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Executive function, attention, and memory deficits in antisocial personality disorder and psychopathy

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ABSTRACT

Antisocial personality disorder (ASPD) and psychopathy attempt to represent individuals demonstrating callousness and disregard for others. ASPD has been criticized for capturing a heterogeneous population whilst missing the essence of the diagnosis by neglecting interpersonal/affective deficits which measures of psychopathy include. This heterogeneity in operationalizations has led to diverse findings without clear understanding of what characterizes this broader population. This study sought to clarify the neuropsychological profiles of ASPD and psychopathy. The Cambridge Neuropsychological Test Assessment Battery was administered to 85 adult male offenders in a personality disorder secure service and to 20 healthy controls. Of patients with ASPD, 46% met criteria for psychopathy. Of those with psychopathy, 89% met criteria for ASPD. There were two sets of comparisons: ASPD versus other personality disorders versus controls and psychopathy versus other personality disorders versus controls. ASPD showed deficits across executive functions, visual short-term and working memory, and attention (compared with controls). Psychopathy showed deficits limited to attention, complex planning, inhibitory control, and response reversal. Response reversal and visual search deficits appeared specific to ASPD and psychopathy versus other personality disorders and may underpin antisocial traits. Additional deficits in inhibitory control and working memory appeared to distinguish ASPD from other personality disorders.

1. Introduction

It has long been recognized that a small group of individuals, characterized by callousness and a lack of concern for others as well as antisocial behavior, is responsible for a disproportionate amount of crimes committed in society (Dolan and Doyle, 2000; Salekin, 2008). Attempts to characterize this group in common diagnostic manuals have produced various iterations of disorders, focusing on social, affective or behavioral aspects of the presentation. The Diagnostic and Statistical Manual (DSM; American Psychiatric Association [APA], 1952) first included “sociopathic personality disturbance”, a disorder identified in terms of lack of conformity with the prevailing milieu. DSM-II (APA, 1968), described a similar condition, “antisocial personality”, instead with a broadly similar description though it was recognized that deviant behavior was in itself insufficient to diagnose the disorder but that emotional deficits had to also exist. The term “antisocial personality disorder” (ASPD) first appeared in DSM-III (APA, 1980) and has since been retained. The diagnostic criteria for this disorder have focused mainly on observable behavior at the expense of emotional features.

A parallel development in the psychological literature saw the term “psychopathy” develop, based originally on Cleckley's (1941) description which emphasized affective and interpersonal traits culminating in the operationalization of the concept in Hare's psychopathy Checklist, since redeveloped and now in its current form, the PCL-R (Hare, 2003). This has been subjected to various analyses, identifying a Factor 1, containing affective and interpersonal features and Factor 2 with mainly antisocial characteristics, the latter overlapping with DSM criteria. The current DSM (APA, 1994, 2000, 2013) diagnosis of ASPD has been criticized for capturing a heterogeneous population and for not sufficiently representing interpersonal and affective deficits (such as callousness, lack of empathy, grandiosity, etc.) thought to be important features of individuals presenting with persistent antisocial, and in particular violent, behavior (Millon et al., 1998; Widiger et al., 1996; Widiger and Corbitt, 1993). In the fifth edition of DSM, a proposal was introduced for revising the diagnostic criteria of ASPD in line with the

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criteria defining psychopathy though ultimately ASPD criteria were retained unchanged.

Though sometimes used interchangeably, the two concepts, ASPD and psychopathy, are therefore distinct. This is not only demonstrated by the fact that the criteria differ but also as most people with a diagnosis of psychopathy also meet criteria for ASPD while the reverse is not the case (Hare, 2003). Together with the additional interpersonal and affective deficits and more severe offending characterizing psychopathy, this has led some authors to suggest that psychopathy represents a more severe end of ASPD (De Brito and Hodgins, 2009). As psychopathy is a risk factor for violent recidivism (Hare, 2003), it's presence is often assessed in addition to ASPD, particularly in forensic settings.

The heterogeneity in operationalizations has led to significant variation in findings across studies without clear understanding of what underpins antisocial traits. This highlights a need for better understanding of the specific subgroups in this broader population. To this end, the literature has drawn increasingly on neuroscience.

Neuropsychological studies on ASPD provide evidence of deficit in cognition across executive functions involving planning and set shifting (ability to work on a conceptual level), inhibitory control, and response reversal, especially where more challenging or specialized tasks or conditions are used (Barkataki et al., 2005, 2008; De Brito et al., 2013; Dolan, 2012; Dolan and Park, 2002). Other than in executive functions, deficits have been noted in sustained attention, short-term, and long-term memory (Barkataki et al., 2005, 2008; Dolan and Park, 2002). There are often inconsistencies in the deficits detected among studies or unusual patterns such as no deficit in working memory (e.g., Barkataki et al., 2005; De Brito et al., 2013) when other, related functions in tasks that involve overlapping neural substrates such as planning and short-term memory are impaired (Goel et al., 2001; Owen, 2000; Phillips, 1999; Smith and Jonides, 1999; Welsh et al., 1999). Such discrepancies may be partly due to differences in how specialized the tests used are (e.g., the set-shifting subtest of the CANTAB which reinforces a set rather than the Wisconsin Card Sorting Test [WCST], or a cancellation task versus a continuous performance task; Barkataki et al., 2005; Dolan and Park, 2002) or less sensitive assessment (e.g., use of non-computerized tests where random error may be higher). This highlights a need for better assessment in this regard. Overall, deficits in ASPD implicate tasks thought to be sensitive to frontotemporal lobe functioning (planning, set shifting, inhibitory control, memory, etc.; De Brito et al., 2013; Dolan, 2012; Dolan and Park, 2002).

In an attempt to understand the pattern of deficits in ASPD to explain its broadly erratic presentation and a possible role for psychopathy in defining subtypes in this population, theorists have attempted to distinguish between “cool” and “hot” executive functions (De Brito et al., 2013). The former involves cognitive effort and reasoning (e.g., planning, set-shifting, response inhibition) with focus on the lateral and dorsolateral prefrontal cortex. The latter refers to behavior regulation based on contingencies of reinforcement (e.g., response reversal) with focus on the ventromedial circuitries (De Brito et al., 2013; Zelazo and Carlson, 2012) and is thought to be particularly relevant to features of psychopathy (De Brito et al., 2013). However, as findings have been mixed for most neuropsychological functions and deficits in ASPD have spanned “cool” and “hot” executive functions regardless of psychopathy (e.g., De Brito et al., 2013), it seems that ASPD is characterized by a range of neuropsychological deficits, though replication is needed to establish this further.

The literature on psychopathy has focused on affective functions and “hot” executive functions. Studies have indicated deficits in complex tasks integrating cognitive and motor regulation such as planning in Porteus Mazes (Dolan and Park, 2002; Gao et al., 2009; Herpertz and Sass, 2000; Kiehl, 2006; Pham et al., 2003) together with deficits in the “hot” functions of response control such as response reversal and reinforcement-based decision-making (Blair, 2013). However, support for generalized frontal deficits or those more specific to dorsolateral prefrontal cortex and “cool” executive functions such as those relating to perseveration, set-shifting, fluency, visuomotor control, and working memory (in tests such as the Trail Making Test, set-shifting tests, WCST, Digit Span) has been inconsistent, with less support from studies using clinical samples and relatively more rigorous control (Blair, 2004, 2005; Gao et al., 2009; Hart et al., 1990; Ishikawa et al., 2001; Maes and Brazil, 2013; Navas Collado and Munoz Garcia, 2004; Pham et al., 2003; Seguin, 2004; Smith et al., 1992; Sommer et al., 2006). Memory deficits without emotional processing have not been supported either but testing may have not been sufficiently sensitive (e.g., Hart et al., 1990; Ishikawa et al., 2001).

The diversity in the findings may also reflect the heterogeneity of psychopathy as a population (Blackburn, 2009). For example, “cool” executive function deficits may be specific to individuals with psychopathy who score lower on anxiety or higher on the social deviance factor (Delfin et al., 2018; Maes and Brazil, 2013; Smith et al., 1992). Furthermore, studies have even reported better “cool” executive performance in non-offenders with psychopathy (“successful psychopaths”) or those scoring highly on the interpersonal/affective rather than the social deviance factors (Ishikawa et al., 2001; Maes and Brazil, 2013; Weidacker et al., 2017). Conversely, impairment in “hot” executive functions such as response reversal and impairment in attention to peripheral information has been associated with the interpersonal/affective features of psychopathy more strongly than with social deviance features and has been associated with the ventromedial/limbic circuitry (Dargis et al., 2017; Hamilton et al., 2015).

Interestingly, psychopathy has also been associated with broader neuropsychological deficits involving language especially with conceptual use, with deficits including semantic processing and comprehension of abstract words and metaphors, aspects of verbal expression such as slower speech, and so forth (Blair, 2005; Brites, 2016; Gao et al., 2009; Kiehl, 2006). Overall, the deficits in psychopathy primarily implicate tasks that are thought to be sensitive to ventromedial, orbitofrontal, and limbic regions (Baliousis et al., 2018; Blair et al., 2005) in a pattern that has led theorists to suggest that psychopathy is characterized by impaired integration among neural networks during complex operations such those drawing on combinations of cognitive, emotional, and attentional processes (Hamilton et al., 2015). However, this relatively new theory requires further testing.

The divergence in neuropsychological findings between ASPD and psychopathy indicate that these labels may reflect largely different populations. However, there are some overlapping deficits and these may underpin common features between the two operationalizations such as social deviance. However, ASPD and the social deviance factor of psychopathy do not appear to reflect the same population in light of findings such as “hot” executive deficits (that are associated less with social deviant features) in individuals with ASPD with and without psychopathy in probabilistic response reversal (or similar performance between patients with ASPD with and without psychopathy in non-probabilistic response reversal; De Brito et al., 2013; Dolan, 2012). This might suggest that ASPD and psychopathy are different constructs though this is difficult to establish as studies on either operationalization have focused on different functions with different assessment methods. Whilst some studies have examined psychopathy within ASPD and report impaired affective processing in the former but not the latter (De Brito et al., 2013; Dolan, 2012; Kosson et al., 2006), this may be specific to the subset of individuals with ASPD and psychopathy rather than the broader population of each of these operationalizations. Establishing the neuropsychological profiles of ASPD and psychopathy could help validate the two constructs and their potential relationship further.

There is also considerable overlap between deficits in ASPD and psychopathy and other clinical populations. For example, planning, inhibitory control, cognitive flexibility, attention, and memory difficulties have also been detected in Borderline Personality Disorder though “hot” executive functions such as response reversal have not been emphasized (Hagenhoff et al., 2013; Rentrop et al., 2008; Ruocco,

2005) whilst executive function deficits have been found in offenders generally sometimes with stronger effects than in ASPD (Ogilvie et al., 2011). Clarifying what neuropsychological deficits characterize ASPD and psychopathy relative to other clinical populations could improve understanding of needs and help develop interventions.

Following from the above, this project aimed to further elucidate what cognitive deficits underlie antisocial traits and delineate the neuropsychological profiles of the ASPD and psychopathy subgroups of this broader population, with focus on cognitive functions that have been most contested (i.e., frontotemporal rather than affective deficits), via a parallel examination of ASPD and psychopathy versus other personality disorders and healthy controls. We used the extensively validated and sensitive Cambridge Neuropsychological Test Assessment Battery (CANTAB; Strauss et al., 2006) to select tests relevant to the deficits we expected to find.[1] Based on the previous literature, we expected that ASPD will be associated with extensive frontotemporal deficits. We expected deficits in psychopathy consistent with the view of deficits in more complex operations requiring greater information integration such as planning solutions to complex problems and "hot" executive functions such as response reversal. We did not expect deficits in attention or memory or inhibitory control in psychopathy as these have not been supported in most rigorous studies. We expected the deficits in response reversal to be unique to antisocial patients (ASPD or psychopathy) versus patients with other personality disorders. Consequently, the hypotheses regarding ASPD and psychopathy could be delineated as:

1 ASPD will show deficits in planning, set shifting, inhibitory control, response reversal, sustained attention, and memory. Response reversal will only be found in patients with ASPD.
2 Psychopathy will show deficits in planning for complex problems only, response reversal, and attention. Response reversal will only be found in patients with psychopathy.

2. Materials and methods

2.1. Participants

Participants were male patients in the personality disorder service in a medium secure hospital in the UK ($n = 102$) and a healthy control group of male staff from the same hospital ($n = 20$). All patients had forensic histories and were transferred under restrictions either from prisons on a voluntary basis or from high secure hospitals. The most frequent offence was robbery or assault with intent to rob (27%), followed by assault with actual bodily harm (24%), burglary or attempt to burgle (19%), grievous bodily harm (15%), and arson or related threat (14%), with other offences occurring less frequently. Participant characteristics are shown in Table 1.

Patients were recruited from consecutive admissions (1999–2011) and had mean age of 30.8 years and mean IQ of 88.3 (Wechsler Adult Intelligence Scale, version 3; Wechsler, 1997). Patients were divided into those with and without ASPD and those with and without psychopathy on the basis of standardized preadmission clinical assessments by appropriately trained senior healthcare professionals (three patients were not assessed for psychopathy). These assessments included the DSM module from the International Personality Disorder Examination (IPDE; Loranger, 1999) and the Psychopathy Checklist – Revised (PCL-R, European cut off of 25; Hare, 2003), the latter using information from both patient interviews and file reviews. We excluded patients with psychosis or bipolar disorder at preadmission using standardized instruments (Schedule for Affective Disorders and Schizophrenia and Structured Clinical Interview for DSM-IV Disorders-Axis I; First et al., 2002; Spitzer and Endicott, 1978). Patients were free of substance misuse (regular drug screens). We also excluded patients with IQ denoting impairment (IQ < 70, Wechsler Adult Intelligence Scale, version 3; Wechsler, 1997) or serious traumatic brain injury (unconsciousness exceeding 30 min following head trauma; American Congress of Rehabilitation Medicine, 1993).

Of the 102 patients assessed on the CANTAB, 17 did not meet inclusion criteria. Reasons were traumatic brain injury ($N = 10$), psychosis and bipolar disorder unrecognized at the time of admission ($N = 4$), or IQ < 70 ($N = 3$). In the final patient sample ASPD and psychopathy overlapped. Of patients with ASPD, 46% ($N = 24$) also met criteria for psychopathy whereas of those without ASPD, 9% ($N = 3$) met criteria for psychopathy. Of patients with psychopathy, 89% ($N = 24$) met criteria for ASPD whereas of those without psychopathy, 47% ($N = 26$) met criteria for ASPD. Fig. 1 shows the level of overlap among the patients.

For the control group, from ancillary staff at the service, inclusion criteria were: (a) age at least 18 years; and, (b) absence of a history of personality disorder, major mental illness, neurological condition, traumatic brain injury, and learning disability. Current or past substance misuse and current psychotropic medication were emphasized as exclusions at recruitment but ethical approval did not permit direct questioning. Controls were assessed for Axis I and II diagnoses (Mini International Neuropsychiatric Interview and IPDE Screening Questionnaire; Loranger, 1999; Sheehan et al., 1998), psychopathy (Psychopathy Checklist: Screening Version; Hart et al., 1995), and intellectual functioning (IQ ≥ 70, Quick Test; Ammons and Ammons, 1962) by a trained researcher.

The study was approved by the appropriate National Health Service Health Research Authority ethics committee. Participants gave informed consent prior to participation.

2.2. Materials

The CANTAB was used for neuropsychological assessment. It is a comprehensive computerized neuropsychological assessment battery of functions associated primarily with the frontotemporal lobe with sensitivity and selective focus of tests (Strauss et al., 2006). The CANTAB has adequate to high internal consistency, adequate or high test-retest reliability for most tests, and good ability to discriminate between cognitive functions, cerebral regions, and neurological conditions (Cambridge Cognition, 2008; Fray et al., 1996; Sahakian and Owen, 1992; Strauss et al., 2006). The battery has shown good validity in ASPD and psychopathy with sensitive measurement in several functions (e.g., planning, set-shifting, inhibitory control; De Brito et al., 2013; Dolan and Park, 2002; Mitchell et al., 2002). We administered the subtests detailed below (see manual, Cambridge Cognition, 2006) to target attention, the executive functions (working memory, planning, response reversal, set shifting, inhibitory control), and memory (visual and verbal) involved in the hypotheses. Following the existing conceptualization of "hot" and "cool" executive functions (De Brito et al., 2013), we considered response reversal as "hot" with the remaining executive functions as "cool".

2.2.1. Attention

Sustained attention – Rapid Visual Processing (RVP): A continuous performance test (Lezak et al., 2012), with a small working memory component in which the participant has to identify and respond to three different three-digit sequences (targets) in a series of single-digit numbers. Outcomes were the total number of hits (correct responses).

Visual search – Matching to Sample (MTS): Participants select a pattern that matches a target in color and shape among two, four, and eight alternatives. We assessed attention via the total number of errors. We recorded reaction time to control for effects on performance.

[1] Decision-making is considered a "hot" cognitive function (De Brito et al., 2013) but the relevant CANTAB subtest, Cambridge Gambling Task (CGT) was introduced later into our battery and the sample was insufficient for analysis.

Table 1
Sample characteristics (means with standard deviations and frequencies) for Antisocial Personality Disorder (ASPD), psychopathy (Psy), and healthy controls, with different superscripts indicating significantly different means for each set of comparisons.

Study groups	ASPD	Non-ASPD	Healthy control	Psy	Non-psy
N	52	33	20	27	55
Additional PDs					
N	1.7(2.2)	1.7(1.1)	(none)	2.6(1.4)	2.2(1.2)
ASPD	(all)	(none)		89%	46%
Borderline	67%	36%		56%	55%
Paranoid	35%	30%		30%	33%
Avoidant	29%	30%		22%	33%
Other	<12%	<6%		<15%	<6%
Age (years)	30.3(8.9)*	37.8(9.2)*	33.9(10.7)	34.3(10.9)	31.5(8.8)
IQ	87.8(10.1)	93.7(18.1)	87.6(13.0)	86.5(13.9)	91.0(13.1)
Education (years)					
Basic	9.8(2.0)	10.1(2.4)	11	9.7(2.5)	9.9(2.0)
Advanced	0.2(0.5)**	0.2(0.7)**	3.4(2.2)**	0.1(0.3)**	0.1(0.6)**
PCL-R	24.0(4.9)***	15.9(6.4)***	–	28.0(2.7)***	17.7(5.2)***
N of additional PDs	1.7(1.2)	1.7(1.1)	(none)	2.6(1.4)	2.2(1.2)
SRD	55.1%*	27.1%*	(none)	30.8%*	51%*
Medication			(none)		
Antidepressant	14.8%*	19.3%*		9.4%	25.9%
Antipsychotic	21.6%*	5.7%*		11.8%	14.1%
Mood stabilizer	9.1%	4.5%		5.9%	7.1%

Notes. PDs = Personality disorders; PCL-*R* = Psychopathy Checklist-Revised; SRD = Substance-related disorder.
* $P < 0.05$.
** $P < 0.01$.
*** $P < 0.001$.

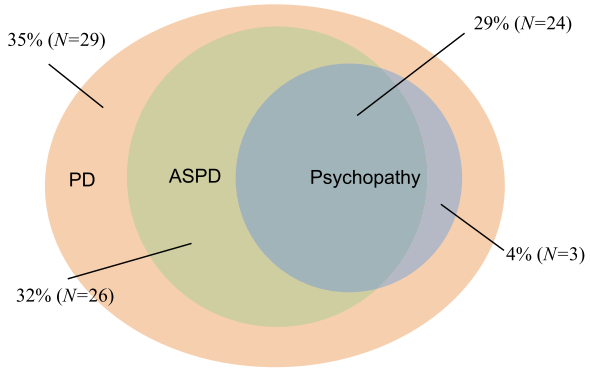

Fig. 1. Venn diagram showing the degree of overlap between Antisocial Personality Disorder (ASPD/PD) and psychopathy in the project's sample. Percentages reflect proportions of the total sample of patients ($N = 82$ rather than 85 as three patients had missing Psychopathy Checklist – Revised).

2.2.2. Executive functions

Spatial Working Memory (SWM). A self-ordering task targeting ability to hold and manipulate visuospatial information. Stimuli are presented in four blocks of increasing number of stimuli (four, six, and eight). We recorded number of errors.

Planning – Stockings of Cambridge (SOC; Fray et al., 1996): A problem-solving task analogue to the Tower of London. There are four levels of increasing difficulty where problems require two, three, four, or five moves to reach a solution. We examined perfect solutions (fewest moves), average number of moves per solution, initial thinking times (prior to executing solution), and subsequent thinking times.

Response reversal and set shifting – Intra/Extradimensional Set Shift (IED; Fray et al., 1996): A WCST analogue where participants respond based on attentional set (category; e.g. purple shapes vs. white lines) with computer feedback. During the task, participants have to shift focus either within the same (intradimensional shift) or a different set (extradimensional shift; EDS). Response reversal is required four times before the extradimensional shift and once afterwards. We assessed response reversal via total reversal errors (pre-EDS to avoid systematic loss of participants who did not progress beyond this stage) and ability to shift set via EDS errors (cf., Mitchell et al., 2002).

Inhibitory control – Affective Go/NoGo (AGN; Schulz et al., 2007). Participants respond to either emotionally positive (e.g., joyful) or negative target words (e.g., hopeless) in blocks. The rule is reversed every two blocks. We assessed difficulty inhibiting inappropriate responses via the number of commission errors (responding to non-targets). We recorded response latencies to control for quicker responding.

2.2.3. Memory

Visual immediate and delayed short-term memory – Delayed Matching to Sample (DMS). This test requires pattern recognition. The patterns and target are presented either simultaneously, in immediate succession, and after a four- and a twelve-second delay. We assessed memory via the number of correct responses. We recorded response latency to control for quicker responding.

Visual short-term memory capacity – Spatial Span (SSP). This is a visuospatial analogue of the Digit Span test. We recorded span length: the longest sequence of boxes which participants recalled.

Verbal short- and long-term memory – Verbal Recognition Memory (VRM). Free recall and immediate and delayed recognition of 12 words. Long-term recognition is assessed after a 20-min interval (after the DMS). We assessed memory via number of correct responses.

2.3. Procedure

Assessments were administered in a standardized manner according to manuals. The CANTAB tests were administered over five sessions in the standard battery order on an IBM compatible computer with Intel Pentium 4 processor (1.7 GHz), 256 MB RAM, Windows XP, touch screen monitor and press pad as appropriate (Cambridge Cognition, 2006). The VRM was administered before and after the DMS to enable the required delay in testing. The cognitive assessment lasted approximately 1.5 h in total. Diagnostic assessments for patients were completed at preadmission. For the control group, these were completed prior to CANTAB testing. Control participants received a modest fee following participation. Patients did not receive any benefits for participation.

The Affective Go/NoGo (AGN) and Verbal Recognition Memory (VRM) were introduced later in the personality disorder service and had smaller samples (38 ASPD vs. 16 non-ASPD; 22 individuals with psychopathy vs. 29 without).

2.4. Design and data analysis

In this quasi-experimental design, group was the between-subjects variable and CANTAB outcome measures were dependent variables. Some tests involved within-subjects factors (SOC, DMS, & SWM). The effects of mediating variables such as participant characteristics (Table 1) and attention were evaluated and controlled for only where these were significantly different between groups and correlated with outcomes. We also examined performance validity by establishing that outcome measures changed in line with task difficulty in tasks with within-subjects factors, though details are not reported for economy.

We screened for outliers (z-scores greater than 3.29; Tabachnick and Fidell, 2013) and excluded them. We assessed assumption violations according to standard practice. We examined normality via histogram inspection, Kolmogorov-Smirnov test, and skewness and kurtosis (significant at $\alpha = 0.001$). We assessed linearity visually via bivariate scatter plots, homogeneity of variance (ANOVA) via Levene's test, and sphericity (repeated-measures) via Mauchly's test (Field, 2013). Multicollinearity (MANOVA) was considered present in bivariate correlations with $r > 0.07$ and tolerance approaching zero or condition index exceeding 30 coupled with variance proportions greater than 0.5 for at least two different variables (Belsey et al., 1980; Tabachnick and Fidell, 2013).

Analyses involved two parallel sets of comparisons, each with three groups: (a) patients with ASPD versus patients with personality disorders (not ASPD) versus healthy controls, and (b) patients with psychopathy versus patients without psychopathy (but with personality disorders including ASPD) versus healthy controls. In essence, the first comparison focused on deficits in ASPD as one construct and the other comparison focused on deficits in psychopathy as another construct. The total sample size of 90–100 individuals (among three groups) was in line with sample requirements for small to medium effect sizes in ANOVA with three groups ($f = 0.33$, $\alpha = 0.05$, power = 0.80), determined using G*Power (Buchner et al., 2009; Faul et al., 2007).

Supplementary analyses were conducted after removing individuals with psychopathy from the group with ASPD (ASPD-only group vs. other personality disorders vs. healthy controls). The converse was not possible (psychopathy without ASPD) due to small samples. In some cases, we also removed individuals with ASPD from the group without psychopathy when these performed worse than individuals with psychopathy to gain clarity on the role of ASPD in these deficits. ANOVA methods were employed to test for between and within-groups effects and control for covariates (Field, 2013; Tabachnick and Fidell, 2013). Where heterogeneity of regression slopes failed, multilevel modeling (MLM) was used to control for covariates (Goldstein, 2003). Results from controlling for covariates and for the ASPD-only analyses were reported only when different to the main analysis, for economy. MANOVA was conducted for IED reversal and EDS errors. Bonferroni post hoc comparisons were selected to limit Type I error (Field, 2013). Test effects (e.g., task difficulty) were examined for validity of manipulation but not reported (beyond the study's scope). Where variance or normality assumptions were violated, Pillai's Trace (relatively robust) and the Kruskal-Wallis test (nonparametric) were used (Field, 2013; Tabachnick and Fidell, 2013). We used partial η^2 as effect size in ANOVA. As this reflects variance across groups, we also used Cohen's *d* with pooled standard deviation (Cohen, 1992) to represent the magnitude of deficit in ASPD and psychopathy compared specifically to healthy controls (Table 3). For all statistical tests, α was 0.05 unless otherwise specified.

3. Results

3.1. Sample characteristics

Patients with ASPD were younger: $F(2, 72) = 3.8$, $P < 0.05$; had higher PCL-R scores: $F(1, 80) = 37.2$, $P < 0.001$, and were more frequently diagnosed with substance-related disorder (SRD) compared to the non-ASPD group, $\chi^2(1) = 4.2$, $P < 0.05$. Furthermore, they were prescribed antidepressants less frequently, $\chi^2(1) = 6.2$, $P < 0.05$ but received antipsychotics more often than the non-ASPD group, $\chi^2(1) = 4.4$, $P < 0.05$. The healthy control group had completed more years in further education compared to patients with and without ASPD, $F(2, 102) = 55.9$, $P < 0.01$. The ASPD, non-ASPD and healthy control groups were comparable on IQ, $F(2, 102) = 1.2$, $P > 0.05$, years in basic education, $F(2, 102) = 2.97$, $P > 0.05$, number of additional personality disorders (patients only), $F(1, 74) = 0.03$, $P > 0.05$, and prescription of mood stabilizers (patients only), $\chi^2(1) = 0.2$, $P > 0.05$.

The groups with and without psychopathy were different (by definition) on PCL-R, $F(1, 80) = 88.8$, $P < 0.01$, and history of SRD, $\chi^2(1) = 5.2$, $P < 0.05$. Again, healthy controls completed more years in further education than patients with and without psychopathy, $F(2, 99) = 55.9$, $P < 0.01$. The groups with psychopathy, without psychopathy, and healthy controls were comparable on age, $F(2, 69) = 1.0$, $P > 0.05$, IQ, $F(2, 99) = 0.9$, $P > 0.05$, years in basic education, $F(2, 99) = 2.97$, $P > 0.05$, number of additional disorders (patients only), $F(1, 74) = 1.3$, $P > 0.05$; and prescription of antidepressants, $\chi^2(1) = 0.8$, $P > 0.05$, antipsychotics, $\chi^2(1) = 2.1$, $P > 0.05$, and mood stabilizers (again, patients only), $\chi^2(1) = 0.9$, $P > 0.05$.

Excluding individuals with psychopathy from the ASPD group resulted in a group of 26 patients with ASPD only (for supplementary analyses). Healthy controls had received significantly more years of basic education than this ASPD-only group, $F(2, 57) = 3.9$, $P < 0.05$, but groups were comparable otherwise, as above.

Missing data due to patients declining to complete tasks were between 6–8% for all tests except for RVP where the rate was 15%. Table 2 shows the relevant means and statistics (between-subjects) for all neuropsychological tests regarding the primary analyses. The results of interactions and supplementary analyses are summarized below. For all tests with within-subjects factors, performance changed significantly as expected according to task difficulty. An overview of neuropsychological difficulties in ASPD and psychopathy is presented in Table 3 with effect sizes for deficits specifically compared to healthy controls and shading to indicate uniqueness of deficit in ASPD and psychopathy.

3.2. Attention

Table 2 shows the results on the RVP and MTS subtests (using Kruskal-Wallis tests and then supplementary ANOVA to determine post hoc differences). There were no significant differences between ASPD, non-ASPD, and control groups in correct, $\chi^2(2) = 2.51$, $P > 0.05$, and error reaction times, $\chi^2(2) = 0.71$, $P > 0.05$. There were also no significant group differences between patients with and without psychopathy and controls in correct, $\chi^2(2) = 2.46$, $P > 0.05$, and error reaction times, $\chi^2(2) = 0.18$, $P > 0.05$.

3.3. Executive functions

3.3.1. Spatial working memory (SWM)

For the ASPD analysis, there was no significant main effect of group (Table 2) but there was a significant group x stage interaction, *Pillai's Trace* = 0.13, $F(4, 184) = 3.24$, $P < 0.05$, partial $\eta^2 = 0.07$. Unpacking the interaction suggested no significant group differences for four-box problems, $F(2, 93) = 0.09$, $P > 0.05$, partial $\eta^2 = 0.002$, and marginal effects for six, $F(2, 96) = 3$, $P = 0.05$, partial $\eta^2 = 0.06$, and eight-box problems, $F(2, 97) = 3.09$, $P = 0.05$, partial $\eta^2 = 0.06$, where the ASPD group potentially performed significantly worse than healthy controls

Table 2
Overall performance (means and standard errors) on neurocognitive tests for Antisocial Personality Disorder (ASPD) and psychopathy (Psy).

Neurocognitive outcomes	ASPD	Non-ASPD	HC	Psy	Non-Psy	Tests ASPD	Psy	Significant differences
Sustained attention (RVP hits)	12.4(0.8)	14.0(0.9)	18.6(1.2)	11.4(1.1)	13.8(0.7)	$\chi^2(2) = 16.2^{***}$ $F(2, 90) = 9.85^{***}$ partial $\eta^2 = 0.18$	$\chi^2(2) = 17.2^{***}$ $F(2, 87) = 10.65^{***}$ partial $\eta^2 = 0.20$	ASPD & Non-ASPD < HC Psy & Non-Psy < HC
Visual search (MTS errors)	46.2(0.2)	46.5(0.3)	47.3(0.3)	45.4(0.4)	46.2(0.3)	$\chi^2(2) = 8.85^{*}$ $F(2, 92) = 4.78^{*}$	$\chi^2(2) = 9.97^{**}$ $F(2, 93) = 4.86^{*}$	ASPD < HC Psy < HC
Spatial working memory (SWM errors)	8.37(0.80)	8.12(0.98)	5.13(1.20)	7.26(1.10)	8.72(0.78)	$F(2, 92) = 2.71$ partial $\eta^2 = 0.06$	$F(2, 89) = 3.2^{*,‡}$ partial $\eta^2 = 0.07$	(ASPD-only > HC) Non-Psy > HC
Planning (SOC)								
Perfect solutions	8.13(0.22)	8.52(0.27)	9.7(0.34)	8.46(0.31)	8.13(0.21)	$\chi^2(2) = 13.1^{**}$ $F(2, 97) = 7.49^{**}$ partial $\eta^2 = 0.13$	$\chi^2(2) = 13.6^{**}$ $F(2, 94) = 7.8^{**}$ partial $\eta^2 = 0.14$	ASPD & Non-ASPD < HC Psy & Non-Psy < HC
Average moves	4.39(0.06)	4.33(0.07)	4.04(0.09)	4.28(0.08)	4.41(0.06)	$F(2, 93) = 4.96^{**}$ partial $\eta^2 = 0.10$	$F(2, 90) = 5.90^{**}$ partial $\eta^2 = 0.12$	ASPD & Non-ASPD > HC Non-Psy > HC
Initial thinking time (ms)	3492.0 (393.8)	4976.9 (467.0)	6501.3 (590.7)	4023.5 (580.0)	4214.0 (385.8)	$F(2, 94) = 9.46^{***}$ partial $\eta^2 = 0.17$	$F(2, 92) = 5.65^{**}$ partial $\eta^2 = 0.11$	ASPD < HC (ASPD-only < Non-ASPD & HC) Psy & Non-Psy < HC
Subsequent thinking time (ms)	402.7(75.2)	662.8(87.9)	400.9(113.5)	452.7(110.9)	566.2(73.3)	$F(2, 86) = 2.92$ partial $\eta^2 = 0.06$	$F(2,84) = 0.84$ partial $\eta^2 = 0.02$	None
Set-shifting (IED EDS errors)	15.9(1.5)	14.0(1.9)	7.75(2.3)	12.4(2.1)	16.3(1.5)	$\chi^2(2) = 6.97^{†}$ $F(2, 94) = 4.45^{*}$ partial $\eta^2 = 0.09$	$\chi^2(2) = 9.97^{**}$ $F(2, 91) = 5.00^{**}$ partial $\eta^2 = 0.10$	ASPD > HC Non-Psy > HC
Response reversal (IED reversal errors)	4.06(0.17)	3.27(0.21)	3.30(0.26)	4.28(0.24)	3.53(0.17)	$\chi^2(2) = 9.94^{***}$ $F(2, 94) = 5.70^{**}$ partial $\eta^2 = 0.11$	$\chi^2(2) = 6.37^{†}$ $F(2, 91) = 4.36^{*}$ partial $\eta^2 = 0.09$	ASPD > Non-ASPD & HC (no effect for ASPD-only) Psy > HC
Inhibitory control (AGN commissions)	11.8(1.1)	9.2(1.7)	4.5(1.7)	12.6(1.5)	10.3(1.3)	$\chi^2(2) = 16.3^{***}$ $F(2, 67) = 6.62^{**}$ partial $\eta^2 = 0.17$	$F(2, 64) = 6.58^{**}$ partial $\eta^2 = 0.17$	ASPD > HC Psy & Non-Psy > HC
Visual immediate & delayed STM (DMS correct responses)	8.54(0.12)	8.61(0.15)	9.43(0.19)	8.52(0.17)	8.55(0.12)	$F(2, 98) = 8.63^{***}$ partial $\eta^2 = 0.15$	$F(2, 95) = 8.83^{***}$ partial $\eta^2 = 0.16$	ASPD & Non-ASPD < HC Psy & Non-Psy < HC
Visual STM capacity (SSP)	6.20(0.19)	6.09(0.23)	7.15(0.29)	6.50(0.25)	5.98(0.18)	$\chi^2(2) = 6.90^{*}$ $F(2, 96) = 4.64^{*}$ partial $\eta^2 = 0.09$	$\chi^2(2) = 9.44^{**}$ $F(2, 93) = 6.10^{**}$ partial $\eta^2 = 0.12$	ASPD & Non-ASPD < HC Non-Psy < HC (& Psy, after controlling for attention)
Verbal STM & LTM (VRM)								
Correct recalls	6.68(0.30)	6.75(0.39)	7.2(0.35)	6.63(0.37)	6.82(0.34)	$\chi^2(2) = 1.77$	$\chi^2(2) = 1.85$	None
Correct recognitions (overall)	22.9(0.2)	22.7(0.3)	22.9(0.3)	22.4(0.3)	22.9(0.3)	$F(2, 58) = 0.13$ partial $\eta^2 = 0.04$	$F(2, 56) = 1.07$ partial $\eta^2 = 0.04$	None

Notes. RVP = Rapid Visual Processing; MTS = Matching to Sample; SWM = Spatial Working Memory; SOC = Stockings of Cambridge; IED = Intra/Extradimensional Set Shifting; EDS = Extra-dimensional shift; AGN = Affective Go/NoGo; S/LTM = Short-/long-term memory; DMS = Delayed Matching to Sample; SSP = Spatial Span; VRM = Verbal Recognition Memory.

† Not significant after Bonferroni correction for multivariate testing, $P > 0.025$.

‡ Effect not maintained after controlling for RVP hits.

* $P < 0.05$.

** $P < 0.01$.

*** $P < 0.001$.

during eight-box problems only, post hoc $P < 0.05$. Excluding individuals with psychopathy from the ASPD group resulted in a significant main effect of group, $F(2, 69) = 3.27$, $P < 0.05$, partial $\eta^2 = 0.09$, where the ASPD-only group performed worse than healthy controls, but no significant group x stage interaction, *Pillai's Trace* = 0.17, $F(4, 138) = 2.94$, $P > 0.05$, partial $\eta^2 = 0.08$.

Regarding psychopathy, there was a significant main effect of group (Table 2) but no significant group x difficulty interaction, *Pillai's Trace* = 0.08, $F(4, 178) = 1.77$, partial $\eta^2 = 0.04$. The apparent deficit in the group without psychopathy may be attributed to presence of ASPD, as removal of those individuals resulted in a non-significant effect, $F(2, 67) = 1.72$, $P > 0.05$, partial $\eta^2 = 0.05$. Attention (RVP hits) appeared to contribute to these effects, $\Delta\chi^2 = 4.03$, $\Delta df = 1$, $P < 0.05$, which were not maintained after controlling for it.

3.3.2. Planning (SOC)

3.3.2.1. Perfect solutions and number of moves. All patient groups performed fewer perfect solutions compared to healthy controls (Table 2, using Kruskal-Wallis tests and then supplementary ANOVA to determine post hoc differences). Patients with and without ASPD performed more moves compared to controls (Table 2) but there was no group x difficulty interaction, *Pillai's Trace* = 0.12, $F(6, 184) = 1.89$, $P > 0.05$, partial $\eta^2 = 0.06$.

In the analysis with patients with ASPD only (no psychopathy), patient groups also made fewer perfect solutions, $F(2, 73) = 8,43$, $P < 0.05$, partial $\eta^2 = 0.19$, and more moves compared to healthy controls, $F(2, 71) = 5.90$, $P < 0.05$, partial $\eta^2 = 0.14$, and there was no group x difficulty interaction, *Pillai's Trace* = 0.15, $F(6, 140) = 1.84$, partial $\eta^2 = 0.07$.

Regarding patients with psychopathy versus patients without psychopathy versus controls, patients without psychopathy performed more moves than controls (Table 2). There was also a significant group x difficulty interaction, *Pillai's Trace* = 0.17, $F(6, 178) = 2.79$, $P < 0.05$, partial $\eta^2 = 0.09$. All participants performed 2-moves at the 2-move problems and there were no group differences for three-move problems, $F(2, 90) = 0.32$, $P > 0.05$, partial $\eta^2 = 0.01$. Individuals

Table 3
Summary of impairments and effect sizes in Antisocial Personality Disorder (ASPD) and psychopathy, shaded to illustrate the uniqueness of deficits in these populations.

Function	ASPD	Psychopathy
Attention		
Sustained attention (RVP)	1.34	1.38
Visual search (MTS)	1.38	2.43
"Cool" Executive functions		
Working memory (SWM)	† 0.77	0.39
Planning (SOC)	1.09	(Most challenging problems only) 0.89
Set shifting (IED)	0.82	0.50
Inhibitory control (AGN)	1.05	1.22
"Hot" Executive function		
Response reversal (IED)	0.59	1.40
Memory		
Visual STM (DMS & SSP)	1.06 & 0.75	1.07 & 0.50
Verbal (recall & recognition)	0.29 & 0.07	0.31 & 0.25

Notes. The effect size statistic is Cohen's *d* with pooled standard deviation (Cohen, 1992) to represent the magnitude of deficit in ASPD and psychopathy specifically compared to healthy controls. Dark shading indicates unique deficits where only patients with ASPD or psychopathy showed a deficit vs. healthy controls.
CANTAB = Cambridge Neuropsychological Test Assessment Battery; RVP = Rapid Visual Processing; MTS = Matching to Sample Visual Search; SWM = Spatial Working Memory; SOC = Stocking of Cambridge; IED = Intra/Extra-Dimensional Set Shifting; AGN = Affective Go/NoGo; DMS = Delayed Matching to Sample; SSP = Spatial Span; VRM = Verbal Recognition Memory.
†=ASPD-only group.

with personality disorders but not psychopathy performed worse than healthy controls during four-move problems, $F(2, 92) = 4.54$, $P < 0.05$, partial $\eta^2 = 0.09$, while both patient groups performed significantly worse than the healthy control group for five-move problems, $F(2, 92) = 4.39$, $P < 0.05$, partial $\eta^2 = 0.09$. The performance of patients without psychopathy did not appear related to ASPD as removal of patients with ASPD from that group led to comparable results.

3.3.2.2. Thinking times. The ASPD group showed less initial thinking time than controls (Table 2). A significant group x difficulty interaction, *Pillai's Trace* = 0.17, $F(6, 186) = 2.92$, $P < 0.05$, partial $\eta^2 = 0.09$, suggested that the ASPD group spent significantly less time planning compared to healthy controls for three, $F(2, 95) = 5.16$, $P < 0.01$, partial $\eta^2 = 0.10$, four, $F(2, 94) = 6.87$, $P < 0.01$, partial $\eta^2 = 0.13$, and five-move problems, $F(2, 96) = 5.96$, $P < 0.01$, partial $\eta^2 = 0.11$ (no differences for two-move problems, $F(2, 96) = 1.66$, $P > 0.05$, partial $\eta^2 = 0.03$). There were no significant differences on subsequent thinking times (Table 2) and the interaction was not significant, *Pillai's Trace* = 0.03, $F(6, 170) = 0.44$, $P > 0.05$, partial $\eta^2 = 0.02$.

Supplementary analyses indicated that the ASPD-only group spent significantly less time planning compared both to non-ASPD and healthy controls (who performed similarly to each other post hoc), $F(2, 70) = 12.41$, $P < 0.001$, partial $\eta^2 = 0.26$. There was a significant group x difficulty interaction, *Pillai's Trace* = 0.27, $F(6, 138) = 3.53$, $P < 0.05$, partial $\eta^2 = 0.13$. The ASPD-only group spent less time planning compared to non-ASPD and healthy controls during two-move, $F(2, 72) = 3.38$, $P < 0.05$, partial $\eta^2 = 0.09$, three-move, $F(2, 72) = 9.79$, $P < 0.001$, partial $\eta^2 = 0.21$, four-move, $F(2, 72) = 6.24$, $P < 0.01$, partial $\eta^2 = 0.15$, and five-move problems, $F(2, 73) = 6.33$, $P < 0.05$, partial $\eta^2 = 0.15$.

Regarding patients with psychopathy versus patients without psychopathy versus controls, both patient groups showed less initial thinking time than controls (Table 2) but the group x difficulty interaction for initial thinking times was not significant, *Pillai's Trace* = 0.09, $F(6, 182) = 1.44$, $P > 0.05$, partial $\eta^2 = 0.05$. There were no significant differences on subsequent thinking times (Table 2) and the interaction was not significant either, *Pillai's Trace* = 0.02, $F(6, 166) = 0.31$, $P > 0.05$, partial $\eta^2 = 0.01$.

3.3.3. Set shifting and response reversal (IED)

One outlier (with psychopathy, not ASPD) was removed due to extreme performance across this task. The ASPD group performed marginally more EDS errors compared to controls and more reversal errors compared to patients without ASPD and controls (Table 2). The effect relating to EDS errors was strengthened in the ASPD-only group, where Kruskal-Wallis tests revealed a significant main effect of group in the same direction for EDS errors, $\chi^2(2) = 8.94$, $P < 0.025$. However, the difference was marginally significant for reversal errors, $\chi^2(2) = 7.25$, $P = 0.027$. This marginal result may be due to loss of power as the group with psychopathy had also exhibited this deficit (Table 2).

Patients with personality disorder but not psychopathy made significantly more EDS errors than healthy controls (Table 2). This may be related to presence of ASPD in this patient group, as removal of these individuals resulted in a non-significant effect, $\chi^2(2) = 5.26$, $P > 0.05$, and, $F(2, 68) = 2.61$, $P > 0.05$, partial $\eta^2 = 0.07$. Patients with psychopathy performed more reversal errors compared to healthy controls (Table 2).

3.3.4. Inhibitory control (AGN)

Supplementary nonparametric testing was conducted for psychopathy due to deviations from normality. The ASPD group performed significantly more commission errors compared to controls (Table 2). Patients with and without psychopathy performed significantly more

commission errors compared to controls (Table 2).

There were no significant differences between groups in response latency, $F(2, 71) = 0.22$, $P > 0.05$, partial $\eta^2 = 0.06$, for ASPD versus non-ASPD versus controls; and $F(2, 68) = 0.09$, $P > 0.05$, partial $\eta^2 = 0.03$, for psychopathy versus no psychopathy versus controls. There was also no interaction with emotional valence of words, *Pillai's Trace* = 0.03, $F(2, 71) = 1.23$, $P > 0.05$, partial $\eta^2 = 0.03$, for ASPD versus non-ASPD versus controls; and *Pillai's Trace* = 0.04, $F(2, 68) = 1.32$, $P > 0.05$, partial $\eta^2 = 0.04$, for psychopathy versus no psychopathy versus controls.

3.4. Memory

3.4.1. Visual immediate and delayed short-term memory (DMS)

There was a significant main effect of group and a group x delay interaction for both sets of analysis, *Pillai's Trace* = 0.14, $F(6, 194) = 2.39$, $P < 0.05$, partial $\eta^2 = 0.07$, and *Pillai's Trace* = 0.17, $F(6, 188) = 2.39$, $P < 0.01$, partial $\eta^2 = 0.09$, for ASPD and psychopathy respectively. All patient groups performed significantly worse than healthy controls (Table 2).

For the ASPD analysis, unpacking the interactions revealed no significant differences in simultaneous presentation, $F(2, 98) = 1.20$, $P > 0.05$, partial $\eta^2 = 0.02$. However, patients with ASPD performed worse than healthy controls in immediate recognition, $F(2, 99) = 4.34$, $P < 0.05$, partial $\eta^2 = 0.08$, 4 s delayed recognition, $F(2, 99) = 5.41$, $P < 0.01$, partial $\eta^2 = 0.10$, and 12 s delayed recognition, $F(2, 99) = 6.2$, $P < 0.01$, partial $\eta^2 = 0.11$. At post hoc, patients without ASPD also performed worse than healthy controls during recognition after the 12 s delay only. Regarding response latencies, there were no significant differences between groups, $F(2, 97) = 0.83$, $P > 0.05$, partial $\eta^2 = 0.02$, and no interactions with task difficulty, *Pillai's Trace* = 0.08, $F(6, 192) = 1.33$, $P > 0.05$, partial $\eta^2 = 0.04$.

Regarding psychopathy, unpacking the interactions revealed no significant differences in simultaneous presentation, $F(2, 95) = 1.71$, $P > 0.05$, partial $\eta^2 = 0.04$. The group without psychopathy performed significantly worse than healthy controls during immediate recognition, $F(2, 96) = 4.72$, $P < 0.05$, partial $\eta^2 = 0.09$. The group with psychopathy performed significantly worse than the healthy control group during the 4 s delayed recognition, $F(2, 96) = 5.93$, $P < 0.01$, partial $\eta^2 = 0.11$. Finally, both patient groups performed significantly worse than healthy controls during 12 s delayed recognition, $F(2, 96) = 6.86$, $P < 0.01$, partial $\eta^2 = 0.13$. Attention (RVP hits) appeared to contribute to these effects, $\Delta\chi^2 = 48.1$, $\Delta df = 5$, $P < 0.001$. Controlling for attention indicated that only the group without psychopathy performed significantly worse than healthy controls, $\beta = -0.51$, $SE = 0.20$, $P < 0.05$. Individuals with psychopathy did not perform significantly differently to controls, $\beta = -0.42$, $SE = 0.26$, $P > 0.05$. The performance of those without psychopathy did not appear related to ASPD as removal of patients with ASPD from the group led to comparable results.

3.4.2. Visual short-term memory span (SSP)

Both individuals with and without ASPD performed worse than controls (Table 2). However, only individuals without psychopathy but not those with psychopathy performed worse than controls (Table 2). Controlling for attention (RVP hits) was a significant improvement, $\Delta\chi^2 = 4.42$, $\Delta df = 1$, $P < 0.05$, and suggested that patients without psychopathy performed worse than both healthy controls, $\beta = -0.93$, $SE = 0.36$, $P < 0.01$, and the group with psychopathy, $\beta = -0.83$, $SE = 0.33$, $P < 0.01$. The performance of those without psychopathy did not appear related to ASPD, as removal of patients with ASPD from the group led to comparable results.

3.4.3. Verbal short- and long-term memory (VRM)

One multivariate outlier was removed from the non-ASPD group. There were no significant differences between groups (Table 2). There was also no significant group x recognition delay interaction for the ASPD, *Pillai's Trace* = 0.08, $F(2, 58) = 0.77$, $P > 0.05$, partial $\eta^2 = 0.03$, or psychopathy analysis, *Pillai's Trace* = 0.12, $F(2, 56) = 0.33$, $P > 0.05$, partial $\eta^2 = 0.01$.

4. Discussion

Individuals with ASPD and psychopathy pose considerable social and treatment challenges highlighting a need for better understanding to inform intervention. They have been associated with a range of neuropsychological deficits but their characterization has been unclear in light of divergent findings and overlaps with other clinical populations. This study aimed to further elucidate the neuropsychological deficits by comparing in parallel offenders with ASPD and psychopathy to offenders with other personality disorders and healthy controls.

ASPD was associated with difficulties in attention, executive functions, and visual memory. The deficits in visual search, inhibitory control, response reversal, and working memory may characterize ASPD as they appeared pronounced in ASPD relative to patients with other personality disorders. More moves together with shorter thinking times during planning suggested impulsiveness. Results extend previous literature, especially research that used similarly sensitive measurement (e.g., De Brito et al., 2013; Dolan and Park, 2002). Deficits in planning, set shifting, response reversal, memory, and sustained attention were supported further using the SOC, IED, DMS, and RVP tasks (Dolan and Park, 2002). Notably, the planning deficit in ASPD was pervasive and not related to problem difficulty (in contrast to the deficit in psychopathy). These findings, however, are in contrast with studies which have not supported deficits in planning, set shifting, attention, and working memory using the Porteus Mazes, WCST, and simple continuous performance tasks, and the Digit Span (Barkataki et al., 2005; De Brito et al., 2013; Stevens et al., 2003). Further, whilst the lack of deficit in visual search/matching was replicated using the DMS (Dolan and Park, 2002), such a deficit emerged during the MTS task.

There may be several explanations for these differences. Together with more impulsiveness, working memory impairments, as highlighted by the present findings, may have contributed to the relatively more pervasive deficit in planning in ASPD, due to the role of working memory in the planning paradigm of the SOC test (Goel et al., 2001; Owen, 2000; Phillips, 1999; Smith and Jonides, 1999; Welsh et al., 1999). Further, the pattern of results during the SWM suggested deficits potentially depending on increasing level of difficulty and task complexity as the test has an element of strategy development, unlike the Digit Span. The SWM task was computerized and without contextual information compared to a similar task in a real-world context (Executive Golf Task) that did not reveal a deficit in ASPD previously (Barkataki et al., 2005), though the current study also had a larger sample. Regarding the deficit in set shifting, the requirement to shift category in the IED comes after strong reinforcement of the existing set amidst additional elements and an intradimentional shift (Fray et al., 1996), thus potentially reflecting greater challenge than the WCST (Berg, 1948). The MTS is also substantially more complex in the array of information presented compared to the DMS.

Psychopathy was associated with deficits in attention, complex planning, inhibitory control, and response reversal. As with ASPD, visual search and response reversal difficulties appeared pronounced relative to patients with other personality disorders. The planning deficit was not pervasive as it emerged for more complex problems only, as expected. It also emerged in absence of a deficit in working memory. These suggest that the planning deficit in psychopathy may arise for different reasons to ASPD, such as when more integration is required between neuropsychological functions as task complexity increases. Interestingly, individuals with personality disorder and psychopathy emerged as less impaired on planning overall, set-shifting, and short-term memory (the deficit in working memory was not reliable due to association with both ASPD and impaired attention). These patterns are consistent with the notion that psychopathy may be partly underpinned

by enhanced attentional focus *in goal-directed behavior* (impaired attention to peripheral information; Vitale and Newman, 2009) and potentially fewer deficits, at least relative to patients with other personality disorders.

Attentional deficits, in sustained attention, visual search, and inhibitory control, also emerged in psychopathy. Deficits in attention were not expected especially in light of literature suggesting potentially superior selective attention in psychopathy (Vitale and Newman, 2009). However, deficits have been observed in psychopathy under conditions of divided attention and top-down processing (Hoppenbrouwers et al., 2015; Kosson and Newman, 1986), which may reflect impairment when integration of information is needed. It is difficult to conclude from the present results whether deficits on the RVP and MTS reflect impaired integration or impaired attention per se as both tasks involve integration (e.g., RVP draws significantly on working memory and MTS involves considerable visual processing, some working memory, and sustained attention) and sub-functions were not tested in relative isolation. Regarding inhibitory control, deficits may be related to a relative overrepresentation of social deviance in the psychopathy group (Weidacker et al., 2017). However, the AGN is unique among Go/NoGo tasks in that it involves affective stimuli; therefore, deficits may also have arisen due to the additional complexity from the need to integrate this information with the task process and connections with the affective circuitry thought to be impaired in psychopathy (Blair et al., 2005).

Deficits in complex attention (MTS) and response reversal that were present in ASPD and psychopathy but not in other groups may underpin similarities between ASPD and psychopathy. Difficulties with reversing responding when outcomes change and difficulty focusing attention amidst complex information and distractors *when this is not goal-directed* may reflect the persistently misdirected behavior observed in both presentations. Conversely, neuropsychological deficits shared with other personality disorders such as those in sustained attention and planning indicates that these deficits may underpin criminal behavior generally. This is in line with findings of broader deficits of this kind in antisocial populations beyond ASPD and psychopathy (Ogilvie et al., 2011).

Frontotemporal deficits in ASPD appeared more widespread compared to psychopathy, though this does not mean that ASPD is associated with more severe deficits overall (see literature on extensive deficits in limbic circuitry in psychopathy; Blair et al., 2005). The range of frontotemporal deficits in ASPD suggests a role for this circuitry whilst those in psychopathy indicate a role for inferior prefrontal regions. Altered function in these areas alongside the limbic circuitries has been documented (Baliousis et al., 2018; Koenigs, 2012) with the present findings suggesting impairment. Lack of impairment in set-shifting and working memory in psychopathy further suggests that planning deficits may result from connections between inferior prefrontal areas and the dorsolateral prefrontal cortex rather than impairment in the latter (see Clark et al., 2010, for review of functions associated with various brain areas). However, fewer deficits in psychopathy may also reflect smaller power (smaller sample).

The findings may explain some of the differences in the presentation/symptoms of ASPD and psychopathy. Extensive executive difficulties may underpin the generally erratic behavior often thought to characterize ASPD (De Brito and Hodgins, 2009). Characteristics of psychopathy such as inability to respond to punishment, instrumental aggression, callousness and earlier and more violent offending may relate to deficits in response reversal and integrating complex new information, alongside the well-documented affective and other "hot" executive impairments (Baliousis et al., 2018; Blair, 2013) which were not in focus in this study.

The findings need to be viewed in light of limitations. Comparison groups were not firmly equivalent on clinical and diagnostic measures, education, prior substance abuse, and medication. Though groups were comparable on IQ, educational differences may still have contributed to the observed effects (Heaton et al., 2009). Patients were offenders and, together with controls, came from a single site so results may not generalize to other samples. The number of tasks may have contributed to Type I error and the study was only sufficiently powered to detect medium-large effect sizes for the main analyses (i.e., not the ASPD-only, etc.), calling for replication with larger samples. Offenders with other personality disorders performed between controls and offenders with ASPD or psychopathy in many tasks so that it was not possible to establish uniqueness of deficits in ASPD or psychopathy firmly. ASPD and psychopathy were confounded. Though the ASPD-only analysis partly mitigated this, a psychopathy-only analysis was not possible, neither was a direct comparison between the two, which calls for caution when interpreting results and a need for replication accordingly.

Regarding the neuropsychological assessment, the study was limited to the CANTAB tests we possessed data for so that aspects of executive functions remained untested (e.g., decision-making, interference control, verbal working memory). We also did not include performance validity tests; therefore, it is not possible to exclude the possibility that deficits reflect less effort. Finally, the AGN stimuli were affective words which may have influenced inhibitory control in light of affective difficulties (esp. in psychopathy). As we did not screen for history of Attention Deficit-Hyperactivity Disorder (ADHD) in patients with ASPD (as epidemiological studies demonstrate; Storebø and Simonsen, 2016), it was not possible to determine the effect of ADHD in this study. However, conclusions are strengthened by sensitive measurement, control for several confounders (e.g., comparable groups on IQ), a parallel examination of ASPD and psychopathy to identify characteristic cognitive profiles, two comparison groups, and sampling from consecutive admissions. In spite of threatening internal validity, including patients with a history of substance abuse led to a sample that was more relevant to clinical practice and external validity (De Brito and Hodgins, 2009; Hare, 2003).

In conclusion, findings suggested widespread deficits in ASPD and psychopathy and revealed largely different neuropsychological profiles between the two operationalizations. This adds evidence suggesting that ASPD and psychopathy are likely to reflect divergent populations. Nonetheless, the two operationalizations showed similarities in some attentional and executive deficits with common deficits in complex, top-down attention and response reversal setting them apart from patients with other personality disorders, which could explain some of their common features. However, ASPD showed wider deficits spanning "hot" and "cool" executive functions and memory (thus implicating the frontotemporal network). Inhibitory control and working memory deficits also appear to characterize ASPD relative to other personality disorders. Psychopathy demonstrated deficits in attention and executive functions potentially more specific to the "hot" category implicating the inferior medial prefrontal cortex. However, the pattern of results suggesting impairment in integration of cognitive processes, as discussed above, appears more consistent with the impaired integration hypothesis of psychopathy (Hamilton et al., 2015) rather than hypotheses of focal impairment in distinct clusters of cognitive function.

Future research should further explore the neuropsychological profiles distinguishing ASPD and psychopathy relative to each other and other personality disorders. In light of indications regarding the role of task complexity and integration, it will be important to examine impairments in tests drawing more strongly on specific neuropsychological functions versus impairments in tests requiring integration among functions, to test the hypothesis of impaired integration further. Replication with other, larger samples, individuals with psychopathy but not ASPD, and examining the differential relationship of the deficits with the two factors of psychopathy are important in helping to understand the neuropsychological underpinnings of these presentations further.

Declaration of Competing Interest

None.

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