Neurocognitive Deficits Associated with Antisocial Personality Disorder in Non-treatment-seeking Young Adults

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Antisocial personality disorder (ASPD) is a relatively common problem, but the neuropsychological profile of affected individuals has seldom been studied outside of criminal justice recruitment settings. Non–treatment-seeking young adults (18–29 years) were recruited from the general community by media advertisements. Participants with ASPD ($n = 17$), free from substance use disorders, were compared with matched controls ($n = 229$) using objective computerized neuropsychological tasks tapping a range of cognitive domains. Compared with controls, individuals with ASPD showed significantly elevated pathological gambling symptoms, previous illegal acts, unemployment, greater nicotine consumption, and relative impairments in response inhibition (Stop-Signal Task) and decision-making (less risk adjustment, Cambridge Gamble Task). General response speed, set-shifting, working memory, and executive planning were intact. ASPD was also associated with higher impulsivity and venturesomeness on the Eysenck Questionnaire. These findings implicate impaired inhibitory control and decision-making in the pathophysiology of ASPD, even in milder manifestations of the disorder. Future work should explore the neural correlates of these impairments and use longitudinal designs to examine the temporal relationship between these deficits, antisocial behavior, and functional impairment.

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With a lifetime prevalence rate of 3.6 percent, antisocial personality disorder (ASPD) is a common personality disorder characterized by disregard for, and violation of, the rights of others that begins in childhood and continues into adulthood.[1] ASPD has been associated with elevated risk for the development of substance use disorders and a range of other psychiatric comorbidities (e.g., depression, attention-deficit hyperactivity disorder, gambling disorder, and paraphilic disorders).[2–4]

Given the high prevalence of ASPD, it is important to question whether the disorder is associated with impairments in dissociable cognitive functions dependent on the integrity of frontostriatal circuitry. Knowledge of any cognitive deficits associated with ASPD would be valuable, not only in working toward understanding the neurobiology of this disorder and its relationship with other conditions, but also in developing more targeted treatment interventions. Antisocial symptoms have been significantly associated with cognitive control deficits; attentional problems; abnormalities in decision-making; deficits in aspects of flexible responding, such as reversal learning; planning impairments; and abnormalities in neural regions governing inhibitory control.[5–10]

Many of those studies examined cognitive functioning in participants recruited from forensic settings or those who had sought treatment for other mental health problems, which may not be reflective of younger adults in the general community; many

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also used pen-and-paper cognitive tests, assessed one or two cognitive domains, and included participants with substance use disorders. Inclusion of individuals with substance use disorders in previous cognitive research is potentially problematic in view of the well-established association between substance dependence and executive dysfunction.[11,12] We therefore examined dissociable cognitive functions by using a previously validated battery of computerized tests in participants recruited from the general community who were free of substance use disorders. Potential advantages of these cognitive paradigms include their validation in animal models and in human studies involving focal lesions and neuroimaging and sensitivity to neuropsychiatric sequelae.[13] On the basis of the existing literature, we hypothesized that participants with ASPD versus control subjects would exhibit impairments on decision-making, impulse control, set-shifting, memory, and executive planning, consistent with underlying dysregulation of frontostriatal circuitry, including both the orbitofrontal and the more dorsolateral portions of the frontal cortices.

Method

Participants

Participants were non–treatment-seeking individuals aged 18–29 years, who were recruited from two large urban environments by media advertisements for a single study examining impulsivity in young adults. Media advertisements used the following text: "Do you gamble? Do you feel impulsive? Study for young adults aged 18–29 years who have gambled five times during the past year." Before inclusion, participants underwent a detailed psychiatric evaluation (details later). Individuals with ASPD were entered into the study if they were free of substance use disorders. Controls, free of ASPD and substance use disorders, were similarly recruited as part of the same study and matched to the ASPD group in age, gender, and overall rate of one or more Axis I disorders. Because we wished to match groups in demographic characteristics, ASPD participants and controls were recruited from the same pool. This has the advantage of reducing risk of confounding group differences that have been potentially problematic in interpreting results of other ASPD studies. To maximize power, we included all available participants with ASPD and targeted a control sample size of at least 200 participants.

The study procedures were performed in accordance with the Declaration of Helsinki. The Institutional Review Boards of the Universities of Chicago and Minnesota approved the study and the consent statement. After all study procedures were explained to the participants, voluntary written informed consent was obtained. Participants were compensated $50 U.S. for their time.

Assessments

Raters

The raters were experienced research coordinators who had completed adequate training in the various instruments enumerated below (including clinical and neurocognitive assessment). Raters were supervised by a board-certified psychiatrist.

Psychiatric Evaluation

Raters assessed each participant using the Mini International Neuropsychiatric Inventory (MINI),[14] which is a well-validated structured clinical interview designed to meet the need for prompt but accurate screening for mainstream psychiatric disorders, on the basis of Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR) criteria. Raters also assessed each participant with the structured Clinical Interview for Pathological Gambling (SCI-PG),[15] and with a semistructured instrument to examine a range of behaviors including nicotine and alcohol consumption. Diagnoses of ASPD were made on the basis of the relevant module from the MINI: participants were first asked about six specific childhood misbehaviors. If two or more were endorsed, then participants were asked about six antisocial behaviors occurring since the age of 15 years, with three or more endorsements being required for the diagnosis. This method of screening for ASPD has been used in forensic settings and found to be associated with negative outcomes, including elevated risk of suicidality, comorbidity, and worse quality of life.[16]

Impulsivity Questionnaires

Barratt Impulsivity Scale, Version 11 (BIS-11)

This instrument is a 30-item, self-report measure that assessed general impulsivity.[17] Subscales of the Review of Attention-Deficit/Hyperactivity Disorder Comorbid With Oppositional Defiant Disorder

(Barratt Impulsivity Scale, Version 11; BIS-11) include attentional impulsivity (inability to concentrate attention), motor impulsivity (acting without thinking), and nonplanning impulsivity (being present in the moment, lack of future thinking).

Eysenck Impulsivity Questionnaire

This questionnaire is a 54-item, self-report measure that assessed 3 facets of personality, including impulsivity (failure to evaluate risk), venturesomeness (consciousness and acceptance of risk), and empathy (ability to identify with other peoples' experiences).[18]

Cognitive Assessments

Participants completed the following selected cognitive paradigms from the Cambridge Neuropsychological Test Automated Battery (CANTABeclipse, version 3; Cambridge Cognition Ltd., UK), in a fixed order. Maintaining a fixed task order across all participants using CANTAB was undertaken to minimize noise and for pragmatic purposes. This approach is widely used both in studies comparing cognition between groups and in clinical trials using this battery. Testing was conducted in a quiet room with a trained administrator present. Domains of interest were selected on the basis of their hypothesized role in ASPD (i.e., impulsivity, decision-making), their dissociable nature, and the afore-described existing literature on cognition in ASPD, described in the introductory section, suggesting a broad range of prefrontal deficits.[8,19]

Cambridge Gambling Task

This task[20] measured dissociable aspects of decision-making and has been shown to be sensitive to orbitofrontal and insula lesions.[21] On each trial, participants viewed a mix of red and blue boxes (10 in total) and were told that the computer has hidden a "token" behind one of them. They had to choose what color of box they believed the token was hidden behind and the number of accumulated points they wanted to gamble on having made the correct color choice. The key outcome measures for this task were risk adjustment (a measure of the tendency to modulate the amount of points gambled contingent on risk), overall proportion bet, and quality of decision-making (the proportion of rational decisions made). For risk adjustment, greater scores indicated greater sensitivity to risk, which related to more conservative gambling; for overall proportion bet, higher scores indicated preference for putting more points at risk, a form of risk-seeking; for quality of decision-making, higher scores were better, indicating more rational choices.

One-Touch Stockings of Cambridge Task

This task measured executive planning, a cognitive function dependent on the dorsolateral prefrontal cortices.[22] Participants attempted to work out in their heads the minimum number of moves it would take to rearrange a set of balls in pockets shown to meet a goal arrangement indicated by the computer. They then indicated this estimated minimum number of moves by pressing a button on-screen. The key outcome measure was the number of problems solved at the first attempt. A higher number of problems solved at the first attempt was indicative of better executive planning performance.

Spatial Working Memory

This task quantified strategy and working memory. Participants attempted to locate tokens hidden underneath boxes and tried to avoid returning to boxes that had previously yielded such tokens. The key outcome measures comprised the "total number of errors" (inappropriately returning to boxes that had yielded tokens; higher errors indicated worse spatial working memory performance), and strategy scores (a lower score equated to use of superior strategies).

Intradimensional/Extradimensional Set-Shift Task

The Set-Shift Task[23] explored aspects of rule learning and behavioral flexibility. On each trial, participants were presented with two stimuli and attempted to work out an underlying rule about which stimulus was correct, based on feedback. The primary outcome measure on the task was the total number of errors, adjusted for stages that were failed/not attempted. Higher errors equated to worse cognitive rule-learning and flexibility.

Stop-Signal Task

This task tested[24,25] response inhibition. Participants responded quickly to a series of directional arrows appearing on the screen (for a left arrow, they pressed a left button, and vice versa).[23] On a subset of trials, an auditory stop signal occurred, indicating that the participant should try to suppress the motor response for the corresponding trial. This task estimated the time taken by each volunteer's brain to

Table 1 Demographics and Clinical Variables of Young Adults With Antisocial Personality Disorder

Variable	Antisocial Personality Disorder (n = 17)	Controls (n = 229)	p	d
Age, years	23.8 (3.9)	23.6 (3.1)	0.763	
Gender, male, n (%)	10 (58.8)	141 (61.6)	0.823c	
Education score[a]	2.7 (0.8)	2.9 (0.8)	0.087	
Unemployed? yes, n (%)	6 (35.3)	28 (12.2)	0.008c	0.170
Married? yes, n (%)	1 (5.9)	9 (4.9)	0.694c	
Previous illegal acts, yes, n (%)	16 (94.1)	12 (5.2)	<0.001c	0.710
SCI-PG score	3.2 (3.1)	1.4 (1.9)	<0.001	0.70
Current alcohol use, drinks per week	1.1 (0.9)	1.6 (1.6)	0.219	
Current nicotine use, packs per day	0.43 (0.59)	0.17 (0.31)	0.002	0.55
Any current psychiatric comorbidity (besides substance use disorder), n (%)[b]	5 (29.4)	36 (15.7)	0.144c	

All data are expressed as the mean (SD) except where indicated. d = Cohen's d effect size, except for chi-square when phi is used to denote effect size. Statistical significance was determined by independent-samples t tests except where indicated 'c' (chi-square). Significance was set at $p < 0.05$.

[a] 1, below high-school; 2, high-school graduate; some college; 4, college graduate; and 5, higher than college level education.

[b] The number of participants with the given Axis I disorders in the ASPD group were: n = 2, major depressive disorder; n = 3, agoraphobia; n = 1, social phobia; n = 2, posttraumatic stress disorder (PTSD); n = 1, psychosis; n = 1, bulimia; n = 1, general anxiety disorder and for controls; n = 9, major depressive disorder; n = 2, hypomanic episode; n = 4, panic disorder; n = 8, agoraphobia; n = 11, social phobia; n = 6, obsessive-compulsive disorder; n = 1, PTSD; n = 6, bulimia; n = 12, general anxiety disorder.

suppress an already triggered command (the stop-signal reaction time). Longer stop-signal reaction times corresponded to worse inhibitory control. Median reaction times for go-trials were also recorded, with longer reaction times being indicative of psychomotor slowing.

Data Analysis

Differences between ASPD participants and controls were examined using independent-sample t tests (or equivalent nonparametric tests, as indicated in the text). This being a pilot study, significance was defined as $p < .05$ uncorrected. Where significant differences between the study groups were identified on a given measure, the effect size was reported (Cohen's d).

Results

Total 540 subjects were screened. From this pool, 25 ASPD subjects were identified, of whom 8 were excluded because of substance use disorders (SUDs); 229 control subjects were selected on the basis of absence of SUDs and matching to the ASPD group in terms of having similar demographic characteristics. Demographic and clinical characteristics of the final sample are provided in Table 1. There were no withdrawals, as all participants were screened and tested on a single occasion rather than longitudinally. It can be seen that the ASPD and control groups were well matched in age, gender, education, and rates of Axis I psychiatric disorders in general. ASPD did not differ from controls in quantity of alcohol used per week. Compared with controls, ASPD was associated with higher SCI-PG scores (reflecting greater pathological gambling symptoms) and greater quantities of nicotine consumption per day.

Questionnaire scores and performance on the neurocognitive tasks are presented in Table 2, where it can be seen that the ASPD participants manifested significantly elevated impulsivity and venturesomeness on the Eysenck Impulsivity Questionnaire and significant impairment of inhibitory control (stop-signal reaction times, Stop-Signal Task) and one aspect of decision-making (risk adjustment, Cambridge Gamble Task). Overall response speed, set-shifting, spatial working memory, and executive planning were intact in individuals with ASPD versus control subjects. Groups did not differ significantly from each other on the BIS-11 scale.

Discussion

In this study, we examined a range of clinical and cognitive domains in young adults with ASPD, a condition often associated with a host of deleterious long-term outcomes. Unlike many prior studies, we examined ASPD in a representative non–treatment-seeking community sample, rather than, for example, participants recruited from forensic settings (incarcerated populations or those on parole). We also excluded participants with substance use disorder(s). Our sample of ASPD participants can be regarded as being at the milder end of the disease severity spec-

Table 2 Measures of Impulsivity in Young Adults With Antisocial Personality Disorder

Task	Antisocial Personality Disorder (n = 17)	Controls (n = 229)	p	d
BIS				
Attention impulsiveness	17.6 (5.3)	16.4 (4.2)	0.265	
Motor impulsiveness	25.8 (7.3)	23.5 (4.9)	0.081	
Nonplanning impulsiveness	24.4 (7.0)	24.4 (6.0)	0.958	
EIQ				
Impulsivity	13.4 (5.7)	9.8 (5.8)	0.015	0.63
Venturesomeness	18.9 (7.5)	13.9 (7.0)	0.005	0.69
Empathy	18.3 (9.3)	14.8 (7.2)	0.057	
IDED total errors (adjusted)	30.4 (21.5)	26.1 (26.0)	0.516	
SST SSRT	216.6 (82.0)	182.2 (61.9)	0.032	0.47
SST median correct RT on GO trials	529.0 (255.1)	475.8 (152.1)	0.190	
CGT Risk adjustment	0.73 (0.86)	1.40 (1.21)	0.025	0.64
CGT overall proportion bet	0.580 (0.162)	0.547 (0.138)	0.352	
CGT quality of decision making	0.931 (0.068)	0.934 (0.102)	0.878	
SWM strategy	33.2 (5.1)	30.7 (6.2)	0.105	
SWM total errors	27.5 (20.9)	20.2 (18.7)	0.129	
OTS Problems solved on first choice	15.9 (4.5)	17.4 (3.9)	0.128	

All data are expressed as the mean (SD). Significance was set at $p < 0.05$. BIS, Barratt Impulsivity Scale; EIQ, Eysenck Impulsivity Questionnaire; IDED, Intradimensional/Extradimensional Set-Shift Task; SST, Stop-Signal Task; CGT, Cambridge Gamble Task; SWM, Spatial Working Memory Task; OTS, One-Touch Stockings of Cambridge Task; *d*, Cohen's *d* effect size.

trum. The key cognitive findings were that ASPD was associated with impaired response inhibition on the Stop-Signal Task, and impaired risk adjustment on the Cambridge Gamble Task, both with medium-large effect size, but intact performance on the other domains considered (general response speed, set-shifting, working memory, and executive planning). ASPD was also associated with significantly elevated rates of previous illegal acts (not surprisingly), gambling problems and nicotine use (medium-large effect size), unemployment (small effect size), but not alcohol use (the latter being consistent with our exclusion of substance use disorders). Of all these findings, elevated rates of previous illegal acts had the largest effect size, followed by gambling symptoms.

It is potentially informative to contrast the cognitive results reported here to those of previous studies in people with ASPD. In a meta-analysis conducted in 2000, antisocial groups showed, on average, worse performance than controls on composite measures of executive function (medium to large effect size)[8]; however, that meta-analysis included various operationalizations of antisocial behavior, including not only ASPD, but also psychopathic personalities, criminality, delinquency, and conduct disorder. When the authors restricted their meta-analysis to studies that had included ASPD specifically, the composite executive function deficit was statistically significant, but with a negligible effect size. This result suggests that many of the cognitive problems associated in the literature with antisocial behavior are not evident to the same degree when only diagnosed ASPD is considered. ASPD is arguably a milder manifestation of antisociality compared with psychopathy. Our results are consistent with this suggestion, as people with ASPD showed a restricted pattern of cognitive impairment only.

Since the meta-analysis by Morgan and Lilienfeld,[8] there have been only a few cognitive studies in people with well-delineated ASPD. In a sample of young noninstitutionalized community-based individuals, ASPD (n = 35 participants) was associated with disadvantageous decision-making versus the control condition on the Iowa Gambling Task, compared with 32 control subjects.[7] Although most ASPD participants had alcohol dependence, alcohol use did not appear to account for the finding. In 34 individuals with ASPD who were on probation or parole but who were recruited from the community setting, significantly slower reaction times on commission error trials for a working memory task were identified versus the reaction times of 30 control subjects.[9] In violent offenders recruited from a national probation service, individuals with ASPD (n = 28) showed significant impairments versus the control participants (n = 21) on reversal learning and aspects of decision-making (slower decisions, lower quality of decision-making); there was a trend toward digit span backward impairment, as well.[26] Spatial alter-

nation was intact. Clearly caution is needed when comparing cognitive findings across studies, since different tests were used. To our knowledge, none of the previous studies used the CANTAB battery. Our results partially support previous research, in that we identified decision-making impairment, albeit on a select measure (risk adjustment) rather than across all aspects of decision-making. Our study extends the existing literature by highlighting response inhibition deficits in ASPD also. Contrary to one or more previous studies, we did not find evidence for psychomotor slowing or reversal learning impairment (reversal learning is indexed by the Set-Shifting Task).

We found elevated questionnaire-based impulsivity and venturesomeness using the Eysenck Impulsivity Questionnaire in participants with ASPD, somewhat consistent with pre-existing literature.[9,27–29] However, contrary to our expectations, ASPD was not linked with significantly elevated Barratt Impulsivity (BIS-11) scores. These findings serve to highlight the multifaceted nature of impulsivity and suggest that the Eysenck Impulsivity Questionnaire may be more sensitive to impulsivity than BIS-11 is, at least when studying personality features of ASPD. The finding that ASPD was linked with gambling problems and nicotine use was predicted.

Although our study is one of very few to examine a spread of dissociable cognitive functions in ASPD, some important limitations should be considered. The sample size provided >90 percent power to detect large effect sizes with $\alpha = .05$ (two-tailed), and therefore we consider it to have been amply powered to identify clinically meaningful differences between the groups, which was the focus of this study. Indeed, we report significant differences on response inhibition and decision-making, for example, highlighting that the study was adequately powered. Because of the sample size, however, power would have been limited to detect more subtle cognitive problems in ASPD; as such, negative findings should be regarded as tentative and in need of replication. We deliberately matched the control group to the ASPD group in salient demographic characteristics and overall occurrence of one or more Axis I disorders, to help minimize potential confounding variables that could otherwise account for cognitive impairments. Nonetheless, this matching of the control to the ASPD group on certain variables means that the influence of these variables on cognition in ASPD cannot be evaluated within the confines of the current study. We did not record histories of conduct disorder, a frequent precursor of ASPD. We did not correct our statistical analyses for multiple comparisons, because this was an exploratory study. The study was neither designed nor powered to explore the influence of gender on the results. This topic would be an important one for future research. Our sample of ASPD volunteers was identified on the basis of a clinical interview using a well-validated instrument; in clinical practice, it would be valuable to confirm the diagnosis with a longitudinal assessment and to undertake more comprehensive assessment, before confirming individuals' diagnoses and commencing treatments. That said, the MINI ASPD module has been used successfully in the forensic setting.[16] Finally, we did not collect information about current medication status or history of neuropsychiatric diagnoses, such as epilepsy and head trauma.

These findings have several implications. The elevated rates of pathological gambling and nicotine use in people with ASPD highlights the importance of screening for these behaviors and providing treatment. Of course, this is in addition to the importance of screening for SUDs, which are common in ASPD, but which were exclusionary for participation in the current study. The response inhibition and decision-making impairments found in ASPD represent candidate treatment targets for novel interventions, and may also be suggestive of possible neural dysfunction implicated in this disorder. Response inhibition is dependent on distributed neural circuitry including the right inferior frontal gyrus.[30] Medications with actions on the norepinephrine system have been found to be capable of improving this function in animal models and in certain patient groups.[31,32] The neural and neurochemical mechanisms mediating risk adjustment are less well studied, but lesions of the insula in humans particularly impair risk adjustment on the same decision-making task as used in the current study.[21] It would be valuable to study the neural underpinnings of response inhibition and risk adjustment deficits in ASPD in future work and to examine whether these can be ameliorated by pharmacological and psychological means. In addition, this study enrolled non–treatment-seeking individuals with ASPD, free from substance use disorders, who were likely to represent the milder end of the ASPD severity spectrum: more pronounced findings

(e.g., in the magnitude and range of cognitive impairments) may occur in more severe ASPD cases.

Because virtually all of the ASPD subjects in this study had histories of illegal behavior, the current findings may have forensic implications as well. A recent study of state court judges found that the introduction of biomechanical evidence (e.g., atypical brain functioning, neurodevelopmental factors) resulted in reduced sentences in the case of psychopathy, because of reduced culpability based on lack of impulse control.[33] Although impaired volitional control may be relevant to sentencing decisions, whether neurocognitive findings such as those in this study would have a similar impact at sentencing is unclear. Presenting evidence of neurocognitive dysfunction may help reframe the ASPD person's potentially detrimental history and mitigate its potentially aggravating effect. Evidence of a dysfunctional brain, however, may be damaging to the defendant as well. Attempts to mitigate responsibility by showing neurocognitive dysfunction may result in a view of the person as permanently unchangeable, leading to a harsher sentence. The success of using this type of neurocognitive evidence may therefore depend on many additional factors including the type of crime and the quality of neuroscience testimony.[34] In addition, although matters of criminal responsibility and treatment are complex, it is conceivable that aspects of criminality in ASPD could arise, in part as a result of cognitive deficits. If so, this raises the question of whether criminal justice systems should consider incorporating treatment specifically for these cognitive deficits, in addition to other types of nonpunitive intervention, for people with ASPD.

Future work should consider cognitive domains beyond those addressed herein, such as emotional processing and other aspects of impulsivity, such as temporal discounting. It is also important that future studies be of longitudinal design, to examine the temporal relationship between ASPD, cognition, functional impairment, and the evolution of more extreme manifestations of antisocial behaviors.

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