can guide clinicians toward the most promising intervention targets for each individual case.

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## 9. Future Directions and Research Gaps

Despite substantial recent progress, the empirical foundation for understanding and treating comorbid ASPD and SUDs remains incomplete. Several priorities for future research emerge from the current evidence base.

### *9.1 Replication in General Population Samples*

Both major contemporary studies synthesized here—Low et al. (2024) and Jarcuskova et al. (2024)—used samples that were not representative of the general population. The Yale-Penn cohort was ascertained specifically for addiction-related traits, and the Jarcuskova et al. sample consisted of hospitalized adults with alcohol dependence. Replication in general population samples is essential to confirm the generalizability of the findings and to establish population-level base rates and association patterns.

### *9.2 Longitudinal and Developmental Research*

The cross-sectional nature of most current studies precludes strong inferences about the temporal and causal dynamics of ASPD–SUD comorbidity. Longitudinal studies tracking individuals from childhood or adolescence through adulthood are needed to clarify how conduct disorder features, early substance experimentation, adverse childhood experiences, and neurobiological vulnerabilities combine to produce the adult comorbid presentation. Such research could identify critical developmental windows during which preventive intervention might interrupt the trajectory toward dual pathology.

### *9.3 Related Externalizing Conditions*

Low and colleagues (2024) note that similar analyses could productively be extended to other externalizing conditions that share features with ASPD, including conduct disorder itself and adult antisocial behavior not meeting full ASPD criteria (Fairchild et al., 2019; Gomez-Smith & Piquero, 2005; Mata & van Dulmen, 2012, as cited in Low et al., 2024). Such extensions would help to clarify whether the association patterns observed for ASPD reflect specific ASPD-related pathology or broader externalizing vulnerability.

### *9.4 Mechanism-Focused Research*

The shared vulnerability model of ASPD–SUD comorbidity would be strengthened by more detailed mechanistic research. Genetic, neuroimaging, and psychophysiological investigations of the specific pathways connecting disinhibition, reward-system dysregulation, and affective processing to both antisocial and substance-related behavior are needed. The recent identification of shared genetic loci—including the *SLCO3A1* signal reported by Li and colleagues (2023, as cited in Jarcuskova et al., 2024)—offers promising leads for such

research.

### *9.5 Treatment Development and Evaluation*

The current state of treatment evidence for ASPD is, as the Cochrane reviews cited by Jarcuskova and colleagues (2024) make clear, deeply unsatisfying. Well-powered randomized trials of psychological and pharmacological interventions for ASPD, particularly in the context of comorbid substance use, are urgently needed. Trials should evaluate not only conventional outcomes such as symptom reduction and abstinence but also functional outcomes including occupational stability, interpersonal functioning, and criminal justice involvement.

### *9.6 Sex- and Gender-Specific Research*

The persistence of sex effects in the Low et al. (2024) analysis, despite careful matching, suggests that sex-specific pathways deserve dedicated attention. Research on female presentations of comorbid ASPD–SUD is particularly needed, given the historical underrepresentation of women in this literature and the likelihood that female presentations differ importantly in phenomenology, comorbidity patterns, and treatment needs.

### *9.7 Cross-Cultural Research*

Finally, the cultural embeddedness of both substance use and antisocial behavior argues for cross-cultural research that examines how these conditions and their comorbidity vary across national and cultural contexts. The Slovak context studied by Jarcuskova and colleagues (2024) represents one important contribution to this literature, but systematic cross-cultural comparisons remain rare and are urgently needed.

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## 10. Conclusion

Comorbid antisocial personality disorder and substance use disorders represent one of the most challenging and consequential presentations encountered in contemporary clinical

practice. The empirical evidence synthesized in this manual, drawing centrally on the contributions of Low and colleagues (2024) and Jarcuskova and colleagues (2024), documents a robust and multiply determined comorbidity that spans substance classes, developmental stages, and cultural contexts. ASPD is strongly and consistently associated with alcohol, cannabis, and tobacco use disorders at the diagnostic level, with additional associations at the level of individual diagnostic criteria—particularly the "hazardous use" criterion, which emerges as a transdiagnostic marker of externalizing vulnerability. The comorbid presentation is characterized by earlier onset, greater severity, higher hospitalization rates, poorer treatment adherence, and elevated rates of cognitive impairment, affective distress, sleep disturbance, and childhood traumatization.

Theoretical understanding of this comorbidity has advanced substantially through frameworks emphasizing shared vulnerability, behavioral disinhibition, bidirectional causation, and shared neurobiological substrates. These frameworks converge on the view that ASPD and SUDs are not independent conditions that happen to co-occur but rather alternative phenotypic expressions of a common underlying diathesis, shaped by genetic vulnerability, adverse developmental environments, and reciprocal reinforcement processes. This reframing has direct implications for clinical practice, inviting a shift from disorder-focused intervention toward mechanism-focused, personalized treatment.

Substantial gaps remain in the empirical foundation and in the treatment armamentarium. Longitudinal, mechanistic, and cross-cultural research is needed to clarify the developmental dynamics of ASPD–SUD comorbidity. Well-designed treatment trials are urgently required to establish evidence-based approaches to a population that has historically been underserved by therapeutic research. Attention to sex differences, cultural context, and the specific mechanisms driving individual presentations should guide both future research and current practice.

For psychologists working with individuals affected by comorbid ASPD and SUD, the immediate implications are clear. Routine screening, comprehensive assessment across cognitive, affective, and trauma domains, careful case formulation attuned to shared mechanisms, and treatment approaches that combine mechanism-focused intervention with attention to engagement dynamics and countertransference all represent essential components of competent practice. The population is challenging; the evidence base is imperfect; the therapeutic work is often protracted and uncertain. Yet the scale of clinical need, and the promise of emerging research on shared mechanisms and personalized

intervention, argue for sustained clinical and scholarly engagement with this consequential comorbidity.

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## 11. References


American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Association.

Alcorn, J. L., 3rd, Gowin, J. L., Green, C. E., Swann, A. C., & Moeller, F. G. (2013). Aggression, impulsivity, and psychopathic traits in combined antisocial personality disorder and substance use disorder. *Journal of Neuropsychiatry and Clinical Neurosciences, 25*(3), 229–232.

Alegria, A. A., Blanco, C., Petry, N. M., Skodol, A. E., Liu, S. M., Grant, B., & Hasin, D. (2013). Sex differences in antisocial personality disorder: Results from the National Epidemiological Survey on Alcohol and Related Conditions. *Personality Disorders, 4*(3), 214–222.

Brennan, G. M., Hyde, L. W., & Baskin-Sommers, A. R. (2017). Antisocial pathways associated with substance use disorders: Characterizing etiological underpinnings and implications for treatment. *Current Opinion in Behavioral Sciences, 13*, 124–129.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*(2), 193–213.

Chamberlain, S. R., Derbyshire, K. L., Leppink, E. W., & Grant, J. E. (2016). Neurocognitive deficits associated with antisocial personality disorder in non-treatment-seeking young adults. *Journal of the American Academy of Psychiatry and the Law, 44*(2), 218–225.

Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants: A proposal. *Archives of General Psychiatry, 44*(6), 573–588.

Cloninger, C. R. (1987). Neurogenetic adaptive mechanisms in alcoholism. *Science, 236*(4800), 410–416.

Compton, W. M., Conway, K. P., Stinson, F. S., Colliver, J. D., & Grant, B. F. (2005). Prevalence, correlates, and comorbidity of DSM-IV antisocial personality syndromes and alcohol and specific drug use disorders in the United States: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Clinical Psychiatry, 66*(6), 677–685.

Conway, K. P., Compton, W., Stinson, F. S., & Grant, B. F. (2006). Lifetime comorbidity of DSM-IV mood and anxiety disorders and specific drug use disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Clinical Psychiatry, 67*(2), 247–257.

DeLisi, M., Drury, A. J., & Elbert, M. J. (2019). The etiology of antisocial personality disorder: The differential roles of adverse childhood experiences and childhood psychopathology. *Comprehensive Psychiatry, 92*, 1–6.

Dugré, J. R., & Potvin, S. (2021). Impaired attentional and socio-affective networks in subjects with antisocial behaviors: A meta-analysis of resting-state functional connectivity studies. *Psychological Medicine, 51*(8), 1249–1259.

Fairchild, G., Hawes, D. J., Frick, P. J., Copeland, W. E., Odgers, C. L., Franke, B., Freitag, C. M., & De Brito, S. A. (2019). Conduct disorder. *Nature Reviews Disease Primers, 5*, 43.

Fergusson, D. M., Boden, J. M., & Horwood, L. J. (2008). Exposure to childhood sexual and physical abuse and adjustment in early adulthood. *Child Abuse & Neglect, 32*(6), 607–619.

Galbraith, T., Heimberg, R. G., Wang, S., Schneier, F. R., & Blanco, C. (2014). Comorbidity of social anxiety disorder and antisocial personality disorder in the National Epidemiological Survey on Alcohol and Related Conditions (NESARC). *Journal of Anxiety Disorders, 28*(1), 57–66.

Gelernter, J., Kranzler, H. R., Sherva, R., Almasy, L., Herman, A. I., Koesterer, R., et al. (2015). Genome-wide association study of nicotine dependence in American populations: Identification of novel risk loci in both African-Americans and European-Americans. *Biological Psychiatry, 77*(5), 493–503.

Goldstein, R. B., Chou, S. P., Saha, T. D., Smith, S. M., Jung, J., Zhang, H., Pickering, R. P., Ruan, W. J., Huang, B., & Grant, B. F. (2017). The epidemiology of antisocial behavioral syndromes in adulthood: Results from the National Epidemiologic Survey on Alcohol and Related Conditions-III. *Journal of Clinical Psychiatry, 78*(1), 90–98.

Gonzalez, E., Arias, F., Szerman, N., Vega, P., Mesias, B., & Basurte, I. (2019). Coexistence between personality disorders and substance use disorder: Madrid study about prevalence of dual pathology. *Actas Espanolas de Psiquiatria, 47*(6), 218–228.

Goodwin, R. D., & Hamilton, S. P. (2003). Lifetime comorbidity of antisocial personality disorder and anxiety disorders among adults in the community. *Psychiatry Research, 117*(2), 159–166.

Gorenstein, E. E., & Newman, J. P. (1980). Disinhibitory psychopathology: A new perspective and a model for research. *Psychological Review, 87*(3), 301–315.

Grant, B. F., Stinson, F. S., Dawson, D. A., Chou, S. P., Ruan, W. J., & Pickering, R. P. (2004). Co-occurrence of 12-month alcohol and drug use disorders and personality disorders in the United States: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry, 61*(4), 361–368.

Hasin, D. S., O'Brien, C. P., Auriacombe, M., Borges, G., Bucholz, K., Budney, A., Compton, W. M., Crowley, T., Ling, W., Petry, N. M., Schuckit, M., & Grant, B. F. (2013). DSM-5 criteria for substance use disorders: Recommendations and rationale. *American Journal of Psychiatry, 170*(8), 834–851.

Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. *Psychological Bulletin, 112*(1), 64–105.

Helle, A. C., Watts, A. L., Trull, T. J., & Sher, K. J. (2019). Alcohol use disorder and antisocial and borderline personality disorders. *Alcohol Research: Current Reviews, 40*(1), arcr.v40.1.05.

Holzer, K. J., Vaughn, M. G., Loux, T. M., Mancini, M. A., Fearn, N. E., & Wallace, C. L. (2022). Prevalence and correlates of antisocial personality disorder in older adults. *Aging & Mental Health, 26*(1), 169–178.

Jarcuskova, D., Pallayova, M., Carnakovic, S., Frajka, M., Fidmik, J., & Bednarova, A. (2024). Clinical characteristics of adults with alcohol dependence syndrome comorbid with antisocial personality disorder: A cross-sectional study. *Frontiers in Psychiatry, 15*, 1397009.

Kelly, T. M., & Daley, D. C. (2013). Integrated treatment of substance use and psychiatric disorders. *Social Work in Public Health, 28*(3–4), 388–406.

Kilpatrick, D. G., Resnick, H. S., Milanak, M. E., Miller, M. W., Keyes, K. M., & Friedman, M. J. (2013). National estimates of exposure to traumatic events and PTSD prevalence using DSM-IV and DSM-5 criteria. *Journal of Traumatic Stress, 26*(5), 537–547.

Kolla, N. J., Tully, J., & Bertsch, K. (2023). Neural correlates of aggression in personality disorders from the perspective of DSM-5 maladaptive traits: A systematic review. *Translational Psychiatry, 13*, 330.

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry, 56*(10), 921–926.

Krueger, R. F., Markon, K. E., Patrick, C. J., Benning, S. D., & Kramer, M. D. (2007). Linking antisocial behavior, substance use, and personality: An integrative quantitative model of the adult externalizing spectrum. *Journal of Abnormal Psychology, 116*(4), 645–666.

Li, W., Zhou, H., Thygesen, J. H., Heydtmann, M., Smith, I., Degenhardt, F., et al. (2023). Genome-wide association study of antisocial personality disorder diagnostic criteria provides evidence for shared risk factors across disorders. *Psychiatric Genetics, 33*(6).

Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour Research and Therapy, 33*(3), 335–343.

Low, A., Stiltner, B., Nunez, Y. Z., Adhikari, K., Deak, J. D., Pietrzak, R. H., Kranzler, H. R., Gelernter, J., & Polimanti, R. (2024). Association patterns of antisocial personality disorder across substance use disorders. *Translational Psychiatry, 14*, 346.

Martin, C. S., Chung, T., & Langenbucher, J. W. (2008). How should we revise diagnostic criteria for substance use disorders in the DSM-V? *Journal of Abnormal Psychology, 117*(3), 561–575.

Mewton, L., Teesson, M., Slade, T., & Cottler, L. (2011). Psychometric performance of DSM-IV alcohol use disorders in young adulthood: Evidence from an Australian general population sample. *Journal of Studies on Alcohol and Drugs, 72*(5), 811–822.

Murray, L., Waller, R., & Hyde, L. W. (2018). A systematic review examining the link between psychopathic personality traits, antisocial behavior, and neural reactivity during reward and loss processing. *Personality Disorders, 9*(6), 497–509.

Nasreddine, Z. S., Phillips, N. A., Bédirian, V., Charbonneau, S., Whitehead, V., Collin, I., Cummings, J. L., & Chertkow, H. (2005). The Montreal Cognitive Assessment, MoCA: A brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society, 53*(4), 695–699.

Pandey, S., Bolstad, I., Lien, L., & Bramness, J. G. (2021). Antisocial personality disorder among patients in treatment for alcohol use disorder (AUD): Characteristics and predictors of early relapse or drop-out. *Substance Abuse and Rehabilitation, 12*, 11–22.

Pierucci-Lagha, A., Gelernter, J., Feinn, R., Cubells, J. F., Pearson, D., Pollastri, A., Farrer, L., & Kranzler, H. R. (2005). Diagnostic reliability of the Semi-Structured Assessment for Drug Dependence and Alcoholism (SSADDA). *Drug and Alcohol Dependence, 80*(3), 303–312.

Pierucci-Lagha, A., Gelernter, J., Chan, G., Arias, A., Cubells, J. F., Farrer, L., & Kranzler, H. R. (2007). Reliability of DSM-IV diagnostic criteria using the Semi-Structured Assessment for Drug Dependence and Alcoholism (SSADDA). *Drug and Alcohol Dependence, 91*(1), 85–90.

Rhee, S. H., & Waldman, I. D. (2002). Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. *Psychological Bulletin, 128*(3), 490–529.

Ross, S., & Peselow, E. (2012). Co-occurring psychotic and addictive disorders: Neurobiology and diagnosis. *Clinical Neuropharmacology, 35*(5), 235–243.

Rounsaville, B. J., Kranzler, H. R., Ball, S., Tennen, H., Poling, J., & Trifflemann, E. (1998). Personality disorders in substance abusers: Relation to substance use. *Journal of Nervous and Mental Disease, 186*(2), 87–95.

Ruiz, M. A., Pincus, A. L., & Schinka, J. A. (2008). Externalizing pathology and the five-factor model: A meta-analysis of personality traits associated with antisocial personality disorder, substance use disorder, and their co-occurrence. *Journal of Personality Disorders, 22*(4), 365–388.

Sargeant, M. N., Bornovalova, M. A., Trotman, A. J., Fishman, S., & Lejuez, C. W. (2012). Facets of impulsivity in the relationship between antisocial personality and abstinence. *Addictive Behaviors, 37*(3), 293–298.

Saunders, J. B., Aasland, O. G., Babor, T. F., de la Fuente, J. R., & Grant, M. (1993). Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption II. *Addiction, 88*(6), 791–804.

Schulte Holthausen, B., & Habel, U. (2018). Sex differences in personality disorders. *Current Psychiatry Reports, 20*(12), 107.

Shmulewitz, D., Greene, E. R., & Hasin, D. (2015). Commonalities and differences across substance use disorders: Phenomenological and epidemiological aspects. *Alcoholism: Clinical and Experimental Research, 39*(10), 1878–1900.

Skodol, A. E., Oldham, J. M., & Gallaher, P. E. (1999). Axis II comorbidity of substance use disorders among patients referred for treatment of personality disorders. *American Journal of Psychiatry, 156*(5), 733–738.

Stiltner, B., Pietrzak, R. H., Tylee, D. S., Nunez, Y. Z., Adhikari, K., Kranzler, H. R., Gelernter, J., & Polimanti, R. (2023). Polysubstance addiction patterns among 7,989 individuals with cocaine use disorder. *iScience, 26*, 107336.

Tarter, R. E., Kirisci, L., Mezzich, A., Cornelius, J. R., Pajer, K., Vanyukov, M., Gardner, W., Blackson, T., & Clark, D. (2003). Neurobehavioral disinhibition in childhood predicts early age at onset of substance use disorder. *American Journal of Psychiatry, 160*(6), 1078–1085.

Trull, T. J., Jahng, S., Tomko, R. L., Wood, P. K., & Sher, K. J. (2010). Revised NESARC personality disorder diagnoses: Gender, prevalence, and comorbidity with substance dependence disorders. *Journal of Personality Disorders, 24*(4), 412–426.

Troyer, A. K., & Rich, J. B. (2002). Psychometric properties of a new metamemory questionnaire for older adults. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 57*(1), P19–P27.

Van Veen, M. M., Karsten, J., & Lancel, M. (2017). Poor sleep and its relation to impulsivity in patients with antisocial or borderline personality disorders. *Behavioral Medicine, 43*(3), 218–226.

Wallinius, M., Nordholm, J., Wagnström, F., & Billstedt, E. (2019). Cognitive functioning and aggressive antisocial behaviors in young violent offenders. *Psychiatry Research, 272*, 572–580.

Watts, A. L., Watson, D., Heath, A. C., & Sher, K. J. (2023). Alcohol use disorder criteria exhibit different comorbidity patterns. *Addiction, 118*(8), 1457–1468.

Werner, K. B., Few, L. R., & Bucholz, K. K. (2015). Epidemiology, comorbidity, and behavioral genetics of antisocial personality disorder and psychopathy. *Psychiatric Annals, 45*(4), 195–199.

Windle, M. (1999). Psychopathy and antisocial personality disorder among alcoholic inpatients. *Journal of Studies on Alcohol, 60*(3), 330–342.

Witkiewitz, K., King, K., McMahon, R. J., Wu, J., Luk, J., Bierman, K. L., et al. (2013). Evidence for a multi-dimensional latent structural model of externalizing disorders. *Journal of Abnormal Child Psychology, 41*(2), 223–237.

World Health Organization. (1993). *The ICD-10 classification of mental and behavioural disorders*. WHO Press.

World Health Organization. (2014). *Adverse Childhood Experiences International Questionnaire (ACE-IQ)*. World Health Organization.

Yalch, M. M., Stewart, A. M., & Dehart, R. M. (2021). Influence of betrayal trauma on antisocial personality disorder traits. *Journal of Trauma & Dissociation, 22*(1), 122–134.

Yoshino, A., Fukuhara, T., & Kato, M. (2000). Premorbid risk factors for alcohol dependence in antisocial personality disorder. *Alcoholism: Clinical and Experimental Research, 24*(1),

35–38.

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