Neurocognitive Assessment of Antisocial Personality Disorder and Psychopathy: Methodological Challenges, Clinical Implications, and Future Directions

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

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Prepared for Continuing Education in Clinical, Forensic, and Neuropsychological Practice

Based on the work of Griem, Kolla, and Tully (2022) and integrated primary literature

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Abstract

Antisocial personality disorder (ASPD) and psychopathy represent among the most clinically challenging, socially costly, and empirically contested conditions encountered in forensic and general clinical psychology. Although accumulating research has begun to reveal characteristic neurocognitive dysfunctions—particularly in the domains of emotion recognition, empathic responding, reinforcement-based learning, and attention regulation—the promise of neurocognitive assessment as a means of clarifying causal mechanisms, guiding stratified intervention, and producing personalized medicine approaches has been persistently constrained by methodological limitations. This continuing education manual synthesizes the conceptual arguments and empirical evidence advanced by Griem, Kolla, and Tully (2022), integrating them with the broader literature on ASPD and psychopathy that they cite. Four principal challenges are examined in depth: (a) inconsistent phenotypic characterization of heterogeneous samples, (b) unreliable and inconsistent task design and selection, (c) poor task engagement among individuals with ASPD and psychopathy, and (d) the paucity of longitudinal neurocognitive research. Each challenge is situated within its historical and

theoretical context, its underlying conceptual mechanisms are explicated, and its practical clinical implications are traced. The manual concludes with a discussion of collaborative, pre-registered, longitudinally informed strategies for advancing neurocognitive science in ASPD and psychopathy, framed with attention to the clinical decisions that psychologists must make in assessment, formulation, and treatment planning.

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1. Introduction

Antisocial personality disorder and psychopathy occupy a distinctive place in the landscape of adult psychopathology. Unlike disorders whose principal costs are borne by the affected individual, ASPD and psychopathy generate substantial harm to others, communities, and public systems. Individuals with conduct disorder (CD) in youth and antisocial personality disorder in adulthood are responsible for a large proportion of violent crime, with a widely cited Swedish population study indicating that roughly one percent of the population accounts for a majority of violent crime convictions (Falk et al., 2014; Martinez et al., 2017). Within this already high-risk population, a substantial subgroup—approximately one third—meets criteria for callous-unemotional (CU) traits in youth and psychopathy in adulthood, and these individuals offend earlier, more widely, and more severely than their counterparts without psychopathy (Kosson et al., 2006).

The clinical, social, and financial burdens associated with ASPD, with or without psychopathy (hereafter ASPD+/-P), are correspondingly substantial (Heeks et al., 2018). ASPD is markedly overrepresented in forensic and penal settings, where prevalence rates far exceed those observed in general population samples (Fazel & Danesh, 2002; Coid et al., 2009b). Despite this, effective psychological and pharmacological treatments remain elusive. Systematic reviews of both psychological and pharmacological interventions for ASPD have concluded that the evidence base is thin, methodologically inconsistent, and insufficient to support clear recommendations for practice (Gibbon et al., 2020; Khalifa et al., 2020). Understanding of the causal mechanisms underlying ASPD and its heterogeneous presentations remains limited, and this scientific gap directly constrains clinical progress.

Against this backdrop, neurocognitive assessment has emerged as a particularly promising avenue of investigation. If the affective, motivational, and cognitive dysfunctions that

characterize ASPD and psychopathy can be identified with precision, tied to specific neural systems, and mapped onto developmental trajectories, then treatment targets can be defined, interventions can be tailored to particular biocognitive profiles, and stratified care can replace the diffuse and often ineffective interventions now available. Baskin-Sommers and colleagues (2015) have already demonstrated that cognitive remediation targeted at specific attentional and affective dysfunctions can produce meaningful change in externalizing offender subtypes, providing proof of principle that neurocognitive science can inform intervention.

Yet the neurocognitive literature on ASPD and psychopathy is also marked by inconsistency, contradiction, and interpretive difficulty. Studies of empathy, executive function, decision-making, and reinforcement learning have produced findings that vary substantially in direction and magnitude across samples that are, on the surface, comparable. As Griem, Kolla, and Tully (2022) have observed, this incoherence is not merely a reflection of nature's complexity; it is, in significant measure, a product of methodological choices that can and should be improved.

This continuing education manual is organized around the framework advanced by Griem et al. (2022). It examines four key challenges that constrain the potential of neurocognitive testing in ASPD and psychopathy: (1) inconsistent phenotypic characterization of heterogeneous samples; (2) unreliable and inconsistent task design and selection; (3) poor task engagement, itself a product of the very pathology being studied; and (4) the lack of longitudinal studies capable of tracing developmental trajectories of neurocognitive dysfunction. Each is examined in depth, situated within the empirical literature, and translated into implications for psychologists engaged in assessment, formulation, and treatment planning. The manual then considers future directions and concludes with reflections on the ways in which improved neurocognitive science can serve the personal, clinical, and social imperatives associated with these disorders.

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2. Historical and Theoretical Foundations

The construct of psychopathy has undergone repeated conceptual revision across the twentieth and twenty-first centuries, and the current landscape of assessment and research reflects the residue of these successive reformulations. Modern operational definitions are

dominated by the Psychopathy Checklist–Revised (PCL-R; Hare, 1991), a clinician-administered instrument that scores individuals on a set of interpersonal, affective, lifestyle, and antisocial features. The PCL-R is the most widely used assessment tool in clinical forensic and penitentiary populations and has served as the anchor for the majority of neurocognitive research programs.

However, the field is not unified around a single conceptualization. Considerable debate persists about the most appropriate construct of psychopathy, with alternative models emphasizing different combinations of interpersonal, affective, and behavioral features (Cooke & Michie, 2001; Hare & Neumann, 2008). These theoretical disagreements are not merely academic. They translate directly into the choice of assessment tools, the setting of cut-off scores, and the operational definitions used to compose research samples. As Griem et al. (2022) note, the practical consequence is that samples labeled as "psychopathic" in different studies may in fact reflect meaningfully different clinical populations.

ASPD, as defined in the Diagnostic and Statistical Manual of Mental Disorders, is broader and more behaviorally focused than psychopathy. It encompasses a heterogeneous group of individuals whose common feature is a persistent pattern of disregard for the rights of others, but who may differ substantially in the affective and interpersonal features that distinguish psychopathy from a purely behavioral pattern of antisocial conduct (First et al., 2015). This heterogeneity has motivated a stratification approach in which ASPD is subdivided according to the presence or absence of psychopathy—yielding the ASPD+P and ASPD-P designations that structure much of the current literature (Brazil et al., 2018).

The developmental antecedents of ASPD and psychopathy are increasingly well characterized. Conduct disorder, with or without CU traits, represents the childhood and adolescent precursor of the adult syndrome. Landmark longitudinal cohort studies have followed the trajectories of youth with CD across decades and have identified early cognitive risk factors for differential pathways toward life-course-persistent antisocial behavior (Frick & Viding, 2009; Frick et al., 2013; Assink et al., 2015; Jolliffe et al., 2017; Moffitt, 2018; Carlisi et al., 2020; Farrington, 2020; Lasko & Chester, 2021). Evidence for substantial heritability of psychopathic traits, observable as early as age seven (Viding et al., 2005), together with a marked male preponderance (Baron-Cohen et al., 2011; Yildirim & Derksen, 2012), has led some authors to argue that ASPD+P bears meaningful similarities to neurodevelopmental disorders and should be conceptualized within that framework.

The theoretical models that guide contemporary neurocognitive research on ASPD and psychopathy draw on this developmental foundation and integrate it with more focused claims about specific dysfunctions. Blair's work on emotion recognition and empathic responding has emphasized the role of amygdala-based affective processing deficits, particularly in the recognition and internalization of others' distress cues (Blair, 2007, 2018; Marsh & Blair, 2008; Dawel et al., 2012). The response modulation hypothesis, associated with Newman and colleagues, has proposed that psychopathic individuals suffer from an exaggerated attentional bottleneck that impairs the integration of contextual information once attention has been captured by a dominant goal (Hamilton & Newman, 2018; Baskin-Sommers & Brazil, 2022). Reinforcement-based learning accounts, drawing on both behavioral and neuroimaging data, have documented characteristic impairments in reversal learning, punishment sensitivity, and probabilistic decision-making (Budhani et al., 2006; Dolan, 2012; De Brito et al., 2013; Gregory et al., 2015; Hughes et al., 2016; Glimmerveen et al., 2022a). Together, these frameworks form the theoretical context within which the challenges discussed in the remainder of this manual arise.

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3. Conceptual Models and Mechanisms

To appreciate the significance of the methodological challenges facing neurocognitive research on ASPD and psychopathy, it is helpful to first outline the conceptual models that dominate the field and to consider the mechanisms they propose. Three broad classes of model are especially prominent: affective and empathic dysfunction models, reinforcement learning and decision-making models, and attentional models.

3.1 Affective and Empathic Dysfunction Models

Empathy is a foundational clinical construct in ASPD and, most centrally, in psychopathy. Impaired empathy is a defining criterion of ASPD (First et al., 2015) and a critical component of the psychopathy construct as operationalized by the PCL-R (Hare, 1991). Empathic dysfunction, however, is not a monolithic phenomenon. Contemporary theoretical work distinguishes at minimum between cognitive empathy—the capacity to represent and reason about others' mental states, encompassing mentalizing and theory of mind—and affective

empathy—the capacity to share, resonate with, or be moved by others' emotional experiences, encompassing emotional contagion and experience sharing (Bird & Viding, 2014; Decety & Ickes, 2011; Zaki & Ochsner, 2012; Blair, 2018).

Even this binary distinction masks considerable conceptual complexity. Different research programs use terms such as mentalizing, theory of mind, and perspective-taking in ways that overlap incompletely (Choi-Kain & Gunderson, 2008). The consequence is that studies claiming to assess "empathy" may in fact be probing distinct neurocognitive systems, and apparent disagreements in the literature may reflect genuine differences in what is being measured rather than genuine differences in the populations under study.

The empirical picture that has emerged, notwithstanding these definitional challenges, suggests a partially dissociated pattern. Adults with ASPD, whether or not they also meet criteria for psychopathy, tend to show relatively preserved performance on many measures of cognitive empathy (Blair et al., 1996; Dolan & Fullam, 2004; Shamay-Tsoory et al., 2010). Yet individuals with ASPD+P have been shown to fail at automatic perspective-taking, suggesting that spontaneous, non-effortful engagement of others' viewpoints may be selectively impaired even when explicit reasoning about mental states remains intact (Drayton et al., 2018).

In the domain of affective empathy, ASPD+P and ASPD-P appear to diverge more sharply. Brain regions involved in implicit responsivity to others' pain and to emotional faces, including regions of anterior insula and anterior cingulate cortex, show hyporesponsivity in individuals with ASPD+P during tasks that elicit empathic engagement (Decety et al., 2013, 2014; Contreras-Rodríguez et al., 2014). In contrast, individuals with ASPD-P are frequently found to be hyperresponsive to emotional stimuli, particularly those signaling fear and threat (Schönenberg & Jusyte, 2014; Hodgins et al., 2018). This dissociation is theoretically important: it suggests that the affective abnormalities that underlie violence in ASPD-P may be nearly opposite in direction to those that underlie violence in ASPD+P. Reactive, threat-driven aggression in ASPD-P and instrumental, callous aggression in ASPD+P thus appear to arise from meaningfully different affective substrates, and treatments must accordingly be tailored.

3.2 Reinforcement Learning and Decision-Making Models

A second family of models focuses on how individuals with ASPD and psychopathy process rewards and punishments and how they use these signals to adjust behavior. Impairments in reinforcement-based learning have been documented across a range of tasks, including probabilistic reversal paradigms and passive avoidance tasks (Budhani et al., 2006; Gregory et al., 2015; Hughes et al., 2016). Gregory and colleagues (2015), in a case-control functional MRI investigation, demonstrated that violent men with antisocial personality disorder and psychopathy showed characteristic neural signatures during punishment-based learning, differentiating them from ASPD-P and healthy control groups.

Glimmerveen and colleagues (2022a) have integrated these findings under the broader concept of maladaptive learning, arguing that psychopathy is associated with a systematic difficulty in updating behavior in response to changing contingencies, particularly when doing so requires the integration of punishment signals. Importantly, this framework has practical implications: interventions that rely on the delivery of aversive consequences to shape behavior may fail in populations characterized by punishment-processing deficits.

3.3 Attentional Models

The response modulation hypothesis, developed by Newman and colleagues over several decades, proposes that individuals with psychopathy exhibit an exaggerated attentional bottleneck: once a goal-relevant stimulus captures attention, the integration of peripheral, contextual, or emotional information is unusually impaired (Hamilton & Newman, 2018; Baskin-Sommers & Brazil, 2022). This model has been influential in part because it offers a mechanistic account of features that other models struggle to explain, including the apparent situational specificity of empathic and affective deficits in psychopathy. If emotional information is not being processed only because attention is elsewhere, then apparent affective abnormalities may in fact be secondary to a more fundamental attentional constraint.

Attentional deficits have been documented not only in psychopathy narrowly defined but also in ASPD more broadly, with additional evidence of executive function and memory impairments (Baliousis et al., 2019). The relative contributions of attention, executive function, and affective processing to observed behavioral deficits remain a central and unresolved empirical question, and as this manual argues below, the answer depends critically on task design.

3.4 Toward Biocognitive Stratification

Brazil, van Dongen, Maes, Mars, and Baskin-Sommers (2018) have advanced a proposal that draws these threads together: rather than treating ASPD as a homogeneous behavioral category, the field should stratify antisocial individuals according to their underlying biocognitive profiles. Different profiles—characterized by different combinations of affective, motivational, and attentional dysfunctions—may respond to different interventions. This stratification approach represents a compelling scientific and clinical aspiration, but its realization depends on the resolution of the methodological challenges to which this manual now turns.

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4. Empirical Findings Across Studies

Before examining the methodological challenges in detail, it is useful to consider the empirical landscape as it currently stands. The picture is one of substantial promise mixed with substantial inconsistency.

In the domain of emotion recognition, meta-analytic evidence supports the conclusion that antisocial populations, including those with psychopathy, exhibit pervasive deficits in recognizing facial and vocal expressions of emotion (Dawel et al., 2012; Marsh & Blair, 2008). These deficits extend beyond fear and sadness, contrary to earlier views that psychopathy might be characterized by a selective impairment for distress cues.

In the domain of empathy, the picture is more equivocal. Marsden, Glazebrook, Tully, and Völlm (2019), in a systematic review of empathy and emotion processing in adult men with ASPD (with and without psychopathy), concluded that ASPD+/-P was not consistently associated with deficits on neurocognitive paradigms of empathy. This is a striking finding given the centrality of empathy impairment to the clinical concept of the disorder. As Griem et al. (2022) argue, however, this null pattern may reflect not the absence of an empathy deficit but rather the inconsistency of task design and the heterogeneity of samples. Winter, Spengler, Bermpohl, Singer, and Kanske (2017), using the EmpaToM paradigm developed by Kanske, Böckler, Trautwein, and Singer (2015), demonstrated deficits in affective empathy but intact theory of mind in male violent offenders—an example of how more targeted,

validated tasks can recover findings that broader, less specific paradigms obscure.

In reinforcement learning, findings are somewhat more consistent, particularly with regard to reversal learning impairments in psychopathy (Budhani et al., 2006; Gregory et al., 2015). Structural neuroimaging studies have further identified characteristic differences in ASPD+P samples, including alterations in gray matter volume in regions implicated in emotion processing and decision-making (Gregory et al., 2012). Griffiths and Jalava (2017), in a comprehensive review of neuroimaging in PCL-R–defined psychopathy, noted that despite converging evidence for structural and functional abnormalities, considerable heterogeneity of findings persists across studies.

Executive function findings similarly show variability. De Brito, Viding, Kumari, Blackwood, and Hodgins (2013) demonstrated distinct patterns of "cool" and "hot" executive function impairments in violent offenders with ASPD, distinguishing those with and without psychopathy. Pera-Guardiola and colleagues (2016) reported modulatory effects of psychopathy on Wisconsin Card Sorting Test performance in male offenders with ASPD, providing additional evidence for the biocognitive dissociation of ASPD subtypes.

Table 1 offers a synthetic overview of the empirical findings across the domains reviewed above, organized to highlight both convergences and divergences.

Table 1

Synthesis of Neurocognitive Findings in ASPD+P and ASPD-P

Domain	ASPD+P	ASPD-P	Consistency of Findings
Facial and vocal affect recognition	Pervasive deficits	Deficits also observed, less specific pattern	Moderate; meta-analytic support (Dawel et al., 2012; Marsh & Blair, 2008)
Cognitive empathy / theory of mind	Largely intact on explicit tasks; automatic perspective-taking impaired	Largely intact	Moderate to high (Blair et al., 1996; Drayton et al., 2018)

Domain	ASPD+P	ASPD-P	Consistency of Findings
Affective empathy	Hyporesponsivity to others' pain and emotional faces	Hyperresponsivity, particularly to threat	Moderate; task-dependent (Decety et al., 2013; Schönenberg & Jusyte, 2014)
Reversal / reinforcement learning	Consistent impairments	Variable	Moderate to high (Gregory et al., 2015; Hughes et al., 2016)
Executive function	"Hot" executive function deficits prominent	"Cool" deficits more variable	Moderate (De Brito et al., 2013; Baliousis et al., 2019)
Attention / response modulation	Exaggerated attentional bottleneck	Less studied	Moderate (Baskin-Sommers & Brazil, 2022)

The pattern that emerges from Table 1 supports two broad conclusions. First, ASPD+P and ASPD-P are meaningfully distinct at the neurocognitive level, with dissociations that are theoretically coherent and clinically relevant. Second, findings are consistent enough to support cautious inference but inconsistent enough to demand methodological scrutiny. It is to that scrutiny that this manual now turns.

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5. Psychological Pathways and Stress Responses

Before examining the specific methodological challenges that Griem et al. (2022) identify, it is worth pausing to consider how the neurocognitive dysfunctions described above map onto psychological pathways and stress-response mechanisms that clinicians encounter in practice.

Individuals with ASPD-P, characterized by high reactive aggression, low tolerance to frustration, and heightened responsivity to threat (Hodgins et al., 2018; Schönenberg & Jusyte, 2014), can be understood as occupying a stress-response profile in which ambiguous social cues are readily perceived as threatening. The hostile attribution bias documented by Schönenberg and Jusyte (2014) is a paradigmatic example: ambiguous facial expressions are

more likely to be interpreted as angry or threatening, and this misinterpretation shapes downstream affective and behavioral responses. Under stress, the ASPD-P individual may thus enter a loop in which perceived threat elicits reactive aggression, which in turn elicits genuine threat from others, which further reinforces the interpretive bias.

Individuals with ASPD+P present a different profile. Diminished implicit responsivity to others' distress cues, coupled with characteristic reinforcement-learning impairments, produces a pattern in which the ordinary social feedback that shapes prosocial behavior in most individuals fails to register with typical force. Punishment, in particular, does not deter as effectively as one would expect. Callous-unemotional traits, present in the childhood precursor of ASPD+P, index this affective and motivational profile from an early age (Frick et al., 2013; Viding et al., 2005).

These pathways have implications for how psychologists conceptualize the presentations they encounter. The individual with ASPD-P may be misread as callous when in fact they are reacting to perceived threat with poorly regulated affect; the individual with ASPD+P may be misread as merely impulsive when in fact their reinforcement-processing profile makes ordinary behavioral consequences less impactful. The neurocognitive framework helps to disambiguate these presentations, and it does so with implications for treatment: interventions targeting threat-processing may be indicated in ASPD-P, whereas interventions targeting reinforcement processing and affective engagement may be indicated in ASPD+P (Baskin-Sommers et al., 2015).

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6. Mental Health Outcomes and Severity Spectrum

The clinical severity of ASPD+/-P varies substantially, both across individuals and across the lifespan of any given individual. The prevalence of subclinical psychopathic traits in community samples is nontrivial (Coid et al., 2009a), and this fact has important methodological consequences that are discussed further in Section 7.1. Clinically, the severity spectrum encompasses individuals whose antisocial features have led to repeated, serious violent offending and long-term incarceration, individuals whose features have produced substantial interpersonal and occupational impairment without formal criminal justice involvement, and individuals whose subclinical traits have relatively modest life

consequences.

The distribution of these outcomes is shaped by developmental factors, environmental factors, and, importantly for this manual, by the specific configuration of neurocognitive strengths and weaknesses that the individual possesses. Lasko and Chester (2021) have shown that longitudinal trajectories of antisocial behavior and impulse control differ as a function of psychopathy, with implications for understanding what has sometimes been called the "successful" psychopath—an individual whose psychopathic traits do not translate into criminal outcomes. Understanding why some individuals with pronounced psychopathic traits achieve relatively favorable life outcomes, while others do not, is a central question for developmental psychopathology and one that neurocognitive assessment across the lifespan is uniquely positioned to address.

Comorbidity is common. Substance use disorders, mood disorders, and other personality disorders frequently co-occur with ASPD, and these comorbidities complicate both assessment and treatment. Serious mental disorder is markedly overrepresented in incarcerated populations (Fazel & Danesh, 2002), and psychopathy is prevalent among prisoners in England and Wales (Coid et al., 2009b), underscoring the clinical urgency of improved assessment methods for these populations.

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7. System-Level and Contextual Psychological Effects: The Four Key Challenges

We now turn to the central analytic contribution of Griem, Kolla, and Tully (2022): a structured analysis of four key methodological and contextual challenges that constrain neurocognitive assessment in ASPD and psychopathy. Each is examined in turn, with attention to its conceptual basis, its empirical manifestations, and its implications for the field.

7.1 Inconsistent Phenotypic Characterization of Heterogeneous Samples

The first and arguably most fundamental challenge concerns the definition and characterization of the populations under study. ASPD is inherently heterogeneous, and this heterogeneity is not merely quantitative variation on a single dimension but reflects

meaningful biocognitive differences. Brazil and colleagues (2018) have argued that a pragmatic response to this heterogeneity is stratification into more biologically homogeneous subtypes. The distinction between ASPD+P and ASPD-P is the most widely used such stratification, and, as Section 4 illustrated, it is supported by dissociations across a wide range of neurocognitive domains (Kosson et al., 2006; Gregory et al., 2012, 2015; De Brito et al., 2013; Pera-Guardiola et al., 2016; Marsden et al., 2019).

Stratification, however, requires a consistent definition of psychopathy—and here the field encounters difficulty. Although the PCL-R (Hare, 1991) remains the most widely used tool in clinical forensic and penitentiary populations, considerable debate persists about the appropriate construct of psychopathy (Cooke & Michie, 2001; Hare & Neumann, 2008). This debate has translated into the proliferation of alternative assessment tools, including self-report questionnaires. Self-report measures, however, may have lower reliability in this population than in others, since individuals with ASPD+/-P may not tell the truth or may lack the insight required to accurately report their own features (Brinkley, Schmitt, Smith, & Newman, 2001; Sellbom, Ben-Porath, & Stafford, 2007; Gonsalves, McLawsen, Huss, & Scalora, 2013). Brinkley and colleagues' (2001) construct validation work on the Levenson Self-Report Psychopathy scale exemplifies the difficulty: even carefully constructed self-report tools may not measure the same constructs as clinician-administered instruments.

A related concern is the widespread study of community-dwelling individuals with subclinical psychopathic traits. Given the nontrivial prevalence of such traits in the general population (Coid et al., 2009a), the study of subclinical samples is not inherently problematic; indeed, dimensional approaches to psychopathology encourage such inquiry (Esser & Eisenbarth, 2021; Friedman, Rhee, Ross, Corley, & Hewitt, 2021). Yet as Griem et al. (2022) caution, findings from subclinical samples may not generalize to clinical populations in which more severe neurocognitive deficits may have qualitatively different underpinnings. The extrapolation from community samples with mild trait elevations to incarcerated samples with clinical-level psychopathy is thus an inferential leap that requires careful justification.

Even within studies that use the PCL-R, cut-off scores vary. Cooke and Michie (1999) demonstrated cross-cultural differences in PCL-R scores between North American and Scottish samples, and their work supports the use of a cut-off of 25 in European samples as opposed to the traditional 30 used in United States samples. This recommendation, however, is not universally followed. Furthermore, as Griem et al. (2022) point out, some studies refer to their samples as "high" on psychopathy despite scores that in fact indicate only subclinical

levels of the construct (e.g., Domes, Hollerbach, Vohs, Mokros, & Habermeyer, 2013; Weidacker, Snowden, Boy, & Johnston, 2017). This terminological drift muddies the interpretation of findings and contributes to the incoherence of the cumulative literature.

The consequence of these inconsistencies is that samples labeled comparably across studies may differ in ways that materially affect the interpretation of neurocognitive findings. Griem et al. (2022) argue that ongoing research into the most appropriate construct of psychopathy remains important, but they also propose a pragmatic next step: the development of a large-scale collaborative protocol that agrees on the most appropriate tools to measure psychopathy within clinical forensic research. Such a protocol need not preclude the use of multiple metrics; indeed, sufficiently well-powered, pre-registered neurocognitive studies could then evaluate which constructs correlate best with potential biomarkers. The move toward pre-registration and collaborative standardization aligns with the broader open-science reforms advocated by Munafò and colleagues (2017) in their manifesto for reproducible science.

For psychologists engaged in assessment practice, the implications of this challenge are direct. The choice of instrument in a forensic assessment is not merely a technical decision; it embeds assumptions about the construct being measured and about the population to which findings will be compared. Clinicians should be transparent about which construct their instruments target, explicit about the cut-off scores they apply, and cautious about generalizing from research conducted in samples that may differ in important respects from the individual before them.

7.2 Unreliable and Inconsistent Task Design and Selection

The second challenge concerns the neurocognitive tasks themselves. For any given neurocognitive function—empathy, executive function, reinforcement learning, attention—there exist multiple tasks claiming to provide reliable metrics. These tasks often differ subtly from one another in stimuli, timing, response demands, and instructions. These subtle differences can produce meaningfully different findings, and this variability in turn contributes to the incongruence of the literature.

Empathy provides a particularly instructive example. Despite being both a clinical feature of ASPD (First et al., 2015) and a critical component of the psychopathy construct (Hare, 1991),

the systematic review by Marsden and colleagues (2019) found that ASPD+/-P was not consistently associated with deficits on neurocognitive paradigms of empathy. As Griem et al. (2022) argue, this striking result likely reflects at least two factors: underpowered studies in a population in which recruitment is difficult, and inconsistency in the way empathy is operationalized and measured.

The conceptual complexity of empathy compounds this problem. As reviewed in Section 3, empathy encompasses multiple, partially dissociable components, and even careful researchers may use the same term to refer to different processes (Bird & Viding, 2014; Decety & Ickes, 2011; Zaki & Ochsner, 2012; Blair, 2018). Mentalization, in particular, is used variably across research programs (Choi-Kain & Gunderson, 2008). When a study reports intact empathy or impaired empathy, the reader must ask which component of empathy is being measured, what task is being used to measure it, and whether the measurement is validated in the population being studied.

The empirical evidence reviewed in Section 3 illustrates the payoff of more precise task design. When empathy is decomposed into cognitive and affective components, and when tasks are used that can validly distinguish them, a coherent pattern emerges: individuals with ASPD+/-P show largely preserved performance on cognitive empathy and theory-of-mind measures (Blair et al., 1996; Dolan & Fullam, 2004; Shamay-Tsoory et al., 2010), while diverging in affective empathy, with ASPD+P showing hyporesponsivity and ASPD-P showing hyperresponsivity (Decety et al., 2013, 2014; Contreras-Rodríguez et al., 2014; Schönenberg & Jusyte, 2014; Hodgins et al., 2018). The development of the EmpaToM by Kanske and colleagues (2015)—a validated tool that delineates cognitive and affective empathy within a single paradigm—represents a methodological advance, and Winter and colleagues (2017) have used it to demonstrate the expected dissociation in male violent offenders.

Griem et al. (2022) argue that disentangling the relative contributions of deficits in cognitive and affective empathy through consistent and reliable task designs may be crucial in developing targeted treatments. This principle generalizes beyond empathy: for each neurocognitive domain of interest, the field would benefit from convergence around validated tasks whose psychometric properties are well established and whose construct validity has been demonstrated in the relevant populations. Blair, Mathur, Haines, and Bajaj (2022) have made a related argument in advocating for biomarker design based on rigorous test–retest reliability work.

For the practicing psychologist, the practical implication is that the choice of task matters, and that findings from different tasks should not be pooled uncritically. A clinician synthesizing evidence for or against an empathy deficit in a particular patient should consider what tasks were used in the underlying research, whether those tasks measured the components of empathy most relevant to the clinical question, and whether the tasks have demonstrated psychometric adequacy in comparable populations.

7.3 Poor Task Engagement

The third challenge is one that is not shared with most other clinical populations and that arises directly from the pathology being studied. Poor attention, lack of interest or motivation to participate in activities that are not self-beneficial, irresponsibility, and a propensity for lying are inherent pathological features of ASPD and, particularly, of psychopathy. These features complicate assessment procedures and can produce both false positive and false negative findings.

Consider first the risk of false positives. A lack of effort due to poor motivation may produce performance on accuracy and reaction time measures that is substantially poorer than would be observed in a real-life scenario in which the individual is genuinely invested. If such data are interpreted as evidence of a neurocognitive deficit, the deficit may be inferred where none exists, or its magnitude may be overestimated.

The risk of false negatives is subtler but equally consequential. Individuals with psychopathy, for whom pathological lying is a defining feature, may report inaccurate responses that do not reflect their true emotional reactions in tasks measuring explicit responses to emotional stimuli. A task that requires the participant to self-report their emotional experience may thus fail to detect an underlying abnormality that would be evident on implicit measures.

Griem et al. (2022) offer an illustrative example from Tully's (2021) functional MRI study of cooperation and retaliation, which used the Dealmaking game developed by White and colleagues (2016). The sample comprised male violent offenders with ASPD-P, clinically characterized by high reactive aggression and low tolerance to frustration and threat. Based on prior findings in comparable populations (White, Brislin, Meffert, Sinclair, & Blair, 2013; White, Brislin, Sinclair, & Blair, 2014), the a priori prediction was that these participants would reject and punish unfair financial offers at high rates. Instead, they typically accepted unfair

offers and punished less frequently than healthy non-offending controls, though the findings did not reach statistical significance. Neuroimaging further revealed no activation of threat circuitry.

One interpretation of this pattern is that ASPD-P is not, in fact, associated with the retaliatory decision-making that the clinical profile would suggest. Griem et al. (2022) argue, however, that other interpretations are more plausible. The absence of actual monetary incentives may have left participants insufficiently motivated; the length of the task may have produced boredom; participant feedback comments, collated separately, hinted at disengagement. If task engagement failed, then the observed pattern may not reflect the underlying neurocognitive profile at all but rather the failure of the task to elicit the relevant psychological states in the first place.

This case study highlights the importance of building engagement checks into neurocognitive assessment protocols. Griem et al. (2022) advance several concrete recommendations. First, experimental manipulations that explicitly measure attention should be incorporated, allowing investigators to distinguish true task effects from the impact of inherent clinical symptoms. Second, strategies to increase motivation should be considered, including the use of adequate and tangible incentives or personalized rewards. Glimmerveen, Maes, Bulten, Scheper, and Brazil (2022b) have demonstrated the impact of individualized rewards on associative learning in psychopathic offenders, providing empirical support for this recommendation. Third, control items and performance validity tests offer a time- and cost-effective way to identify data that should be excluded from analysis due to poor engagement (Greher & Wodushek, 2017; Sweet et al., 2021). Fourth, precise and uniform instructions must be delivered to all participants, since effort may fluctuate depending on whether participants are prioritizing speed or accuracy (Ging-Jehli, Ratcliff, & Arnold, 2021). Finally, task difficulty must be calibrated: tasks that are too simple may promote boredom, while tasks that are too difficult may hinder effort (Cornacchio, Pinkham, Penn, & Harvey, 2017).

The clinical implications of this challenge extend beyond research. In neuropsychological evaluation of individuals with ASPD or psychopathic features, performance validity testing is not merely a technical safeguard but an essential component of interpretive rigor. The American Academy of Clinical Neuropsychology's 2021 consensus statement on validity assessment (Sweet et al., 2021) underscores that validity testing has become a standard of care in neuropsychological practice, and its application to forensic populations is particularly

important given the incentives for both intentional and unintentional distortion of performance.

7.4 Lack of Longitudinal Studies

The fourth challenge concerns the developmental dimension of neurocognitive research on ASPD and psychopathy. Given that conduct disorder with or without callous-unemotional traits is the developmental precursor of ASPD+/-P, longitudinal research is essential for understanding the mechanisms by which early risk factors translate into adult outcomes. Several pivotal large cohort studies have followed the trajectories of youth with CD over years and even decades, providing crucial insights into differential pathways toward life-course-persistent antisocial behavior and improving understanding of protective and promotive factors (Frick & Viding, 2009; Frick et al., 2013; Assink et al., 2015; Jolliffe et al., 2017; Moffitt, 2018; Carlisi et al., 2020; Farrington, 2020; Lasko & Chester, 2021).

Yet as Griem et al. (2022) note, these landmark studies have been limited by a relative lack of thorough neurocognitive testing. Behavioral outcomes and, in some cases, structural neuroimaging (Carlisi et al., 2020) have been measured, but the specific neurocognitive mechanisms that mediate developmental trajectories remain poorly characterized. As a result, the understanding of how mechanisms such as reinforcement-based learning, empathic responsivity, and attentional regulation change over time in this population is still limited.

The parallel to research in other neurodevelopmental disorders is instructive. In autism spectrum disorder, longitudinal research has revealed that neurobiological underpinnings continue to develop distinctively throughout adulthood, underscoring the importance of lifespan-wide assessment (Magiati, Tay, & Howlin, 2014). Given that ASPD+P has been argued to share features with neurodevelopmental disorders—including substantial heritability and a marked male preponderance (Viding et al., 2005; Baron-Cohen et al., 2011; Yildirim & Derksen, 2012)—the case for longitudinal neurocognitive study is particularly strong.

Longitudinal studies could also illuminate important sex differences. Tully, Frey, Fotiadou, Kolla, and Eisenbarth (2022) have argued that psychopathy in women has been understudied and that trajectories of neurocognitive dysfunction in females may differ in important ways from those in males. Personalized medicine approaches, as Blair and colleagues (2022) have argued, will require not only cross-sectional neuropsychological markers but also longitudinal characterization of how these markers change over time.

Encouragingly, large-scale collaborations focused on antisocial youth have begun to adopt this approach. The Adolescent Brain Cognitive Development (ABCD) study is designed to acquire imaging and behavioral data across multiple sites and time points during adolescence (Casey et al., 2018), and the FemNAT-CD consortium has been assembled to address conduct disorder in adolescent females with a longitudinal, multi-site design (Freitag et al., 2018). These developments align with the broader move toward replicability and transparency in psychological research articulated by Munafò and colleagues (2017).

The clinical implication of this challenge is that the developmental picture available to psychologists is currently incomplete. Assessment of an adult with ASPD or psychopathy is inevitably a cross-sectional snapshot, and inferences about the developmental origins of the observed neurocognitive profile must be made cautiously and with awareness of the limits of the underlying research.

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8. Clinical Implications for Psychologists

The methodological challenges reviewed above have practical consequences that reach into everyday clinical practice. This section considers those consequences across three broad clinical activities: assessment, formulation, and treatment planning.

8.1 Assessment

Psychologists engaged in the assessment of individuals with suspected or diagnosed ASPD, particularly in forensic contexts, are called upon to render judgments about the presence, severity, and specific configuration of the disorder. The methodological literature reviewed in this manual suggests several principles of practice.

First, the choice of instrument should be deliberate and transparent. The PCL-R remains the most widely used and best-validated clinician-administered instrument in forensic and penitentiary populations (Hare, 1991), and its use is generally to be preferred over self-report measures when the assessment context and the individual's clinical presentation permit. When self-report measures are used, their limitations in this population must be borne in mind (Brinkley et al., 2001; Sellbom et al., 2007; Gonsalves et al., 2013), and their findings should

be integrated with information from other sources.

Second, cut-off scores must be applied with attention to context. The Cooke and Michie (1999) demonstration of cross-cultural variation in PCL-R scores supports the use of a lower cut-off for European samples than for United States samples, and clinicians should be explicit about the threshold they are applying and the rationale for it. Terminological drift—describing subclinical elevations as "high" psychopathy, for example—should be avoided both in written reports and in clinical communication.

Third, performance validity testing should be considered a standard component of neuropsychological evaluation in these populations. The AACN 2021 consensus statement (Sweet et al., 2021) offers detailed guidance, and its recommendations apply with particular force to a population whose clinical features include the very traits that raise concern about the validity of test performance.

Fourth, task selection in neuropsychological evaluation should reflect awareness of the conceptual and psychometric issues reviewed in Section 7.2. When empathy or emotional processing is a focus of assessment, tasks capable of distinguishing cognitive from affective components should be preferred where available.

8.2 Formulation

Case formulation in ASPD and psychopathy benefits from the biocognitive stratification framework advanced by Brazil and colleagues (2018). Rather than treating ASPD as a homogeneous entity, the clinician can attend to the specific pattern of affective, motivational, and attentional dysfunctions that the individual presents, and can use this pattern to generate hypotheses about the mechanisms that maintain the presenting problems.

Distinguishing ASPD+P from ASPD-P is a first, essential step. The former is characterized by hyporesponsivity to others' distress, reinforcement-learning impairments, and callous-unemotional interpersonal features; the latter, by hyperresponsivity to threat, reactive aggression, and a more clearly stress-driven behavioral profile (Kosson et al., 2006; De Brito et al., 2013; Hodgins et al., 2018; Schönenberg & Jusyte, 2014). These profiles imply different formulations of what drives the presenting problems and, correspondingly, different formulations of what might change.

Within each of these broad profiles, further refinement is possible. Attention to specific reinforcement-learning capacities, to the automaticity of perspective-taking, and to the individual's response to different types of emotional stimuli can inform a more granular formulation. The available evidence base does not yet support fully individualized biocognitive profiling in clinical practice, but the direction of movement in the research literature makes this an increasingly realistic future goal.

8.3 Treatment Planning

Evidence for successful treatment of ASPD remains limited (Gibbon et al., 2020; Khalifa et al., 2020). This sobering fact should not, however, discourage careful, mechanism-informed intervention. The demonstration by Baskin-Sommers, Curtin, and Newman (2015) that cognitive remediation targeted at specific cognitive-affective dysfunctions can produce meaningful change in externalizing offender subtypes offers proof of principle that stratified, mechanism-informed intervention is possible.

The neurocognitive framework has several concrete implications for treatment planning. First, interventions that rely heavily on the delivery of aversive consequences may fail in populations characterized by punishment-processing impairments (Glimmerveen et al., 2022a), and their use should be complemented or replaced by interventions that engage reward-based learning. Second, the use of individualized, tangible rewards may enhance engagement and learning in this population (Glimmerveen et al., 2022b). Third, for individuals with ASPD-P whose behavior is driven by threat-based reactivity, interventions targeting hostile attribution biases and stress-response regulation may be particularly indicated. Fourth, for individuals with ASPD+P, interventions that target automatic perspective-taking and affective responsivity to others' distress may be more relevant than interventions focused on abstract reasoning about mental states, which is typically preserved.

Across all of these considerations, the fundamental principle is that treatment for ASPD and psychopathy should be informed by mechanism, and mechanism should be identified through careful, psychometrically rigorous neurocognitive assessment.

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9. Future Directions and Research Gaps

Griem, Kolla, and Tully (2022) advance a coherent program for future research that emerges naturally from the four challenges they identify. This section elaborates on that program and considers its implications for the trajectory of the field.

The most encompassing recommendation is for a shift toward large-scale, collaborative, pre-registered research designs. The advantages of such designs are multiple. Sample sizes adequate to detect the moderate effects that characterize this literature can be assembled by pooling across sites. Standardization of instruments and tasks across sites reduces the between-study heterogeneity that has plagued the field. Pre-registration constrains the flexibility of analytic decisions that can otherwise inflate false-positive rates. The reforms advocated by Munafò and colleagues (2017) in their manifesto for reproducible science apply with particular force to a field in which findings have been notably inconsistent.

Within this collaborative framework, the field would benefit from convergence around a small number of well-validated tasks for each neurocognitive domain of interest. Task selection should be informed by rigorous test–retest reliability work of the kind advocated by Blair and colleagues (2022). Where competing conceptualizations of a construct exist—as is the case for empathy, mentalizing, and psychopathy itself—multiple metrics can be included in the same study, allowing empirical adjudication among constructs on the basis of their relations to biomarkers and clinical outcomes.

Longitudinal designs should be prioritized. The ABCD study (Casey et al., 2018) and the FemNAT-CD consortium (Freitag et al., 2018) provide models. Future longitudinal work would benefit from thorough neurocognitive characterization at each time point, not merely behavioral or structural neuroimaging measurement. Sex differences in developmental trajectories should be a focus rather than an afterthought (Tully et al., 2022).

Task engagement should be systematically addressed in the design of neurocognitive protocols. This includes the use of tangible or personalized rewards, the incorporation of attention manipulation checks and performance validity indices, careful calibration of task difficulty, and uniform delivery of instructions (Glimmerveen et al., 2022b; Greher & Wodushek, 2017; Sweet et al., 2021; Cornacchio et al., 2017; Ging-Jehli et al., 2021). Where possible, incentive structures that align with the participant's own motivational profile should be explored.

Several specific empirical gaps warrant attention. The relative contributions of cognitive and affective empathy dysfunctions across the ASPD+/-P spectrum require further study using tools such as the EmpaToM (Kanske et al., 2015). The developmental trajectory of the exaggerated attentional bottleneck posited by response modulation theory (Baskin-Sommers & Brazil, 2022) is largely uncharacterized. The trajectories of "successful" psychopathy—patterns of trait elevation without commensurate life impairment—require longitudinal characterization (Lasko & Chester, 2021). And, as noted, sex differences across the developmental course of ASPD and psychopathy demand focused inquiry (Tully et al., 2022).

Ultimately, these lines of work converge on a common goal: the development of a personalized medicine approach to ASPD and psychopathy in which neurocognitive markers serve as treatment targets. This goal is ambitious but not implausible. The demonstration by Baskin-Sommers and colleagues (2015) that targeted cognitive remediation can alter cognitive-affective dysfunction in externalizing offender subtypes suggests that mechanism-informed intervention can produce meaningful change. Realizing this goal at scale, however, requires the resolution of the methodological challenges reviewed in this manual.

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10. Conclusion

Antisocial personality disorder and psychopathy are among the most challenging conditions that clinical, forensic, and neuropsychological practitioners encounter. Their prevalence in forensic and penal settings, their substantial personal, social, and financial costs, and the limited evidence base for effective treatment together create both an urgent clinical need and a substantial scientific opportunity. Neurocognitive assessment offers a route to progress by identifying the specific affective, motivational, and attentional dysfunctions that characterize these disorders and by translating that identification into targeted intervention.

Yet, as Griem, Kolla, and Tully (2022) have persuasively argued, the promise of this approach has been constrained by four interlocking methodological challenges: the inconsistent characterization of phenotypically heterogeneous samples, the unreliable and inconsistent design and selection of neurocognitive tasks, the difficulty of ensuring adequate task

engagement in a population whose pathology directly undermines it, and the paucity of longitudinal research capable of characterizing developmental trajectories. Each of these challenges is tractable. Standardization through collaborative protocols, convergence around validated tasks with established psychometric properties, careful attention to engagement through incentive design and performance validity testing, and the extension of large-scale longitudinal designs to include thorough neurocognitive assessment can, together, transform the field.

For the practicing psychologist, awareness of these challenges is not merely of academic interest. It shapes the way findings from the research literature are interpreted, the way assessment instruments are selected and applied, the way case formulations are constructed, and the way interventions are planned. A psychologist who understands why the empathy literature in ASPD is inconsistent is better equipped to evaluate the significance of a null finding on a particular task. A psychologist who understands why performance validity testing matters in this population is better equipped to render defensible neuropsychological opinions. A psychologist who understands the biocognitive dissociation between ASPD+P and ASPD-P is better equipped to formulate a case and select an intervention.

The path forward, as Griem et al. (2022) conclude, requires a consistent, collaborative approach guided by scientific discourse among experts. It requires the discipline of pre-registration, the transparency of open science, and the humility to acknowledge that the current evidence base does not yet support the personalized medicine approach that the field aspires to. It also requires the ambition to pursue that goal seriously, because the personal, social, and financial burdens associated with ASPD and psychopathy demand nothing less. Neurocognitive science can be part of the answer, but only if it is done well.

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