Neurocognitive function in antisocial personality disorder

Wayne M. Dinn*, Catherine L. Harris

Department of Psychology, Boston University, 64 Cummington Street, Boston, MA 02215, USA

Abstract

Recent neuroimaging studies and neuropsychological test findings support the contention that prefrontal dysfunction is associated with psychopathic personality traits and antisocial behavior. However, conflicting results have arisen regarding performance on measures of frontal executive function. We administered a neuropsychological test battery consisting of measures sensitive to frontal lobe dysfunction and a battery of personality questionnaires and clinical scales sensitive to antisocial personality disorder (APD) subjects presenting with prominent psychopathic personality features and matched control subjects. We also monitored the subjects' electrodermal activity during the presentation of emotionally charged stimuli. APD subjects showed greater neuropsychological deficits on measures sensitive to orbitofrontal dysfunction in comparison to control participants. Moreover, APD subjects were electrodermally hyporesponsive to aversive stimuli relative to control group members. APD subjects did not demonstrate performance deficits on classical tests of frontal executive function. Participants also underwent clinical assessment. As expected, APD subjects were less conscientious, self-reproaching, guilt-prone, and socially anxious than matched control subjects. Moreover, the scores indicated that APD subjects were more venturesome and uninhibited relative to control subjects. Contrary to expectations, APD subjects and community control subjects did not differ on a self-report measure of sensitivity to specific phobic situations. © 2000 Elsevier Science Ireland Ltd. All rights reserved.

Keywords: Psychopathy; Frontal lobe; Orbitofrontal; Dorsolateral–prefrontal; Neuropsychological; Electrodermal

* Corresponding author. 42 Washington Terrace, Whitman, MA 02382, USA. Tel.: +1-617-353-1362.
  E-mail address: dinn@bu.edu (W.M. Dinn).

1. Introduction

1.1. Prefrontal dysfunction and psychopathy

Recent neuroimaging studies and neuropsychological test findings support the contention that prefrontal dysfunction (particularly orbitofrontal) is associated with psychopathic personality traits and antisocial behavior (Davidson et al., 2000; Raine et al., 1998, 2000; Lapierre et al., 1995). However, conflicting results have arisen regarding the performance of psychopathic subjects on measures of frontal executive function.

Several studies suggest that a select deficit involving the orbitofrontal system may underlie psychopathy and antisocial behavior. Lapierre et al. (1995) found that incarcerated psychopathic subjects were significantly impaired on tasks considered sensitive to orbitofrontal/ventromedial−prefrontal dysfunction including a visual go/no−go discrimination task, Porteus Maze Q-scores (i.e., rule-breaking errors), and an odor identification task in comparison to matched control subjects (non-psychopathic inmates). Lapierre et al. (1995) also found that psychopathic subjects did not display performance deficits on measures sensitive to dorsolateral−prefrontal (DLPF) and posterorolandic function (i.e., the Wisconsin Card Sorting Test (WCST) and the Mental Rotation Task). Moreover, Deckel et al. (1996) reported that performance on tests assessing frontal executive functioning (e.g. the WCST, controlled oral word fluency test, and trail-making test) failed to predict antisocial personality disorder classifications. These tasks are considered sensitive indicators of DLPF dysfunction (i.e., tasks which require the employment of organizational strategies for efficient performance). These findings suggest that a select orbitofrontal deficit may be associated with psychopathy.

However, Gorenstein (1982) reported that psychopathic subjects demonstrated performance deficits on tests of frontal executive function including the WCST (i.e., perseverative errors), Necker cube task, and a sequential matching memory task. A recent meta-analytic review of 39 studies by Morgan and Lilienfeld (2000) lends strong support to the contention that executive function deficits are associated with antisocial personality (APD). Morgan and Lilienfeld (2000) found a significant relationship between executive function deficits and antisocial behavior.

In response to Gorenstein's study, Hare (1984) assigned inmates to low-, medium-, and high-psychopathy groups and administered the aforementioned frontal executive function tasks. No significant group differences were observed. Hare was unable to replicate Gorenstein's findings. Sutker and Allain (1987) also found that psychopathic subjects and control subjects performed similarly on measures of concept formation, abstraction, and planning. How can we account for these conflicting findings? One possibility is that the executive function deficits among psychopathic subjects are associated with the presence of comorbid psychiatric conditions, while the core interpersonal and affective characteristics associated with psychopathy (e.g., egocentricity, callousness, manipulativeness, guile, lack of empathy and remorse) may result from orbitofrontal dysfunction.

1.2. Executive function and APD

Several studies examining neurocognitive function in psychopathy and APD reveal broadly frontal deficits, but if the prefrontal cortex can be fractionated into separate frontal subsystems, these may be differentially engaged in APD subtypes. One possibility is that APD subjects demonstrating core psychopathic traits and a lack of foresight (e.g., difficulty anticipating negative consequences), planning, and goal-directed behavior (e.g., disorganized offending) will show greater neuropsychological deficits on tasks considered sensitive to orbitofrontal dysfunction and on measures of frontal executive function. Specifically, core psychopathic personality characteristics may result from orbitofrontal dysfunction. The additional involvement of DLPF dysfunction may lead to antisocial behavior that combines core psychopathic characteristics with poor planning and organization, and difficulty keeping in mind diverse future consequences. Motivation for this hypothesis is that the DLPF cortex mediates executive functions (Smith and Jonides, 1999), while the

orbitofrontal cortex mediates sensitivity to dynamically changing reinforcement contingencies, and thus may be particularly important for modulating individuals' response to the social world and other threat-laden situations.

1.3. Is orbitofrontal dysfunction the key to psychopathy?

Numerous case reports describe the emergence of psychopathic behavior following orbitofrontal damage. Striking alterations in personality have been well-documented. These patients demonstrate social disinhibition, shallow affect, decreased empathy, and impulsive, antisocial behavior (Blumer and Benson, 1975; Cummings, 1993; Damasio, 1994; Damasio and Van Hoesen, 1983; Grattan et al., 1994; Martzke et al., 1991; Meyers et al., 1992; Stuss et al., 1992). Meyers et al. (1992) reported post-operative behavioral changes which "strongly resembled" APD in a 33-year-old male with left orbitofrontal damage following surgery for a pituitary tumor. It is noteworthy that cognitive abilities are generally preserved in these patients, suggesting a highly selective disruption.

A significant body of research suggests that the orbitofrontal system plays a major role in regulating the individual's emotional response to aversive stimuli. Cummings (1993) described the emergence of a disinhibition syndrome following damage to the orbitofrontal region. Moreover, he noted that damage to subcortical structures (e.g., caudate nucleus) is associated with the development of disinhibited behaviors similar to those manifested by orbitofrontal lesion patients. Cummings (1993) suggested that disruption of a basal ganglia−thalamocortical circuit underlies the characteristic changes. He concluded that these personality alterations constitute a circuit-specific neurobehavioral syndrome. Malloy et al. (1993) also described an orbitomedial frontal behavioral syndrome distinguished by disinhibition, confabulation, anosmia, and Go/no−go deficits. Interestingly, Lapierre et al. (1995) reported that psychopathic subjects were less accurate at identifying odors in comparison to matched control subjects. They used the Modular Smell Identifi-

cation Test to investigate olfactory function. Olfactory identification tasks have been used to assess the functional integrity of orbitofrontal cortex. The orbitofrontal region plays a major role in odor discrimination, and olfactory agnosia has been reported in patients with damage to the orbitofrontal aspect of the prefrontal cortex. Jones-Gotman and Zatorre (1988) reported that patients with focal surgical brain lesions involving orbitofrontal cortex were impaired on an odor identification task.

The fact that orbitofrontal lesion patients undergo a dramatic personality change (alterations which strongly resemble primary psychopathy) is an intriguing line of evidence which lends support to the contention that orbitofrontal dysfunction is associated with psychopathy. However, we are not suggesting that an orbitofrontal lesion is an etiologic factor in psychopathy or APD. Rather, we support the hypothesis that orbitofrontal hypoactivity, in the absence of gross lesions, underlies primary psychopathy (Lapierre et al., 1995). Neuropsychological test performance patterns and atypical electrodermal responses among psychopathic subjects are consistent with the notion that orbitofrontal hypometabolism may underlie psychopathy.

1.4. Psychophysiological testing, psychopathy, and the orbitofrontal hypothesis

Although atypical electrodermal responses is associated with damage to non-frontal regions (Tranel and Damasio, 1994), converging lines of evidence demonstrate that the orbitofrontal system contributes to the regulation of autonomic nervous system (ANS) activity including electrodermal activity and the rapid control of arterial pressure (Damasio et al., 1990; Guyton, 1991; Tranel and Damasio, 1994; Tranel et al., 1988). Tranel et al. (1988) reported that human subjects with bilateral orbitofrontal lesions demonstrated less reactive electrodermal responses during exposure to emotionally charged stimuli. Their findings illustrate the importance of the orbitofrontal region in modulating autonomic response to emotionally evocative events.

One possibility is that the reduced electroder-

mal responsiveness exhibited by psychopathic subjects reflects orbitofrontal hypofunction. On multiple measures of ANS activity, psychopathic individuals also exhibited reduced physiological reactions to noxious stimuli. For example, psychopaths demonstrated diminished skin conductance levels and were electrodermally hyporesponsive to aversive stimuli (Hare, 1978; Hare et al., 1978; Hare and Craigen, 1974; House and Milligan, 1976; Levander et al., 1979; Patrick et al., 1994; Schalling et al., 1973; Tharp et al., 1980). Researchers have suggested that the psychopath's diminished sensitivity to aversive stimuli hinders the acquisition of avoidance responses essential to socialization. Moreover, they concluded that the psychopath's profound lack of empathy may reflect an inability to generate appropriate autonomic responses to the pain or distress experienced by another individual (Hare, 1978; Lykken, 1957). Patrick et al. (1994) presented findings which support the contention that psychopaths manifest a "deficit in physiological response during fear imagery, reflecting an impairment of the normal associative processes by which symbolic stimuli (in this case, language cues) prompt affect" (p. 524). This is consistent with the somatic marker hypothesis of Damasio (1994). Damasio and colleagues found that ventromedial prefrontal lesion patients (orbital and lower mesial frontal regions) were electrodermally hyporesponsive to emotionally charged stimuli. If electrodermal hyporesponsiveness reflects orbitofrontal hypofunction, then APD subjects exhibiting core psychopathic personality traits should display reduced physiological reactions to aversive stimuli.

1.5. Research goals

The principal objectives of this study were:

1. an examination of the neurocognitive profiles of APD subjects presenting with prominent psychopathic personality features. Work on APD has also revealed broadly frontal deficits, but if the prefrontal cortex can be fractionated into separate frontal subsystems, these may be differentially engaged in APD;

2. an assessment of autonomic reactivity among APD subjects. We monitored the subjects' electrodermal activity during the presentation of emotionally charged stimuli;

3. an investigation of the relationship between clinical presentation and neuropsychological test performance.

2. Methods

We administered a neuropsychological test battery consisting of measures sensitive to frontal lobe dysfunction and a battery of personality questionnaires and clinical scales to antisocial personality disorder (APD) subjects and control subjects recruited from the general population. We employed the community recruitment technique developed by Widom (1977) to obtain the APD sample. Widom (1977) placed newspaper advertisements seeking individuals exhibiting specific personality characteristics to participate in a research study. The advertisement described psychopathic personality traits in a non-pejorative manner. Widom (1977) reported that this method was an effective means of recruiting non-institutionalized psychopathic subjects.

Recruitment Advertisement

"Wanted charming, aggressive, carefree people who are impulsively irresponsible, but are good at handling people and at looking after number one." (Widom, 1977, p. 675).

The APD group consisted of 12 male subjects and their ages ranged from 19 to 34 years ($M =$ 27.8; SD = 4.0). The mean educational level of the APD group was 13.9 years (SD = 1.7). All APD subjects were right-handed as determined by self-report. The APD subjects met the *Diagnostic and Statistical Manual of Mental Disorders 4th ed.) (DSM-IV)* diagnostic criteria for APD (American Psychiatric Association, 1994). In addition, we administered the Hare psychopathy checklist: screening version (PCL:SV) (Hart et al., 1995) to APD subjects. Upon completion of the formal testing session, we conducted a semi-struc-

tured interview with APD subjects designed to elicit information regarding educational and vocational history, family background, prior psychoactive substance use or abuse, and juvenile and adult antisocial behavior. Eleven APD subjects exceeded the recommended cutoff score on the PCL:SV ($\geq = 18$) (Hart et al., 1995). We included one additional subject who obtained a marginal score of 16 on the PCL:SV. The mean PCL:SV score for the APD group was 18.5 (SD = 1.24). It is important to emphasize that APD and psychopathy may be discrete syndromes which often coexist in the same individual. Moreover, the study of psychopathic individuals who present with or without a history of behavioral dyscontrol is clearly warranted. While this distinction may have merit, we were unable to address it since all APD subjects in our study reported a long-standing pattern of antisocial behavior and behavioral dyscontrol dating back to late childhood or early adolescence, and displayed prominent psychopathic characteristics. None of the APD subjects or control participants were receiving psychoactive drugs or met diagnostic criteria for substance abuse or dependence disorders at the time of testing, although several APD subjects reported a history of substance abuse. Individuals were excluded if they were currently abusing alcohol or other psychoactive substances. However, a history of substance abuse or dependence among APD subjects did not constitute exclusion criteria. We compared the performance patterns of APD subjects who met criteria (lifetime) for alcohol or substance abuse to the neurocognitive profiles of APD subjects who did not meet criteria for substance abuse or dependence.

Ten male control subjects were recruited from the general population via advertisements seeking individuals interested in participating in research examining the neuropsychology of personality. Control subjects were matched to APD subjects for age, educational level, handedness, and gender. Their ages ranged from 21 to 41 (M = 28.9; SD = 6.9). The mean educational level of the control group was 13.9 years (SD = 1.7). All control subjects were right-handed, as determined by self-report. All participants received financial compensation ($25 per session). Written informed consent was obtained from all participants.

Approximately 65 individuals responded to the APD recruitment advertisements and 30 individuals responded to control recruitment advertisements. Potential participants contacted our neuropsychology lab in response to the newspaper advertisement. The lead author or a trained research assistant contacted the respondent and administered a brief screening interview (based on the PDQ-4 and the PCL:SV) over the telephone. Individuals were excluded if they were currently using psychotropic medications, reported a history of electroconvulsive treatment, or if they reported a history of traumatic head injury (with loss of consciousness or cognitive sequelae) or central nervous system pathology. Subsequent group assignment was based on PDQ-4 and PCL:SV scores. APD subjects exceeded the recommended symptom threshold on the PDQ-4 (APD subscale) (Hyler, 1994). In addition, 11 of 12 APD subjects exceeded the recommended cutoff scores on the psychopathy checklist: screening version (PCL:SV) ($\geq = 18$) (Hart et al., 1995). The mean score on the APD subscale for the control group was 1.4, but was 6.6 for the APD sample. The mean score on the PCL:SV for the control group was 3.4, but was 18.5 for the APD sample. Clearly, APD subjects exceeded recommended cutoff scores on the PCL:SV. Control group scores on the PCL:SV were comparable to published norms. Therefore, we are confident that our control subjects were not psychopathic.

2.1. Procedure

We administered neuropsychological measures considered sensitive to orbitofrontal dysfunction including computer versions of the object alternation test (Freedman, 1990), the Stroop color–word test, and a visual go/no–go discrimination task (based on Lapierre et al., 1995). All computerized tasks were administered on a Macintosh IIci using PsyScope, experimental design software developed by Cohen et al. (1993). Reaction time and voice onset latencies were collected using a mil-

lisecond timer that interfaces with the PsyScope software. In addition, we administered tests assessing frontal executive functioning including the controlled word fluency test (FAS test) (Goodglass and Kaplan, 1972) and a divergent thinking task [based on Guilford and Hoepfner (1971)].

As noted previously, if electrodermal hyporesponsiveness reflects orbitofrontal hypofunction, then APD subjects exhibiting core psychopathic personality traits should display reduced physiological reactions to aversive stimuli. We tested this prediction by monitoring subjects' electrodermal activity during the presentation of emotionally charged stimuli. Subjects viewed 30 words possessing positive, negative, or neutral emotional connotations. Subjects rated the words (displayed one at a time on the monitor) in terms of pleasantness (1–7 scale) while the experimenter monitored the subjects' electrodermal activity (tonic and phasic) with the Davicon C2A custom skin conductance monitor (NeuroDyne Medical Corporation, Cambridge, MA). The resolution of the Davicon C2A custom skin conductance system is 0.01 micromhos. Electrodes were attached to the distal phalanges of the first and second fingers of the subject's dominant hand. While conductive gel is used with Ag–AgCl electrodes, the contact surface of the electrodes employed in our study was gold, and gold electrodes do not require conductive paste. The combined effective surface area of the sensors was 1 cm^2. The subject's skin was not prepared. Data collection was divided into 10-s intervals following the presentation of each stimulus. Davicon psychophysiological assessment software provided the basepoint and the maximum point during each interval and subtracted the basepoint from the maximum score, yielding a measure of SCR in micromhos. That is, the software automatically calculated the amplitude of the phasic response. We calculated the mean phasic response to each stimulus class (i.e., positive, negative, or neutral stimuli) for each subject. In addition, a research technician observed the subject during the word-rating task and noted if the participant moved during electrodermal monitoring. These trials were excluded from subsequent analysis. We used the *Handbook of Semantic Word Norms* (Toglia and Battig, 1978) to categorize words (see Appendix A).

We also administered a battery of personality questionnaires and clinical scales. Administered measures included Cattell's 16PF questionnaire (factors G, H, and O); personality diagnostic questionnaire (PDQ-4; antisocial personality disorder) subscale (Hyler, 1994), and a modified version of the fear survey schedule (Wolpe and Lang, 1964) consisting of three subscales. During the fear survey, participants were instructed to indicate the degree of avoidance behavior associated with specific situations or stimuli presented on the computer screen because of fear or anxiety. Stimuli were classified in the following manner: (1) social situations; (2) specific phobic situations; and (3) agoraphobic concerns. We also administered subscales from the 16PF questionnaire (factors G, H, and O) which reflect personality characteristics (e.g., lack of threat-sensitivity, guilt, conscientiousness, and behavioral inhibition) associated with APD and psychopathy. We anticipated that APD subjects would obtain low scores on factors O and G, reflecting a lack of guilt and concern for social conventions, and achieve high scores on factor H, reflecting social disinhibition.

2.2. Description of neurocognitive tests and clinical scales

2.2.1. Object alternation test

Performance on the object alternation test is determined by number of trials required to induce the solution. Subjects view two distinct stimulus objects (a red cup and a blue cup) on a computer monitor. The computer 'hides' a coin in one of the cups. The subjects are asked to determine which cup contains the coin. The coin moves to the unoccupied cup following a correct response. Subjects receive immediate feedback from the computer regarding the accuracy of response following each choice. Participants reach criterion when they correctly predict the coin location on 12 consecutive trials. The subjects' score is the trial number of their last wrong response before the onset of their run of 12

correct trials. A low score indicates superior performance. The score of participants who never reach the solution is set to 50, the maximum number of trials (Freedman, 1990).

2.2.2. Stroop color−word test

During our computer version (administered on a Macintosh IIci computer using the experimental design software PsyScope), subjects are asked to read words as quickly as possible (displayed one at a time on the computer monitor) describing a color. The ink color may be inconsistent with the given word. During the first block (non-conflict block), the subject is asked to read the word displayed on the monitor and ignore the ink color (40 trials). During the second block (conflict block), the participant is asked to identify the ink color (40 trials). Response latencies were recorded using a voice-activated millisecond timer. Each trial ends when the subject responds verbally into a microphone. All participants completed both blocks. The dependent measure was response time.

2.2.3. Go / no−go task

This test was based on Lapierre et al. (1995). Subjects press the space bar as quickly as possible when a 2×2-cm blue square appears (against a white background) on a computer monitor. During the first block (50 trials), only blue squares are displayed. During the second block (50 trials), subjects are instructed to respond when the blue square appears and refrain from responding when a 2×2-cm blue cross is displayed. During the third block (50 trials), subjects are instructed to respond when the blue cross is displayed and refrain from responding when the square appears. The blue square or blue cross appears at random locations across the computer screen. The inter-stimulus interval is also randomized with intervals of 100, 250, 400, 500, 750, 1000, and 2000 ms.

2.2.4. Assessment of autonomic activity

Subjects view 30 words possessing positive, negative, or neutral emotional connotations. The subjects rate the words (displayed one at a time on the monitor) in terms of aversiveness (1−7 scale) while the experimenter monitors subjects' electrodermal activity.

2.2.5. Word fluency test (FAS test)

During the word fluency test (FAS test), the subject is asked to write down as many words as possible that begin with a specific letter (F, A, or S) during three 1-min trials (Goodglass and Kaplan, 1972).

2.2.6. Divergent thinking task

This task is based on Guilford and Hoepfner (1971). During the divergent thinking task, subjects are asked to name as many different uses of a newspaper as possible during a 1-min trial. They are provided with the following example: one use is rolling up the newspaper to swat a mosquito.

2.2.7. Personality diagnostic questionnaire (PDQ-4) (APD subscale)

The PDQ-4 is a true−false questionnaire which yields subscale scores reflecting *DSM-IV* diagnostic criteria for axis-II disorders. Questions were adapted from DSM-IV diagnostic criteria (Hyler, 1994).

2.2.8. Psychopathy checklist: screening version (PCL:SV)

The PCL:SV is designed to yield a total score and subscale scores reflecting two correlated factors. Factor 1 reflects interpersonal and affective characteristics associated with psychopathy such as egocentricity and profound lack of empathy, while factor 2 reflects behavioral dyscontrol, impulsivity, and antisocial conduct (Hart et al., 1995).

3. Results

Since multiple comparisons were planned, we controlled for type 1 errors with the Bonferroni procedure. Thus, the alpha level was set to 0.003.

3.1. Neuropsychological testing

We conducted a multivariate analysis of variance (MANOVA). Significant MANOVA findings

were followed up with post hoc comparisons using the Tukey test of significance. MANOVA indicated that the APD group was impaired on tasks considered sensitive to orbitofrontal dysfunction relative to control subjects: Wilks' Lambda = 0.136; $F_{9,12} = 8.439$; $P < 0.001$. Subsequent univariate analyses yielded significant group differences on the object alternation test ($F_{1,20} = 26.462$, $P < 0.001$), and conflict blocks of the Stroop color−word test ($F_{1,20} = 12.011$, $P < 0.003$ and $F_{1,20} = 13.446$, $P < 0.001$). APD subjects exhibited performance deficits on the object alternation test and conflict blocks of the Stroop task in comparison to community control subjects. The mean number of trials to solve the object alternation test was 11.8 (SD = 4.7) for the community control group, but was 33.9 (SD = 12.8) for the APD group.

Relative to the community control group, APD subjects demonstrated slower reaction times on the color-naming blocks of the Stroop color−word test (see Table 1). When required to inhibit a previously learned response pattern (i.e., conflict blocks), APD subjects displayed greater reaction times in comparison to control subjects. Group differences on the non-conflict blocks of the Stroop (wordnaming) ($Ps > 0.35$) and go/no-go (block 1) ($P > 0.11$) tasks were not statistically significant. Contrary to expectation, group differences on the conflict blocks of the go/no−go task were not significant, although group differences on the third block of the go/no−go task approached significance ($P < 0.06$). APD subjects did not demonstrate performance deficits on classical tests of frontal executive function. Interestingly, the APD subjects generated significantly more responses on the divergent thinking task than control participants ($F_{1,20} = 14.051$, $P < 0.001$). APD subjects did produce fewer words on the word fluency test relative to control subjects,

Table 1
Neuropsychological test performance[a]

	APD		Mean (SD) Control subjects		Univariate analysis $F_{1,20}$	P
n	12		10			
Age	27.8	(4.0)	28.9	(6.9)	−0.202	0.658
Education	13.9	(1.72)	13.9	(1.74)	0.006	0.938
Handedness	100%	Right	100%	Right		
OAT	**33.9**	**(12.8)**	**11.8**	**(4.7)**	**26.462**	**< 0.001**
Stroop color−word test						
Stroop Word-c	479 ms	(50)	505 ms	(81)	−0.843	0.369
Stroop Word-i	521 ms	(79)	549 ms	(95)	−0.606	0.445
Stroop Color-c	**948 ms**	**(223)**	**674 ms**	**(121)**	**12.011**	**< 0.003**
Stroop Color-i	**1214 ms**	**(334)**	**811 ms**	**(103)**	**13.446**	**< 0.001**
Go/no−go task						
Go/no−go — 1	322 ms	(61.5)	284 ms	(38.9)	2.746	0.113
Go/no−go — 2	495 ms	(71.9)	468 ms	(55.7)	0.943	0.345
Go/no−go — 3	505 ms	(56.3)	459 ms	(45.9)	4.083	0.056
FAS Test	38.0	(5.6)	41.6	(9.0)	−1.294	0.268
DvT	**9.5**	**(1.9)**	**6.8**	**(1.2)**	**14.051**	**0.001**
SCR (micromhos)	**0.02**	**(0.01)**	**0.14**	**(0.08)**	**−24.333**	**< 0.001**

    [a]*Note:* OAT = object alternation test; Stroop = Stroop color−word test (blocks 1 and 2 = word naming, blocks 3 and 4 = color naming), c = congruent, i = incongruent; Go/no−go = go/no go task (blocks 1, 2, 3); FAS = Controlled word fluency test (FAS test); DvT = divergent thinking task; ms = milliseconds; SCR = mean amplitude of skin conductance response (micromhos).

although this difference did not approach significance ($P > 0.25$).

The mean amplitudes of skin conductance response (SCR) to the three categories of words (positive, neutral, and negative) were compared across the subject groups. As expected, APD subjects were electrodermally hyporesponsive to aversive stimuli relative to control group members ($F_{1,20} = -24.333$, $P < 0.001$). In addition, all participants, regardless of group membership, categorized words in the expected manner, demonstrating that they were aware of the emotional connotations of the stimuli.

3.2. Clinical profile

MANOVA, followed by post hoc analyses, revealed highly significant group differences on the clinical scales and personality measures. As expected, APD subjects achieved significantly lower scores on factors O ($F_{1,20} = -18.768$, $P < 0.001$) and G ($F_{1,20} = -12.814$, $P < 0.001$) from the 16PF questionnaire, and on the social anxiety subscale ($F_{1,20} = -34.246$, $P < 0.001$) in comparison to control subjects. In addition, they displayed significantly higher scores on the APD subscale of the PDQ-4 ($F_{1,20} = 67.663$, $P < 0.001$), the PCL:SV ($F_{1,20} = 631.315$, $P < 0.001$) and on factor H ($F_{1,20} = 18.782$, $P < 0.001$). This clinical profile indicates that APD subjects, as expected, were less conscientious, self-reproaching, guilt-prone, and socially anxious than matched control subjects. Moreover, the scores indicated that APD subjects were more venturesome and uninhibited relative to control subjects. However, the scores of APD subjects on the specific phobic situations subscale did not differ significantly from the scores achieved by control participants ($P > 0.78$) (see Table 2). Group comparison revealed a marginally significant difference on the agoraphobia subscale ($F_{1,20} = -6.364$, $P < 0.02$).

Five of 12 APD subjects reported a history of substance abuse. To examine the impact of prior substance abuse on neuropsychological test performance, we compared the performance patterns of APD subjects who met criteria (lifetime) for alcohol or substance abuse to the neurocognitve profiles of APD subjects who did not meet criteria for substance abuse or dependence. The clinical and neurocognitive profiles of these groups were remarkably similar. Groups did not differ significantly on the object alternation test ($P >$

Table 2
Personality and clinical findings[a]

Clinical Characteristics	Mean (SD)				Univariate analysis	
	APD		Control subjects		$F_{1,20}$	P
N	12		10			
16 PF Subscales						
Factor G	**6.4**	**(3.7)**	**11.3**	**(2.4)**	**−12.814**	**< 0.001**
Factor H	**20.5**	**(2.7)**	**13.3**	**(4.9)**	**18.782**	**< 0.001**
Factor O	**7.8**	**(2.8)**	**12.6**	**(2.3)**	**−18.768**	**< 0.001**
Fear Survey Subscales						
Agoraphobia	1.9	(3.2)	5.8	(4.0)	−6.364	< 0.02
Social anxiety	**3.9**	**(3.3)**	**15.5**	**(5.8)**	**−34.246**	**< 0.001**
Specific phobia	13.2	(8.8)	14.1	(4.6)	−0.075	0.787
APD (PDQ)	**6.6**	**(1.78)**	**1.4**	**(0.96)**	**67.663**	**< 0.001**
PCL:SV	**18.5**	**(1.2)**	**3.4**	**(1.5)**	**631.315**	**< 0.001**

[a]*Note:* 16 PF Questionnaire — factors G, H, and O; Fear survey schedule — agoraphobia subscale; specific phobia subscale; social anxiety subscale); Personality diagnostic questionnaire (PDQ-4); APD = antisocial personality disorder subscale (PDQ-4); Psychopathy checklist: screening version (PCL:SV).

0.45), Stroop color−word test (all $Ps > 0.15$), go/no−go task (all $Ps > 0.10$), verbal fluency task ($P > 0.12$), and divergent thinking task ($P > 0.15$). In addition, groups did not exhibit significantly different patterns of autonomic reactivity ($P > 0.25$).

4. Discussion

4.1. Neurocognitive test findings

APD subjects showed greater neuropsychological deficits on measures sensitive to orbitofrontal dysfunction in comparison to control participants. Performance deficits on the object alternation test may reflect an inability to effectively process feedback information regarding reward and punishment (i.e., the inability to successfully employ punishment cues to guide behavior). Rolls (1995) suggested that the orbitofrontal region determines the reinforcing value of stimuli.

In our prior unpublished work, performance deficits on the go/no−go and Stroop tasks (conflict blocks) correlated strongly with object alternation test performance, suggesting that the orbitofrontal system is involved in the inhibition of a dominant or prepotent response pattern, which is consistent with its role in modifying behavior in response to changing contingencies. In addition, a number of APD subjects were unable to identify the correct alternation pattern. The score of participants who never reached the solution was set at 50, the maximum number of trials. Interestingly, several of these subjects employed 'superstitious' response strategies. For example, one APD subject stated that 'everything goes in threes' and he would make three perseverative responses before shifting to the alternate choice (i.e., the correct location). We should note that we did not screen APD subjects for schizotypal personality traits. It would be interesting to determine if the presence of schizotypal features among APD or psychopathic subjects is associated with performance deficits on the object alternation test. Moreover, several APD subjects exhibited extreme perseverative behavior. For example, one APD subject made a correct choice on the initial

trial. The coin shifted to the unoccupied cup after the correct response. The subject continued to choose the cup which originally contained the coin and made 49 consecutive incorrect responses (despite the fact that the research technician pointed out that he was free to choose the other cup).

APD subjects did not demonstrate performance deficits on classical tests of frontal executive function. Our findings are consistent with the contention that a highly select deficit involving the orbitofrontal system is associated with psychopathy and APD.

Contrary to prediction, group differences on the go/no−go Task were not statistically significant. We anticipated that APD subjects would exhibit performance deficits (e.g., reaction time slowing) on the go/no−go Task. This finding does not support our central hypothesis. Unexpectedly, APD subjects performed significantly better than control subjects on the divergent thinking task. To our knowledge, the divergent thinking task has never been administered to APD or psychopathic subjects. However, it is interesting to note that several studies found that individuals psychometrically defined as extraverted, or exhibiting low levels of anxiety, obtained significantly higher scores on divergent thinking tasks.

4.2. Do the neuropsychological measures employed possess localizing value?

Patients with prefrontal damage perform similarly to control subjects on measures of general cognitive ability (i.e., standard intelligence test scores are typically in the normal range) and on verbal and non-verbal memory tasks (e.g., paired associate learning). However, careful neuropsychological examination will reveal subtle cognitive deficits. Researchers have come to appreciate that the prefrontal region is not a unitary structure; rather, it is fractionable into anatomically and functionally distinct subsystems. Converging lines of evidence suggest that dorsolateral-prefrontal cortex mediates executive functions (Smith and Jonides, 1999), while orbitofrontal cortex modulates sensitivity to reinforcement contingencies (Rolls, 1995).

Functional neuroimaging research and human lesion studies suggest that the tasks employed in our protocol are sensitive measures of prefrontal dysfunction. Of course, we must proceed cautiously when we argue that variations in brain function correspond to patterns of neuropsychological impairment and clinical presentation. Several lines of evidence suggest that many of the experimental tasks employed in this study possess localizing value.

Performance deficits on the object alternation test are associated with orbitofrontal dysfunction in human and non-human primates (Freedman et al., 1998; Mishkin, 1964; Mishkin et al., 1969; Pribram and Mishkin, 1956). Following prior work in the neuropsychiatric literature, we assume that the orbitofrontal cortex mediates sensitivity to dynamically changing reinforcement contingencies, and thus may be particularly important for modulating individuals' response to the social world and other threat-laden situations. Therefore, impaired performance on the object alternation test may reflect an inability to effectively process feedback information regarding reward and punishment. Meunier et al. (1997) found that the formation of object–reward associations was impaired following orbitofrontal lesions in non-human primate subjects, and Rolls (1995) suggested that the orbitofrontal region determines the reinforcing value of the stimuli. Poor performance on the object alternation test among APD subjects may reflect a deficit involving the inability to successfully employ punishment cues to guide behavior.

The object alternation test is a moderately difficult induction task which requires the subject to process positive and negative feedback information. Animal lesion studies may provide insight into the relation between performance deficits on the object alternation test and orbitofrontal dysfunction. Kesner (1992) noted that "orbitofrontal cortex-damaged animals have difficulty changing their behavior when the value of rewards is not consistent with expectations based on prior experiences. Thus, animals with orbitofrontal cortex lesions display prolonged extinction of a previously rewarded response." (p. 393).

Difficulty inhibiting a prepotent response during the go/no–go and Stroop tasks may indicate ventral/orbitofrontal dysfunction. Several studies demonstrated that performance deficits on go/no–go tasks (e.g., reaction time slowing and frequency of false alarms) were associated with orbitofrontal lesions (e.g. Malloy et al., 1993). However, it is important to note that imaging studies document activation in orbitofrontal as well as dorsolateral–prefrontal regions during go/no–go task performance in children and adults (Casey et al., 1997). Subjects undergoing positron emission tomography (PET) while participating in the Stroop color–word test demonstrated right orbitofrontal activation as well as increased activity in bilateral parietal structures (Bench et al., 1993). During a second experiment, Bench et al. (1993) documented right frontal polar and right anterior cingulate activation during Stroop task performance. Carter et al. (1997) reported that subjects with schizophrenia exhibited reduced anterior cingulate activation relative to matched control subjects during the Stroop task performance. Efficient performance on the Stroop and go/no–go tasks requires sustained attention and impulse control. Therefore, the go/no–go task and the Stroop color–word test should be considered broadly frontal tasks. Nevertheless, the orbitofrontal or ventral prefrontal systems may be implicated when presenting symptoms including social cognitive deficits, disinhibition, and impaired performance on neurocognitive tasks which require the subject to suppress a prepotent response pattern.

Impaired verbal fluency may reflect DLPF dysfunction. Regional cerebral blood flow (rCBF) and PET studies revealed significant flow augmentation and increased activity in DLPF cortex during word fluency tasks (Cantor-Graae et al., 1993; Warkentin et al., 1991). Moreover, patients with damage to the dorsolateral aspect of the prefrontal region display performance deficits on the FAS test. Warkentin and colleagues (1991) reported that volunteers demonstrated significant flow augmentation in DLPF cortex during a verbal fluency task. Frith et al. (1991) employed PET and documented increased DLPF activity during a verbal fluency task.

In summary, the imaging and human lesion

literature is broadly consistent with the notion that the prefrontal region is fractionable into anatomically and functionally distinct subsystems. However, our findings must be interpreted with caution. It is important to bear in mind that neurocognitive tests are only indirect measures of neurophysiological function and the localizing value of such tasks is uncertain.

4.3. Clinical presentation

Contrary to expectations, APD subjects and community control subjects did not differ on a self-report measure of sensitivity to specific phobic situations. During the fear survey, participants are instructed to indicate the degree of avoidance behavior associated with specific situations or stimuli. APD subjects scored significantly lower on the social anxiety subscale relative to matched control subjects, demonstrating a profound lack of social anxiety. Unexpectedly, APD and matched control subjects obtained similar scores on the subscale reflecting specific phobic concerns (e.g., heights). However, this finding does not indicate that APD or control subjects were describing irrational fears or phobias. The control group mean on the social anxiety subscale was 15.5, indicating low to moderate avoidance behavior, while the APD group mean was 3.9, indicating an almost complete absence of social anxiety. However, the groups achieved similar scores on the specific phobic situation subscales, with control subjects obtaining a mean score of 14.1, while the APD group mean was 13.2. These scores are not clinically significant; rather, they suggest a low to moderate level of avoidance behavior and are not indicative of irrational fears or phobias. This finding is inconsistent with the contention that the principal deficit underlying core psychopathic personality traits is a diminished (or absent) fear response. For example, Cleckley (1941, 1955, 1982) concluded that an absence of anxiety and psychoneurotic manifestations was a defining characteristic of the psychopathic personality. Lykken (1957, 1995) maintained that psychopaths possess a "low-fear quotient" and suggested that "fearlessness" was an etiologic factor in psychopathy. Lykken (1957, 1995) suggested that the socializa-

tion and conscience development of the psychopath is stunted because of a reduction in general fearfulness. The psychopath's diminished sensitivity to aversive stimuli hinders the acquisition of avoidance responses essential to socialization and the inhibition of antisocial impulses. Hare (1978) suggested that the 'psychopath's apparent inability to anticipate the negative consequences of his own behavior is a reflection of the failure of physical, symbolic and other cues to generate sufficient anticipatory fear for the instigation and reinforcement of avoidance behavior' (p. 120).

In our study, APD and control groups did not differ on a *single* self-report measure of phobic avoidance. Obviously, this finding must be replicated. Nevertheless, it was surprising that individuals displaying core psychopathic personality traits differed on measures of social anxiety and conscientiousness, but did not differ on a measure of specific phobic concerns. This argues against a global reduction in fearfulness in psychopathy. Moreover, we found a strong association between performance on orbitofrontal tasks and on measures of social anxiety among clinical and community samples (unpublished research). Similarly, performance deficits on tasks sensitive to orbitofrontal dysfunction were associated with elevated scores on measures of social anxiety. However, there was no association between performance on executive function tasks and scores on social anxiety measures (Dinn and Harris, 2000; Dinn et al., 2000). In addition, there was no significant association between performance on orbitofrontal tasks and scores on measures of generalized anxiety.

These findings are consistent with the claim that orbitofrontal cortex is an essential component of the 'social brain' (Baron-Cohen and Ring, 1994) and plays a major role in social cognition. This corpus of work suggests that orbitofrontal cortex is directly involved in social cognition and the formation of a theory of mind. In fact, the study of psychopathy may help researchers assess the validity of two influential models of frontal lobe function: (1) Damasio's somatic marker hypothesis; and (2) the social cognition model of Baron-Cohen and colleagues.

Damasio and colleagues concluded that the profound impairments in the social domain exhibited by ventromedial prefrontal lesion (VMPF) patients (orbital and lower mesial frontal regions) stem from an inability to experience somatic states associated with both positive and negative affect (Bechara et al., 1997, 1994; Damasio, 1994; Damasio et al., 1990). Without emotional coloring to guide action, decision-making, particularly in the social domain, becomes problematic. A considerable body of research examining autonomic arousal among psychopathic subjects lends support to a theory of psychopathy based on the somatic marker hypothesis. Damasio and colleagues reported that social cognition is preserved in VMPF lesion patients. That is, individuals with VMPF lesions possess 'abstract social knowledge', yet fail to effectively employ such knowledge. Thus, the somatic marker hypothesis maintains that social cognitive processes are preserved, while the emotional component of this reasoning process is diminished or absent. While this model has intuitive appeal and empirical support, Baron-Cohen and colleagues found that patients with orbitofrontal lesions exhibited subtle social cognition deficits in comparison to matched control subjects (i.e., patients with DLPF damage) (Stone et al., 1998). To address this issue, researchers should investigate social cognitive ability by administering advanced theory of mind tasks to psychopathic subjects.

Does the orbitofrontal system mediate what Simon (1990) described as '*docility*' or receptivity to social influence? Baron-Cohen and colleagues suggested that a cortical–subcortical circuit involving the orbitofrontal cortex, the superior temporal sulcus, and the amygdala plays a significant role in social cognition and underlies the ability to form mental state representations of others' beliefs and desires (i.e., theory of mind) (Baron-Cohen et al., 1994; Baron-Cohen, 1995; Baron-Cohen and Ring, 1994). Orbitofrontal system dysfunction may underlie the gross distortion of processes which serve to integrate the individual into the social group witnessed in psychopathy. Psychopaths appear indifferent to group censure, display a profound lack of empathy and guilt, and are described as pathologically egocentric. The

psychopath is sensitive to punishment when it involves the loss of a reinforcer (e.g., money) (Schmauk, 1970), yet appears relatively indifferent to aversive social cues typically employed to modify behavior such as rejection, criticism, or loss of social status. Our findings and a recent study examining psychophysiological response patterns among psychopathic individuals during exposure to threat-related stimuli (Blair et al., 1997) suggest that the psychopath's characteristic hyporesponsiveness to emotionally arousing stimuli may be domain-specific. This conclusion represents a departure from traditional theoretical approaches which maintain that psychopathic subjects are insensitive to threat-related cues and manifest a marked lack of emotion.

Blair et al. (1997) monitored electrodermal activity (SCRs) of psychopathic and non-psychopathic subjects during the presentation of threatening and neutral stimuli and reported no significant differences between psychopathic and non-psychopathic subjects in SCR to threatening cues (e.g., color slide of a coiled snake), while groups did differ when exposed to distress cues (e.g., color slide of a child crying) in the expected direction. That is, psychopathic subjects were hyporesponsive to distress cues, but not threatening stimuli. Blair et al. (1997) suggested that reduced electrodermal responses to distress cues among psychopathic subjects reflect dysfunction of a violence inhibition mechanism and may account for the psychopath's characteristic lack of empathy. However, these findings also support a social anxiety deficit hypothesis and are consistent with our results demonstrating that APD subjects did not differ from community control subjects on measures of specific phobic avoidance.

4.4. Future research directions

Future research may help us account for conflicting findings in the neuropsychological literature regarding performance deficits among APD and psychopathic subjects on tests assessing frontal executive function. Specifically, we predict that core psychopathic personality characteristics will be associated with performance deficits on orbitofrontal measures, while impaired perfor-

mance on tests assessing frontal executive functioning will be associated with a lack of foresight, planning, and goal-directed behavior (e.g., disorganized offending). Clearly, APD is a heterogeneous disorder and different aspects of its complex clinical presentation may reflect dysfunction in discrete neural systems. Hart et al. (1995) suggest that two distinct factors underlie the clinical presentation of psychopathy. Factor 1 reflects interpersonal and affective characteristics such as egocentricity and profound lack of empathy, while factor 2 reflects behavioral dyscontrol, impulsivity, and antisocial behavior. It is important to emphasize that APD and psychopathy represent discrete syndromes which may coexist in the same individual. Researchers have come to appreciate that the prefrontal region is not a unitary structure; rather, it is fractionable into anatomically and functionally distinct systems and these subsystems may be differentially engaged in APD. An examination of the relationship between clinical phenomenology and neurocognitive profile is warranted. The following neurobehavioral classification scheme may help us understand the phenomenology and underlying pathophysiology of APD and psychopathy:

1. Individuals who display core psychopathic personality traits (e.g., pathological egocentrism, lack of empathy, superficiality, and so forth), but do not exhibit behavioral dyscontrol and a lack of foresight will demonstrate a select orbitofrontal deficit. Core psychopathic personality characteristics are likely to result from orbitofrontal dysfunction.
2. APD subjects demonstrating core psychopathic traits, and a lack of foresight (e.g., difficulty anticipating negative consequences), planning, and goal-directed behavior (e.g., disorganized offending) will show greater neuropsychological deficits on tasks considered sensitive to orbitofrontal dysfunction and on measures of executive function (i.e., tasks which require the employment of organizational strategies for efficient performance) which are considered sensitive indicators of DLPF dysfunction.

4.5. Limitations

One obvious limitation of this study is sample size and the number of statistical comparisons carried out. Therefore, findings must be interpreted with caution. However, we attempted to address these concerns during statistical analysis (i.e., employing the Bonferroni procedure) and by carefully matching APD and control subjects for age, gender, and educational level. Moreover, the recruitment of individuals meeting diagnostic criteria for APD or psychopathy is problematic. Such individuals do not typically present for treatment. Many researchers obtain access to forensic samples and identify incarcerated APD or psychopathic subgroups. However, exclusive reliance on forensic samples is problematic. Issues involving informed consent and the effects of institutionalization may render the interpretation of neuropsychological test performance and the generalization of results to non-incarcerated populations problematic. Therefore, we employed the community recruitment technique developed by Widom (1977). Nevertheless, we found that the neuropsychological test performance patterns of APD subjects recruited from the general population are similar to patterns demonstrated by incarcerated psychopathic subjects (e.g. Lapierre et al., 1995). However, the APD subjects participating in our study may not represent a prototypical APD sample. That is, they displayed prominent psychopathic personality characteristics and exhibited long-standing patterns of antisocial conduct and may, therefore, represent an atypical sample. Additional limitations of this study include the following: although control subjects were closely matched to APD subjects for age, educational level, handedness, and gender, we did not determine the socioeconomic status of participants or their families. Also, the lead experimenter (W. Dinn) was not blind to the clinical status of participants at the time of testing.

4.6. Conclusion

We assessed neuropsychological functions, autonomic reactivity, and clinical presentation and generated a detailed neurocognitive profile of

APD subjects presenting with prominent psychopathic personality traits. To our knowledge, this study represents the first administration of the object alternation task, a robust marker of orbitofrontal dysfunction in prior research, and the divergent thinking task to APD or psychopathic subjects. Of course, it is important to emphasize that the striking group differences on clinical and personality measures were expected. Indeed, they are a function of our inclusion criteria. APD subjects exceeded symptom thresholds on the APD subscale (PDQ-4) and on the PCL:SV. One purpose of the clinical/personality assessment component of our protocol was to ensure that we obtained a robust clinical sample. Contrary to expectation, APD subjects and matched control subjects did not differ on a self-report measure of sensitivity to specific phobic situations. This finding is not consistent with the hypothesis that the principal deficit underlying psychopathy is a greatly diminished fear response.

Of greater interest were the neuropsychological test findings. As predicted, APD subjects displayed performance deficits on two putative orbitofrontal measures. Moreover, they did not exhibit impaired performance on two classical tests of frontal executive function and on the go/no−go Task. The latter finding was not expected given that prior research found that psychopathic subjects displayed performance deficits on a similar visual go/no−go discrimination task (Lapierre et al., 1995). Contrary to expectations, APD subjects achieved significantly higher scores on the divergent thinking task.

Researchers have come to appreciate that the prefrontal region can be fractionated into anatomically and functionally distinct subsystems (i.e. orbitofrontal and DLPF systems). Following Lapierre and colleagues, we attempted to address this distinction by including neurocognitive tasks considered sensitive to dysfunction in discrete prefrontal subsystems (i.e. orbitofrontal and DLPF), although, as noted previously, the localizing value of such tasks remains controversial. As recently as 1995, Lykken could write, "Taken all together, it seems fair to say that the search for neuropsychological evidence of frontal lobe or verbal-left hemisphere defect in psychopaths has been unsuccessful" (p. 178). However, recent neuropsychological and neuroimaging studies suggest that prefrontal dysfunction is, indeed, associated with psychopathy.

Acknowledgements

Appendix A

Happiness
Grave
Envelope
Cruel
Friend
Disease
Blossom
Death
Mile
Pain
Wisdom
Lice
Magnet
Murder
Lemonade
Sadist
Waist
Rabies
Music
Kill
Chair
Suffocate
Happy
Tuberculosis
Softly

Cancer
Table
Humiliation
Laugh
Poison

References

American Psychiatric Association, 1994. Diagnostic and Statistical Manual of Mental Disorders (4th ed.). APA, Washington, DC.

Baron-Cohen, S., 1995. Mindblindness: An Essay on Autism and Theory of Mind. MIT Press, Cambridge, MA.

Baron-Cohen, S., Ring, H., 1994. A model of the mindreading system: neuropsychological and neurobiological perspectives. In: Lewis, C., Mitchell, P. (Eds.), Children's Early Understanding of Mind: Origins and Development. Lawrence Erlbaum Associates, Hove, UK, pp. 183−207.

Baron-Cohen, S., Ring, H., Moriarty, J., Schmitz, B. et al., 1994. Recognition of mental state terms: clinical findings in children with autism and a functional neuroimaging study of normal adults. British Journal of Psychiatry 165, 640−649.

Bechara, A., Damasio, A.R., Damasio, H., Anderson, S.W., 1994. Insensitivity to future consequences following damage to human prefrontal cortex. Cognition 50, 7−15.

Bechara, A., Damasio, H., Tranel, D., Damasio, A.R., 1997. Deciding advantageously before knowing the advantageous strategy. Science 275, 1293−1295.

Bench, C.J., Frith, C.D., Grasby, P.M., Friston, K.J., Paulesu, E., Frackowiak, R.S.J., Dolan, R.J., 1993. Investigations of the functional anatomy of attention using the Stroop test. Neuropsychologia 31, 907−922.

Blair, R.J.R., Jones, L., Clark, F., Smith, M., 1997. The psychopathic individual: a lack of responsiveness to distress cues? Psychophysiology 34, 192−198.

Blumer, D., Benson, D.F., 1975. Personality changes with frontal and temporal lobe lesions. In: Benson, D.F., Blumer, D. (Eds.), Psychiatric Aspects of Neurologic Disease. Grune & Stratton, New York, pp. 151−170.

Cantor-Graae, E., Warkentin, S., Franzen, G., Risberg, J., 1993. Frontal lobe challenge: a comparison of activation procedures during rCBF measurements in normal subjects. Neuropsychiatry, Neuropsychology and Behavioral Neurology 6, 83−92.

Carter, C.S., Mintun, M., Nichols, T., Cohen, J.D., 1997. Anterior cingulate gyrus dysfunction and selective attention deficits in schizophrenia: [^{15}O]H$_2$O PET study during single-trial Stroop task performance. American Journal of Psychiatry 154, 1670−1675.

Casey, B.J., Trainor, R.J., Orendi, J.L., Schubert, A.B., Nystrom, L.E., Giedd, J.N., Castellanos, F.X., Haxby, J.V., Noll, D.C., Cohen, J.D., Forman, S.D., Dahl, R.E., Rapoport, J.L., 1997. A developmental functional MRI study of prefrontal activation during performance of a go−no−go task. Journal of Cognitive Neuroscience 9, 835−847.

Cleckley, H., 1941, 1955, 1982. The Mask of Sanity. Mosby, St. Louis.

Cohen, J.D., MacWhinney, B., Flatt, M.R., Provost, J., 1993. PsyScope: an interactive graphic system for designing and controlling experiments in the psychology laboratory using Macintosh computers. Behavior Research Methods, Instruments and Computers 25, 257−271.

Cummings, J.L., 1993. Frontal-subcortical circuits and human behavior. Archives of Neurology 50, 873−880.

Damasio, A.R., 1994. Descartes' Error: Emotion, Reason and the Human Brain. Putnam, New York.

Damasio, A.R., Tranel, D., Damasio, H., 1990. Individuals with sociopathic behavior caused by frontal damage fail to respond autonomically to social stimuli. Behavioural Brain Research 41, 81−94.

Damasio, A.R., Van Hoesen, G.W., 1983. Emotional disturbances associated with focal lesions of the limbic frontal lobe. In: Heilman, K.M., Satz, P. (Eds.), Neuropsychology of Human Emotion. Guilford Press, New York, pp. 85−110.

Davidson, R.J., Putnam, K.M., Larson, C.L., 2000. Dysfunction in the neural circuitry of emotion regulation: a possible prelude to violence. Science 289, 591−594.

Deckel, A.W., Hesselbrock, V., Bauer, L., 1996. Antisocial personality disorder, childhood delinquency, and frontal brain functioning: EEG and neuropsychological findings. Journal of Clinical Psychology 52, 639−650.

Dinn, W.M., Harris, C.L., 2000. Obsessive−compulsive disorder: neuropsychological correlates and clinical presentation (under review).

Dinn, W.M., Harris, C.L., Cooke, G.L., 2000. Orbitofrontal dysfunction in a non-clinical population: evidence for a neurobehavioral continuum (under review).

Freedman, M., 1990. Object alternation and orbitofrontal system dysfunction in Alzheimer's and Parkinson's disease. Brain and Cognition 14, 134−143.

Freedman, M., Black, S., Ebert, P., Binns, M., 1998. Orbitofrontal function, object alternation and perseveration. Cerebral Cortex 8, 18−27.

Frith, C.D., Friston, K.J., Liddle, P.F., Frackowiak, R.S., 1991. A PET study of word finding. Neuropsychologia 29, 1137−1148.

Goodglass, H., Kaplan, E., 1972. The Assessment of Aphasia and Related Disorders. Lea & Febiger, Philadelphia.

Gorenstein, E.E., 1982. Frontal lobe functions in psychopaths. Journal of Abnormal Psychology 91, 368−379.

Grattan, L.M., Bloomer, R.H., Archambault, F.X., Eslinger, P.J., 1994. Cognitive flexibility and empathy after frontal lobe lesion. Neuropsychiatry, Neuropsychology and Behavioral Neurology 7, 251−259.

Guilford, J.P., Hoepfner, R., 1971. The Analysis of Intelligence. McGraw-Hill, New York.

Guyton, A.C., 1991. Textbook of Medical Physiology (8th ed.). W.B. Saunders, Philadelphia.

Hare, R.D., 1984. Performance of psychopaths on cognitive tasks related to frontal lobe function. Journal of Abnormal Psychology 93, 133−140.

Hare, R.D., 1978. Electrodermal and cardiovascular correlates of psychopathy. In: Hare, R.D., Schalling, D. (Eds.), Psychopathic Behavior: Approaches to Research. Wiley, New York, pp. 107–143.

Hare, R.D., Craigen, D., 1974. Psychopathy and physiological activity in a mixed-motive game situation. Psychophysiology 11, 197–206.

Hare, R.D., Frazelle, J., Cox, D.N., 1978. Psychopathy and physiological responses to threat of an aversive stimulus. Psychophysiology 15, 165–172.

Hart, S.D., Cox, D.N., Hare, R.D., 1995. The Hare Psychopathy Checklist: Screening Version (PCL:SCV). Multi-Health Systems Inc, Toronto.

House, T.H., Milligan, W.L., 1976. Autonomic responses to modeled distress in prison psychopaths. Journal of Personality and Social Psychology 34, 556–560.

Hyler, S.E., 1994. Personality Diagnostic Questionnaire (PDQ-4). New York, NY.

Jones-Gotman, M., Zatorre, R.J., 1988. Olfactory identification deficits in patients with focal cerebral excision. Neuropsychologia 26, 387–400.

Kesner, R.P., 1992. Learning and memory in rats with an emphasis on the role of the amygdala. In: Aggleton, J.P. (Ed.), The Amygdala: Neurobiological Aspects of Emotion, Memory and Mental Dysfunction. Wiley-Liss, New York, pp. 379–399.

Lapierre, D., Braun, C.M.J., Hodgins, S., 1995. Ventral frontal deficits in psychopathy: neuropsychological test findings. Neuropsychologia 33, 139–151.

Levander, S.E., Lidberg, L., Schalling, D., Lidberg, Y., 1979. Skin conductance, finger pulse volume and heart rate measures as related to real-life stress and psychopathy. Reports from the Laboratory for Clinical Stress Research 100, 22.

Lykken, D.T., 1957. A study of anxiety in the sociopathic personality. Journal of Abnormal and Social Psychology 55, 6–10.

Lykken, D.T., 1995. The Antisocial Personalities. Lawrence Erlbaum, Hillsdale, NJ.

Malloy, P., Bihrle, A., Duffy, J., Cimino, C., 1993. The orbitomedial frontal syndrome. Archives of Clinical Neuropsychology. 8, 185–201.

Martzke, J.S., Swan, C.S., Varney, N.R., 1991. Post traumatic anosmia and orbital frontal damage: neuropsychological and neuropsychiatric correlates. Neuropsychology 5, 213–225.

Meunier, M., Bachevalier, J., Mishkin, M., 1997. Effects of orbital frontal and anterior cingulate lesions on object and spatial memory in rhesus monkeys. Neuropsychologia 35, 999–1015.

Meyers, C.A., Berman, S.A., Scheibel, R.S., Hayman, A., 1992. Case report: acquired antisocial personality disorder associated with unilateral left orbital frontal lobe damage. Journal of Psychiatry and Neuroscience 17, 121–125.

Mishkin, M., 1964. Perseveration of central sets after frontal lesions in monkeys. In: Warren, J.M., Akert, K. (Eds.), The Frontal Granular Cortex and Behavior. McGraw-Hill, New York, pp. 219–241.

Mishkin, M., Vest, B., Waxler, M., Rosvold, H.E., 1969. A re-examination of the effects of frontal lesions on object alternation. Neuropsychologia 7, 357–363.

Morgan, A.B., Lilienfeld, S.O., 2000. A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. Clinical Psychology Review 20, 113–156.

Patrick, C.J., Cuthbert, B.N., Lang, P.J., 1994. Emotion in the criminal psychopath: fear image processing. Journal of Abnormal Psychology 103, 523–534.

Pribram, K.H., Mishkin, M., 1956. Analysis of the effects of frontal lesions in monkeys. III. Object alternation. Journal of Comparative and Physiological Psychology 49, 41–45.

Raine, A., Lencz, T., Bihrle, S., LaCasse, L., Colletti, P., 2000. Reduced prefrontal gray matter volume and reduced autonomic activity in antisocial personality disorder. Archives of General Psychiatry 57, 119–127.

Raine, A., Stoddard, J., Bihrle, S., Buchsbaum, M., 1998. Prefrontal glucose deficits in murderers lacking psychosocial deprivation. Neuropsychiatry, Neuropsychology and Behavioral Neurology 11, 1–7.

Rolls, E.T., 1995. A theory of emotion and consciousness, and its application to understanding the neural basis of emotion. In: Gazzaniga, M.S. (Ed.), The Cognitive Neurosciences. MIT Press, Cambridge, MA, pp. 1091–1106.

Schalling, D., Lidberg, L., Levander, S.E., Dahlin, Y., 1973. Spontaneous autonomic activity as related to psychopathy. Biological Psychology 1, 83–97.

Schmauk, F.J., 1970. Punishment, arousal, and avoidance learning in sociopaths. Journal of Abnormal Psychology 76, 325–335.

Simon, H.A., 1990. A mechanism for social selection and successful altruism. Science 250, 1665–1668.

Smith, E.E., Jonides, J., 1999. Storage and executive processes in the frontal lobes. Science 283, 1657–1661.

Stone, V.E., Baron-Cohen, S., Knight, R.T., 1998. Frontal lobe contributions to theory of mind. Journal of Cognitive Neuroscience 10, 640–656.

Stuss, D.T., Gow, C.A., Hetherington, C.R., 1992. No longer Gage: frontal lobe dysfunction and emotional changes. Journal of Consulting and Clinical Psychology 60, 349–359.

Sutker, P.B., Allain, A.N., 1987. Cognitive abstraction, shifting, and control: clinical sample comparisons of psychopaths and non-psychopaths. Journal of Abnormal Psychology 96, 73–75.

Tharp, V.K., Maltzman, I., Syndulko, K., Ziskind, E., 1980. Autonomic activity during anticipation of an aversive tone in non-institutionalized sociopaths. Psychophysiology 17, 123–128.

Toglia, M.P., Battig, W.F., 1978. Handbook of Semantic Word Norms. Wiley, New York.

Tranel, D., Damasio, H., 1994. Neuroanatomical correlates of electrodermal skin conductance responses. Psychophysiology. 31, 427–438.

Tranel, D., Damasio, A.R., Damasio, H., 1988. Impaired autonomic reactions to emotional and social stimuli in patients

with bilateral orbital damage and acquired sociopathy. Society of Neuroscience 14, 1288.

Warkentin, S., Risberg, J., Nilsson, A., Karlson, S. et al., 1991. Cortical activity during speech production: a study of regional cerebral blood flow in normal subjects performing a word fluency task. Neuropsychiatry, Neuropsychology and Behavioral Neurology 4, 305−316.

Widom, C.S., 1977. A methodology for studying non-institutionalized psychopaths. Journal of Consulting and Clinical Psychology 45, 674−683.

Wolpe, J., Lang, P.J., 1964. A fear survey schedule for use in behavior therapy. Behaviour Research and Therapy 2, 27−30.