# NEUROPSYCHOLOGICAL DEFICITS IN THE ANTISOCIAL PERSONALITY AND THEIR RELATIONSHIP TO PROGRESS IN TREATMENT

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## ABSTRACT

**Background:** Antisocial personality is characterised by impulsive behaviour and a pervasive disregard for the rights of others. Its consequences are often debilitating and its presentation poses considerable treatment challenges. While it may be associated with a range of neuropsychological deficits, the literature is often contradictory and no research has examined their effect on treatment.

**Method:** A systematic review of the neuropsychological literature on male adults with antisocial personality was conducted to facilitate generation of hypotheses. Pooled evidence from 132 studies suggested robust cognitive deficits in motor regulation, affect recognition, and concept formation. Findings were less consistent for other functions and differences between operationalisations of the antisocial personality were present. To further investigate the neurocognitive deficits and examine their effect on treatment, the Cambridge Neuropsychological Assessment Battery (CANTAB) was administered on 102 adult male offenders (divided into those with antisocial vs. other personality disorders) and on 20 healthy controls in a between-subjects design. Two operationalisations were examined in parallel for the first time: Antisocial Personality Disorder (ASPD) and psychopathy. Progress in treatment was measured using a two-part, standardised instrument – the Progress Rating Schedule (PRS) – developed systematically via thematic analysis as part of the project.

**Results:** ASPD demonstrated impairments in executive, memory, attentional, and visual processing functions while psychopathy showed primarily executive but overall milder deficits. Impairments in motor regulation, set-shifting, working memory, and visual perception appeared present in the antisocial personality (ASPD and psychopathy) but not offenders with other personality disorders. Regarding progress in treatment, the PRS showed good reliability (intra-class correlations: 0.63-0.92; internal consistency: 0.77-0.87) and concurrent and predictive validity. However, cognitive difficulties predicted outcomes only to a limited extent. In ASPD, fronto-temporal deficits predicted poorer progress through the forensic pathway. However, higher risk-taking (Cambridge Gambling Task) predicted better outcomes while intellectual functioning and presence of psychopathy mediated some effects. In psychopathy, only visual short-term memory and planning predicted progress; impairments in the former predicted slower progress but there were inconsistencies for the latter.

**Conclusions:** A range of neuropsychological deficits appeared to characterise the antisocial personality and some may have adverse effects on progress in treatment. Further research is required in other, larger samples and cognitive functions not included in the CANTAB to confirm and extend these findings.

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## LIST OF ACRONYMS

| | |
|---|---|
| AGN | Affective Go/NoGo task |
| APA | American Psychiatric Association |
| ASPD | Antisocial Personality Disorder |
| BAS | Behavioural Activation System |
| BIS | Behavioural Inhibition System |
| CANTAB | Cambridge Neuropsychological Test Assessment Battery |
| CGT | Cambridge Gambling Task |
| COWAT | Controlled Oral Word Association Test |
| CPA | Care Programme Approach |
| CPT | Continuous Performance Task |
| DMS | Delayed Matching to Sample task |
| DPD | Dissocial Personality Disorder |
| DSM | Diagnostic and Statistical Manual of Mental Disorders |
| DSQ | Defence Style Questionnaire |
| DTT | Divergent Thinking Task |
| EA | Equal activation (hemispheric) |
| EDS | Extra-dimensional set shifting |
| fMRI | Functional magnetic resonance imaging |
| FSIQ | Full-scale IQ |
| HA | High-anxious |
| HCR-20 | Historical Clinical Risk – 20 |
| HoNOS | Health of the Nation Outcome Scales |
| ICC | Intraclass correlation |
| ICD | International Classification of Diseases and Related Health Problems |
| IED | Intra/Extra-Dimensional Set Shifting task |
| IES | Integrated Emotion Systems |
| IHA | Interhemispheric advantage |
| IGT | Iowa Gambling task |
| IPDE | International Personality Disorder Examination |
| K-S (test) | Kolmogorov-Smirnov (normality test) |
| LA | Low-anxious |

| | |
|---|---|
| LHA | Left Hemisphere Activation |
| LTM | Long-term memory |
| LVF | Left visual field |
| MAR | Missing at random |
| MCAR | Missing completely at random |
| MINI | Mini International Neuropsychiatric Interview |
| MLM | Multilevel Modelling |
| MTS | Matching to Sample Visual Search task |
| NART | National Adult Reading Test |
| NICE | National Institute for Health and Clinical Excellence |
| NSS | Neurological soft signs |
| OFC | Orbito-frontal cortex |
| PAL | Paired Associates Learning task |
| PCL/-R/:SV | Psychopathy Checklist/-Revised/:Screening version |
| PDS | Personality Disorder Service |
| PIQ | Performance IQ |
| PRM | Pattern Recognition Memory task |
| PRS | Progress Rating Schedule |
| RVF | Right visual field |
| RVP | Rapid Visual Processing task |
| SADS-L | Schedule for Affective Disorders and Schizophrenia-Lifetime |
| SCID-I/: CV | Structured Clinical Interview for DMS Disorders-Axis I/: Clinical version |
| SILS/-R | Shipley Institute of Living Scale/-Revised |
| SOA | Stimulus onset asynchrony |
| SOC | Stockings of Cambridge |
| SPSI-R | Social Problem Solving Inventory-Revised |
| SRD | Substance-related disorder |
| SRM | Spatial Recognition Memory task |
| SSP | Spatial Span task |
| SST | Stop-Signal task |
| STM | Short-term memory |
| SWM | Spatial Working Memory task |
| TMT | Trail-Making Test |

| | |
|---|---|
| ToL | Tower of London |
| VIM | Violence inhibition mechanism |
| VIQ | Verbal IQ |
| VRM | Verbal Recognition Memory task |
| WAIS | Wechsler Adult Intelligence Scale |
| WCST | Wisconsin Card Sorting Test |
| WM | Working memory |
| WRAT | Wide Range Achievement Test |

# 1 INTRODUCTION

Crime poses a significant problem in the society and is associated with considerable costs (Brand & Price, 2000). Among offenders, there is a group who appear to begin their criminal activities from an earlier age than others, offend more extensively and are more likely to engage in violence and aggression. These individuals may present with an antisocial personality (Hare, 2003; Hart & Hare, 1996).

The term antisocial personality in mental health describes individuals who exhibit socially deviant behaviour, disregard for the rights of others, lack empathy and remorse and who pursue personal gain over considerations for others. Its notion as a personality disturbance in psychiatry can be traced as early as the 19th century (Millon, Simonsen, & Birket-Smith, 1998) and nowadays is part of the diagnostic nomenclature (American Psychiatric Association [APA], 2004; World Health Organisation, 1990).

Although the antisocial personality is very common in correctional settings (Alwin et al., 2006), it is as prevalent as schizophrenia in the general population and the impact it has on society is extensive and much beyond crime (Duggan, 2009). Antisocial personality is associated with a poor prognosis and high mortality rates (APA, 2000; Duggan, 2009; Hare, Clark, Grann, & Thornton, 2000). Treatments for this type of personality exist and can be pharmacological, psychological and psychosocial (National Institute for Health and Clinical Excellence [NICE], 2010). Reviews of the evidence, however, reveal that they lack a credible evidence base, are associated with high drop-out rates and re-offending remains a cause for concern (Coid, Hickey, Kahtan, Zhang, & Yang, 2007; McCarthy & Duggan, 2010; McMurran, Huband, & Overton, 2010; NICE, 2009). Even though some psychosocial benefit has been recorded following admission to psychiatric services (McCarthy, Huband, Patel, & Banerjee, 2012), it is clear that further improvements would be desirable.

In recent years, research on the antisocial personality has increasingly drawn on neuroscience with a breadth of findings (e.g. R. Blair, Mitchell, & Blair, 2005; Hare, 2003). However, this line of study has not yet been found to influence treatment or improve outcomes. Considering that the neuropsychological makeup of an

individual can play a major role in affecting his or her personality and behaviour (D. L. Clark, Boutros, & Mendez, 2010; Kolb & Whishaw, 2009; Martin, 2006), investigating how the neurocognitive deficits associated with antisocial personalities may impact on treatment could provide new insights into treatment development. This project aims to delineate these neuropsychological deficits using the Cambridge Neuropsychological Assessment Battery (CANTAB) and to investigate their impact on progress in treatment within a medium-secure hospital.

## 1.1 The Antisocial Personality and Contemporary Operationalisations

Personality is 'the sum total of the behavioural and mental characteristics that are distinctive of an individual' (Colman, 2003, pp.547). When one's personality deviates markedly and persistently from patterns expected within a specific culture, leading to significant distress and impaired functioning, then one is considered to suffer from a personality disorder (Alwin et al., 2006; APA, 2000). There are several personality disorders in contemporary diagnostic nomenclature, one of which is the antisocial type.

The conceptualisation of this personality type has evolved greatly over the years from the earlier notions of 'Manie sans délire', 'Moral insanity' and 'Semantic dementia' to today's understanding which crystallised largely due to the work of Hervey Cleckley who employed discreet diagnostic criteria (Cleckley, 1941, 1976; Millon et al., 1998). Since then, increasingly rigorous research of the antisocial personality has gradually developed three mainstream operationalisations: the Antisocial Personality Disorder (ASPD) of the Diagnostic and Statistical Manual of Mental Disorders, or DSM (DSM-III, DSM-III-R, DSM-IV, and DSM-IV-TR; APA, 1980, 1987, 1994, 2000), the Dissocial Personality Disorder (DPD) of the tenth version of the International Classification of Diseases and Related Health Problems, or ICD-10 (World Health Organisation, 1990), and psychopathy according to Hare's Psychopathy Checklist and its revision, or PCL and PCL-R (Hare, 1980, 1991, 2003).

### 1.1.1 ASPD

Recent versions of the DSM place emphasis on antisocial and deviant conduct while elements such as lack of empathy and grandiosity are considered associated features of the disorder. Diagnosis requires presence of conduct disorder prior to the age of 15. This requirement was influenced by research showing continuity from conduct disorder to ASPD (De Brito & Hodgins, 2009; R. B. Goldstein, Grant, Ruan, Smith, & Saha, 2006; Lahey, Loeber, Burke, & Applegate, 2005; Loeber, Burke, & Lahey, 2002; Moffitt, Caspi, Harrington, & Milne, 2002; Robins, 1966; Robins, Tipp, & Przybeck, 1991; Simonoff et al., 2004; Washburn et al., 2007). Prognosis is poor with even up to 72% of individuals still meeting the criteria 9 years after diagnosis while experiencing high rates of morbidity and mortality (Coid, Yang, Roberts, & Ullrich, 2006; Guze, 1976; NICE, 2009; Robins et al., 1991; Swanson, Bland, &

Newman, 1994; Torgensen, Kringlen, & Cramer, 2001). A summary of the DSM-IV-TR criteria for ASPD can be found in Table 1.1.

The focus on behavioural manifestation has been one of the major criticisms of ASPD as it appears to neglect core underlying personality features (Widiger & Corbitt, 1993). In addition, although ASPD is sometimes considered as one of the most reliable diagnostic categories (Coid, 2003) it defines a very heterogeneous population, often with features overlapping with other personality disorders (L. A. Clark, 2007; L. A. Clark, Livesley, & Money, 1997; Lykken, 1995; R. Rogers, 2000; Tyrer et al., 2007; Westen & Arkowitz-Westen, 1998; Widiger et al., 1996).

Table 1.1. *Summary of DSM-IV-TR criteria for ASPD*

| |
|---|
| A. A pervasive pattern of disregard for and violation of the rights of others occurring since age of 15, indicated by three of the following: |
| 1. Failure to conform to social norms |
| 2. Deceitfulness |
| 3. Impulsivity or failure to plan ahead |
| 4. Irritability or aggressiveness |
| 5. Reckless disregard for safety of self or others |
| 6. Consistent irresponsibility |
| 7. Lack of remorse |
| B. Current age at least 18 |
| C. Evidence of Conduct disorder before the age of 15 |
| D. The occurrence of antisocial behaviour is not exclusively during the course Schizophrenia or a manic episode |

*Note.* DSM-IV-TR=Diagnostic and Statistical Manual of Mental Disorders, 4th edition, text revision; ASPD=Antisocial Personality Disorder.

### 1.1.2 DPD

The World Health Organisation's (1990) ICD- 10 operationalisation places more emphasis on features of the antisocial personality and interpersonal impairments alongside the behavioural manifestations, thus being closer to Cleckley's (1941, 1976) criteria (

Table **1**.**2**). Furthermore, it does not require presence of conduct disorder in childhood.

Table 1.2. *Summary of ICD-10 criteria for DPD*

| |
|---|
| 1. Callous unconcern for the feelings of others and lack of empathy |
| 2. Gross and persistent irresponsibility and disregard for social norms, rules and obligations |
| 3. Incapacity to maintain enduring relationships |
| 4. Very low tolerance to frustration and low threshold for discharge of aggression, including violence |
| 5. Inability to experience guilt and to profit from experience |
| 6. Marked proneness to attribute blame on others or to rationalise behaviour bringing the subject into conflict with society |
| 7. Persistent irritability |

*Note.* ICD-10=International Classification of Diseases, 10th edition; DPD=Dissocial Personality Disorder.

### 1.1.3 Psychopathy

Unlike ASPD and DPD, psychopathy is not a clinical diagnosis and the term is most often associated with Hare's (1980, 2003) PCL and PCL-R. It was initially developed for research purposes on the basis of Cleckley's (1941, 1976) criteria and contains both behavioural and personality criteria in a two/four-factor structure (Table 1.3). The first factor represents interpersonal (1a) and affective deficits (1b) while the second factor includes antisocial (2a) and impulsive lifestyle (2b) characteristics of psychopathy (Hare, 2003; Harpur, Hare, & Hakstian, 1989; Neumann, Hare, & Newman, 2007). An alternative model has also been suggested, incorporating three factors: arrogance & deceitfulness, affective deficits and impulsivity/irresponsibility (Cooke & Michie, 2001). Scores on the PCL-R range from 0-40 and scores of 30 or above indicate psychopathy in Northern-American populations while the cut-off is 25 for European populations (Hare, 2003). An abbreviated version of the PCL-R containing 12 items is also available and is known as the screening version ([PCL:SV], Hare, 2003; Hart, Cox, & Hare, 1995).

Table 1.3. *Summary of PCL-R criteria for psychopathy*

I. Factor 1 – Interpersonal and affective features:
- a. Interpersonal features
  - 1. Glibness/superficial charm
  - 2. Grandiose sense of self-worth
  - 4. Pathological lying
  - 5. Conning/manipulative
- b. Affective features
  - 6. Lack or remorse or guilt
  - 7. Shallow affect
  - 8. Callous/lack of empathy
  - 16. Failure to accept responsibility for own actions

B. Factor 2 – Antisocial and impulsive lifestyle features:
- a. Antisocial features
  - 10. Poor behavioural controls
  - 12. Early behavioural problems
  - 18. Juvenile delinquency
  - 19. Revocation of conditional release
- b. Impulsive lifestyle features
  - 3. Need for stimulation/proneness to boredom
  - 9. Parasitic lifestyle
  - 13. Lack of realistic, long-term goals
  - 14. Impulsivity
  - 15. Irresponsibility

C. Independent items
- 11. Promiscuous sexual behaviour
- 17. Many short-term marital relationships
- 20. Criminal versatility[a]

*Note.* PCL-R=Psychopathy Checklist-Revised.

[a] Athough not associated with Factor 2 overall, this item forms part of the "antisocial features" cluster (Hare, 2003).

The three contemporary operationalisations of the antisocial personality are not equivalent, with discrepancies being largest between ASPD and psychopathy (Hare, 1991, 2003; Hare, Hart, & Harpur, 1991; Hodgins, 2007). The discrepancy between ASPD and psychopathy is further reflected on asymmetrical presence in correctional settings. Although both operationalisations are more prevalent in correctional settings than in the general population, ASPD is more prevalent than psychopathy in the latter (Table 1.4). Furthermore, while most offenders with psychopathy are also likely to receive a diagnosis of ASPD, only a small proportion of offenders with ASPD may

meet criteria for Hare's psychopathy (Hare, 2003; Hart & Hare, 1989). Table 1.4 shows prevalence rates for ASPD and psychopathy in Europe (Coid, Yang, Ullrich, Roberts, & Hare, 2009; Fazel & Danesh, 2002; Hare et al., 2000).

Table 1.4. *Prevalence rates in Europe for ASPD and psychopathy*

| Prevalence (Europe) | ASPD | Psychopathy |
|---|---|---|
| General population | | |
| Males | 1-1.3% | <<1% (PCL:SV > 18) |
| Females | 0-0.2% | |
| Correctional institutions (males) | 47% | 4.5% |

*Note.* ASPD=Antisocial Personality Disorder; PCL:SV=Psychopathy Checklist: Screening version.

Though the three operationalisations show some agreement in terms of the behaviours associated with the antisocial personality, overall discrepancies suggest that the range of features attributed to this concept may not be very specific. They are also rather different to Cleckley's (1941, 1976) original conceptualisation. Even though PCL-R psychopathy represents a more homogeneous group than ASPD, it too has sometimes been criticised for containing many subtypes (Blackburn, 2009). In an attempt to distinguish a group more in line with Cleckley's definition and to provide a better account for the affective deficit that is often attributed to individuals with psychopathy, considerable amount of research has incorporated a self-report negative affect scale in conjunction with the PCL-R (Hare, 2003; Newman & Lorenz, 2002) – most often the Welsh Anxiety Scale (Welsh, 1956). Thus, this research claims that it is possible to focus only on those groups which experience low negative affect/anxiety as closer to Cleckley's psychopathy. However, such a method implies motivational and self-report biases (Hare, 2003).

## 1.2 Neuropsychological theories of antisocial personality

There are several theories that attempt to provide an account of antisocial personality though they are primarily concerned with psychopathy. Most of the theories also focus on a limited subset of neuropsychological functions but there are often overlaps between them.

### 1.2.1 The Behavioural Inhibition/Activation System (BIS/BAS) and Fear dysfunction

This theory on psychopathy utilises J. A. Gray's (1987) BIS/BAS model. It suggests that, via the mechanisms of conditioning, punishment results in behavioural inhibition while reward results in behavioural activation. Some researchers have placed greater emphasis on the BIS deficit in psychopathy (Fowles, 1980; Lykken, 1995) and others on a BAS overactivation (Scerbo et al., 1990) while more recently these systems have been associated with different aspects of psychopathy (Wallace, Malterer, & Newman, 2009). The theory is relatively old and has generated considerable amount of research. Experimental evidence has shown that individuals with psychopathy appear unable to avoid punished responses, especially when these were previously rewarded and they demonstrate a hypersensitivity to reward (Arnett, 1997).

The mechanism of the BIS is further elaborated on by some researchers (Lykken, 1995; Patrick, 1994) as a fear mechanism. The theory assumes that punishment results in fear, reflected in autonomic responses during aversive conditioning, thus inhibiting future conditioned behaviours. If this BIS is deficient in individuals with psychopathy, they may not learn to avoid punished behaviours. Empirical evidence for this theory overlaps with the BIS/BAS account. Additional evidence has demonstrated impaired aversive conditioning and abnormalities in autonomic arousal and startle reflex following threat in individuals with psychopathy (Levenston, Patrick, Bradley, & Lang, 2000; Lykken, 1995; Ogloff & Wong, 1990). Although there is some evidence for the fear hypothesis, R. Blair et al. (2005) have pointed out that the theory assumes a unitary fear system, contrary to empirical evidence from neuroscience, which demonstrates distinct processes (primarily aversive conditioning and instrumental learning). Thus, both fear dysfunction and

BAS/BIS hypotheses do not explain how these specific operations might operate in psychopathy.

In sum, the BIS/BAS and fear hypotheses explains psychopathy in terms of sensitivity to reward and punishment and an inability to learn from the latter. Some weaknesses exist in the neurological underpinning of these theories. Ultimately, however, they remain limited to behavioural aspects of the antisocial personality and thus fail to explain core interpersonal and affective/empathic deficits.

### 1.2.2 Response modulation

The response modulation hypothesis is an attention-based theory of psychopathy. It describes an inherent inability to shift the focus of attention to peripheral information, thus failing to adjust ongoing behaviour appropriately (Newman, 1998; Patterson & Newman, 1993). The theory draws on both the executive functions of self-regulation and selective attention resources (cf. Lezak et al, 2004) and has received considerable experimental support. Supportive evidence includes impaired passive avoidance learning in psychopathy and an inability to adjust responding according to shifting balance of reward and punishment.

Although the response modulation hypothesis is another well-established theory of psychopathy, the evidence it has drawn upon is often what also supports the BIS/BAS hypothesis, for instance passive avoidance and reward/punishment contingencies. As a result, the response modulation hypothesis appears limited in its ability to provide a unique account of these phenomena. Furthermore, it attempts to explain maladaptive responses to a stimulus when learning ought to occur due to shifting reinforcement and not in relation to contextual information (Newman, 1998; Patterson & Newman, 1993). R. Blair et al. (2005) argue that an attention-based theory may not explain such a phenomenon adequately. Finally, the theory also attempts to explain the evidence that individuals with psychopathy do not appear to benefit from emotional content during lexical decision tasks in attentional terms. However, this explanation adds little in light of the much more extensive affective interpersonal/affective impairments observed in psychopathy.

### 1.2.3 Frontal lobe dysfunction and the Somatic Marker Hypothesis

This hypothesis suggests that the antisocial personality may result from a dysfunction of the frontal lobe (Gorenstein, 1982; Moffitt, 1993; Raine, 2002). It relies on evidence from frontal lesion studies evidencing acquired sociopathy and antisocial personality traits (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; R. Blair & Cipolotti, 2000; Burgess & Wood, 1990; Grafman, Schwab, Warden, Pridgen, & Brown, 1996; Pennington & Bennetto, 1993), neuropsychological findings showing executive impairments in individuals exhibiting antisocial behaviour (Dawel, O'Kearney, McKone, & Palermo, 2012; Kandel & Freed, 1989; Morgan & Lilienfeld, 2000; Ogilvie, Stewart, Chan, & Shum, 2011; Wilson, Juodis, & Porter, 2011) and imaging studies highlighting structural and functional abnormalities in the frontal lobe of antisocial populations (Koenigs, Baskin-Sommers, Zeier, & Newman, 2011; Plodowski, Gregory, & Blackwood, 2009). The breadth of the evidence this theory is based on provides a high level of credibility. However, although the theory identifies a neurological substrate of antisocial behaviour, it does not attempt to explain the mechanism by which frontal lobe dysfunction translates into the observed behaviours (R. Blair et al., 2005). Furthermore, frontal lesions appear to be more closely associated with reactive than instrumental aggression (R. Blair et al., 2005). In addition to this, the evidence for the theory emerges from a variety of populations thus making it unable to capture the more complex interpersonal and criminal pathology of antisocial personalities.

Research focusing on frontal lesion studies has revealed that the orbital and ventromedial parts are more closely associated with an increased risk of antisocial behaviour (Bechara, Tranel, & Damasio, 2000; L. Clark et al., 2008; Damasio, 2000; Damasio, Tranel, & Damasio, 1990; Saver & Damasio, 1991). This is captured by the Somatic Marker Hypothesis which postulates that antisocial behaviour may result from an impaired decision-making process arising from lack of conditioned physiological responses, a function linked to orbitofrontal/ventromedial prefrontal areas (e.g. Damasio, 1994; Damasio et al., 1990). The theory is based on evidence showing that ventromedial lesions result in loss of autonomic reactivity during passive viewing of affectively-laden stimuli and during reduction of implicit learning of reward and punishment associations (e.g. Iowa Gambling Task [IGT]). The Somatic Marker Hypothesis may be able to explain some features of the antisocial personality

but it does not attempt to offer a specific account. There is also conflicting evidence. For instance, some research has revealed impairment in decision-making without loss of conditioned autonomic function in individuals with psychopathy (R. Blair et al., 2005). In addition, Heims et al. (Heims, Critchley, Dolan, Mathias, & Cipolotti, 2004) did not find decision-making impairments on the IGT in patients with pure autonomic failure. Such findings indicate that the Somatic Marker Hypothesis is limited in its account.

In conclusion, both the frontal dysfunction theory and the Somatic Marker Hypothesis place the locus of antisocial behaviour in the frontal lobe, the latter proposing a mechanism of how ventromedial prefrontal function may result in the observed behaviours. However, both theories have drawn on a heterogeneous pool of evidence and seem to present more general accounts of antisocial behaviour than specific theories of antisocial personality. Conflicting evidence also limits the applicability of the Somatic Marker Hypothesis.

### 1.2.4 Left Hemisphere Activation (LHA)

Moving away from traditional frontal or affective models of antisocial behaviour, the LHA theory is another hypothesis focusing on psychopathy. It postulates that there is an unusual lateralisation in the brain of individuals with psychopathy so that left hemisphere processing is impaired, especially for the processing of language (R. Blair et al., 2005; Hare, 2003; Kosson, 1998). This view is based on findings from language studies and paradigms which draw on left hemisphere resources, often using lateralised presentation of stimuli. For instance, evidence has shown that the performance of individuals with psychopathy was worse during abstract semantic processing for right visual field (RVF) stimuli (Hare & Jutai, 1988), right-ear targets during dichotic listening (Hare & McPherson, 1984) or during conditions which activated the left hemisphere (Kosson, 1998). Although the model highlights involvement of the left hemisphere in psychopathy, it does not explain how this results in the aggressive, interpersonal, impulsive or affective features of antisocial personalities.

### 1.2.5 The Violence Inhibition Mechanism (VIM) and the Integrated Emotion Systems (IES) Model

The VIM theory hypothesises an operant conditioning process in which distress in ones conspecifics act as aversive stimuli thereby shaping to moral socialisation. The model is based on the observation that distress cues in conspecifics inhibit one's own aggressive responses. According to the VIM, this process is impaired in psychopathy (R. Blair et al., 2005). Evidence for this view suggests that individuals with psychopathy may show impaired autonomic arousal and startle reflex reaction to distressing stimuli (R. Blair, Jones, Clark, & Smith, 1997; Levenston et al., 2000). R. Blair et al. (2005) indicate that reduced sadness and fear recognition in individuals with psychopathy also supports the theory. Once again, this is a focused theory of psychopathy and therefore attempts to explain only a small subset of features of the antisocial personality whereas it does not account for its more complex antisocial and impulsive behaviours and diverse interpersonal/affective features. As with the BIS/BAS and fear dysfunction models, its neurological basis also lacks clarity.

A later theory developed by R. Blair et al. (2005) is the IES model. This integrative theory attempts to explain the complexities of the antisocial personality and in particular psychopathy. Its focus is on the amygdala and the orbito-frontal cortex (OFC) suggesting that functions primarily associated with these areas are impaired resulting in the affective and behavioural deficits of antisocial personality (R. Blair et al., 2005). The amygdala plays a major role in conditioning basic emotional reactions (e.g. startle reflex) and affective processes (e.g. emotion recognition) and have many connections to several other brain areas, primarily including the prefrontal cortex and in particular the orbitofrontal part, cingulate gyrus, ventral striatum, brainstem, hypothalamus, hippocampus, superior temporal sulcus and fusiform cortex (D. L. Clark et al., 2010). The OFC, on the other hand, is involved in self-regulation with some input from the amygdala, especially in connection with prior conditioning (D. L. Clark et al., 2010). As we have seen, these operations appear impaired in psychopathy. Imaging studies on psychopathy have also provided structural and functional evidence supporting the IES model; yet the neurological abnormalities observed in antisocial populations seem far from being confined to the amygdala and involve a wider fronto-temporal network (Koenigs et al., 2011; Plodowski et al.,

2009). Although the IES suggests that many abnormalities may result from amygdala input, additional research is required to further qualify this proposal. For instance, it is yet uncertain to what extent the dysfunction may be due to genetic/biochemical or lifestyle factors such as alcohol abuse (which has also been associated with OFC dysfunction, e.g. Bechara et al., 2001) and how these might interact.

In sum, the IES theory of psychopathy is a development from the VIM model. The VIM was an account limited to violence inhibition in response to others' distress cues with a lack of neurological underpinning. On the other hand, the IES is an integrative theory suggesting that dysfunction of the amygdala and the OFC results in an impaired neuropsychological mechanism incorporating both behavioural and affective processes. Although the IES is a comprehensive account of psychopathy, further research is required to further its ability to explain many observations on the antisocial personality.

In conclusion, there are several neuropsychological theories concerned with the antisocial personality. The theories of BIS/BAS, fear activation, response modulation, LHA, VIM and IES focus on psychopathy whereas the frontal dysfunction model and the Somatic Marker Hypothesis seem more suitable to address antisocial behaviour in more general terms. No models have been developed based on ASPD or DPD and the extent to which current theories would generalise to these diagnoses remains uncertain.

An additional observation is that the majority of the neuropsychological theories appear to focus on a subset of antisocial personality features with the interpersonal and affective aspects being often neglected. The IES appears to be the only integrative theory and is currently the most comprehensive account of psychopathy. It highlights key neural substrates and at the same time provides a psychological mechanism in which their (dys)function results in observed behaviours. Yet, many questions in terms of its ability to explain wider neurological findings remain unanswered.

It appears that individuals with an antisocial personality may experience a breadth of neuropsychological difficulties with effects likely manifesting in almost every aspect of their lives for instance in poor occupational and social functioning, apart from crime (Duggan, 2009). It would not be unreasonable, therefore, to suspect

that they may also affect the way in which these individuals respond to psychological treatment.

### 1.3 Treatment for ASPD

The present research is concerned with the manner in which treatment progress may be affected by the neuropsychological deficits of antisocial personality within a medium-security setting. Antisocial personality, however, is a complex condition to treat as it encompasses an array of behavioural and interpersonal difficulties and co-morbid conditions (NICE, 2009). There is considerable variation in the interventions available which can be psychological, pharmacological, and psychosocial but many have been developed in other areas of mental health and several have not yet been tailored to the antisocial personality (Howard & Howells, 2010). Some examples include treatments specific to ASPD (e.g. Davidson et al., 2008), therapies focused on particular features of the antisocial personality (e.g. Vannoy & Hoyt, 2004), interventions seeking to address co-morbid conditions such as substance abuse (e.g. Austin, Robinson, Elms, & Chan, 1997; Dugan & Everett, 1998; G. Johnson & Hunter, 1995; Kinlock, O'Grady, & Hanlon, 2003) and treatments targeting offending behaviour (e.g. Armstrong, 2003; Liau et al., 2004; Porporino & Robinson, 1995; Ross, Fabiano, & Ewles, 1988).

Although there is a range of potentially suitable interventions for the antisocial personality, the evidence from randomised clinical trials on the effectiveness of pharmacological methods has not permitted firm conclusions (Khalifa et al., 2010). On the other hand, psychological interventions based on cognitive-behavioural models have shown some positive results but randomised clinical trial research remains limited (Duggan, Huband, Smailagic, Ferriter, & Adams, 2007; Gibbon et al., 2010). Following a wider examination of the existing evidence, the national guidelines for the treatment of the antisocial personality in the UK (NICE, 2009), where the present research project took place, concluded that treatments within a multi-agency framework (e.g. medicine, psychology, nursing, social work, occupational therapy, etc.) using a group-based cognitive behavioural format and additional one-to-one support may be recommended.

The planning of such treatment programmes in the UK follows the general Care Programme Approach (CPA) to cater for a patient's mental health, social, and risk management needs (McMurran, Khalifa, & Gibbon, 2009). The CPA involves comprehensive assessments of the patient's needs and an agreed care plan to address them. There are regular reviews and delivery is monitored by the care coordinator.

Although steps have been taken to develop a framework of intervention in antisocial personality, much remains to be investigated. At present, treatment programmes face various barriers including premature disengagement and limited effectiveness (Coid et al., 2007; Davies, Clarke, Hollin, & Duggan, 2007; McCarthy & Duggan, 2010; McMurran et al., 2010; O'Brien, Fahmy, & Singh, 2009). Costs associated with the treatments are also high (Duggan, 2009; NICE, 2009) thus placing additional emphasis on the need for more efficient interventions.

In conclusion, current treatments for ASPD are primarily psychological comprising group-based cognitive behavioural interventions with supportive individual sessions and medication if necessary. The evidence-base is still limited and more research is needed in several domains in this area, from treatment retention strategies to improving various outcomes. Since neuropsychological research in the antisocial personality has increasingly facilitated understanding of the condition, relating its findings to treatment progress may generate relevant new insights. In order to facilitate this, it will be important to provide an accurate and comprehensive summary of the findings to date.

## 1.4 Aims

The present research endeavoured to investigate the neurocognitive deficits associated with the antisocial personality (ASPD & psychopathy) and evaluate their impact on treatment. Since neuropsychological research in the antisocial personality has been extensive over recent years, a systematic review appeared necessary in order to gain a good understanding of the field and thus generate specific, testable hypotheses. Having examined and summarised the relevant literature in this manner, the project then sought to investigate the neuropsychological function in ASPD and psychopathy empirically using the CANTAB to assess a range of neuropsychological functions for the hypothesised deficits. Finally, the project sought to explore the effect of the anticipated neuropsychological impairments on treatment using a structured instrument to measure progress.

The project is presented in seven chapters. Following the introduction to the general field in Chapter 1, Chapter 2 is focused on the systematic review with meta-analyses. Chapter 3 outlines the aims and specific hypotheses regarding the neurocognitive deficits in the antisocial personality and their relationship to progress in treatment. Chapter 4 describes the development and validation of an instrument to measure progress in treatment in personality disorders. This is followed by Chapter 5 which focuses on the cognitie abilities in the antisocial personality. Chapter 6 details the investigation into the relationship between neuropsychological deficits and progress in treatment. Finally, Chapter 7 presents the discussion of the findings and relevant conclusions.

# 2 SYSTEMATIC LITERATURE REVIEW AND META-ANALYSES OF STUDIES EXAMINING COGNITIVE FUNCTIONS IN THE ANTISOCIAL PERSONALITY

## 2.1 Brief introduction

A systematic review and meta-analyses of the extensive neuropsychological research in the field of antisocial personality was necessary in order to summarise the evidence and then generate testable hypotheses with reference to specific deficits and the relation of cognitive functions to treatment outcome. Existing reviews in the area tend to include very heterogeneous antisocial samples and focus on specialised areas of neurocognition such as executive functions or affective processing (Dawel et al., 2012; Kandel & Freed, 1989; Marsh & Blair, 2008; Morgan & Lilienfeld, 2000; Ogilvie et al., 2011; Wilson et al., 2011). As a result, there is little clarity as to what neurocognitive deficits are associated specifically with the antisocial personality usually encountered in clinical settings. In order to gain an accurate picture of this vast field, a systematic review examined the full spectrum of neurocognitive functions (organised hierarchically) using strict criteria to define the antisocial personality. It also sought to delineate differences between the various definitions of this type of personality.

## 2.2 Method

### 2.2.1 Literature search

#### 2.2.1.1 Search strategy

Major medical and psychological literatures databases were searched. MEDLINE and EMBASE are considered as two representative medical databases for this purpose (Khan, Kunz, Kleijnen, & Antes, 2003) with moderate overlap (Egger & Smith, 2001). PsycINFO was the third database utilised here, as a major resource on psychological publications (Khan et al., 2003; McBurney & White, 2007). Reference lists of all included publications were hand-searched for additional relevant literature.

The year 1987 was selected as a starting date for the literature search being the point when DSM-III-R ASPD diagnosis was introduced (APA, 1987). Research using the PCL will have gained considerable assessment validity and reliability by that year also, following the circulation of its essential scoring criteria in a mimeograph form in 1985 (Hare, 1985, 2003). MEDLINE, EMBASE, and PsycINFO were searched separately using the OvidSP interface from 1987 until March 2010. Electronic searches utilised both medical subject heading terms and other terms of interest. Although the key words were the same across all databases, the medical subject heading term or equivalent indexing terms were only applicable to their specific database. For each database, indexing terms relevant to the key words were identified through OvidSP.

The search script consisted of two parts (Appendix A). The first part used search terms relevant to antisocial personality. The script had been used previously in the development of the national guidance for the treatment of ASPD (NICE, 2009). Only steps 1-4 were employed, containing variant terms for ASPD, DPD and psychopathy. Steps 5-7 described personality disorder or dysfunction more generally and also included DSM-II and were therefore not of interest. The NICE script contained specific field identifiers (e.g. ‘sh’ for Subject Heading). However, where a field identifier was not supported by the database, it was replaced by an equivalent or else broader term.

The second part of the script consisted of neuropsychological terms. Textbooks of neuropsychology (Kolb & Whishaw, 2009; Martin, 2006), neuropsychological assessment (Lezak, Howieson, & Loring, 2004; Strauss, Sherman,

& Spreen, 2006), and neuroscience (Gazzaniga, Ivry, & Mangun, 2009; Rosenzweig, Breedlove, & Watson, 2007) were utilised in order to identify neuropsychological operations, their divisions and tests for assessing them. The search followed a hierarchical structure and included term variants. Because neuropsychological tests are many but not of equal standard and validity, only extensively substantiated and researched tests or batteries were included. At each stage of the script, the added terms were evaluated for usefulness on the basis of the additional studies they contributed to the list to result in a comprehensive yet economical script. More general terms added towards the end for completion (e.g. cognition) made unique contributions.

#### 2.2.1.2 Inclusion criteria

A study was included in the review if:

i. ***It was empirical and published in a peer-reviewed journal or they were doctoral dissertations with a published abstract.*** This was to ensure a high quality of included research and selection of studies in as systematic a manner as possible. However, studies without statistically significant results tend to be underrepresented in journals (Egger, Dickersin, & Smith, 2001; Khan et al., 2003), a form of publication bias evaluated using appropriate methods (described below).

ii. ***It utilised clinical/diagnostic assessments during sample selection.*** Because the review is concerned with clinical forms of antisocial personality, focus was on the contemporary operationalisations of the DSM, ICD-10 and PCL/-R identified by standardised, clinician-administered instruments (rather than self-report) in the interests of validity and reliability (Spitzer, Endicott, & Robins, 1975).

iii. ***Samples consisted of male adults.*** The DSM cautions against diagnosing personality disorders prior to adulthood (APA, 1987, 2000) and Hare (2003) suggests that psychopathy in adolescence and in adulthood may not be equivalent. Thus, only adult samples were included. Three studies on

psychopathy (Kosson et al., 2007; Llanes & Kosson, 2006; Mayer et al., 2006) invited participants of 17-39 years of age. The younger participants, on the one hand, are unlikely to be of substantial numbers and, on the other hand, will be close to 18 years of age. Therefore, it was decided to include these studies. Male-only samples were included due to the relative lack of research on the female antisocial personality (Hare, 2003) and because evidence suggests there may be neuropsychological gender differences, thus not allowing integration (Kolb & Whishaw, 2009).

iv. ***Participants were not selected from populations systematically diagnosed with mental illness (psychosis and depression) or substance related disorders.*** These, can be serious confounders when assessing neuropsychological function (Lezak et al., 2004). However, samples with unsystematic diagnoses of substance-related disorders (SRDs) were included, as these are often comorbid with the antisocial personality (De Brito & Hodgins, 2009; Hare, 2003).

v. ***They utilised cognitive-behavioural tasks for evaluating neuropsychological function (Gazzaniga et al., 2009; Kolb & Whishaw, 2009; Lezak et al., 2004; Martin, 2006; Rosenzweig et al., 2007; Strauss et al., 2006).*** This also included studies with a primary neuroimaging or electrophysiological focus but which nevertheless provided neuropsychological data. Assessment methods not using cognitive-behavioural tasks, such self-report measures (e.g. emotional intelligence, emotional valence, alexithymia scales, etc.), were not included.

#### 2.2.1.3 A special case for intelligence

The present review has included intelligence data from all the neuropsychologically relevant studies identified via the literature search strategy described above. However, it is likely that other, non-neuropsychologically relevant studies may have measured intelligence but were not identified.

### 2.2.2 Evaluation of study quality

Not all studies identified during a literature search could be equivalent in terms of quality and this may introduce bias (Jüni, Altman, & Egger, 2001; Khan et al., 2003). Studies differed in a variety of dimensions including their design, conduct and analysis and bias may be introduced at any stage including sample selection, administration, and measurement of confounders (Jüni et al., 2001; Khan et al., 2003). A quality scale was therefore developed for this review focusing on all these characteristics in the context of neuropsychological assessment, as shown in Table 2.1. A range of confounders were included based on the relevant literature (Gazzaniga et al., 2009; Kolb & Whishaw, 2009; Lezak et al., 2004; Martin, 2006; Strauss et al., 2006). Studies received points when they fulfilled the criteria of sampling, standardised procedure, and confounder control. The points-weighting for each category was finalised via consensus between the student and the PhD supervisors.

Table 2.1. *The Quality Rating Scale including details on scoring*

| **Quality category** | **Points given** |
|---|---|
| *Sample* | |
| Adequate sample description (procedure of how the final sample came to be selected) | /1 |
| *Procedure* | |
| Standardised administration of tests | /1 |
| Standardised administration of diagnostic instruments (e.g. use of both file review and interview on the PCL/-R) | /½ |
| *Control of neuropsychological confounders: Studies gain points for having controlled (statistically, sampling, or otherwise) for:* | |
| Age | /1 |
| Key mental illnesses, especially psychotic, bipolar & depressive disorders | /1 |
| Handedness & lateral asymmetry | /½ |
| Intelligence | /1 |
| Substance abuse/dependence | |
| Current | /1 |
| Past | /½ |
| Traumatic brain injury & neurological condition | /1 |
| Education/literacy | /½ |
| Current medication | /1 |
| **Total** | /10 |

*Note.* PCL/-R=Psychopathy Checklist/-Revised.

Following rating of all included studies, the distribution of scores appeared normal, as assessed by standard methods (Pallant, 2005). The mean was $M$=5.57, $SE$=0.15 and the 5% trimmed mean was very close with $M_{5\%}$=5.55, demonstrating little deviation of the extreme values (Pallant, 2005). Skewness and Kurtosis were not

significant at $P$=0.01 level. The Kolmogorov-Smirnov normality test (K-S test) with Lilliefors significance correction indicated normality, with a statistic of 0.075, $df$=130, $P$>0.05. Visual inspection of plots also suggested a normal distribution. These included the histogram, an almost linear Q-Q plot and a detrended normal plot showing some deviation towards the higher values only (Appendix B, Figure 10.1 and Figure 10.2). A normal distribution allowed for a tertile split of scores so that studies fell in one of three groups: low, medium and high quality. Final study ratings are shown in Appendix C (Table 11.1). It is important to highlight that the rating resulted from the information in the study reports which may not be complete.

### 2.2.3 Data analysis and reporting

Details of the comparison groups, sample sizes, populations, age, IQ, education, method of assessment for antisocial personality, neuropsychological tests used, special conditions and results are shown in summary tables. Study results were considered as significant when $P<0.05$, which was applied strictly for consistency, even where a study used a different *alpha* level. Potentially overlapping samples (e.g. from the same research group and recruitment location) complicated estimations of sample totals. Where possible overlaps were detected, the largest sample was selected for the calculation of a minimum total number of participants (e.g. "at least 100 individuals with ASPD..."). In addition, where antisocial and control groups were divided in subgroups (e.g. high and low-anxious – HA and LA respectively), results were reported for these subgroups when significant differences were present or when no overall comparisons were conducted.

#### 2.2.3.1 Quantitative synthesis

Descriptive statistics including means, standard deviations, and sample sizes were recorded for both the antisocial and control groups. For the synthesis, the software Review Manager, 5$^{th}$ version (The Cochrane Collaboration, 2008) was employed. The *alpha* level of significance was 0.05 unless otherwise specified. Inverse variance analysis was conducted using standardised mean group differences with the DerSimonian and Laird variation for random effects (Deeks, Altman, & Bradburn, 2001; Deeks, Higgins, & Altman, 2008; DerSimonian & Laird, 1986). The standardised mean group difference is also known as Hedges' adjusted *g* (Hedges & Olkin, 1985). Hedges' adjusted *g* contains a different calculation of pooled standard deviation than Cohen's (1988) *d* adjusting for small sample bias (Deeks et al., 2001; Hedges & Olkin, 1985), appropriate for the studies in this review. Smaller standard errors and larger sample sizes attract larger weights, which dictate each study's contribution in the pooled effect size estimate (Hedges & Olkin, 1985). According to Cohen's guidelines, an effect size is small when in the range of 0.2, medium when in the range of 0.5 and large when it is in the range of 0.8.

Fixed effects models assume that the true group differences are the same across studies, whereas random effects assume that these differences will vary

between clusters around the grand mean (Deeks et al., 2001; Deeks et al., 2008; Tabachnick & Fidell, 2007). Random effects generally produce wider confidence intervals and are considered to be more realistic when there is considerable (and unexplained) heterogeneity (Everitt, 2003; Khan et al., 2003). They have become the norm in dealing with this issue, especially in observational research where it is difficult to assume that the effect size is the same across studies (Hunter & Schmidt, 2000). Although often thought of as conservative, random effect models are not always so, since they can exaggerate the contribution of smaller studies the results of which may be subject to many biases and thus proving misleading (Deeks et al., 2008; Poole & Greenland, 1999). Therefore, care should be exercised in exploring sources of heterogeneity when applying random effects models to accommodate for it and model selection should be made a priori (Khan et al., 2003). Because of the strict diagnostic inclusion criteria, fixed effects models were employed when examining the same neuropsychological function with equivalent methods (e.g. variations of the Wisconsin Card Sorting Test [WCST] for set shifting ability), when there were no outliers in the sample of effect sizes and where heterogeneity was small. On the other hand, random effects were applied where heterogeneity was significant or outliers in the sample of effect sizes were observed, in order to compute a more conservative estimate.

#### *2.2.3.1.1 Multiple sample comparisons*

Where there were multiple comparison groups from the same study, extreme groups only were included in the meta-analyses (e.g. high vs. low psychopathy), with priority for those originating from the same population (e.g. prisoners) in order to minimise sampling bias. It was also important to make the best effort in identifying overlapping samples. Inclusion of overlapping samples can result in inflated total sample size estimations and systematically bias the pooled effect estimate (Khan et al., 2003). Where sample overlaps were encountered, the largest sample was preferred.

#### *2.2.3.1.2 Incompatible outcome measures*

The outcome measures from neuropsychological tasks can indicate either how good (e.g. number of correct responses) or how poor (e.g. number of errors)

performance was and these cannot be pooled simultaneously. Where these needed to be entered into the same meta-analysis, the direction good performance indicators was reversed (multiplied by -1).

#### *2.2.3.1.3 Sensitivity analysis*

Often, more than one set of data are available in the same study or for the same sample. Where this occured in the present review, sensitivity analysis was employed which involved repeating the analysis with different sets of assumptions or alternative sets of data (Khan et al., 2003). In some cases, a single study or group of studies provided more than one sets of data for the same sample or overlapping samples. In this case, two meta-analyses were conducted, one including the strongest effects and another one including the weakest ones. This method resulted in indentifying the margins of the pooled effect size estimate.

### 2.2.3.2 Heterogeneity of effect sizes

Because of different methods employed by the studies, considerable heterogeneity was anticipated, especially for broad neuropsychological functions. To evaluate heterogeneity, a $Chi^2$ – or $Q$ statistic – test (Deeks et al., 2008; Hedges & Olkin, 1985; Higgins, Thompson, Deeks, & Altman, 2003) was employed. A significantly large $Chi^2$ suggests heterogeneity. Because this statistic has low power with smaller sample sizes and few studies, an *alpha* level of significance of 0.10 is recommended. Furthermore, heterogeneity may be quantified by the $I^2$ statistic (Deeks et al., 2008; Hedges & Olkin, 1985; Higgins et al., 2003). Generally, $I^2$ up to 40% represents relatively inconsequential, 30%-60% moderate, 50%-90% substantial, and 75%-100% considerable heterogeneity. Where there was heterogeneity and sufficient data, studies were stratified according to possible sources of heterogeneity (Deeks et al., 2008; Khan et al., 2003; G. D. Smith & Egger, 2001). When examining subgroup differences during stratification, the $I^2$ represents the size of variability due to genuine subgroup differences (Deeks et al., 2008).

#### 2.2.3.3 Publication bias

One of the major sources of bias in a meta-analysis is the publication bias due to editors' preference to select studies reporting significant results and because authors may not submit a report with no significant findings (Khan et al., 2003; Sterne, Egger, & Smith, 2001). This problem becomes even more pronounced when the focus of the review is on peer-reviewed publications, although in principle they would generally reflect more rigorous, valid, and reliable research. There are a variety of ways to explore publication bias including funnel plots and the failsafe *N*.

##### *2.2.3.3.1 Funnel plots*

According to Khan et al. (2003) and Sterne et al. (2001), funnel plots are essentially scatter plots of effect size against sample size. The result is a funnel-shaped distribution with greater variability between effect sizes of smaller studies. In a broadly inclusive selection of studies, the funnel plot should appear symmetrical. Asymmetry in the funnel plot may provide evidence for studies missing due to publication or other selection bias, for considerable heterogeneity, or for overestimation of effect size. To use this method of assessment of bias, a large enough number of studies is required, since the conclusions are based on visual inspection of the funnel plot. In this review, funnel plots were examined where a pooled effect size estimate has reached significance and five or more studies were available.

##### *2.2.3.3.2 Failsafe N*

Another way to investigate publication bias is to calculate how many studies with zero effect size would be required so that the pooled effect size estimate does not reach significance ($P>0.05$). The required number of studies with a zero effect size is known as the 'failsafe *N*' (Rosenthal, 1979). If the failsafe *N* exceeds a critical value representing the number of filed away studies (e.g. due to null results) then publication bias is less probable. The critical value equals $5k+10$ where $k$ is the number of studies in the meta-analysis. Although generally considered a valid way of exploring robustness of meta-analytic results against publication bias, because the failsafe *N* relies on rejection of the null hypothesis using critical probability levels of

significance, it should be considered in conjunction with effect size estimates and related confidence intervals (Sterne et al., 2001).

#### 2.2.3.4 Assessing the influence of confounding variables

Although most included studies addressed possible confounding/moderator variables in neuropsychological performance, including age, intellectual functioning and years in education (Lezak et al., 2004), when pooling the results for a meta-analysis, the increased sample size may reveal a different picture. Study quality may also affect results. In order to assess the effect of the possible moderator variables (age, intelligence, prior education and study quality) in the meta-analyses, bivariate correlations (Spearman's) were conducted between standardised mean group differences and the moderator variables using SPSS Statistics, version 17.0 (SPSS Inc, 2009). In order to conduct these correlations meaningfully, a minimum sample is necessary. Cohen (1988, 1992) suggested that a statistical power of 0.80 (20% probability for Type II error) may be used as standard and has described a correlation coefficient of 0.50 as large. For an *alpha* of 0.05, by using a reduced power of 0.70 (more conservative) and a correlation coefficient of 0.70 (very strong) in a two-tailed test, meaningful Spearman's correlations would require a sample of 11 cases or above (exact distribution). This was calculated using the software GPower, Ver. 3.1.2 (Erdfelder, Faul, & Buchner, 1996). Therefore, correlations between the SMDs and moderator variables were computed only if a minimum of 11 studies were available in a meta-analysis.

## 2.3 Results

### 2.3.1 Studies

Electronic searches produced a total of 7,083 abstracts. Following initial screening of titles and abstracts for relevance and according to the inclusion criteria, full text papers were obtained for further examination. This resulted in a selection of 142 publications/dissertations. Hand searching the reference lists of these studies revealed two additional publications (Klaver, Lee, & Hart, 2007; Kosson, Smith, & Newman, 1990). Of the studies, 30 were doctoral dissertations of which 12 were not obtainable either through interlibrary loan schemes or via the authors. Thus the final list contained 132 publications/dissertations (Appendix C, Table 11.1). All of these were in English the exceptions being one German (Weber, Sommer, Hajak, & Muller, 2004), one French (Pham, Philippot, & Rime, 2000) and one Portuguese (Jozef & da Silva, 1999) which were translated in order to enable use of their data in the review. Not all publications reported data for meta-analysis resulting in loss of participants in pooling. There were also occasions where no group comparisons were conducted, for example when a Wechsler Adult Intelligence Scale (WAIS) assessment was employed to illustrate the groups' IQ but no comparisons were reported in subtests. Such missing information is highlighted as n/a (=Not Available) in study tables. Tabled details of all studies are presented in Appendix C.

### 2.3.2 Neuropsychological functions

Studies examined most neuropsychological functions. An outline of functions, and their descriptions are provided in Table 2.2. Summative Forest plots from meta-analyses (strongest effects only, where applicable) are presented in Figure 2.1 for ASPD and Figure 2.2 for psychopathy. Tables 2.31 to 2.36 provide additional statistical detail.

Table 2.2. *Neuropsychological functions examined by studies on antisocial personality with relevant tasks and pages for corresponding commentary.*

| Function | Tasks | Description |
|---|---|---|
| **1. Executive (pp. 62-103)** | | Complex, subtle, higher-order functions that facilitate goal-oriented behaviour. They are necessary for generation of adaptive responses and intentional actions. Theory suggests the following distinct elements of executive functions: volition, planning, purposive action, self-regulation and effective performance [1,2]. There were no studies on volition and purposive action identified. |
| **Planning (pp. 62-64)** | Towers of London<br>Stockings of Cambridge<br>Porteus Mazes<br>Executive Golf Task<br>N-back Task<br>Digit Span Backwards | A step-wise approach to goal-oriented behaviour [2]. |
| **Self-regulation (pp. 65-90)** | | Operations that involve the ability to direct one's actions within one's environment and achieve objectives. |
| Productivity (p. 65) | Controlled Oral Word Association | Ability to demonstrate activity. Tests often include item generation such as design and verbal fluency [2]. |
| Cognitive flexibility (pp. 65-73) | Wisconsin Card Sorting Test<br>Intra/Extra-Dimensional Set Shifting<br>Differential reward & | Capacity to shift or change course of action including attentional set shifting, decision-making, response reversal, etc. Failure in cognitive flexibility may result in perseveration and stimulus-bound behaviour [1,2, 12]. |

| | | |
|---|---|---|
| | punishment learning | |
| | Iowa Gambling Task | |
| | Card Playing Task | |
| | Baloon Analogue Risk Task | |
| | Object Alternation | |
| | Space Alternation | |
| | Divergent Thinking Task | |
| | Train Making Test, Part B | |
| Motor regulation (pp. 74-89) | Luria Motor Tasks 22 & 23 | Operations of motor control including tasks of alternating responses, finger sequencing, hand sequencing and response inhibition [2]. |
| | Go/NoGo (including passive avoidance) | |
| | Response/gratification delay | |
| | Stop-Signal Task | |
| | Stroop (colour-word, box, semantic, picture-word, number) | |
| **Effective performance (pp. 91-99)** | Stroop (as above) | Processes of monitoring, self-correction and conflict resolution in order to minimise errors [2]. |
| | Flanker | |
| | Simon | |
| | Invalid respone cue | |
| | Dichotic Listening Task | |
| | Picture-word interference | |

| | | |
|---|---|---|
| **2. Abstraction (pp. 104-114)** | | Abstraction describes the ability to reason in and grasp non-concrete terms and generalisations. Attentional set shifting paradigms also involve concept formation, apart from executive function [2]. |
| **Concept formation (pp. 108-110)** | Similarities<br>Proverbs<br>Set-shifting tasks as above<br>Short Category Test<br>Raven's Matrices<br>Shipley Abstraction<br>Abstraction (Dureman-Sälde) | The ability to think in abstract terms. Performance can differ between paradigms and between sensory modalities (verbal vs. visual stimuli) [2]. |
| **Reasoning (p. 111)** | Comprehension<br>Interpretation of metaphors<br>Picture Completion<br>Picture Arrangement | The process of thinking using logic in order to reach conclusions [2]. |
| **Mathematical procedures (p. 112)** | Arithmetic | Reasoning in mathematical terms [2]. |
| **Semantic abstraction (p. 112)** | Abstract semantic processing | Tasks in this group involved semantic processing of abstract versus concrete terms. |

| | | |
|---|---|---|
| **3. Affect & social cognition (pp. 115-146)** | | Affect may include feelings experienced subjectively about a stimulus whereas emotions are relatively short-lived inner feelings, often inferred from affect [3,4]. Affect and emotion may involve cognitive appraisals [5,6] and contribute to social cognition [3]. |
| **Affective operations (pp. 115-135)** | | All affect/emotion-related paradigms. |
| Affective processing (pp. 115-121) | Lexical Decision Task<br>Affective induction<br>Affective discrimination<br>Affective priming<br>Flanker<br>Abstract discrimination<br>Oddball<br>Go/NoGo | Paradigms involving affective induction and measurement of its impact on cognitive task performance. |
| Affect recognition (pp. 122-129) | Affect recognition (forced-choice, open-ended)<br>Morphed faces<br>Dichotic Listening Task | Paradigms evaluating the ability to identify affective content. |
| Affect & memory (pp. 130-133) | Uncued/cued recall<br>Uncued/cued recognition | Memory for affectively-laden content. |
| Affect & language (p. 134) | Written and oral narratives | Expression of affective content and emotion in speech. |
| **Social cognition (pp. 136-142)** | | Being able to understand others' actions or minds and to interpret interpersonal behaviour are all aspects of social cognition [3]. This complex function goes beyond the |

| | | |
|---|---|---|
| | | basics of emotion recognition and abstract thinking and recruits several brain areas [1,3]. |
| Theory of Mind (pp. 136-140) | 1st & 2nd order inference<br>Mentalising | Describes the ability to understand one's own and others' minds or mental states [3,4]. |
| Moral reasoning (pp. 140-141) | | This subgroup of tasks involved moral perception and decision-making. |
| Social interpretation & knowledge (pp. 142-143) | | Tasks involving interpretations of social interactions, attitudes and knowledge. |
| **4. Memory (pp. 147-161)** | | A broad neuropsychological function encompassing many modules including STM and LTM, declarative and non-declarative and emotional memory, depending on the type of information and the duration of storage [1,2,3,7]. Access to stored information can also vary, e.g. recall and recognition [2]. |
| **STM (p. 153 & pp. 156-157)** | Continuous Performance – Identical pairs<br>Logical Memory I<br>Delayed Matching to Sample<br>Digit Span (Forward & Backward)<br>Visual Retention Test<br>Auditory-Verbal Learning Test<br>Matching | Describes storage of a relatively short duration, up to a few minutes, and is often referred to as 'working' memory when its contents are manipulated [3]. |

| | | |
|---|---|---|
| | Visual Reproduction I | |
| | Paired Associate Learning | |
| | Other uncued or cued recall | |
| | Other uncued or cued recognition | |
| **LTM (pp. 153-154 & 156-157)** | Logical Memory II<br>Emotional Memory Task<br>Visual Reproduction II<br>Paired Associate Learning-Delayed | Refers to the system storing information for longer than approximately fifteen minutes and includes multiple other systems. These are, primarily, a conscious (declarative memory), an unconscious (non-declarative) and a part encompassing affect, conditioning and emotion (emotional memory). Declarative memory has also been divided in episodic (personal and autobiographical) and semantic (facts and knowledge) while non-declarative memory includes item-specific and procedural stores [1,2,3]. |
| **WM (p. 154 & 157)** | Digit Span Backward<br>N-back | The term WM is sometimes used interchangeably with STM [3] but here it refers to short-term storage of items when these are subjected to mental manipulations [1]. |
| **Implicit memory: priming (p. 155)** | Lexical Decision Task<br>Stroop<br>Flanker | Tasks in this group examined implicit memory in the form of facilitation or inhibition due to a preceding prime [1,11]. They were not included with selective attention paradigms because stimuli are not competing in simultaneous presentation. |
| **5. Attention (pp. 162-180)** | | Mechanism which channels the finite mental resources to particular stimuli or focal points in the environment. It can be regulated automatically as well as consciously [1,3,7]. |
| **Sustained (pp. 162-168)** | Continuous Performance – Identical pairs<br>Continuous Performance – Oddball<br>Cancellation | Ability to focus onto the same set of stimuli over a prolonged period of time [1,3,7]. |

| | | |
|---|---|---|
| | Game | |
| | Target discrimination | |
| **Selective (pp. 169-170)** | As for effective performance | Process of focusing onto a set of stimuli against competing ones [1,3,7]. |
| **Divided (pp. 171-174)** | Dichotic Listening Task | Concurrent allocation of attentional resources between two or more sets of competing stimuli [1,3,7]. |
| | Continuous Performance – Oddball vs. Game | |
| | Dual task – target descrimination | |
| **Complex (pp. 175-176)** | Trail Making Test, Parts A & B | A group of visuographic tasks which draw on various neuropsychological resources [2,11]. |
| | Digit Symbol | |
| **Reaction time (p. 177)** | Reaction time | This category includes tasks solely recording response latency under forced conditions. |
| **6. Language (pp. 181-199)** | | A higher function for communication involving several operations and substrates [1,3]. |
| **Verbal expression (pp. 181-188)** | Controlled Oral Word Association | Oral and written expression such as discourse, fluency and vocabulary [2]. |
| | Vocabulary | |
| | Narratives | |
| **Non-verbal expression (pp. 188-189)** | Oral narratives | Tasks including gestural and body language accompanying verbal expression. |
| **Academic skills (reading & writing) (pp. 190-192)** | National Adult Reading Test | Learned verbal skills including writing and spelling [2]. Reading tasks were also included. |
| | Word naming | |

| | | |
|---|---|---|
| | Wide Range Achievement Test | |
| | Transcribing | |
| **Semantic processing (pp. 192-197)** | Lexical Decision Task | Tasks evaluating the ability to access semantic content of verbal stimuli. |
| | Concrete &/or abstract discrimination | |
| | Semantic Stroop | |
| | Sentence comprehension | |
| **Knowledge acquisition and retention (p. 197)** | | Lezak et al. (2004) have considered acquisition and retention of general knowledge as a subset of language operations, with tests such as the Information subtest (general knowledge) of the WAIS. |
| **7. Perception (pp. 200-205)** | | Although all cognitive tasks are dependent on perception, this category includes targeted perceptual operations which are often addressed in neuropsychological assessments [2]. |
| **Visual (pp. 200-204)** | Matching | A cluster of functions including visual (in)attention (awareness of visual stimuli as opposed to the attentional processes discussed earlier), scanning/search, colour perception, recognition (identifying features of visual stimuli as opposed to recognition in connection with memory) and organisation (making sense of distorted visual stimuli) [1,2]. |
| | Judgement of Line Orientation Task | |
| | Hooper's Visual Recognition Task | |
| | Target discrimination | |
| | Cancellation | |
| **Auditory (pp. 204-205)** | Dichotic Listening Task | A cluster of functions including acuity, discrimination (for phonemes, sounds, etc), auditory attention (analogous to visual attention), rhythm, music, emotion, etc. [2] |
| | Target discrimination | |
| **Olfaction (pp. 204-205)** | Odour detection | Another form of perception with neuropsychological interest including tests such as odour detection and smell identification [1,2,3]. |

| | | |
|---|---|---|
| | Smell identification | |
| **8. Interhemispheric integration (pp. 206-210)** | Banich's Letter-name Identity Task<br>Poffernberger's paradigm<br>Consonant-Vowel-Consonant<br>Handedness | Here, this refers to connectivity between the two hemispheres and lateralisation [1,3]. |
| **9. Construction & visuospatial skills (pp. 211-212)** | Block Design<br>Block (Dureman-Sälde)<br>Object Assembly<br>Mental rotation | Operations constituting a synergy between motor skills and visuospatial perception on a conceptual level. They do not involve processes of abstraction or mental flexibility to the extent of falling into the category of concept formation tasks and do not represent the composite of volition, planning, purposive and effective action characterising executive tasks [2]. |
| **10. NSS & Motor skills (pp. 213-214)** | Neurological evaluation scale<br>NSS assessment<br>Finger tapping<br>Luria Motor Tasks 22 & 23 | NSS involve minor neurological impairments likely due to lack of sensory or motor integration [8] potentially caused by subcortical abnormalities [9]. Motor tasks involve functions of the motor cortex such as manual dexterity [2]. |
| **11. Intelligence (pp. 215-224)** | Wechsler Adult Intelligence Scales (Revised & 3rd editions) | Although a broad concept [4], intelligence in neuropsychology describes general cognitive ability [3]. Nowadays it is considered by some to be an archaic concept with little practical meaning [2]. It is commonly measured by a score known as the IQ where the value of 100 represents the population mean [2,3]. It may also be distinguished to VIQ or PIQ depending |

| | | |
|---|---|---|
| | National Adult Reading Test | on whether it draws on verbal or other cognitive resources [10]. |
| | Shipley Institute of Living Scale | |
| | Quick Test | |
| | Dureman-Sälde battery | |

[1]Gazzaniga et al. (2009); [2]Lezak et al. (2004); [3]Kolb and Whishaw (2009); [4]Colman (2003); [5]Damasio (1994); [6]LeDoux (2000); [7]Martin (2006); [8]Griffiths, Sigmundsson, Takei, Rowe, and Murray (1998); [9]Heinrichs and Buchanan (1988); [10]Wechsler (1955, 1981, 1997); [11]Strauss et al., (2006); [12]Rahman, Sahakian, Hodges, Rogers, & Robbins, 1999; Rolls, 2000; Rolls, Hornak, Wade, & McGrath, 1994.

STM/LTM=Short/long-term memory; WM=Working memory; VIQ=Verbal IQ; PIQ=Performance IQ.

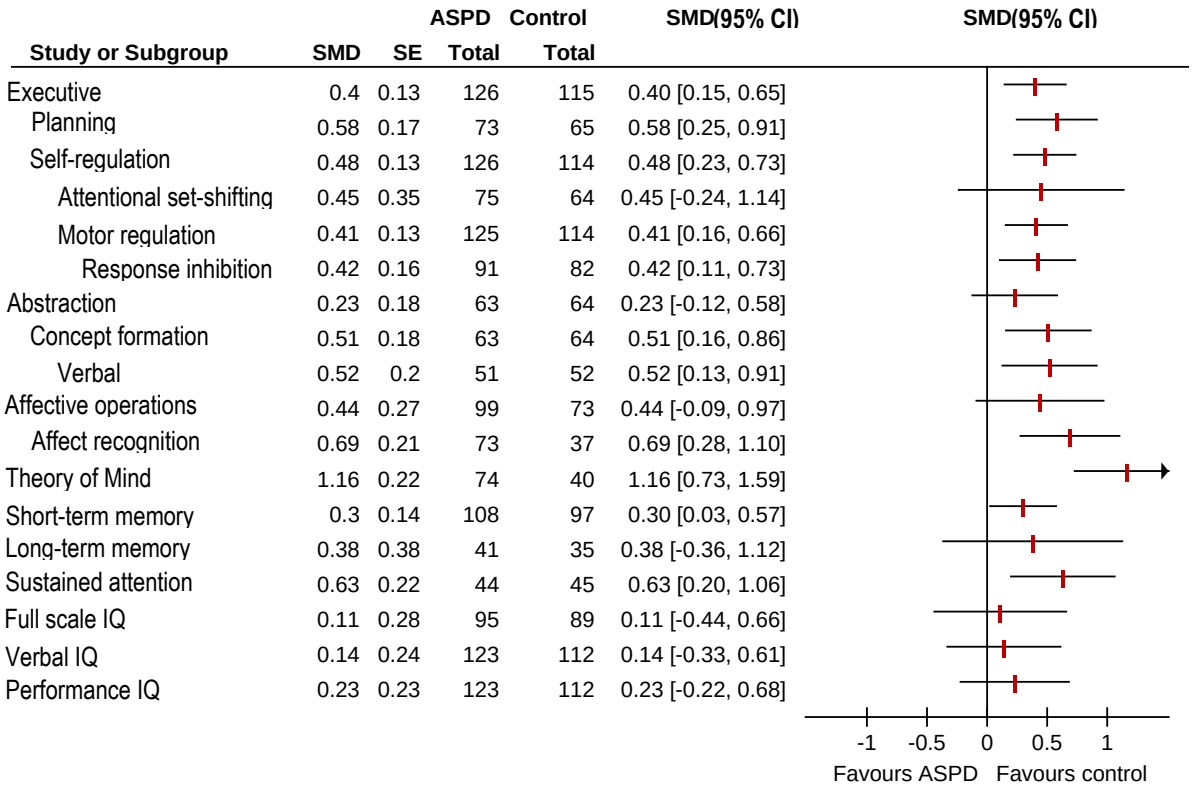


*Figure 2.1.* Summative Forest plot of pooled effect sizes (standard mean differences [SMDs]) from meta-analyses on neuropsychological findings in Antisocial Personality Disorder (ASPD) using strongest effects where applicable.

SE=Standard error; CI=Confidence intervals.

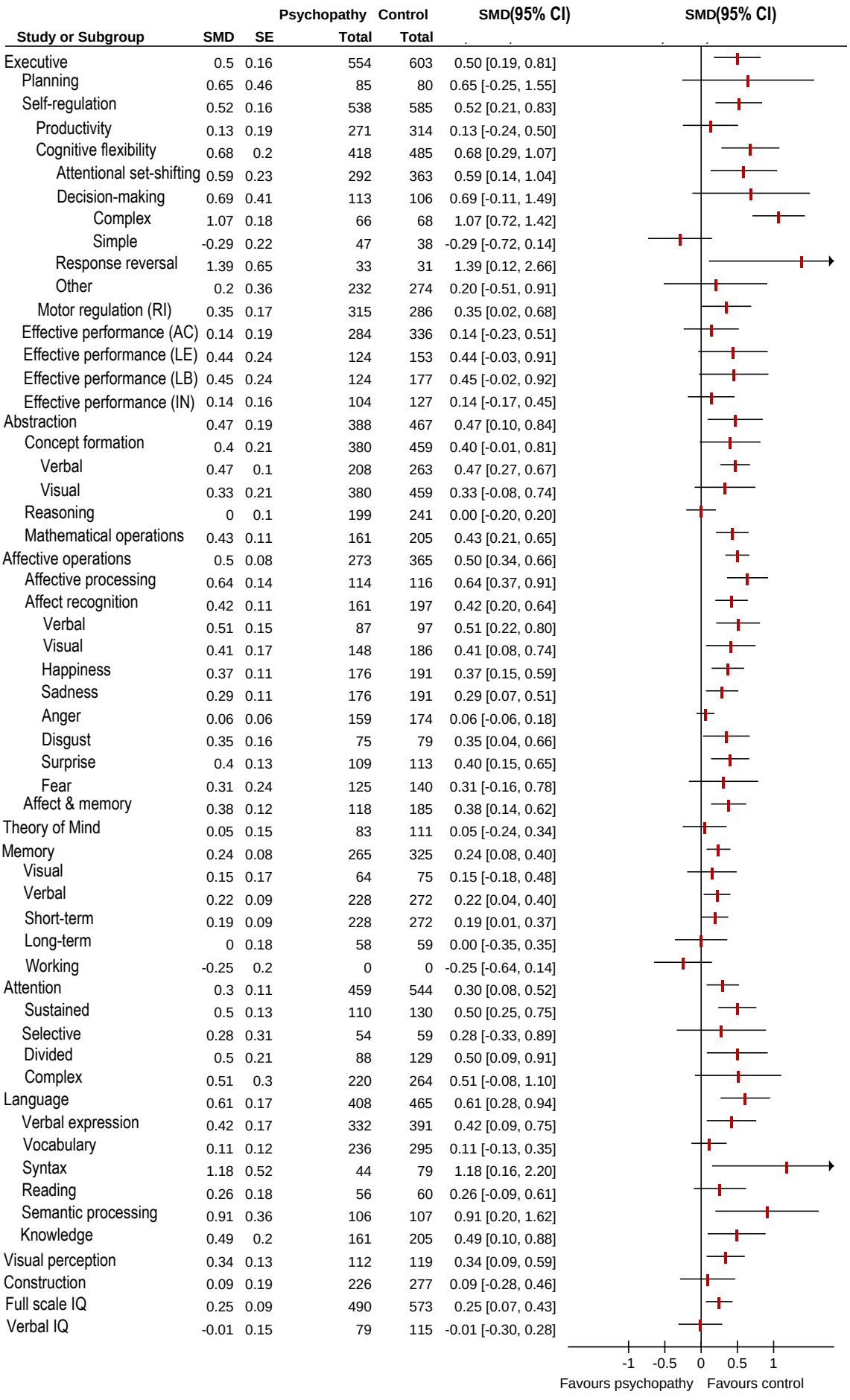
Psychopathy
Control
SMD(95% CI)
SMD(95% CI)
Study or Subgroup
SMD
SE
Total
Total
Executive 0.5 0.16 554 603 0.50 [0.19, 0.81]
Planning 0.65 0.46 85 80 0.65 [-0.25, 1.55]
Self-regulation 0.52 0.16 538 585 0.52 [0.21, 0.83]
Productivity 0.13 0.19 271 314 0.13 [-0.24, 0.50]
Cognitive flexibility 0.68 0.2 418 485 0.68 [0.29, 1.07]
Attentional set-shifting 0.59 0.23 292 363 0.59 [0.14, 1.04]
Decision-making 0.69 0.41 113 106 0.69 [-0.11, 1.49]
Complex 1.07 0.18 66 68 1.07 [0.72, 1.42]
Simple -0.29 0.22 47 38 -0.29 [-0.72, 0.14]
Response reversal 1.39 0.65 33 31 1.39 [0.12, 2.66]
Other 0.2 0.36 232 274 0.20 [-0.51, 0.91]
Motor regulation (RI) 0.35 0.17 315 286 0.35 [0.02, 0.68]
Effective performance (AC) 0.14 0.19 284 336 0.14 [-0.23, 0.51]
Effective performance (LE) 0.44 0.24 124 153 0.44 [-0.03, 0.91]
Effective performance (LB) 0.45 0.24 124 177 0.45 [-0.02, 0.92]
Effective performance (IN) 0.14 0.16 104 127 0.14 [-0.17, 0.45]
Abstraction 0.47 0.19 388 467 0.47 [0.10, 0.84]
Concept formation 0.4 0.21 380 459 0.40 [-0.01, 0.81]
Verbal 0.47 0.1 208 263 0.47 [0.27, 0.67]
Visual 0.33 0.21 380 459 0.33 [-0.08, 0.74]
Reasoning 0 0.1 199 241 0.00 [-0.20, 0.20]
Mathematical operations 0.43 0.11 161 205 0.43 [0.21, 0.65]
Affective operations 0.5 0.08 273 365 0.50 [0.34, 0.66]
Affective processing 0.64 0.14 114 116 0.64 [0.37, 0.91]
Affect recognition 0.42 0.11 161 197 0.42 [0.20, 0.64]
Verbal 0.51 0.15 87 97 0.51 [0.22, 0.80]
Visual 0.41 0.17 148 186 0.41 [0.08, 0.74]
Happiness 0.37 0.11 176 191 0.37 [0.15, 0.59]
Sadness 0.29 0.11 176 191 0.29 [0.07, 0.51]
Anger 0.06 0.06 159 174 0.06 [-0.06, 0.18]
Disgust 0.35 0.16 75 79 0.35 [0.04, 0.66]
Surprise 0.4 0.13 109 113 0.40 [0.15, 0.65]
Fear 0.31 0.24 125 140 0.31 [-0.16, 0.78]
Affect & memory 0.38 0.12 118 185 0.38 [0.14, 0.62]
Theory of Mind 0.05 0.15 83 111 0.05 [-0.24, 0.34]
Memory 0.24 0.08 265 325 0.24 [0.08, 0.40]
Visual 0.15 0.17 64 75 0.15 [-0.18, 0.48]
Verbal 0.22 0.09 228 272 0.22 [0.04, 0.40]
Short-term 0.19 0.09 228 272 0.19 [0.01, 0.37]
Long-term 0 0.18 58 59 0.00 [-0.35, 0.35]
Working -0.25 0.2 0 0 -0.25 [-0.64, 0.14]
Attention 0.3 0.11 459 544 0.30 [0.08, 0.52]
Sustained 0.5 0.13 110 130 0.50 [0.25, 0.75]
Selective 0.28 0.31 54 59 0.28 [-0.33, 0.89]
Divided 0.5 0.21 88 129 0.50 [0.09, 0.91]
Complex 0.51 0.3 220 264 0.51 [-0.08, 1.10]
Language 0.61 0.17 408 465 0.61 [0.28, 0.94]
Verbal expression 0.42 0.17 332 391 0.42 [0.09, 0.75]
Vocabulary 0.11 0.12 236 295 0.11 [-0.13, 0.35]
Syntax 1.18 0.52 44 79 1.18 [0.16, 2.20]
Reading 0.26 0.18 56 60 0.26 [-0.09, 0.61]
Semantic processing 0.91 0.36 106 107 0.91 [0.20, 1.62]
Knowledge 0.49 0.2 161 205 0.49 [0.10, 0.88]
Visual perception 0.34 0.13 112 119 0.34 [0.09, 0.59]
Construction 0.09 0.19 226 277 0.09 [-0.28, 0.46]
Full scale IQ 0.25 0.09 490 573 0.25 [0.07, 0.43]
Verbal IQ -0.01 0.15 79 115 -0.01 [-0.30, 0.28]
-1
-0.5
0
0.5
1
Favours psychopathy
Favours control

*Figure 2.2.* Summative Forest plot of pooled effect sizes (standard mean differences [SMDs]) from meta-analyses on neuropsychological findings in psychopathy using strongest effects where applicable.
SE=Standard error; CI=Confidence intervals; RI=Response inhibition; AC=Accuracy; LE=Latency-experimental; LB=Latency-baseline; IN=Interference.

### 2.3.3 Executive functions

Seven studies were identified on ASPD which reported data on at least 126 individuals with the disorder and 115 without. There was also one study on DPD whereas a total of 42 studies examined psychopathy including at least 564 individuals with psychopathy and 616 without.

#### 2.3.3.1 Planning

##### *2.3.3.1.1 ASPD*

There were four studies on ASPD (Barkataki et al., 2005; Dolan & Park, 2002; Kumari et al., 2006; Stevens, Kaplan, & Hesselbrock, 2003) including at least 77 individuals with ASPD and 67 without (Table 2.3). All ASPD samples originated from forensic inpatients except those of Stevens et al. which was recruited from the general public. Healthy controls were from the general public. This population difference is potentially a confounder. Significant group differences were reported only by Dolan and Park where controls performed better on the more challenging problems of the SOC task. An overview of studies is shown in Table 2.3.

A meta-analysis yielded a significant, medium pooled effect size estimate with no evidence of heterogeneity with the strongest effect from Kumari et al. (2006) who provided breakdown data. Results were comparable and significant with the weakest effect from that study. Failsafe *N*s of 10.15 and 6.82 for the two sets of data respectively were under the critical cut-off of 25, suggesting possible publication bias.

##### *2.3.3.1.2 Psychopathy*

There were four studies on psychopathy (Lapierre, Braun, & Hodgins, 1995; Mercer, Selby, & McClung, 2005; Pham, Vanderstukken, Philippot, & Vanderlinden, 2003; S. S. Smith, Arnett, & Newman, 1992), including 246 individuals with psychopathy and 285 without, all from prisons (Table 2.3). Those with psychopathy performed worse on the executive measure of the Porteus Mazes (qualitative score) on every occasion. They also performed worse on planning measures of the Tower of London (ToL), while no significant differences were reported for Digits Backward (result available for S. S. Smith et al., 1992 only).

Using strongest effects, an overall meta-analysis yielded a medium to large but non-significant pooled effect size estimate with considerable heterogeneity. The pooled effect size estimate was small and non-significant with weakest effects, 0.19[-0.73,1.11], and comparable heterogeneity.

Table 2.3. *Studies which examined planning*

| Reference | Task | Primary outcomes | Result favoured |
|---|---|---|---|
| ***ASPD*** | | | |
| Barkataki 2005 | ToL (computerised) | Moves | ns |
| | | Initial thinking time | ns |
| | | Subsequent execution time | ns |
| | Executive Golf[a] | Within-search errors | ns |
| | | Between-search errors | ns |
| | | Strategy score | ns |
| Dolan 2002 | SOC | Perfect solutions | Control (4 & 5-move problems) |
| | | Excess moves | Control (4-move problems only) |
| | | Initial thinking time | ns |
| | | Subsequent thinking time | n/a |
| Kumari 2006 | n-back[a] | Accuracy | ns |
| | | Response latency | ns |
| Stevens 2003 | Porteus mazes | Highest mental age | ns |
| ***Psychopathy*** | | | |
| Lapierre 1995 | Porteus mazes | Quantitative score | ns |
| | | Qualitative score | Control |
| Mercer 2005 | Digit Span Backward (WAIS-R)[a] | n/a | n/a |

| | | | |
|---|---|---|---|
| Pham 2003 | ToL:<br>Three conditions of difficulty – facilitated (easiest), neutral & misleading (most difficult) | Excessive moves | Control (difficult condition only) |
| | | Broken rules | ns |
| | | Initial thinking time | ns |
| | | Execution time | Control |
| | Porteus mazes | Quantitative score | ns |
| | | Qualitative score | Control |
| | Digit Span Backward (WAIS)[a] | Performance score | n/a |
| Smith 1992 | Digit Span Backward (WAIS-R)[a] | Performance score | ns |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; ToL=Tower of London; SOC=Stockings of Cambridge; WAIS=Wechsler Intelligence Scale; n/a=Not available; ns=Not significant.

[a]Task involved WM.

#### 2.3.3.2 Self-regulation

For self-regulation, there were eight publications on ASPD involving at least 135 individuals with the disorder and 122 without. Furthermore, 38 publications and dissertations on psychopathy included at least 551 individuals with psychopathy and 585 controls.

##### *2.3.3.2.1 Productivity*

Of six studies in this category (Dinn & Harris, 2000; D. S. Goldstein, 1998; Hart, Forth, & Hare, 1990; Mercer et al., 2005; S. S. Smith et al., 1992; Stevens et al., 2003), only Stevens et al. (2003) investigated ASPD (general public), failing to find a significant effect. In the remaining studies (Dinn & Harris, 2000; D. S. Goldstein, 1998; Hart et al., 1990; Mercer et al., 2005; S. S. Smith et al., 1992) there were over 271 individuals with psychopathy and over 314 without. No significant group differences were observed. Dinn and Harris (2000) recruited from the general public. Participants in other studies came from prisons.

A meta-analysis (psychopathy) yielded a small and non-significant pooled effect size estimate with substantial heterogeneity**Error! Reference source not found.**, mostly due to the contribution of Mercer et al. (2005) This was the only study of medium quality in the group (others were of high quality). Without this study, results were comparable but heterogeneity was relatively inconsequential. Sensitivity analysis revealed comparable results with either sample from Hart et al. (1990).

##### *2.3.3.2.2 Cognitive flexibility*

Several studies were found in this category and relevant tasks involved attentional set shifting, response reversal, decision-making, and identifying alternative item uses. A summary of studies in this category can be found in Table 2.4 (studies which employed the Trail-Making Test [TMT], Part B, can be found in Table 2.19). The three publications on ASPD included 75 individuals with the disorder and 64 controls. Barkataki et al. (2005) and Dolan and Park (2002) included forensic inpatient ASPD samples whereas all other group were from non-forensic sources. There were 16 publications on psychopathy with at least 428 individuals with the

disorder and 505 controls. All samples originated from prisons except that of Dinn and Harris (2000) and Ishikawa et al. (2001) who recruited from the general public.

Table 2.4. *Studies which employed paradigms of cognitive flexibility including attentional set shifting, decision-making, response reversal, and divergent thinking*

| Reference | Function | Test | Primary outcomes | Result favoured |
|---|---|---|---|---|
| ***ASPD*** | | | | |
| Barkataki 2005 | Attentional set shifting | WCST Computerised | Perseverative errors | ns |
| | | | Categories achieved | ns |
| Dolan 2002 | Attentional set shifting | IED | EDS errors | Control |
| | Response reversal | | Reversal errors | ns |
| Stevens 2003 | Attentional set shifting | WCST Non-computerised | Perseverative errors | ns |
| | | | Categories achieved | ns |
| ***Psychopathy*** | | | | |
| Blair, Morton 2006 | Decision-making | Differential Reward & Punishment Learning | Errors | Control |
| Blair, Newman 2006 | Response reversal | Object alternation | Errors | Control |
| | | Space alternation | Errors | ns |
| Budhani 2006 | Response reversal | Object alternation with varying reward ratios | Errors | Control |
| Dinn 2000 | Response reversal | Object alternation | Last error trial | Control |
| | Alternative uses | DTT | Number of responses | Antisocial |
| Goldstein 1998 | Attentional set shifting | WCST Computerised | Perseverative errors | ns |
| | | | Categories achieved | ns |

| | | | | |
|---|---|---|---|---|
| Ishikawa 2001 | Attentional set shifting | WCST Computerised | Perseverative errors | ns |
| | | | Categories achieved | Successful individuals with psychopathy only; ns overall |
| Lapierre 1995 | Attentional set shifting | WCST Non-computerised | Perseverative errors | ns |
| | | | Categories achieved | ns |
| Lösel 2004 | Decision-making | IGT | Disadvantageous responses | ns overall<br>Control (vs. low-attention antisocial) |
| Mercer 2005 | Attentional set shifting | WCST Computerised | Perseverative errors | Control |
| | | | Categories achieved | ns |
| Mitchell 2002 | Attentional set shifting | IED | EDS errors | ns |
| | Response reversal | | Reversal errors | Control |
| | Decision-making | IGT | Disadvantageous responses | Control |
| Mol 2009 | Attentional set shifting | WCST Computerised | Perseverative errors | ns |
| | | | Categories achieved | ns |
| Moltó 2007 | Decision-making | Card Playing | Number of cards played | Control |
| Newman 1987 | Decision-making | Card Playing | Number of cards played | Control (overall); ns when following a reflective delay |
| Pham 2003 | Attentional set | WCST | Perseverative | ns |

| | | | | |
|---|---|---|---|---|
| | shifting | Computerised | errors | |
| | | | Categories achieved | ns |
| Schmitt 1999 | Decision-making | IGT | Disadvantageous responses | ns |
| Swogger 2006 | Decision-making | Balloon Analogue Risk Task | Number of responses (adjusted) | ns |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; WCST=Wisconsin Card Sorting Test; IED=Intra/Extra-Dimensional Set shifting task; EDS=Extra-dimensional set shifting; DTT=Divergent Thinking Task; IGT=Iowa Gambling Task; ns=Not significant.

*2.3.3.2.2.1 Attentional set shifting*

ASPD. Of the three studies on ASPD, only Dolan and Park (2002) found a significant group difference in favour of the control group. A meta-analysis using strongest effects revealed a non-significant, medium pooled effect size estimate with substantial heterogeneity. Weakest effects produced comparable results.

Psychopathy. There were seven studies (D. S. Goldstein, 1998; Ishikawa, Raine, Lencz, Bihrle, & Lacasse, 2001; Lapierre et al., 1995; Mercer et al., 2005; Mitchell, Colledge, Leonard, & Blair, 2002; Mol, Van Den Bos, Derks, & Egger, 2009; Pham et al., 2003) including a total of 305 individuals with psychopathy and 363 without. Ishikawa et al. (2001) divided their community sample of individuals with psychopathy into successful and unsuccessful groups, the latter reflecting criminal history. Mercer et al. (2005) found that individuals with psychopathy committed more perseverative errors on the WCST. Ishikawa et al. reported that successful persons with psychopathy achieved more categories than the other groups on this task but there were no further significant group differences.

A meta-analysis was conducted using strongest effects (initially with data from unsuccessful individuals with psychopathy from Ishikawa et al., 2001). This resulted in a medium and significant pooled effect size estimate. Heterogeneity was considerable while study quality rating did not appear to affect distribution of data.

The major contributor to this heterogeneity appeared to be the study by Mercer et al. (2005), since removal of this dataset, resulted in a reduction to heterogeneity, with a significant small to medium pooled estimate, 0.39[0.17,0.62], $P<0.001$. Results were comparable with data by successful individuals with psychopathy from Ishikawa et al. (2001) instead of the unsuccessful group, except that the pooled estimate was not significant prior to removing the data of Mercer et al. Visual inspection of the funnel plot (Appendix D, Figure 12.1) showed Mercer et al. to be an outlier. The failsafe *N* of 140.2 exceeded the critical value of 45 suggesting robust results against publication bias.

With weakest effects (unsuccessful individuals with psychopathy from Ishikawa et al., 2001), the pooled effect estimate remained medium and significant, 0.45[0.09,0.81],$P<0.05$, with comparable heterogeneity. Removing the data of Mercer et al. (2005) had a similar effect as before. Although the failsafe *N* was smaller at 82.3, it exceeded the critical value once more.

*2.3.3.2.2.2 Decision-making*

All studies (K. Blair, Morton et al., 2006; Lösel & Schmucker, 2004; Mitchell et al., 2002; Moltó, Poy, Segarra, Pastor, & Montanes, 2007; Newman, Patterson, & Kosson, 1987; Schmitt, Brinkley, & Newman, 1999; Swogger, 2006) focused on psychopathy within offender samples and included at least 132 individuals with psychopathy and 153 without. Most studies suggested that controls performed better than the group with psychopathy. Lösel and Schmucker (2004) reported that controls performed significantly better than low-attention participants with psychopathy only. Swoger (2006) did not report significant group differences.

Data for a meta-analysis were available for four of the studies on decision-making. There was a non-significant, medium to large pooled effect size estimate with considerable heterogeneity, the latter mostly due to the Swogger's (2006) data on the relatively simple paradigm. Data from more complex decision making tasks produced a large and highly significant pooled effect size estimate with no heterogeneity. The failsafe *N* of 31.3 was above the critical value of 25, indicating robustness against publication bias. When the weakest effects (reflective condition) from Newman et al. (1987) were included instead, the pooled estimate for complex

decision-making was medium to large but did not reach significance, 0.61[-0.13,1.34], with considerable heterogeneity. With weakest effects, the overall meta-analysis yielded a small to medium but non-significant pooled effect size estimate, 0.35[-0.27,0.97], with substantial heterogeneity.

*2.3.3.2.2.3 Response reversal*

Of the five studies investigating this function, only one was on ASPD (Dolan & Park, 2002) whereas the remaining four focused on psychopathy (K. Blair, Newman et al., 2006; Budhani, Richell, & Blair, 2006; Dinn & Harris, 2000; Mitchell et al., 2002). Sample totals included at least 33 individuals with psychopathy and 31 without. Studies are presented in Table 2.4. Dolan and Park (2002) and Mitchell et al. (2006) employed the response reversal component of the IED from the CANTAB. Budhani et al. (2006), however, adopted a relatively sophisticated response reversal task, with probabilistic reinforcement. Dolan and Park did not find a significant effect for ASPD but the studies on psychopathy reported that controls outperformed individuals with psychopathy. Mitchell et al. reported significant effects in conditions where reversal took place on novel stimuli. On the other hand, reinforcement in space alternation shifted between two stimulus locations and K. Blair, Newman et al. (2006) did not find a significant effect in psychopathy.

A meta-analysis (psychopathy) with strongest effect from Mitchell et al. (2002), produced a large and significant pooled effect size estimate with substantial heterogeneity. The failsafe *N* of 13.77 was below the critical value of 20. With weakest effects, the pooled estimate was not significant,1.01[-1.07,3.09], and heterogeneity was considerable.

*2.3.3.2.2.4 Other studies on cognitive flexibility*

Dinn and Harris (2000) examined divergent thinking, and another six studies (Hart et al., 1990; Jozef & da Silva, 1999; Mercer et al., 2005; Pham et al., 2003; S. S. Smith et al., 1992; Stevens et al., 2003) investigated mental flexibility tasks with the TMT, Part B (because of alternating responses and moderate correlations with the

WCST[1]; Lezak et al., 2004). An overview of studies is shown in Table 2.4 (DTT) and Table 2.19 (TMT). Stevens et al. (2003) did not report any significant group differences for ASPD. The studies on psychopathy included over 241 individuals with psychopathy and over 290 individuals without. Statistical comparisons between groups were not conducted by all studies and only three reported significant group differences. Jozef and da Silva suggested that fewer participants with psychopathy demonstrated impaired performance on the TMT, Part B, compared to controls while S. S. Smith et al. indicated that LA controls performed better than individuals with psychopathy. Dinn and Harris (2000), on the other hand, highlighted superior performance by those with psychopathy on the DTT.

A meta-analysis of the available data (strongest effects) revealed a non-significant overall pooled effect size estimate of small magnitude. For TMT, Part B the pooled estimate was medium but did not reach significance either. Heterogeneity was considerable and performance on the TMT, Part B and DTT tasks was significantly different compared to each other. Weakest effects resulted in a small and non-significant pooled effect size estimate, 0.03[-0.73,0.78], with comparable heterogeneity.

#### *2.3.3.2.2.5 Meta-analysis of cognitive flexibility tests*

Since the TMT taxes complex functions and not mental flexibility alone, other tasks were preferred for the same sample, where available. Response reversal data were not reported for ASPD and therefore a meta-analysis would not contribute anything further to the previous results on attentional set shifting. On the other hand, meta-analysis was possible with additional data for psychopathy.

Meta-analysis for psychopathy with strongest effects was conducted with sensitivity analysis for data associated with unsuccessful and successful individuals with psychopathy from Ishikawa et al. (2001). Strongest effects (and data for unsuccessful individuals with psychopathy from Ishikawa et al., 2001) resulted in a significant, medium to large pooled effect size estimate with considerable

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[1] A third test, the Category Test has been classed as an executive task previously (Morgan & Lilienfeld, 2000). However, it appears to be more of a measure of abstraction than mental flexibility (Lezak et al., 2004); thus, it was included in the section on concept formation.

heterogeneity. Low quality studies appeared to have yielded weaker and non-significant results compared to medium and high quality studies. However, study subgroup differences were not significant and study quality was not correlated with effect size, *rho*=0.35,*n*=12. For the overall pooled estimate, the failsafe *N* of 357.34 was above the critical value of 70, although the funnel plot did not appear symmetrical (Appendix D, Figure 12.1). This may be expected in light of considerable heterogeneity and results may be robust against publication bias on account of the high failsafe *N*. Results were comparable for successful individuals with psychopathy from Ishikawa et al.

With weakest effects and with data from unsuccessful individuals with psychopathy from Ishikawa et al. (2001), the pooled effect size estimate was small, 0.22[-0.13,0.58], and did not reach significance with comparable heterogeneity. Pooled effect estimates of study quality subgroups were also not significant. Results were comparable with data from successful individuals with psychopathy from Ishikawa et al. for either strongest or weakest effect meta-analyses.

#### *2.3.3.2.3 Motor regulation*

Seven publications on ASPD included 135 individuals with the diagnosis and 122 without. On the other hand, there were 27 publications and dissertations on psychopathy including at least 517 individuals meeting criteria for psychopathy and 560 controls.

##### *2.3.3.2.3.1 Response alternation*

There was only one study, on ASPD (Stevens et al., 2003; Table 2.29) reporting no significant differences between groups.

##### *2.3.3.2.3.2 Response inhibition*

Response delay. There were only two studies in this category (Newman, Kosson, & Patterson, 1992; Swann, Lijffijt, Lane, Steinberg, & Moeller, 2009). An overview of studies is presented in Table 2.5. Swann et al. (2009) focused on ASPD while Newman et al. (1992) examined psychopathy. They were no significant performance differences between groups in either study.

Go/NoGo paradigms. An overview of the studies is presented in Table 2.5. Four studies examined Go/NoGo operations in ASPD and included a minimum of 68 individuals with the disorder and 59 controls (Barkataki et al., 2008; Dolan & Park, 2002; Howard, Payamal, & Neo, 1997; Völlm et al., 2010). Most studies recruited the ASPD group from forensic psychiatric samples except Howard et al. (1997; all samples were from prisons). Only two studies reported a significant performance difference between groups (Barkataki et al. 2008; Dolan & Park, 2002), both in favour of the control group. Although Dolan and Park only reported a trend for commission errors ($P$=0.05), there was a significant difference on probability of inhibition. These results do not seem to have been affected by the different manipulations in the tasks while offending may have been a confounder.

A meta-analysis of the available data on ASPD (strongest effects) produced a significant medium pooled effect size estimate with relatively inconsequential heterogeneity. The failsafe $N$ of 6.72 was below the critical cut-off of 25, indicating susceptibility to publication bias. Using weakest effects (second task from Völlm et

al., 2010) resulted in a non-significant, medium pooled estimate, 0.38[-0.19,0.95], with substantial heterogeneity.

Thirteen publications examined Go/NoGo operations in psychopathy and included at least 327 individuals with this personality and 303 controls (Arnett, Howland, Smith, & Newman, 1993; R. Blair, Mitchell, Leonard et al., 2004; Dinn & Harris, 2000; D. S. Goldstein, 1998; Howard et al., 1997; Iria & Barbosa, 2009; Kiehl, Smith, Hare, & Liddle, 2000; Kosson et al., 1990; Lapierre et al., 1995; Newman, Patterson, Howland, & Nichols, 1990; Newman & Schmitt, 1998; A. M. Smith, 1999; Swogger, 2006). Most studies recruited from prison settings except Dinn and Harris (general public), Iria and Barbosa (offenders and general public and A. M. Smith who included a second control group from the general public..

Results in favour of the control group were reported by R. Blair et al. (2004), Lapierre et al. (1995) and in the studies employing response dominance but in possibly overlapping samples (Newman et al., 1990, first study only; Newman & Schmitt, 1998, Caucasian sample only). The variable reward/punishment association manipulation of R. Blair et al. (2004) did not seem to affect the performance of the group with psychopathy as much as the control group, who performed worse for lower levels of punishment. A meta-analysis of Go/NoGo commission errors in psychopathy was conducted. There was a non-significant, medium pooled effect size estimate, with considerable overall heterogeneity particularly among studies associated with medium and low quality ratings**Error! Reference source not found.**.

Stop-signal paradigms. Both publications employing this paradigm (Arnett, Smith, & Newman, 1997; Drugge, 1998) were on psychopathy with a total of 81 offenders with psychopathy and 66 without. Although no overall deficit in response inhibition was highlighted in any study, Arnett et al. (1997) reported that the LA control group made more commission errors than LA individuals with psychopathy in the second task. They also observed that participants with psychopathy were faster than controls during the reward-only phase and that LA individuals of those without psychopathy showed some additional evidence of increased sensitivity to rewards.

A meta-analysis of the available data produced a non-significant and small pooled effect size estimate with marginally significant moderate heterogeneity. Heterogeneity was likely introduced by the different patterns of LA and HA groups

with psychopathy which produced effects in opposite directions. Arnett et al. (1997) reported that their samples did not overlap**Error! Reference source not found.**.

Table 2.5. *Studies which investigated response inhibition (non-Stroop paradigms)*

| Reference | Task | Go/NoGo stimuli | Task features | Primary outcomes | Result favoured |
|---|---|---|---|---|---|
| ***ASPD*** | | | | | |
| Barkataki 2008 | Go/NoGo | 1/1 | No stimulus learning<br>Response dominance: Go only, 40% NoGo and 20% NoGo conditions<br>No Go/NoGo reversal | Commission errors | Control |
| Dolan 2002 | Go/NoGo | 1/1 | No stimulus learning<br>No response dominance<br>No Go/NoGo reversal | Commission errors<br>Response latency<br>Probability of inhibition | ns<br>ns<br>Control |
| Howard 1997 | Go/NoGo | 2/2 | Cued Go/NoGo task<br>Response dominane: Initial Go only block, then 20% NoGo frequency<br>Go/NoGo reversal | Commission errors<br>Response latency | ns<br>n/a |
| Swann 2009 | Response delay: Single key | | Delayed response | Total responses<br>Shortest delay<br>Longest delay | ns<br>ns<br>ns |
| | Response delay: Two-key | | Forced-choice delayed response | % Immediate response | ns |

| | | | | | |
|---|---|---|---|---|---|
| | | | | Max consecutive delayed responses | ns |
| Völlm 2010 | Go/NoGo | 1st Task: Many/1 | No stimulus learning<br>Response dominance: Go and Go/NoGo blocks interlaced<br>No Go/NoGo reversal | Commission errors | ns |
| | | 2nd Task: 2/Many | No stimulus learning<br>No response dominance<br>Special manipulation: Two Go stimuli, one associated with reward while the other did not. Trial blocks contained either but never both, alternating<br>No Go/NoGo reversal | Commission errors | ns |
| ***Psychopathy*** | | | | | |
| Arnett 1993 | Go/NoGo (PA) | 4/4 | Stimulus learning<br>No response dominance<br>No Go/NoGo reversal | Commission errors | ns |
| Arnett 1997 - Exp. 1 | SST visual analogue | | Response dominance: Interlaced reward-only phase (no stop-signal) | Commission errors | ns |
| | | | | Response latency | ns overall (greater sensitivity to reward among individuals with psychopathy in some instances) |

| | | | | | |
|---|---|---|---|---|---|
| - Exp. 2 | SST visual analogue | | Active avoidance | Commission errors<br>Response latency | LA antisocial/ ns overall<br>ns |
| Blair, Mitchell, Leonard 2004 | Go/NoGo (PA) | 4/4 | Stimulus learning<br>No response dominance<br>Special manipulation: Graded reward and punishment for Go & NoGo stimuli respectively.<br>No Go/NoGo reversal | Commission errors | Control |
| Dinn 2000 | Go/NoGo | 1/1 | No stimulus learning<br>Response dominance: Initial Go only block<br>Go/NoGo reversal | Response latency | ns |
| Drugge 1998 | SST | | Variable stop-signal delay | Stop-signal reaction time | ns |
| Goldstein 1998 | Go/NoGo | Many/1 | No stimulus learning<br>Response dominance: 30% NoGo target frequency<br>No Go/NoGo reversal | Commission errors | ns |
| Howard 1997 | Go/NoGo | 2/2 | Cued Go/NoGo task<br>Response dominance: Initial Go only set, then 20% NoGo frequency | Commission errors<br>Response latency | ns[a]<br>ns |
| Iria 2009 | Go/NoGo | 1/Many | No stimulus learning<br>No response dominance | Commission errors | ns |

| | | | | | |
|---|---|---|---|---|---|
| | | | No Go/NoGo reversal | | |
| Kiehl 2000 | Go/NoGo | 1/1 | No stimulus learning<br>No response dominance<br>Go/NoGo reversal | Commission errors<br>Response latency | ns<br>ns |
| Kosson 1990 | Go/NoGo (PA) | 4/4 | Stimulus learning<br>No response dominance<br>Special manipulation: Reward & Punishment/Punishment only conditions<br>No Go/NoGo reversal | Commission errors | ns |
| Lapierre 1995 | Go/NoGo | 1/1 | No stimulus learning<br>Response dominance: Initial Go only block<br>Go/NoGo reversal | Commission errors<br>Response latency | Control<br>ns |
| Newman 1992 | Gratification Delay | | Dual stimulus: Immediate infrequent or delayed frequent rewards<br>Three conditions: Reward only, reward & punishment, control | % Delayed response/ Earnings | n/a |
| Newman 1990 - Study 1 | Go/NoGo (PA) | 4/4 | Stimulus learning<br>Response dominance: Initial block with higher Go ratio<br>No reinforcement association reversal | Commission errors<br>Reflection on feedback prior to next trial | Control<br>ns |

| | | | | | |
|---|---|---|---|---|---|
| - Study 2 | Go/NoGo (PA) | 4/4 | Stimulus learning<br><br>No response dominance<br>Special manipulation: Go/NoGo stimuli were always paired with a neutral stimulus<br>No Go/NoGo reversal | Commission errors<br><br>Reflectivity | ns<br><br>ns |
| Newman 1998 | Go/NoGo (PA) | 5/5 | Stimulus learning<br><br>Response dominance: Initial block with higher Go ratio<br>Go/NoGo reversal | Commission errors | Control (Caucasians only) |
| Smith 1999 | Go/NoGo | 1/Many (& reversed) | No stimulus learning<br><br>No response dominance<br>Go/NoGo reversal | Commission errors<br><br>Response latency | ns<br><br>ns |
| Swogger 2006 | Go/NoGo (PA) | 5/5 | Stimulus learning<br><br>No response dominance<br>No Go/NoGo reversal | Commission errors | ns |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; PA=Passive avoidance; SST=Stop-Signal Task; LA=Low-anxious; n/a=Not available; ns=Not significant.

[a] The authors mentioned a post hoc analysis with the more traditional 12/18 PCL:SV cut-offs. They claim that individuals with psychopathy performed better at not responding to the imperative stimulus in NoGo trials in this arrangement, however, details of statistical analysis were not provided.

Stroop paradigms. An overview of studies is provided in Table 2.6. There were eleven publications of which one focused on ASPD (Barkataki et al., 2005) while the others investigated psychopathy (K. Blair, Newman et al., 2006; Brinkley, Schmitt, & Newman, 2005; Dinn & Harris, 2000; Drugge, 1998; Dvorak-Bertsch, Sadeh, Glass, Thornton, & Newman, 2007; Harpur, 1991; Hiatt, Schmitt, & Newman, 2004; Mercer et al., 2005; Pham et al., 2003; Schmitt, 2000; S. S. Smith et al., 1992). Schmitt's (2000) contribution was a doctoral dissertation and its data on Caucasian participants were included later in the publications by Hiatt et al. (2004) and Brinkley et al. (2005) with some adjustments in analysis. Thus, only data in connection to the African-American sample were taken into account from Schmitt. Except for Dinn and Harris' (2000) samples, which originated from the general public, participants in the other studies were offenders. In total, samples included over 260 individuals with psychopathy and over 287 without.

In terms of the Colour-word Stroop task, Mercer et al. (2005) reported that the control group performed better than individuals with psychopathy. Pham et al. (2003) also found that the group with psychopathy performed significantly worse than controls, but their analysis was across all conditions and thus may reflect an overall performance difference. Dinn and Harris (2000) and Drugge (1998) did not report on error rates. Regarding Hiatt et al. (2004) and Schmitt (2000) who used the Box Stroop, although the Caucasian samples of the former did not produce any significant differences, Schmitt's African-American sample yielded a significant interaction according to which LA controls and HA individuals with psychopathy appeared to perform better than their counterparts. Post hoc comparisons were not reported. During the semantic Stroop task employed by Brinkley et al. (2005) and by Schmitt (2000), no significant differences between individuals with and without psychopathy were found. During the Picture-word Stroop, Hiatt et al. reported better performance for LA individuals with psychopathy but no significant differences between those with and without psychopathy were observed overall. Finally, K. Blair, Newman et al. (2006) employed two Number Stroop tasks but the groups with and without psychopathy performed with comparable accuracy.

Stroop task data for meta-analysis were available for a few of studies only, thus any results are likely to be unrepresentative. The data of Hiatt et al.'s (2004)

colour-word task were preferred over Dvorak-Bertsch et al.'s (2007) as they involved a larger sample in a purer format of the task. The meta-analysis resulted in a non-significant, small pooled effect size estimate in favour of controls with moderate heterogeneity. Analyses with either strongest or weakest effects (due to K. Blair, Newman et al., 2006 reporting data for separate task conditions) produced comparable results. Without the number Stroop tasks, leaving the more traditional Stroop paradigms in the analysis, there was no heterogeneity and the pooled estimate became significant and of moderate magnitude, 0.49[0.08,0.89],$P<0.05$. The failsafe $N$ of 2.5 was below the critical value of 20, thus the result may be susceptible to publication bias.

Table 2.6. *Studies which employed Stroop paradigms on response inhibition, effective performance and selective attention*

| Reference | Task | Task features | Primary outcomes | Result favoured |
|---|---|---|---|---|
| ***ASPD*** | | | | |
| Barkataki 2005 | CW Stroop | | Accuracy | ns |
| | | | Processing speed | ns |
| ***Psychopathy*** | | | | |
| Blair, Newman 2006 | Number counting Stroop | Baseline: Congruent trials | Correct response latency | Control, across conditions |
| | | Congruent & incongruent trials | Errors | ns |
| | | | Interference | ns (ANOVA interaction) |
| | Number reading Stroop | Baseline: Congruent trials | Correct response latency | Control, across conditions |
| | | Congruent & incongruent trials | Errors | ns |
| | | | Interference | Antisocial? (ANOVA interaction) |
| Brinkley 2005 | Semantic Stroop | Baseline: Letter strings | Interference (CW) | ns (test on LA groups only) |
| | | Congruent & incongruent trials | Interference (CRW) | ns (test on LA groups only) |
| | | | Facilitation (CW trials) | Antisocial[a] |
| | | | Facilitation (CRW trials) | ns |
| | | | Errors (CW interference trials) | ns |
| | | | Errors (CRW interference trials) | ns |
| | | | Errors (CW facilitation trials) | Antisocial[a, b] |
| | | | Errors (CRW facilitation trials) | Control[a, b] |

| | | | | |
|---|---|---|---|---|
| Dinn 2000 | CW Stroop | Congruent & incongruent trials | Response latency | Control |
| Drugge 1998 | CW Stroop with negative priming | Baseline: Non-letter strings | Interference | ns |
| | | Negative priming | Response latency | ns |
| Dvorak-Bertsch 2007 | CW Stroop with frequency variations | Baseline: Congruent trials | Interference | ns |
| | | Special frequency manipulation | | ns |
| | | | Correct response latency | ns |
| | | | Errors (interference condition) | ns |
| Harpur 1991 - Exp. 3 | CW Stroop with habituation and negative priming | Negative priming | Correct response latency | ns |
| | | Habituation | Accuracy (interference condition) | ns |
| Hiatt 2004 - Exp. 1 | CW Stroop | Baseline: Coloured letter strings | Interference | ns |
| | | | Response latency | n/a |
| | | | Errors (interference condition) | ns |
| - Exp. 2 | Picture-word Stroop | Baseline: Pictures with superimposed non-word trigrams | Interference | LA antisocial, ns overall |
| | | | Response latency | n/a |
| | | | Errors (interference | ns |

| | | | | |
|---|---|---|---|---|
| | | | condition) | |
| - Exp. 3 | Box Stroop | Baseline: Coloured boxes containing letter strings Congruent & incongruent trials | Interference | LA antisocial |
| | | | Facilitation | ns |
| | | | Response latency | n/a |
| | | | Errors | ns |
| Mercer 2005 | CW Stroop | | Accuracy | Control |
| Pham 2003 | CW Stroop | | Corrected errors | ns |
| | | | Uncorrected errors | ns |
| | | | Total errors | Control (main effect, across all conditions) |
| | | | Completion time | ns |
| | | | Interference | ns |
| | | | Corrected errors (interference) | ns |
| | | | Uncorrected errors (interference) | ns |
| Schmitt 2000 - African American sample only - Exp. 1 | Picture-word Stroop | Baseline: Pictures with superimposed non-word trigrams | Interference | ns (test on LA groups only) |
| | | | Response latency | ns (test on LA groups only) |
| | | | Errors (interference trials) | ns |
| - Exp. 2 | CW Stroop | Baseline: Coloured letter strings | Interference | ns |

| | | | | |
|---|---|---|---|---|
| | | | Errors (interference trials) | ns |
| - Exp. 3 | Semantic Stroop | Baseline: Letter strings<br>Congruent & incongruent trials | Interference (CW) | ns (test on LA groups only) |
| | | | Interference (CRW) | ns (test on LA groups only) |
| | | | Facilitation (CW trials) | ns (test on LA groups only) |
| | | | Facilitation (CRW trials) | ns (test on LA groups only) |
| | | | Errors (interference trials) | ns |
| | | | Errors (facilitation trials) | ns |
| - Exp. 4 | Box Stroop | Baseline: Coloured boxes containing letter strings<br>Congruent & incongruent trials | Interference | ns |
| | | | Facilitation | ns |
| | | | Errors (interference) | LA control & HA antisocial (interaction only) |
| | | | Errors (facilitation) | ns |
| Smith 1992 | CW Stroop | | Completion time | ns |
| | | | Errors (interference trials) | ns |
| | | | Response latency | ns |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; CW=Colour-word; CRW=Colour-related word; LA=Low-anxious; n/a=Not available; ns=Not significant.

[a] from Schmitt (2000) - Exp. 3; [b] interaction with no post hoc comparisons.

Meta-analysis on response inhibition tests. As outlined in the method, available data from studies with the largest samples were preferred but where more than one set of data was available within a study, sensitivity analysis was conducted. However, because Stroop tasks also involve functions other than response inhibition, data from other response inhibition tasks were preferred, where available, for greater validity.

For ASPD, adding response delay data from Swann et al. (2009) to the previous Go/NoGo meta-analysis (strongest effects) resulted in a significant small to medium pooled effect size estimate in favour of controls with relatively inconsequential heterogeneity**Error! Reference source not found.**. The failsafe *N* of 9.35 was below the critical value of 30 indicating possible publication bias. Using the weakest effects, there was a marginally significant small to medium pooled estimate, 0.30[-0.01,0.60], *P*=0.05, with non-significant heterogeneity. Swann et al. was the only study with a medium quality rating while the others received a high rating. Nevertheless, quality rating did not appear to be associated with effect size.

For psychopathy, there was a significant small to medium pooled effect size estimate in favour of controls but with substantial heterogeneity, mostly due to the contribution of Lapierre et al. (1995). For the overall result, the failsafe *N* of 65.60 was below the critical value of 80, with asymmetry in the funnel plot originating from the outlier data of Lapierre et al. (Appendix D, Figure 12.3). Thus, the result may be subject to publication bias. Removal of Lapierre et al. resulted in a small pooled estimate, 0.21[-0.03,0.45], which approached significance, *P*=0.08, with moderate heterogeneity. Studies associated with high quality ratings yielded a small pooled estimate as opposed to the large pooled estimate of studies associated with medium ratings. However, study quality and age difference were not correlated with effect size, *rho*=-0.34,*n*=14 , and *rho*=0.34,*n*=12, respectively.

*Meta-analysis on motor regulation.* Due to sample overlaps, a meta-analysis on psychopathy could not include further studies from response inhibition above. Thus, a meta-analysis on motor regulation was conducted for ASPD only. With strongest effects, the result was a significant, medium pooled effect size estimate with no heterogeneity. The failsafe *N* of 15.79 was below the critical cut-off of 35. In

addition, the funnel plot was relatively asymmetrical (Appendix D, Figure 12.2). Thus, the result may be susceptible to publication bias. With weakest effects, the pooled estimate was also significant, of small to medium magnitude, 0.28[0.02,0.54],*P*<0.05, with non-significant heterogeneity. The failsafe *N* of 5.46 was also below the critical cut-off of 35. In addition, the funnel plot was relatively asymmetrical (Appendix D, Figure 12.2). Thus, this result may be susceptible to publication bias also. Quality rating did not appear to influence the result with Swann et al. (2009) being the only study to have received a medium rating.

#### *2.3.3.2.4 Meta-analysis on self-regulation*

Because Stroop and TMT tasks implicate functions other than self-regulation to a large degree, other task data were preferred where available, for greater validity.

##### *2.3.3.2.4.1 ASPD*

Strongest effects resulted in a significant medium pooled effect size estimate with non-significant heterogeneity. The failsafe *N* of 22.61 was below the critical value of 35. In addition, the funnel plot was relatively asymmetrical (Appendix D, Figure 12.4), thus publication bias was possible. With weakest effects, the pooled estimate was small and non-significant, 0.21[-0.15,0.56], while heterogeneity did not reach significance. Study quality did not appear to affect study results, with Swann et al. (2009) the only study to have received a medium rating.

##### *2.3.3.2.4.2 Psychopathy*

With strongest effects, there was a significant medium pooled effect size estimate in favour of controls with considerable heterogeneity. The failsafe *N* of 485.66 exceeded the critical value of 110 and suggested a robust result. However, the funnel plot appeared asymmetrical primarily due to the outlier data of Dinn and Harris (2000), Lapierre et al. (1995) and Mercer et al. (2005) (Appendix D, Figure 12.4). Removing these outliers, which also contributed the strongest effects, the result was a small yet significant pooled estimate, 0.29[0.11,0.47],*P*<0.01, with non-significant heterogeneity. With weakest effects, the resulting small pooled effect size estimate was not significant, 0.09[-0.13,0.32], with substantial heterogeneity. Studies

associated with high or low quality yielded a smaller estimate than studies of medium quality. However, there was no correlation between effect size and study quality, *rho*=-0.13,*n*=20. Age was not correlated with effect sizes either, *rho*=0.28,*n*=15.

#### 2.3.3.3 Effective performance

An overview of studies is provided in Table 2.6 and Table 2.7. In the relevant paradigms, error rates/accuracy and interference (but not facilitation) from incongruent stimuli may reflect conflict resolution processes, albeit in synergy with selective attention (Gazzaniga et al., 2009; Lezak et al., 2004). One study focused on ASPD (Barkataki et al., 2005). There was also one study on DPD in conjunction with psychopathy (Müller et al., 2008; Weber et al., 2004). A total of 18 publications and dissertations on the latter totalled at least 332 individuals with psychopathy and 432 without, recruited mostly from forensic samples. Exceptions were Brazil et al. (2009) who sought their control group among hospital staff, and Dinn and Harris (2000) who recruited both samples from the general public.

##### *2.3.3.3.1 Stroop paradigms*

Regarding the colour-word Stroop, Mercer et al. (2005) and Pham et al. (2003) reported that controls showed better accuracy than the group with psychopathy, the latter conducting their analysis across all conditions. Dinn and Harris (2000) indicated that controls responded faster than individuals with psychopathy in the interference condition. Regarding colour-word interference, no study suggested a significant group difference. Barkataki and colleagues (2005) were the only study investigating ASPD and did not highlight a significant effect.

Regarding the Box Stroop paradigm, Hiatt et al. (2004) reported that LA individuals with psychopathy displayed less interference than controls (the effect approached significance for psychopathy overall). Schmitt (2000) reported than LA individuals with psychopathy and HA controls made more errors in an interaction effect but without post hoc analysis.

Regarding the Semantic Stroop task, its colour-word components revealed group differences. However, groups performed comparably in the semantic trials (colour-related words) as reported by Brinkley et al. (2005) and Schmitt (2000) – with analyses on interference on LA groups only.

The Picture-word variation resulted in no significant group differences on error rates or response latency (only Schmitt conducted statistical analyses here). Hiatt et

al., however, suggested less interference in the LA group with psychopathy compared to LA controls while there were no significant differences overall.

K. Blair, Newman et al. (2006), with the Number Stroop task, did not find any significant group differences in error rates but controls were faster to respond across all conditions in both tasks. There was, however, a significant group x task interaction for interference indicating that individuals with psychopathy may have experienced less interference in the reading task. No post hoc analyses were reported.

*2.3.3.3.1.1 Meta-analysis*

The meta-analysis of error data in the previous section revealed a significant medium pooled effect size estimate in favour of controls when colour-word data were taken into account. However, the result was not significant when the number Stroop data were also included. A meta-analysis with effects for response latency/completion times during the experimental condition produced a significant, medium to large pooled effect size estimate with non-significant heterogeneity. The failsafe *N* of 18.86 was below the critical value of 30, thus results may be subject to publication bias. Only overall task data were available from K. Blair, Newman et al. (2006) and the results were comparable without this study. Conducting a meta-analysis with baseline data produced results comparable to those of the experimental condition **Error! Reference source not found.**, thus the slower response times in psychopathy are likely to be a general occurrence rather than due to a deficit in conflict resolution. Dinn and Harris (2002) provided the strongest effect and this was the only study associated with a high quality rating in the group (others were of medium quality). The pooled effect size estimate for interference was small and it did not reach significance, with no heterogeneity. This was also unrepresentative due to few studies having reported data for meta-analysis.

***2.3.3.3.2 Non-Stroop paradigms***

There were nine publications in this group, all on psychopathy (Brazil et al., 2009; Harpur, 1991; Howland, Kosson, Patterson, & Newman, 1993; Mayer, Kosson, & Bedrick, 2006; Müller et al., 2008; Newman, Schmitt, & Voss, 1997; Weber et al.,

2004; Zeier, Maxwell, & Newman, 2009) with Müller et al. (2008)/Weber et al. (2004) also examining DPD. The samples included over 130 individuals with psychopathy and over 137 without. Müller/Weber et al. and Brazil et al. (2009) recruited antisocial samples from forensic psychiatric facilities while controls were not offenders (although this remains unclear for the former study), thereby introducing confounding. All other participants were recruited from prisons.

Table 2.7. *Studies which examined effective performance (conflict resolution) and selective attention with non-Stroop paradigms*

| Reference | Function | Task | Special features | Modality | Primary outcomes | Result favoured |
|---|---|---|---|---|---|---|
| ***DPD & psychopathy*** | | | | | | |
| Müller et al 2008/ Weber 2004 | Conflict resolution | Simon task | Affective stimuli: positive, negative and neutral | Visual (pictures) | Response latency | ns |
| | | | | | Errors | ns |
| ***Psychopathy*** | | | | | | |
| Brazil 2009 | Conflict resolution & selective attention | Eriksen flanker task | Active error monitoring | Visual (symbol strings) | Response latency | Control |
| | | | | | Errors | ns |
| | | | | | Error signalling | Control |
| Drugge 1998 | Conflict resolution & selective attention | Flanker task: Word naming with priming | Affective: positive, negative & neutral | Visual (words) | Accuracy | ns |
| | | | | | Correct response latency | ns |
| Harpur 1991 - Exp. 1 | Conflict resolution & selective attention | Invalid response cue | Peripheral cueing | Visual | Response latency | ns |
| | | | Early & late SOAs | | Interference | ns |

| | | | | | | |
|---|---|---|---|---|---|---|
| - Exp. 2 | | Invalid response cue | Central & peripheral cueing | Visual | Response latency | ns |
| | | | Early & late SOAs | | Interference | Antisocial (late SOAs only) |
| - Exp. 4 | | Flanker-type with negative priming | Negative or no priming | Visual | Response latency | ns |
| | | | | | Accuracy | ns[a] |
| - Exp. 5 | | Flanker-type with negative priming | Negative or no priming | Visual | Response latency | ns |
| | | | Varied spatial separation of targets and distractors | | Accuracy | ns |
| Howland 1993 | Conflict resolution & selective attention | Invalid response cue | | Visual | Response latency | ns |
| | | | | | Errors | Control (invalidly cued left-sided imperative stimuli/ neutral cue right-sided imperative stimuli) |
| | | | | | Speed & accuracy score | ns |
| Mayer 2006 | Conflict resolution & selective attention | Task 1: Invalid response cue/flanker-type | Early, middle & late SOA | Auditory (words) | Response latency | Control (overall) |
| | | | | | Accuracy Interference | ns |

| | | | | | | |
|---|---|---|---|---|---|---|
| | | | Central cueing | Visual (words) | Response latency | ns |
| | | | Early, middle & late SOA | | Accuracy | ns |
| | | | | | Interference | |
| | Conflict resolution & selective attention | Task 2: Invalid response cue/flanker-type | Cross-modal | Auditory cue/ Visual distractor | Response latency | Control (overall) |
| | | | Early, middle & late SOA | | Accuracy | ns |
| | | | | | Interference | |
| | | | | Visual cue/ Auditory distractor | Response latency | ns |
| | | | | | Accuracy | ns |
| | | | | | Interference | |
| Mills 1995 - Exp. 2 | Selective attention | Dichotic listening tone identification | | Auditory | Accuracy | ns |
| Newman 1997 | Conflict resolution & selective attention | Picture-word interference | Short & long ISIs | Visual (words & pictures) | Interference | LA antisocial (Caucasian & combined samples) |
| | | | | | Accuracy | n/a |
| Zeier 2009 | Conflict resolution & selective attention | Flanker-type | Cued & un-cued trials | Visual (numbers & letters) | Accuracy | ns |
| | | | Central & peripheral cueing | | | |

| | | |
|---|---|---|
| | Response latency | ns |
| | Interference | LA individuals with psychopathy |
| | Facilitation | ns |

*Note.* Highlighted outcomes contributed to meta-analyses; DPD=Dissocial Personality Disorder; SOA=Stimulus onset asynchrony (target stimulus appeared after various delays); ISI=Interstimulus interval; LA=Low-anxious; n/a=Not available; ns= Not significant.

[a] Significant group x condition interaction for accuracy but distributions were heavily skewed and result author suggested it is likely due to outlier data.

Regarding invalid response cueing, all studies reported an effect but results appeared conflicting. Paired comparisons revealed less interference in the group with psychopathy for late SOAs in Harpur's (1991) second experiment. Controls appeared to make fewer errors in some conditions in Howland et al. (1993; including neutrally cued trials) while they responded faster overall in the first task of Mayer et al. (2006).

In terms of flanker tasks, Brazil et al. (2009) indicated that individuals with psychopathy were slower than controls overall with shorter latencies when responding incorrectly. Thus may suggest a possible speed-accuracy trade-off. This study was the only one to examine error monitoring and highlighted that individuals with psychopathy signalled fewer errors than controls. Of the remaining studies, only the LA group with psychopathy of Zeier et al. (2009) showed less interference than controls. There were no further significant group differences.

Of the remaining studies, Newman et al. (1997) found that interference between presented pictures and words was smaller for LA individuals with psychopathy although this did not generalise to the African-American participants. Müller et al. (2008)/Weber et al. (2004) did not find a significant performance differences between groups during the Simon task.

*2.3.3.3.2.4 Meta-analysis*

Mayer et al. (2006) reported data for task conditions individually. With strongest effects for response latencies during 'invalid'/ incongruent trials, there was a small and non-significant pooled effect size estimate with considerable heterogeneity. Results were comparable with weakest effects. However, results from the baseline condition were analogous to the experimental condition (**Error! Reference source not found.**), thus any effects are unlikely to be attributable to cognitive conflict per se. The two publications in the meta-analysis were of different quality with Zeier et al. (2009) to have received a higher rating.

Using available accuracy data, the pooled effect size estimate was very small and did not reach significance with little evidence of heterogeneity. Once again, the study of Zeier et al. (2009) was associated with a higher quality rating.

Meta-analyses were also conducted for available interference data. Two different sets of data were available for Zeier et al.'s (2009) LA groups (cued and uncued conditions). Using the cued condition data (more challenging), there was a significant medium pooled effect size estimate with non-significant heterogeneity. Some asymmetry was present in the funnel plot (Appendix D, Figure 12.5) and the failsafe *N* of 11.35 was below the critical cut-off of 40, suggesting possible publication bias. When uncued condition data were included instead, the pooled effect size estimate was smaller, -0.27[-0.78,0.24], and did not reach significance with substantial heterogeneity.

#### *2.3.3.3.3 Meta-analysis on effective performance*

Only six studies provided overall error/accuracy data. The small pooled effect size estimate did not reach significance and there was substantial heterogeneity with strongest effects. The results were comparable with weakest effects.

A further two studies (non-Stroop) provided response latency data in addition to the earlier meta-analysis on Stroop tasks. There was a medium pooled effect size estimate in favour of controls which approached significance but with substantial heterogeneity. Results were comparable by using either the strongest or weakest data from Mayer et al. (2006). They were also very similar to the results following meta-analysis of baseline response latency data. Studies associated with higher quality ratings produced more heterogeneous results.

For interference, there were additional data from Zeier et al. (2009) in connection to cued and uncued trials. The pooled effect size estimate was small and did not reach significance when cued trial data were used. Heterogeneity was moderate. The result was a small and non-significant pooled effect size estimate, -0.05[-0.44,0.34], with uncued trial data (this time in the opposite direction) with moderate heterogeneity.

#### 2.3.3.4 Meta-analysis on executive functions

Because Stroop and TMT tasks implicate functions other than self-regulation to a large degree, other task data were preferred for greater validity, where available.

##### *2.3.3.4.1 ASPD*

Strongest effects resulted in a significant small to medium pooled effect size estimate and non-significant heterogeneity. The failsafe *N* of 11.94 was below the critical cut-off of 35. In addition, the funnel plot was relatively asymmetrical (Appendix D, Figure 12.6), suggesting possible publication bias. Weakest effects resulted in a small and non-significant pooled estimate, 0.09[-0.17, 0.35], with no heterogeneity. Swann et al. (2009) was the only study to have received with a lower (medium) quality rating.

##### *2.3.3.4.2 Psychopathy*

Strongest effects resulted in a significant, medium pooled effect size estimate in favour of controls with substantial heterogeneity. The failsafe *N* of 500.16 exceeded the critical cut-off of 115 but with an asymmetrical funnel plot (Appendix D, Figure 12.6). By removing the three outlier sets of data (Dinn & Harris, 2000; Lapierre et al., 1995; Mercer et al., 2005), the pooled estimate became small but remained significant, 0.28[0.09,0.48],$P<0.01$ while heterogeneity became moderate. On the other hand, weakest effects resulted in a small pooled effect size estimate which did not reach significance, 0.04[-0.18,0.27], with substantial heterogeneity.

Stratification revealed subgroup differences and studies associated with higher quality produced a small and non-significant pooled estimate. However, study quality was not correlated with effect size significantly, *rho*=-0.21,*n*=21. There was no significant correlation with age either, *rho*=0.19,*n*=15.

#### 2.3.3.5 Summary

##### *2.3.3.5.1 ASPD & DPD*

No impairment in effective performance emerged in the only study on DPD. On the other hand, ASPD was associated with general as well as more localised executive deficits. Overall meta-analyses provided evidence supporting an executive deficit using the strongest effects only. Possible presence of publication bias was also detected. Although heterogeneity was relatively inconsequential overall, results varied across executive functions of planning, self-regulation and effective performance.

No deficits were identified in the very few studies on WM and effective performance. However, individuals with ASPD demonstrated a moderate planning deficit (some publication bias) especially during more challenging conditions. Evidence of medium impairment in overall self-regulation was also present in a meta-analysis of strongest effects only whereas the result approached significance with weakest effects. Publication bias may have been present.

Within self-regulation, evidence indicated impairment in motor regulation but it was limited for cognitive flexibility whereas individuals with ASPD performed comparably to controls on productivity (one study only). Deficits in flexibility may not be general (meta-analysis) and may be more specific to the operations associated with the IED attentional set shifting task but not the WSCT, response reversal or the TMT, Part B. No studies investigated decision-making in ASPD.

Regarding motor regulation, an overall deficit was supported by meta-analyses but publications bias may have influenced this. There was some evidence of more focal ASPD impairment in response inhibition and Go/NoGo operations, again with possible presence of publication bias. There was no evidence of impairment in alternating responses and response delay functions.

In sum, a review of the literature provided some support suggesting an overall executive deficit in ASPD. Evidence was supportive of a moderate difficulty in planning and overall self-regulation. Within self-regulation, evidence was strongest for motor regulation operations (particularly within response inhibition) while it was limited for cognitive flexibility. Publication bias was detected in these results. Findings did not support impairments in WM and effective performance whereas no

studies examined decision-making in ASPD. The only study on DPD (effective performance) did not suggest a deficit.

#### *2.3.3.5.2 Psychopathy*

Overall meta-analyses supported an executive deficit in psychopathy. This was relatively robust for strongest effects whereas the pooled effect size estimate for weakest effects did not reach significance. The study by Mercer et al. (2005) was often an outlier and this was taken into consideration in sensitivity analyses. Although evidence provided some support for an overall deficit, results varied across the spectrum of executive functions. Most studies focused on self-regulation and effective performance compared to planning. In the latter, deficits may be present during operations specific to planning and in more challenging conditions while a deficit was not supported in tasks involving mental tracking and therefore taxing WM more heavily.

Within the executive functions, strongest effects suggested impairment in self-regulation but studies of high and low quality appeared to contribute more conservative results than studies of medium quality. Within self-regulation, there was robust support of a deficit in cognitive flexibility with strongest effects but not weakest effects. However, the performance of individuals with psychopathy on the DTT and TMT, Part B, did not appear impaired but these tasks also involve non-executive processes. Deficits spanned across the areas of attentional set shifting (may not generalise to non-offending samples), decision-making (more complex tasks) and response reversal (object alternation only). Samples were mostly from offender populations thereby limiting generalisation of results. Individuals with psychopathy performed better than controls in the only study on attentional set shifting in which samples were recruited from the community.

Apart from the aspects of self-regulation relevant to cognitive flexibility, the available evidence indicated a possible impairment in motor regulation whereas it did not reveal any impairment in productivity (verbal fluency). The support for motor regulation came from a variety of paradigms with greater focus on response inhibition. However, there was a possible effect of publication bias and studies of greater quality

contributed smaller effects. Response inhibition included the Go/NoGo and Stroop paradigms whereas the SST featured less often. Although a deficit in overall motor regulation and response inhibition may be present in psychopathy, meta-analyses for each task type did not generally produce significant pooled estimates. Although this could result from low power, it could also suggest a subtler deficit in these cognitive operations. This was supported by the patterns of result from Go/NoGo and SST paradigms where group differences generally arose in more complex manipulations. Out of the Stroop paradigms, some but limited support for a deficit in psychopathy emerged by including more traditional task formats. Finally, there were instances where LA individuals with psychopathy performed generally better in the SST and Stroop tasks.

Although some deficits were likely in planning and self-regulation, results were less conclusive for effective performance. Relevant paradigms primarily included the Stroop, invalid response cueing and flanker tasks. Meta-analyses did not reveal significant group differences. Contradictions between results were also present involving less interference but increased errors in psychopathy.

In sum, evidence using strongest effects suggested an overall executive deficit and this appeared robust. There was some support for a deficit in planning and overall self-regulation where the strongest evidence highlighted difficulties in cognitive flexibility particularly in “purer” tasks. Research on motor regulation placed much focus on response inhibition and provided some support of a difficulty in psychopathy but publication bias was detected. Regarding effective performance, findings were weak and inconsistent. No impairment was highlighted in studies examining WM and productivity. In many cases results from the executive functions may not generalise from offenders to the general population.

### 2.3.4 Abstraction

An overview of abstraction studies is provided in Table 2.8 whereas studies on attentional set shifting are presented in Table 2.4. Across five studies, there were 92 individuals with ASPD and 84 without. Four studies recruited individuals with ASPD from forensic samples with non-offending controls (Shamay-Tsoory, Harari, Aharon-Peretz, & Levkovitz, 2010, do not state the source of their control group), thus introducing confounding of criminality. Stevens et al. (2003) recruited all groups from the general public.

There were also 15 publications on psychopathy with at least 534 individuals with psychopathy and 803 without. Although most samples originated from prison populations, Dinn and Harris (2000) and Ishikawa et al. (2001) recruited from the general public. R. Blair's (1995) samples originated from forensic psychiatric settings, while Hare and Jutai (1988) and Kiehl, Smith et al. (2004) recruited control participants from the general public.

Table 2.8. *Studies which examined abstraction in ASPD and psychopathy*

| Reference | Function | Test | Primary outcome | Result favoured |
|---|---|---|---|---|
| ***ASPD*** | | | | |
| Shamay-Tsoory 2010 | Concept formation (verbal) | Similarities (WAIS-R) | Performance score | ns |
| Stevens 2003 | Concept formation (verbal) | Similarities (WAIS-R) | Performance score | Control |
| | Reasoning (visual stimuli) | Picture Arrangement (WAIS-R) | Performance score | ns |
| ***Psychopathy*** | | | | |
| Blair 1995 | Concept formation (visual) | Advanced Matrices | Performance score | ns |
| Blair 2002 | Concept formation (visual) | Advanced Matrices | Performance score | ns |
| Blair, Morton 2006 | Concept formation (visual) | Advanced Matrices | Performance score | ns |
| Blair, Newman 2006 | Concept formation (visual) | Advanced Matrices | Performance score | ns |

| | | | | |
|---|---|---|---|---|
| Blair, Richell 2006 | Concept formation (visual) | Advanced Matrices | Performance score | ns |
| Budhani 2006 | Concept formation (visual) | Advanced Matrices | Performance score | ns |
| Drugge 1998 | Concept formation (visual) | Abstraction (SILS) | Performance score | n/a |
| Goldstein 1998 | Concept formation (visual) | Standard Matrices | Performance score | ns |
| Gillstrom 1995 | Concept formation (verbal) | Similarities (WAIS-R) | Performance score | ns |
| | | Proverbs | Performance score | Control |
| | Concept formation (visual) | Abstraction (SILS) | Performance score | ns |
| | | Short Category Test | Errors | ns |
| | Reasoning (visual stimuli) | Picture Completion (WAIS-R) | Errors | ns |
| Hare 1988 | Semantic abstraction | Abstract vs. concrete categorisation | Errors | Control groups over individuals with psychopathy in RVF targets only |
| | | | Response latency | ns |
| | | | Laterality | ns |
| Hervé 2003 | Reasoning (verbal) | Interpretation of Metaphors | Aptness | ns |
| Howard 2007 | Semantic abstraction | Abstract vs. concrete categorisation | Errors | ns |
| | | | Response latency | ns |
| Johansson 2005 | Concept formation (visual) | Abstraction test | Performance score | ns |
| Jozef 1999 | Concept formation (verbal) | Similarities (WAIS) | Performance score | ns |
| Kiehl 2004 | Semantic abstraction | Lexical Decision (concrete vs. abstract stimuli) | % Correct | ns |

| | | | | |
|---|---|---|---|---|
| | | | Response latency | Control (concrete & abstract stimuli) |
| Kiehl, Hare, McDonald 1999 | Semantic abstraction | Abstract vs. concrete categorisation | % Correct | ns |
| | | | Response latency | ns |
| | | Lexical Decision (concrete vs. abstract stimuli) | % Correct | ns |
| | | | Response latency | ns |
| Kosson 1998 | Concept formation (visual) | Abstraction (SILS) | Performance score | ns |
| Lapierre 1995 | Concept formation (verbal) | Similarities (Ottawa-WAIS) | Performance score | ns |
| Mercer 2005 | Concept formation (verbal) | Similarities (WAIS-R) | Performance score | n/a |
| | Reasoning (verbal) | Comprehension (WAIS-R) | Performance score | n/a |
| | Reasoning (visual stimuli) | Picture Completion (WAIS-R) | Performance score | n/a |
| | | Picture Arrangement (WAIS-R) | Performance score | n/a |
| | Mathematical procedures | Arithmetic (WAIS-R) | Performance score | n/a |
| Mitchell 2002 | Concept formation (visual) | Advanced Matrices | Performance score | ns |
| Mitchell 2006 | Concept formation (visual) | Advanced Matrices | Performance score | ns |
| Pham 2003 | Concept formation (verbal) | Similarities (WAIS) | Performance score | n/a |
| | Reasoning (verbal) | Comprehension (WAIS) | Performance score | n/a |
| | Reasoning (visual stimuli) | Picture Completion (WAIS) | Performance score | n/a |
| | | Picture Arrangement (WAIS) | Performance score | n/a |
| | Mathematical procedures | Arithmetic (WAIS) | Performance score | n/a |
| Raine 1988 | Concept formation (verbal) | Similarities (WAIS-R) | Performance score | n/a |

| | | | | |
|---|---|---|---|---|
| | Reasoning (verbal) | Comprehension (WAIS-R) | Performance score | n/a |
| | Reasoning (visual stimuli) | Picture Completion (WAIS-R) | Performance score | n/a |
| | | Picture Arrangement (WAIS-R) | Performance score | n/a |
| | Mathematical procedures | Arithmetic (WAIS-R) | Performance score | n/a |
| Richell 2003 | Concept formation (visual) | Advanced Matrices | Performance score | ns |
| Richell 2005 | Concept formation (visual) | Advanced Matrices | Performance score | ns |
| Smith 1992 | Concept formation (visual) | Short Category Test | Errors | LA antisocial |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; WAIS/-R=Wechsler Adult Intelligence Scale/-Revised; SILS=Shipley Institute of Living Scale; RVF=Right visual field; LA=Low-anxious; n/a=Not available; ns= Not significant.

#### 2.3.4.1 Concept formation

Four studies on concept formation compared 92 individuals with ASPD to 84 controls. Another 26 studies included at least 529 individuals with psychopathy and 794 controls.

##### *2.3.4.1.1 Verbal format*

Two studies investigated ASPD (Shamay-Tsoory et al., 2010; Stevens et al., 2003) including 51 individuals with ASPD and 52 controls. Only Stevens et al. (2003) observed a significant difference, in favour of the control group. A meta-analysis yielded a significant medium pooled effect size estimate with non-significant heterogeneity. The failsafe *N* of 2.4 was below the critical value of 20 suggesting possible publication bias.

There were seven studies on psychopathy (Gillstrom, 1995; Jozef & da Silva, 1999; Lapierre et al., 1995; Mercer et al., 2005; Pham et al., 2003; Raine & Venables, 1988) including 231 individuals with psychopathy and 290 without, all offenders. Only Gillstrom highlighted a significant difference, in favour of the control group, while three studies did not report any statistical comparisons. A meta-analysis resulted in a significant medium pooled effect size estimate (Similarities), with no heterogeneity. The failsafe *N* of 13.3 was below the critical value of 30, thus results may be susceptible to publication bias. The pooled estimate was larger with Proverbs data from Gillstrom instead of Similarities, -0.47[-0.66,-0.28],$P<0.001$, while heterogeneity was not significant.

##### *2.3.4.1.2 Visual format*

For ASPD, visual format concept formation studies included those using the attentional set shifting tasks as described earlier. On the other hand, visual concept formation paradigms in addition to attentional set shifting were found in 22 studies on psychopathy. These involved at least 496 individuals with psychopathy and 747 controls. Sorting and shifting tasks were discussed earlier in executive functions. None of the remaining tasks revealed a significant difference between the groups and meta-analyses for each did not yield significant pooled effect size estimates either.

*2.3.4.1.2.6 Meta-analysis*

For ASPD, available data further to the attentional set shifting tasks discussed earlier were not available. Therefore, a meta-analysis was conducted for psychopathy only. With strongest effects, there was a non-significant, small to medium pooled effect estimate with considerable heterogeneity. Stratification by study quality did not result in significant subgroup differences. By removing the outlier (and strongest) effect of Mercer et al. (2005), the pooled estimate became small and only approached significance, 0.20[-0.01,0.41],*P*=0.06, with non-significant heterogeneity. Using data associated with either unsuccessful or successful individuals with psychopathy from Ishikawa et al. (2001) led to comparable results.

With weakest effects, the pooled estimate was small and non-significant, 0.22[-0.11,0.56], with comparable heterogeneity as before when Mercer et al. (2005) was included. The pooled estimate decreased further and remained non-significant when Mercer et al. was excluded, 0.13[-0.06,0.31], with comparable heterogeneity as when the study was excluded previously. Using data associated with either unsuccessful or successful individuals with psychopathy from Ishikawa et al. (2001) led to similar results.

***2.3.4.1.3 Meta-analysis on concept formation overall***

For ASPD, data in addition to attentional shifting were available in connection to Similarities by Stevens et al. (2003). A meta-analysis with strongest effects resulted in a significant, medium pooled effect size estimate with no heterogeneity. The failsafe *N* of 4.55 did not exceed the critical value of 25 therefore the result may be susceptible to publication bias. However, with weakest effects, the result was a small and non-significant pooled estimate, 0.04[-0.30,0.39], with no heterogeneity.

For psychopathy, a meta-analysis was conducted by pooling effect sizes associated with verbal and visual concept formation. This resulted in a marginally significant, small to medium pooled effect size estimate with substantial heterogeneity. The differences between study quality subgroups were not significant. Removal of the outlier data of Mercer et al. (2005) resulted in a smaller and also

marginally significant pooled effect size estimate, 0.27[0.00,0.54],*P*=0.05, with moderate heterogeneity. Using data associated with either unsuccessful or successful individuals with psychopathy from Ishikawa et al. (2001) led to comparable results.

Using weakest effects, there was a small and marginally significant pooled effect size estimate, 0.18[0.00,0.36],*P*=0.05, with non-significant heterogeneity. Random effects were retained in order to moderate possible bias introduced by assigning a lower weighting to Mercer et al. (2005), contrary to a fixed effects model, as this study has presented as an outlier often. Using data associated with either unsuccessful or successful individuals with psychopathy from Ishikawa et al. (2001) led to comparable results.

#### 2.3.4.2 Reasoning

Six studies examined reasoning of which one focused on ASPD. Studies on psychopathy included at least 190 individuals with psychopathy and 247 controls.

##### *2.3.4.2.1 Verbal reasoning*

There were four studies including 185 individuals with psychopathy and 229 without. One study reported results from statistical comparisons and did not find a significant group difference (Hervé, Hayes, & Hare, 2003). Pooling the effect sizes resulted in a non-significant, small to medium pooled effect size estimate with marginally significant heterogeneity. Random effects models were used to moderate possible bias by Mercer et al. (2005).

##### *2.3.4.2.2 Reasoning with visual materials*

Four studies included 190 individuals with psychopathy and 247 without. Only one study focused on ASPD (Stevens et al., 2003), reporting no significant group differences. Regarding Picture Completion, only Gillstrom provided details of statistical comparisons and did not highlight any significant group differences. Mercer et al.'s (2005) data report appeared unrealistic – individuals with psychopathy scored a mean of 0.38, *SD*=3.48. This was not in line with other findings in this group of studies and also indicated severe impairment (Wechsler, 1981). For this reason, it was not included in the meta-analysis. The remaining data produced a small, non-significant pooled effect size estimate with no heterogeneity. In connection with Picture Arrangement, no study reported any statistical comparisons. A meta-analysis revealed a very small, non-significant pooled effect size estimate, with no heterogeneity. An overall meta-analysis on reasoning did not reveal a significant pooled effect size estimate with either strongest or weakest effects (excluding the outlier Mercer et al., 2005). There was no evidence of heterogeneity.

#### 2.3.4.3 Mathematical procedures

There were three studies on psychopathy (Mercer et al., 2005; Pham et al., 2003; Raine & Venables, 1988) including 172 individuals with psychopathy and 219 without. No statistical comparisons were reported. A meta-analysis produced a significant medium pooled effect size estimate with no heterogeneity. The significant result was primarily due to the major contribution of Mercer et al (2005). The failsafe *N* of 6.98 was below the critical value of 20, thus, these results may be subject to publication bias.

#### 2.3.4.4 Semantic abstraction

Four publications employed a paradigm of abstract semantic processing (Hare & Jutai, 1988; Howard & McCullagh, 2007; Kiehl, Hare, McDonald, & Brink, 1999; Kiehl et al., 2004). All studies focused on psychopathy, including at least 38 offenders with psychopathy, 39 offenders without psychopathy and 21 healthy controls from the general public. Kiehl et al. (2004) concluded that control participants responded faster to abstract as well as concrete words. Hare and Jutai, however, reported a significant group difference indicating a deficit of abstract processing in the group with psychopathy during RVF stimulus presentation only. Sufficient data were unavailable for a meta-analysis primarily due to many samples potentially overlapping.

#### 2.3.4.5 Meta-analysis on abstraction overall

Regarding ASPD, the only data in addition to concept formation were by Stevens et al. (2003) for Picture Arrangement. A meta-analysis with this set yielded a small and non-significant pooled effect size estimate with no heterogeneity with either dataset from Barkataki et al. (2005). This is in contrast to the previous medium estimate with Similarities but more in line with the results when the WCST was included.

For psychopathy, using strongest effects, the result was a significant medium pooled effect estimate in favour of controls, with considerable heterogeneity. The failsafe *N* of 198.10 was above the critical cut-off of 70. Although this appears robust, a relatively asymmetrical funnel plot (Appendix D, Figure 12.7) may indicate possible publication bias. By removing the outlier data of Mercer et al. (2005), the overall pooled estimate decreased but remained significant, 0.34[0.10,0.58],$P<0.01$, while heterogeneity became moderate

With data from successful individuals with psychopathy from Ishikawa et al. (2001), the pooled estimate was small to medium and approached significance, 0.40[0.00,0.80],$P=0.05$, with similarly high heterogeneity as before. However, when Mercer et al. (2005) was excluded, the pooled estimate decreased but became significant,0.28[0.03,0.53],$P<0.05$, in favour of controls whereas heterogeneity became moderate as before. Overall, study quality score was not correlated significantly with effect size, *rho*=-0.11,$n=11$, and stratification by study quality did not reveal any significant subgroup differences. Using weakest effects, the result was a small and non-significant pooled effect size estimate, 0.05[-0.33,0.19], with no heterogeneity. Results were comparable using data associated with unsuccessful or successful individuals with psychopathy from Ishikawa et al. (2001).

#### 2.3.4.6 Summary

##### *2.3.4.6.1 ASPD*

An overall deficit in abstraction was not supported. Studies examined concept formation and reasoning with some evidence of deficit in the former. A more specific impairment on verbal concept formation was also identified but may be subject to publications bias. In terms of visual concept formation, results did not support impairment whereas no group differences were identified in reasoning.

##### *2.3.4.6.2 Psychopathy*

Research investigated several aspects of concept formation, reasoning, mathematical procedures and semantic abstraction. Evidence suggested an overall deficit in abstraction with strongest effects. In spite of a possible overall deficit, results did not generally support a deficit in more specific operations of abstraction. Mercer et al. (2005) was identified as an outlier. There was some but limited evidence of deficit in mathematical procedures and semantic abstraction, the latter present only for RVF stimuli in one study. Groups did not appear different in their reasoning abilities.

### 2.3.5 Affect and social cognition

#### 2.3.5.1 Affective operations

Affective operations in antisocial personality involved processing, recognition, memory and linguistic expression. Overall, six studies examined ASPD including at least 316 individuals with the diagnosis and 281 controls. A further two studies focused on DPD involving 59 individuals with this diagnosis and 61 controls. Finally, a total of 32 studies examined psychopathy with at least 386 individuals with psychopathy and 567 controls.

##### *2.3.5.1.1 Affective processing*

Fourteen studies employed a paradigm in which the presence of an affectively laden stimulus is anticipated to implicitly affect responding (Table 2.9). Two of the studies investigated ASPD (Kosson, Lorenz, & Newman, 2006; Lorenz & Newman, 2002c). In these, no significant group differences were observed in affective facilitation. Kosson et al. (2006) indicated a significant difference between the ASPD with concurrent psychopathy compared to ASPD-only and control groups, but did not observe a difference between the performance of the ASPD-only and control groups.

Twelve studies explored psychopathy (K. Blair, Richell et al., 2006; Day & Wong, 1996; Drugge, 1998; Howard & McCullagh, 2007; Iria & Barbosa, 2009; Kosson et al., 2006; Lorenz & Newman, 2002a, 2002b; Marshall, 1996; Mitchell, Richell, Leonard, & Blair, 2006; Müller et al., 2008; Weber et al., 2004; Williamson, Harpur, & Hare, 1991) and included at least 207 individuals with psychopathy and 208 controls. Müller et al. (2008) and Weber et al. (2004) reported on the same study. Their sample with psychopathy was also diagnosed with DPD.

Table 2.9. *Studies which examined affective processing in ASPD, DPD and psychopathy*

| Reference | Task | Modality | Stimuli | Relevant outcomes | Result |
|---|---|---|---|---|---|
| ***ASPD*** | | | | | |
| Lorenz & Newman 2002c | Lexical decision | Verbal: semantic (words) | Affective: positive, negative & neutral | Affective facilitation | ns |
| | | | | Response latency | ns |
| | | | | Accuracy | ns |
| ***ASPD & psychopathy*** | | | | | |
| Kosson 2006 | Lexical decision | Verbal: semantic (words) | Affective: positive, negative & neutral | Affective facilitation | Greater in control & ASPD only compared to ASPD+psychopathy |
| ***DPD & psychopathy*** | | | | | |
| Müller 2008/ Weber 2004 | Affective Induction: Simon | Visual: pictorial | Affective: positive, negative and neutral | Response latency | ns |
| | | | | Errors | ns |
| ***Psychopathy*** | | | | | |
| Blair, Richell 2006 | Affective discrimination with affective priming | Verbal: semantic (words) | Affective: positive, negative & neutral | Response latency | Possibly lower in controls (interactions without post hoc comparisons) |
| | | | | Errors | ns |

| | | | | | |
|---|---|---|---|---|---|
| Day 1996 | Affective discrimination | Verbal: semantic (words) | Affective: negative & neutral | Correct response latency | Possibly higher in controls (LVF advantage for control only but no post hoc comparisons) |
| | | | | Accuracy | ns (but significant group x visual field interaction of a mixed profile) |
| | | Visual: facial | Emotional: sadness, anger, fear, disgust & neutral | Correct response latency | ns |
| | | | | Accuracy | ns |
| Drugge 1998 | Modified flanker: Word naming with priming | Verbal: semantic (words) | Affective: positive, negative & neutral | Accuracy | ns |
| | | | | Correct response latency | ns |
| Howard 2007 | Abstract discrimination | Visual: pictorial | Affective: positive, negative and neutral | Response latency | ns |
| | | | | Omission errors | ns |
| | | | | Commission errors | ns |
| | Oddball | Visual: pictorial | Affective: positive, negative and neutral | Response latency | ns |
| | | | | Omission Errors | Favours control in high valence/low arousal and low valence/high arousal conditions |
| | | | | Commission Errors | ns |

| | | | | | |
|---|---|---|---|---|---|
| Iria 2009 | Go/NoGo | Visual: facial | Emotional: happiness, fear, surprise & neutral | Response latency | ns |
| | | | | Total errors | Favours control |
| | | | | Omission errors | Favours control |
| | | | | Commission errors | ns |
| Lorenz & Newman 2002a | Lexical decision | Verbal: semantic (words) | Affective: positive, negative & neutral | Affective facilitation | Favours control (RH responses only) |
| | | | | Response latency | ns |
| | | | | Accuracy | ns |
| Lorenz & Newman 2002b | Lexical decision | Verbal: semantic (words) | Affective: positive, negative & neutral | Affective facilitation | ns |
| | | | | Response latency | n/a |
| Marshall 1996 | Lexical decision | Verbal: semantic (words) | Affective: positive, negative & neutral | Response latency | ns |
| | | | | Accuracy | n/a |
| Mitchell 2006 | Affective induction (during a simple shape discrimination task) | Visual: pictorial | Affective: positive, negative & neutral | Response latency | Greater for control with affective stimuli, especially positive; ns for individual with psychopathy |
| | | | | Errors | Greater for control with negative stimuli; ns for individual with psychopathy |

| | | | | | |
|---|---|---|---|---|---|
| Williamson, Harpur 1991 | Lexical decision | Verbal: semantic (words) | Affective: positive, negative & neutral | Affective facilitation<br><br>Accuracy | Greater for control (no pairwise comparisons)<br>ns |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; DPD=Dissocial Personality Disorder; LVF=Left visual field; RH=right hand; n/a=Not available; ns=Not significant.

Regarding lexical decision tasks, Kosson et al. (2006) reported less affective facilitation in the ASPD group with psychopathy compared to the ASPD-only and control groups, whereas Lorenz and Newman (2002a) reported a similar effect for responses with the right hand only. Williamson et al. (1991) highlighted a significant interaction showing greater emotion facilitation in the control group, however, they did not conduct any post-hoc comparisons. There were no further significant differences. During affective induction, Mitchell et al. (2006) reported a significant interaction in which controls were influenced by affective induction while individuals with psychopathy were not. Müller et al. (2008) did not report a significant effect.

The remaining studies employed various tasks using affective stimuli. In K. Blair, Richell et al. (2006), primes of the same valence as a target which they preceded facilitated responding and there was interference by negative primes to positive targets in the control group (interaction without post hoc comparisons). Day and Wong (1996) requested from their participants to identify the visual hemi-field in which emotional stimuli appeared. Again, there was a significant interaction showing an expected left visual field (LVF) advantage for controls only but without post hoc comparisons. In Drugge's (1998) study, distractors in preceding trials became targets in ensuing ones but there were no group differences in connection to affective processing. In Howard and McCullagh's (2007) oddball task, the group with psychopathy omitted more targets in the high valence/low arousal and in the low valence/high arousal conditions. However, this effect is difficult to interpret. Finally, individuals with psychopathy omitted more targets than controls during an affective Go/NoGo task in Iria and Barbossa's (2009) study.

A meta-analysis was conducted. As only two studies measured affective facilitation directly (Lorenz & Newman, 2002a, 2002b), data from other studies included response latencies and error rates. Lorenz and Newman's samples were Caucasian in one study and African/American in the other and thus not overlapping. Entering strongest effects resulted in a significant medium to large pooled effect size estimate with non-significant heterogeneity. The failsafe *N* of 28.78 did not exceed the critical value of 35 and the funnel plot was not symmetrical (Figure 12.7) thereby suggesting susceptibility to publication bias. In addition, weakest effects resulted in a small and non-significant pooled estimate, 0.23[-0.19,0.65], with moderate

heterogeneity. All studies employed a semantic task except Iria and Barbossa (2009) who examined the impact of affective stimuli interlaced between GoNoGo trials. However, the pooled estimates of the meta-analysis were reduced but remained comparable without this study and heterogeneity was no longer significant for weakest effects.

#### *2.3.5.1.2 Affect recognition*

There were two publications on ASPD (Dolan & Fullam, 2004; Habel, Kuhn, Salloum, Devos, & Schneider, 2002) and one on DPD (Dolan & Fullam, 2006). Habel et al.'s (2002) ASPD group also met criteria for psychopathy and Dolan and Fullam (2004) included groups with and without psychopathy within the DPD diagnosis in both studies. Ten additional publications examined psychopathy (Bagley, Abramowitz, & Kosson, 2009; K. Blair, Richell et al., 2006; R. Blair, Mitchell, Peschardt et al., 2004; R. Blair et al., 2002; Glass & Newman, 2006; Hervé et al., 2003; Hiatt, Lorenz, & Newman, 2002; Kiehl, Hare, McDonald et al., 1999; Kosson, Suchy, Mayer, & Libby, 2002; Mills, 1995). Details are shown in Table 2.10.

Table 2.10. *Studies which examined affect recognition in ASPD, DPD, and psychopathy*

| Reference | Task | Modality | Stimuli | Outcome | Result favoured |
|---|---|---|---|---|---|
| ***ASPD & psychopathy*** | | | | | |
| Dolan 2004 | Affect recognition (forced-2-choice) | Visual: facial | Basic emotional: Happiness, sadness, anger, disgust, surprise, fear & distress | Accuracy | Control (vs. ASPD without psychopathy only) |
| | | Visual: ocular | Basic emotional: as above | Accuracy | Control (vs. ASPD without psychopathy only) |
| Habel 2002 | Affect recognition (open-ended response format) | Visual: facial | Emotional: happiness, sadness, neutral | Accuracy | Control |
| | | | | Sensitivity | ns |
| ***DPD & psychopathy*** | | | | | |
| Dolan 2006 | Morphed faces | Visual: facial | Emotional: happiness, sadness, anger, disgust, surprise & fear | Accuracy | Control (vs. DPD; overall, happiness, sadness, surprise)<br>Non-psychopath vs. individuals with psychopathy (sadness) |
| | | | | Response latency | Control (vs. PD, overall) |
| ***Psychopathy*** | | | | | |
| Bagley 2009 | Affect recognition (forced-5-choice) | Verbal: semantic (sentences)<br>Verbal: prosodic (sentences) | Emotional: happiness, sadness, anger, surprise & neutral | Accuracy | Control (overall, happiness, sadness)<br>Control (surprise) |
| Blair, Richell 2006 | Affective recognition (forced-2-choice) with | Verbal: semantic (words) | Affective: Positive & negative | Errors | ns |

| | | | | | |
|---|---|---|---|---|---|
| | affective priming | | | | |
| | | | | Response latency | ns |
| Blair, Mitchell, Peschardt 2004 | Morphed faces | Visual: facial | Emotional: happiness, sadness, anger, disgust, surprise & fear | Performance score | Control (fear) |
| | | | | Errors | Control (fear) |
| | | | | Stage of recognition | ns |
| Blair 2002 | Affect recognition (forced-5-choice) | Verbal: prosodic (words) | Emotional: happiness, sadness, anger, disgust & fear | Errors | Control (overall, fear) |
| Glass 2006 | Affect recognition (open-ended response format) | Visual: facial | Emotional: happiness, sadness, anger & fear | Accuracy | ns overall, individuals with psychopathy (fear only) when the open-ended response format condition preceded the affect identification condition |
| | | | | Response latency | ns |
| | & affect identification condition | Visual: facial / Verbal: semantic (words) | | Accuracy | ns |
| | | | | Response latency | ns |
| Hervé 2003 | Affective recognition (forced-2-choice) | Verbal: semantic (sentences) | Affective: positive & negative | Errors | Control |

| | | | | | |
|---|---|---|---|---|---|
| Hiatt 2002 | Dichotic listening affect recognition | Verbal: Prosodic (words) | Emotional: happiness, sadness, anger & neutral | Accuracy | ns overall<br>Individuals with psychopathy for right-ear targets |
| | | | | Commission errors | ns |
| | | | | Laterality | ns |
| Kiehl, Hare, McDonald 1999 | Affect recognition (forced-2-choice) | Verbal: semantic (words) | Affective: positive & negative | Accuracy | ns |
| | | | | Response latency | ns |
| Kosson 2002 | Affect recognition (forced-6-choice) | Visual: facial | Emotional: happiness, sadness, anger, disgust, surprise & fear | Accuracy | Control: disgust, both hands; overall, LH<br>Individuals with psychopathy: anger, RH |
| Mills 1995 | Affective recognition/ discrimination | Visual: facial | Emotional: sadness, anger, disgust, fear & neutral | Accuracy | ns |
| | | | | Response latency | ns |
| | | Verbal: semantic (words) | Affective: Negative & neutral | Accuracy | ns |
| | | | | Response latency | ns |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; DPD=Dissocial Personality Disorder; RH/LH=Right/left hand; ns=Not significant.

*2.3.5.1.2.1 ASPD and DPD*

All studies investigated visual emotion recognition. The two publications on ASPD included 101 participants with the diagnosis (prison and forensic psychiatric settings) and 37 controls (general public). Both publications highlighted a deficit in the antisocial group. In these results, it is not possible to associate this effect exclusively with a diagnosis of ASPD due to the antisocial group exhibiting high scores on psychopathy also. Criminality confounded the result in both studies. The group with DPD performed worse in recognising sad, happy and surprised faces and were slower to respond to all emotions in Dolan and Fullam (2006).

In a meta-analysis, ASPD-only group data were used from Dolan and Fullam (2004). Because this study did not provide overall data, facial and ocular data were considered in sensitivity analyses. The resulting pooled effect size estimates were comparable, medium to large and reached significance in both cases with no heterogeneity. The failsafe *N*s were 6.00 and 5.62 for the meta-analysis with facial and ocular data respectively from Dolan and Fullam. Both values were below the critical cut-off of 20 suggesting susceptibility to publication bias.

*2.3.5.1.2.2 Psychopathy*

The three studies on ASPD and DPD also investigated psychopathy. There were ten additional studies on psychopathy, the samples of which originated from prison populations. In total, the studies included at least 207 individuals with psychopathy and 240 controls and examined both verbal and visual affect recognition.

Verbal affect recognition. Seven publications including at least 120 individuals with psychopathy and 107 without. From the priming task of K. Blair, Richell et al. (2006) only the conditions involving affect recognition in connection to a stimulus preceded by a neutral prime were relevant to this section. Significant semantic deficits in psychopathy were reported by Bagley et al. (2009) for emotions overall as well as for happiness and sadness specifically. Hervé et al. (2003) also observed deficits in psychopathy for positive and negative sentences. No significant group differences were reported by K. Blair, Richell et al. (2006), Kiehl et al. (1999) and Mills (1995) in semantic affect recognition.

Regarding prosodic recognition, significant deficits in psychopathy were suggested by Bagley et al. (2009) for surprise and by R. Blair et al. (2002) for emotions overall as well as for fear specifically. Hiatt et al. (2002) concluded that individuals with psychopathy performed more accurately for right ear targets in an emotion recognition dichotic listening task (with a trend for reduced left ear advantage compared to controls).

A meta-analysis of strongest effects (with preference for error rather than response latency data, where available) yielded a significant and medium pooled effect size estimate, 0.51[0.21,0.81],$P<0.001$, with non-significant heterogeneity. The failsafe *N* of 14.90 did not exceed the critical value of 30, indicating possible publication bias. However, the pooled estimate became small and did not reach significance with weakest effects, 0.04[-0.25,0.34]. Heterogeneity remained non-significant.

Visual affect recognition. Seven studies included at least 160 individuals with psychopathy and 198 controls. Mills' (1995) emotion recognition task involved selecting an emotional stimulus against a neutral stimulus thus being less challenging than other paradigms. Glass and Newman (2006) included an additional condition in which participants selected the facial expression matching a pre-specified emotion (e.g. 'locate anger'). This involved semantic processing as well.

There was evidence of impaired performance in participants with psychopathy in the studies by Habel et al. (2002, confounded by ASPD and criminality), R. Blair, Mitchell, Peschardt et al. (2004) for fear, Dolan and Fullam (2006) for sadness and Kosson et al. (2002) for disgust when participants responded with both hands and overall with the left hand. Kosson et al. also concluded that individuals with psychopathy performed better than controls on right-handed responses to expressions of anger. In addition, participants without psychopathy exhibited significant left hand advantage for anger but this was not the case for those with psychopathy. Finally, Glass and Newman (2006) reported that individuals with psychopathy performed better at identifying fear when this condition was presented first, but no overall group differences were detected. There were no further significant differences.

Strongest effects resulted in a significant, small to medium pooled effect size estimate with moderate heterogeneity. The failsafe *N* of 18.01 was below the critical

value of 35. The funnel plots were not symmetrical (Appendix D, Figure 12.8), thus results may be subject to publication bias. Using weakest effects, there was a small and non-significant pooled estimate, 0.02[-0.35,0.38], with comparable heterogeneity. Study quality did not appear to affect distribution of effect sizes.

A meta-analysis on overall affect recognition in psychopathy using strongest effects revealed a significant medium pooled effect size estimate with non-significant heterogeneity. The failsafe *N* of 36.50 did not exceed the critical cut-off of 40. The funnel plot was not symmetrical (Appendix D, Figure 12.8), thus publication bias was possible. Using weakest effects, there was a small and non-significant pooled estimate, 0.09[-0.12,0.31], with non-significant heterogeneity. Study quality did not appear to affect distribution of effect sizes.

A number of studies provided data for individual emotions thus making meta-analyses possible for happiness, sadness, anger, disgust, surprise and fear.

For happiness, strongest effects resulted in a significant small to medium pooled effect size estimate with no heterogeneity. The analysis yielded comparable results with weakest effects. The failsafe *N* of 25.36 was below the critical value of 40 with a relatively asymmetrical funnel plot (Appendix D, Figure 12.9). This indicated possible publication bias.

Regarding sadness, strongest effects resulted in a significant small to moderate pooled effect size estimate with non-significant heterogeneity. The failsafe *N* of 16.29 was below the critical value of 40 with a relatively asymmetrical funnel plot (Appendix D, Figure 12.9), thus the result may be susceptible to publication bias. With weakest effects, the effect size became small and did not reach significance, 0.11[-0.10,0.31], with comparable heterogeneity.

Regarding anger, strongest effects resulted in a small and non-significant pooled effect size estimate, with non-significant heterogeneity. The result was comparable with weakest effects.

For disgust, strongest effects revealed a significant small to medium pooled effect size estimate, with relatively inconsequential heterogeneity. The failsafe *N* of 2.05 was below the critical value of 25, indicating possible publication bias.

However, with weakest effects, the pooled effect size estimate decreased and only approached significance, 0.29[-0.03, 0.61],*P*=0.08, with comparable heterogeneity.

Regarding surprise, strongest effects resulted in a significant small to medium pooled effect size estimate with no heterogeneity. The failsafe *N* of 10.02 was below the critical value of 30. Thus, results may be subject to publication bias. With weakest effects, the result was a smaller pooled effect size estimate which remained significant, 0.31[0.05,0.58],*P*<0.05, with comparable heterogeneity.

Finally, regarding fear, strongest effects resulted in a non-significant small to medium pooled effect size estimate with substantial heterogeneity mostly due to the contribution of a stronger effect by R. Blair et al. (2004). Excluding this study resulted in a small pooled estimate which again did not reach significance, -0.06[-0.38,0.25], while heterogeneity was no longer significant. Results were comparable with weakest effects.

#### *2.3.5.1.3 Affect and memory*

There were four studies in this category (Christianson et al., 1996; Dolan & Fullam, 2005; Glass & Newman, 2009; Kiehl et al., 2001), all of which examined psychopathy (offender samples with and without psychopathy). Dolan and Fullam (2005) also examined ASPD (divided in subgroups according to scores on psychopathy). Dolan and Fullam and Kiehl et al. (2001) included a healthy control group from the general public alongside a group of offenders without psychopathy. All other participants were also offenders. An overview of studies is presented in Table 2.11. The samples included 145 individuals with psychopathy and 250 controls including 28 healthy individuals from the general public. The memory tasks, either targeting visual or verbal memory, varied considerably.

Table 2.11. *Studies which investigated affect-related memory in ASPD and psychopathy*

| Reference | Memory access | Memory | Modality | Stimuli | Outcome | Result favoured |
|---|---|---|---|---|---|---|
| ***ASPD & psychopathy*** | | | | | | |
| Dolan 2005 | Recall | LTM: 2 hours | Visual | Story in pictures with a pre-affective (neutral), affective, and post-affective (neutral) section | Accuracy | Healthy control vs. ASPD with psychopathy for in post-affective condition.<br><br>Healthy control vs. high & low factor 1 scorers. |
| | Recognition, forced-choice | | | | Accuracy | Healthy controls vs. high and medium psychopathy ASPD groups in the pre-affective and affective conditions.<br>Healthy control vs. high & low factor 1 scorers in the pre-affective and affective conditions.<br>Healthy control vs. high & low factor 2 scorers in the pre-affective and affective conditions. |
| ***Psychopathy*** | | | | | | |
| Christianson 1996 | Cued recall | STM: 5 min | Visual | Affective & neutral pictures | Accuracy (central) | ns |
| | | | | | Accuracy (peripheral) | Psychopathy |
| | Cued recognition: forced-4-choice | | | | Accuracy (central) | ns |
| | | | | | Accuracy (peripheral) | ns |

| | | | | | | |
|---|---|---|---|---|---|---|
| Glass 2009 | Recall | STM: 5 min | Verbal: semantic (words) | Affective: positive, negative & neutral | Affective facilitation (central) | ns |
| | | | | | Accuracy (central) | ns |
| | | | | | Affective facilitation (contextual) | Control |
| | | | | | Accuracy (contextual) | ns |
| Kiehl 2001 | Acquisition & recognition | STM/ Learning | Verbal: semantic (words) | Affective: negative & neutral | Accuracy | ns |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; LTM/STM=Long/short-term memory; ns=Not significant.

Two studies examined visual memory. Of these, Dolan and Fullam (2005) reported superior recall performance for the healthy control group compared to offenders with psychopathy and ASPD in post-affective story elements only. In addition, healthy controls performed better on recall of affective elements than both those of the ASPD groups having both high and low psychopathy factor 1 scores. For recognition, healthy controls performed better than the ASPD groups with psychopathy but not the ASPD-only group. In addition, controls performed better than groups with high and low factor 1 and factor 2 scores on pre-affective and affective items. This could indicate an overall, rather than affect-specific, memory deficit. There were no differences between the ASPD-only and healthy control groups. Criminality was a confounder in comparisons between those with offence history and those without. Christianson et al. (1996) observed that individuals without psychopathy exhibited better memory for central information than for peripheral details in the affective condition. However, those with psychopathy did not exhibit this pattern. In fact, they performed better than controls in recalling peripheral information in the affective condition.

Of the two studies on short-term verbal memory, only Glass and Newman (2009) observed a significant effect, where controls demonstrated greater affective facilitation of memory for contextual information compared to individuals with psychopathy. Kiehl et al. (2001) included eight repetitions of an acquisition-recognition procedure, thus likely introducing a ceiling effect.

Meta-analysis with sensitivity analysis was conducted with data for central/peripheral memory from Glass and Newman (2009) and recall/recognition data from Dolan and Fullam (2005). There was a non-significant and small pooled effect size estimate with central stimuli and a significant small to medium pooled effect size estimate with peripheral memory data. There was no heterogeneity. For the significant result, the failsafe *N* of 4.18 was below the critical value of 25, thus there may be some publication bias. Using recognition data from Dolan and Fullam, the results were comparable for each of the two meta-analyses.

#### *2.3.5.1.4 Affective expression*

There was one study on ASPD (Gawda, 2008b) and three on psychopathy (Brinkley, Newman, Harpur, & Johnson, 1999; Louth, Williamson, Alpert, Pouget, & Hare, 1998; Williamson, 1991), with a total of at least 39 individuals with psychopathy and 36 controls, all offenders. An overview is presented in Table 2.21.

##### *2.3.5.1.4.1 ASPD*

The only study on ASPD (Gawda, 2008b), participants identified with an individual in a picture depicting love and composed a narrative on the situation. The study included two groups of offenders, with and without ASPD, and a control group from the general public. Individuals with ASPD included more actors, actor traits, strong emotions, actions and presumptions in their narratives which were also longer compare to those of the other groups. They also made more references to themselves. In absence of an appropriate task control condition (i.e. neutral narrative) it is difficult to draw firm conclusions from these results.

##### *2.3.5.1.4.2 Psychopathy*

Three studies on psychopathy (Brinkley, Newman et al., 1999; Louth et al., 1998; Williamson, 1991) investigated different aspects of discourse (oral narratives) and explored features of speech. Williamson (1991) reported an overall syntax effect (closed to open plot units ratio) in favour of the control group. Further examination revealed that individuals with psychopathy made more incompetent (ambiguous, unclear, etc.) references than controls when relating an angry incident (affective condition). Individuals with psychopathy also demonstrated less cohesion than controls in the non-affective condition. Louth et al. (1998) reported that individuals with psychopathy generally spoke more softly and showed less variation in voice amplitude between negative and neutral words. There were no further significant group differences.

#### *2.3.5.1.5 Meta-analysis on affective operations*

The affective operations examined here were processing, recognition, memory and linguistic expression. For ASPD, available data included accuracy and facilitation. Strongest effects resulted in a non-significant, medium pooled effect size estimate with substantial heterogeneity and were comparable with weakest effects.

For psychopathy, strongest effects resulted in a significant medium pooled effect size estimate in favour of controls. Heterogeneity was not significant. The failsafe *N* of 109.81 exceeded the critical value of 55 and the funnel plot showed some symmetry (Appendix D, Figure 12.10), suggesting indicating robustness of the results against publication bias. However, weakest effects resulted in a small and non-significant pooled effect size estimate, 0.08[-0.08,0.24], with non-significant heterogeneity. Overall, study quality did not appear to affect distribution of effect sizes.

### 2.3.5.2 Social Cognition

#### *2.3.5.2.1 Theory of Mind*

There was one study on ASPD (Shamay-Tsoory et al., 2010), four on psychopathy (R. Blair, Sellars, Strickland, Clark, & et al., 1995; R. Blair et al., 1996; Patterson, 1990; Richell et al., 2003) and one on both operationalisations (Dolan & Fullam, 2004). An overview of studies is presented in Table 2.12.

##### *2.3.5.2.1.1 ASPD*

The studies (Dolan & Fullam, 2004; Shamay-Tsoory et al., 2010) included 106 individuals with ASPD and 40 individuals without. Dolan and Fullam (2004) divided the ASPD group in high and low-psychopathy subgroups. Both studies indicated deficits in ASPD. Dolan and Fullam reported a deficit in understanding the mental states of the individuals in the mentalising (faux pas) task. Shamay-Tsoory et al. (2010) highlighted a deficit during affective (i.e. inferring feelings) but not cognitive (i.e. inferring thoughts) 2$^{nd}$ order inference (there were also physical inferences which did not involve mentalising and functioned as internal control condition). In both studies, the ASPD samples were from prison populations offenders while the control groups were not offenders thereby introducing confounding of criminality.

A meta-analysis with strongest effects resulted in a significant and large pooled effect size estimate in favour of controls with no heterogeneity. The failsafe *N* of 17.49 was just below the critical value of 20, thus there may be some susceptibility to publication bias. However, the pooled effect size estimate was small and in the opposite direction but not significant with the weakest effects, 0.19[-0.32,0.70], while heterogeneity was not significant.

Table 2.12. *Studies which examined Theory of Mind in ASPD and psychopathy*

| Reference | Function/task | Stimuli | Special conditions | Mental states | Primary outcomes | Result favoured |
|---|---|---|---|---|---|---|
| ***ASPD*** | | | | | | |
| Shamay-Tsoory 2010 | 1st order inference | Visual: cartoon drawings | Forced-choice | Cognitive | Accuracy | ns |
| | | | | Affective | | ns |
| | | | | Physical (not mental state) | | ns |
| | 2nd order inference | | | Cognitive | Accuracy | ns |
| | | | | Affective | | Control Antisocial |
| | | | | Physical (not mental state) | | |
| ***ASPD & psychopathy*** | | | | | | |
| Dolan 2004 | 1st order inference (false-belief) | Verbal: stories | Open | False beliefs | Proportions meeting criterion | ns |
| | 2nd order inference (false-belief) | | | | | ns |
| | Mentalising (faux pas) | | | Faux pas | Detections | ns |
| | | | | | Understanding of faux pas | ns |
| | | | | | Speaker mental state | Control (vs. ASPD) |
| | | | | | Listener mental state | Control (vs. ASPD) |
| | | | | | Empathic understanding | Control (vs. ASPD) |
| | 1st order inference (mental state) | Visual: facial | Forced choice | Complex mental states | Accuracy | ns |

| | | | | | | |
|---|---|---|---|---|---|---|
| | | Visual: ocular | | | | ns |
| ***Psychopathy*** | | | | | | |
| Blair 1996 | Mentalising | Verbal: story | Open | Not specific | Performance score | ns |
| | | | | | Mental & physical justifications | ns |
| Blair, Sellars 1995 | 1st order inference | Verbal: story | Open | Happiness | Accuracy | ns |
| | | | | Sadness | | ns |
| | | | | Embarrassment | | ns |
| | | | | Guilt | | Control |
| Patterson 1990 – Exp. 3 | 1st order inference | Visual & auditory (non-verbal): videos | Closed | Presence of affect | Accuracy (active condition) | ns |
| | | | Active/ Reflective conditions | Positive | Accuracy (overall) | Control |
| | | | Favourable/ Unfavourable feedback conditions | Negative | Accuracy (overall) | ns |
| | | | | Amusement | Accuracy (overall) | ns |
| | | | | Happiness | Accuracy (overall) | ns |
| | | | | Interest | Accuracy (overall) | ns |
| | | | | Anger | Accuracy (overall) | ns |
| | | | | Disgust | Accuracy (overall) | ns |
| | | | | Fear | Accuracy (overall) | ns |
| | | | | Global affect | Rating (good vs. bad) | ns |
| Richell 2003 | 1st order inference | Visual: ocular | Forced-choice | Complex mental states | Accuracy | ns |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; ns=Not significant.

*2.3.5.2.1.2 Psychopathy*

The five studies (R. Blair, Sellars et al., 1995; R. Blair et al., 1996; Dolan & Fullam, 2004; Patterson, 1990; Richell et al., 2003) included at least 86 individuals with psychopathy and 134 without. Significant group differences were reported by R. Blair, Sellars et al. (1995) where individuals with psychopathy appeared less likely to appropriately assign guilt overall and more likely to inappropriately assign happiness instead of guilt to actions of intentional harm. Patterson (1990) also reported that participants with psychopathy were less accurate in evaluating affective states when the context was positive in a post hoc analysis following a non-significant interaction, thus the result is not robust. There were no further significant differences.

A meta-analysis with strongest effects was in favour of controls, with a small and non-significant pooled effect size estimate and non-significant heterogeneity. With weakest effects, the pooled effect size estimate was also small and non-significant but in favour of individuals with psychopathy this time, 0.26[-0.05,0.58], with comparable heterogeneity.

***2.3.5.2.2 Moral reasoning***

There were three studies, all on psychopathy (R. Blair, 1995; R. Blair, Jones, Clark, & Smith, 1995; Cima, Tonnaer, & Hauser, 2010). Samples included 34 individuals with psychopathy and 78 without, all from forensic institutions with an additional control group from the general public in the study of Cima et al. (2010). An overview is presented in Table 2.13. Both studies by R. Blair's lab (R. Blair, 1995; R. Blair et al., 1995, likely with overlapping samples) found that individuals with psychopathy made fewer moral/conventional distinctions and were less likely to adjust their views on the basis of authority jurisdiction. Participants with psychopathy were also less likely to justify moral transgressions in terms of others' welfare or make pain-based justifications. However, they judged positive acts as more preferable compared to controls. Cima et al. (2010) did not report any significant differences.

Table 2.13. *Studies which examined moral reasoning in psychopathy*

| Reference | Task | Special conditions | Primary outcomes | Result favoured |
|---|---|---|---|---|
| Blair 1995 | Moral reasoning | Moral & conventional transgressions | Permissibility | ns |
| | | | Seriousness | ns |
| | | | Authority jurisdiction | Control |
| | | | Moral/conventional distinctions | Control |
| | | | Justifications | Individuals with psychopathy less likely to justify in terms of other's welfare (moral transgressions only) |
| Blair, Jones 1995 | Moral reasoning | Moral & conventional transgressions & positive acts | Permissibility | ns |
| | | | Seriousness | ns overall, higher score by individuals with psychopathy for positive acts |
| | | | Authority jurisdiction | ns |
| | | | Moral/conventional distinctions | Control |
| | | | Justifications | Individuals with psychopathy less likely to justify in terms of other's welfare (moral transgressions only) |
| | | | Pain & pleasure justifications (positive acts only) | Control (pain-based justifications only) |
| Cima 2010 | Moral dilemmas | Personal and impersonal<br>Self-serving or other-serving | Endorsements | ns |
| | | Sacrificed person became worse off vs. no change | | |

*Note*. ns=Not significant.

#### *2.3.5.2.3 Social understanding*

There were three publications in this group (N. S. Gray, MacCulloch, Smith, Morris, & Snowden, 2003; Patterson, 1990; Richell et al., 2005; Snowden, Gray, Smith, Morris, & MacCulloch, 2004) (Table 2.14). Snowden and N. S. Gray and their colleagues reported on the same study. In total, there were 73 individuals with psychopathy and 100 without, all from forensic institutions.

Snowden et al. (2004) investigated latent attitudes towards violence in a group of offenders who had committed murder and a group of other offenders. Both groups were divided according to scores on the PCL-R. The study highlighted that the negative association to violence was reduced among individuals who had high psychopathy scores and had committed a murder compared to other offenses. The opposite was observed in individuals with low psychopathy scores.

Of the remaining two studies, Richell et al. (2005) requested from participants to rate the trustworthiness of facial stimuli (no emotions) while Patterson's (1990) studies involved interpretation of an interpersonal situation. In Patterson's second study implicating social understanding (exp. 3), individuals with psychopathy judged the outcome of an interaction between two individuals as more successful in some instances. There were no other significant group differences (Patterson also reported post hoc group differences in a several conditions in experiment 1 but the relevant interaction was not significant).

Table 2.14. *Studies which examined social understanding in psychopathy*

| Reference | Task | Special conditions | Primary outcomes | Result favoured |
|---|---|---|---|---|
| Patterson 1990 - Exp. 1 | Interpersonal interpretation (forced-choice response format) | Visual &/or auditory (non-verbal) video stimuli | Accuracy | ns overall, control in visual stimuli (positive & negative), individuals with psychopathy in positive auditory stimuli |
| | | Positive and negative content | | |
| - Exp. 3 | Interpersonal interpretation | Visual & auditory video stimuli | Successfulness of protagonist | Individuals with psychopathy higher ratings in favourable feedback and negative/favourable/reflective more specifically |
| | | Active/ Reflective conditions Favourable/ Unfavourable feedback | | |
| Richell 2005 | Judgment of trustworthiness | Facial stimuli | Performance score | ns |
| Snowden 2004/ Gray 2003 | Implicit Association | Verbal (written) | Errors | ns |
| | | Congruent & incongruent associations | Response latency | ns overall, murderers without psychopathy (incongruent items) |
| | | | IAT score | murderers without psychopathy & non-murderers |

*Note.* IAT=Implicit Association Task; ns=Not Significant.

#### 2.3.5.3 Summary

##### *2.3.5.3.1 ASPD & DPD*

Only two studies on DPD were available and reported a deficit in affect recognition, which appeared specific to the disorder. No deficits were present in affective processing (one study). Regarding ASPD, seven studies were identified. An overall meta-analysis on affective functions (processing, recognition, memory, expression) did not highlight any significant group differences. Of the various operations, no ASPD-specific deficits were identified in affective processing or memory. Differences in the narratives between individuals with ASPD and controls were highlighted within an affective context but these were not dissociable from language processes. On the other hand, there was evidence of impairment in emotion recognition in ASPD but publication bias appeared likely while sample confounders (criminality & psychopathy) in two studies limited conclusions.

More consistent evidence highlighted deficits in ASPD in connection to social cognition (two studies), although there was still some possibility of publication bias. These deficits appeared present in more complex operations such as mentalising and 2$^{nd}$ order inference during more realistic scenarios whereas no impairments were highlighted for 1$^{st}$ order inference.

In sum, DPD was associated with affect recognition difficulties. In ASPD, although an overall deficit in affective operations was not supported, the diagnosis was associated with difficulties in affect recognition. Publication bias and sampling confounders were detected. Additional findings indicated that affectively laden speech of individuals with ASPD appeared different to controls and there was somewhat robust evidence supporting impairment in social cognition in ASPD.

##### *2.3.5.3.2 Psychopathy*

###### *2.3.5.3.2.1 Affective operations*

Studies examined a diverse pool of affective operations and findings provided some relatively robust evidence for an overall impairment in psychopathy but publication bias was possible. Although variability across affective operations was

observed, deficits were highlighted in virtually all domains including processing, recognition, memory and language. Impairments in overall affective processing and lack of affective facilitation were highlighted in most studies in this category. A meta-analysis further supported a deficit but with strongest effects only whereas publication bias was possible.

Some evidence (strongest effects only) suggested impairment in overall affect recognition. Meta-analyses supported a deficit in happiness, sadness, disgust and surprise recognition (with possible publication bias) but did not support impairment in fear or anger recognition. In terms of different modalities, individuals with psychopathy showed some impairment in visual and verbal affect recognition (particularly for semantic information), but the evidence from the meta-analysis was limited and may be attributable to publication bias. In spite of the significant pooled evidence from meta-analyses, individual studies did not generally report deficits in recognition of specific emotions consistently. Furthermore, there were suggestions contradicting the notion of impairment in psychopathy. For instance, individuals with psychopathy performed better for right-handed responses in one study and during fear recognition in another.

Studies on memory and language were fewer than those on processing and recognition. Evidence suggested that affective content may not enhance verbal memory of individuals with psychopathy as much as controls. Although a meta-analysis supported impairment in visual memory of peripheral stimuli, the overall evidence was inconclusive due to confounding with ASPD in one study and due to presence of contradictory results which were also not available for meta-analysis. On the other hand, the evidence on affective language suggested impaired cohesion in psychopathy during neutral conditions but not anger. By contrast, individuals with psychopathy made more incompetent references when relating an angry incident on another occasion. Finally, participants with psychopathy appeared to speak more softly and showed less variation in voice amplitude during negative emotion rather than neutral speech.

*2.3.5.3.2.2 Social cognition*

This was yet another diverse cluster of operations including theory of mind, moral reasoning and general social understanding. Some deficits were identified in all these functions but with varying strength of evidence. Although no deficits were highlighted in mentalising and 2nd order inference, there was some (but limited) evidence of impaired 1st order inference ability. Evidence was also limited for moral reasoning but individuals with psychopathy appeared less able to make moral/conventional distinctions, were less influenced by authority in their views and were less likely to justify moral transgressions empathically.

In terms of general social understanding, psychopathy appeared to mediate negative associations with violence in offender groups. Evidence suggesting that individuals with psychopathy were different in their understanding of interpersonal situations compared to controls was very limited and no group differences were reported in judging trustworthiness in others' faces.

In sum, there was some robust evidence supporting an overall difficulty in affective operations in psychopathy. This was strongest in affective processing and recognition where difficulties in recognising specific emotions were also observed. In spite of this summative evidence from meta-analyses, some inconsistencies were present between individual studies. Although some evidence suggested group differences in affective memory (psychopathy difficulty) and speech, there were contradictions between studies in both operations. Regarding social cognition, research provided some yet limited evidence suggesting that individuals with psychopathy may experience difficulties during 1st order inference and moral reasoning. Psychopathy may also mediate negative attitudes towards violence and may reflect some differences in understanding social situations.

### 2.3.6 Memory

Studies examined short and long-term memory (STM & LTM respectively) for visual (inc. visuo-spatial) and verbal stimuli using both recall and recognition mechanisms. WM and priming effects were also investigated. An overview of the studies is presented in Table 2.15 and Table 2.16. Memory operations are presented separately for ASPD and psychopathy because differences in study foci between the two operationalisations resulted in different groupings.

Table 2.15. *Studies which examined memory in ASPD and psychopathy*

| Reference | Task | Memory | Memory access | Modality | Stimuli | Outcome | Result favoured |
|---|---|---|---|---|---|---|---|
| ***ASPD*** | | | | | | | |
| Barkataki 2005 | CPT - Identical pairs | STM: immediate | Recognition | Visual | 4-digit numbers | Errors | ns |
| | | | | | | Target/non-target discrimination | ns |
| | Logical Memory I (WMS-III) | STM: immediate | Recall | Verbal (oral) | Story | Performance score | ns |
| | Logical Memory II (WMS-III) | LTM | | | | Performance score | ns |
| Dolan 2002 | DMS | STM: immediate, 4 sec, 12 sec | Recognition | Visual | Patterns | Accuracy | Control (immediate & 4 sec) |
| | | | | | | Response latency | ns |
| Stevens 2003 | Digit Span (WAIS-R) | STM & WM | Recall | Verbal (oral) | Numbers | Performance score | ns |
| Swann 2009 | CPT - Identical pairs | STM: immediate | Recognition | Visual | 5-digit numbers | Correct detections | ns |
| | | | | | | Target/non- | ns |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | | target discrimination | |
| | | | | | | Commission errors | ns |
| | | | | | | Correct responses/ Commission errors ratio | ns |
| ***ASPD & psychopathy*** | | | | | | | |
| Dolan 2005 | Emotional Memory Task | LTM: 2 hours | Recall | Visual | Pictures depicting a story | Accuracy | ns |
| | | | | | | | Healthy control vs. high & low factor 1 scorers. |
| | | | Recognition, forced-choice | | | Accuracy | Healthy controls vs. high and medium psychopathy ASPD groups. |
| | | | | | | | Healthy control vs. high & low factor 1 scorers. |
| | | | | | | | Healthy control vs. high & low factor 2 scorers. |
| ***Psychopathy*** | | | | | | | |
| Bernstein 2000 | Verbal memory | STM | Recall | Verbal (written) | Words | Accuracy | ns |
| | Visuospatial memory | | Cued recall | Visuospatial (contextual) | Word locations | | Control (RVF) |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Christianson 1996 | Visual memory | STM: 5 minutes | Cued recall | Visual | Pictures | Accuracy (central) | ns |
| | | | | | | Accuracy (peripheral) | ns |
| | | | Cued recognition: forced-4-choice | | | Accuracy (central) | ns |
| | | | | | | Accuracy (peripheral) | ns |
| Glass 2009 | Verbal memory | STM: 5 min | Recall | Verbal (written) | Words | Accuracy (central) | ns |
| | Visuospatial memory | | Cued recall | Visuospatial (contextual) | Context & locations | Accuracy (contextual) | ns |
| Hare 1988 | Verbal memory | STM | Recognition | Verbal (written) | Words | Response latency | ns |
| | | | | | | Errors | ns |
| Hart 1990 | Visual Retention Test | STM: immediate | Recall | Visual | Line drawings | Correct reproductions | ns |
| | | | | | | Errors | ns |
| | Auditory-Verbal Learning Test | STM/Learning | Recall | Verbal (oral) | Words | Accuracy | ns |
| | | | | | | Loss | ns |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Ishikawa 2001 | Logical Memory I&II, Visual Reproduction I&II (WMS-R) | STM & LTM | Recall | Visual, Verbal (oral) | Story, Printed designs | Standard score | ns |
| Kiehl 2001 | Verbal memory | STM/Learning | Recognition | Verbal (written) | Words | Accuracy | ns |
| Mercer 2005 | Digit Span (WAIS-R) | STM & WM | Recall | Verbal (oral) | Numbers | Performance score | n/a |
| Newman 1990 - Study 3 | Visual recognition-matching | STM/ Immediate: 2 sec | Recognition | Visual | 6x6 grid pattern | Response latency | LA control in reward-only condition |
| Pham 2003 | Digit Span Forward (WAIS) | STM | Recall | Verbal (oral) | Numbers | Performance score | n/a |
| Raine 1988 | Digit Span Forward (WAIS-R) | STM | Recall | Verbal (oral) | Numbers | Performance score | n/a |
| Smith 1992 | Digit Span Forward (WAIS-R) | STM | Recall | Verbal (oral) | Numbers | Performance score | n/a |
| | Paired Associate Learning (WMS) | STM/Learning | Cued recall | Verbal (oral) | Words | Accuracy | n/a |

| | | | | | |
|---|---|---|---|---|---|
| | Paired Associate Learning (WMS) Delayed | LTM | Recall<br><br>Cued recall | Accuracy | n/a |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; STM/LTM=Short/long-term memory; WM=Working memory; CPT=Continuous Performance Task; DMS=Delayed Matching to Sample task; WAIS/-R=Wechsler Adult Intelligence Scale/-Revised; WMS=Wechsler Memory Scale; RVF=Right visual field; LA=Low-anxious; n/a=Not available; ns=Not significant.

#### 2.3.6.1 ASPD

There were six studies involving ASPD (Barkataki et al., 2005; Dolan & Fullam, 2005; Dolan & Park, 2002; Kumari et al., 2006; Stevens et al., 2003; Swann et al., 2009) including at least 168 individuals with the disorder and 97 without. Barkataki et al. (2005), Kumari et al. (2006), Dolan and Park (2002) and Dolan and Fullam (2005) recruited their ASPD samples from forensic psychiatric settings while the healthy control groups came from non-forensic populations; therefore, criminality was a confounder in these cases. The remaining studies recruited all participants from the general public.

##### *2.3.6.1.1 STM*

Of the four studies in this group (Barkataki et al., 2005; Dolan & Park, 2002; Stevens et al., 2003; Swann et al., 2009), Dolan and Park (2002) reported that healthy controls performed more accurately than forensic patients with ASPD in all recognition delays conditions except for the longest one, where the groups performed comparably. There were no further significant group differences.

A meta-analysis was conducted with the available data for verbal/recall and visual/recognition. Only the medium pooled effect size estimate for visual/recognition memory with strongest effects reached significance, with no heterogeneity. The failsafe *N* of 3.39 was below the critical value of 25, indicating likely publication bias. Weakest effects resulted in a small and non-significant pooled effect size estimate, 0.17[-0.44,0.77], with substantial heterogeneity. For verbal/recall memory, the pooled effect size estimate was small and did not reach significance.

An overall meta-analysis for STM with strongest effects produced a small and significant pooled effect size estimate with non-significant heterogeneity. The pooled effect size estimate was smaller and not significant with weakest effects, 0.12[-0.29,0.52], with marginally significant, moderate heterogeneity.

##### *2.3.6.1.2 LTM*

Dolan and Fullam (2005) used the emotional memory task described earlier in the context of affective memory. However, the focus in this section was on the non-

emotional items. They found an advantage in the healthy control group on recall and recognition against individuals with ASPD and psychopathy. Barkataki et al. (2005) did not report any significant differences.

An overall meta-analysis was conducted with Barkataki et al.'s (2005) verbal recall and Dolan and Fullam's (2005) visual recall and recognition data. Data associated with stimuli presented prior to the affective manipulation in Dolan and Fullam were preferred to avoid possible confounding of emotional influence on stimulus processing and encoding. Inclusion of either recall or recognition data in sensitivity analysis from Dolan and Fullam produced a small and medium pooled effect size estimate respectively, both failing to reach significance. The recall/recognition sample pooling was associated with marginally significant, substantial heterogeneity.

#### *2.3.6.1.3 WM*

The two studies on WM (Barkataki et al., 2005; Kumari et al., 2006) examined overlapping samples. Barkataki et al. adopted a self-ordering WM task in which participants had to remember and not return to previously successful locations. Kumari et al. employed a version of the n-back mental tracking task. Neither study observed a significant difference between groups.

#### *2.3.6.1.4 Overall meta-analysis*

For overall memory function in ASPD, strongest effects included the studies on STM. As discussed before, the result was a significant small to medium pooled effect size estimate with non-significant heterogeneity with the failsafe *N* indicating possible publication bias. On the other hand, weakest effects yielded a small and non-significant pooled effect size estimate, 0.08[-0.35,0.51], while heterogeneity was moderate.

#### 2.3.6.2 Psychopathy

Sixteen studies focused on psychopathy (Bernstein, Newman, Wallace, & Luh, 2000; Christianson et al., 1996; Dolan & Fullam, 2005; Glass & Newman, 2009; Hare & Jutai, 1988; Hart et al., 1990; Ishikawa et al., 2001; Kiehl et al., 2001; Mercer et al., 2005; Newman et al., 1990; Pham et al., 2003; Raine & Venables, 1988; S. S. Smith et al., 1992). They included at least 396 individuals with psychopathy and 595 without. The majority of samples were recruited from prisons except those of Ishikawa et al. (2001) and the healthy control samples of Hare and Jutai (1988) and Kiehl et al. (2001) all of which originated from the general public. Ishikawa et al. included both an unsuccessful (criminal history) and successful (no criminal history) group with psychopathy. Most studies focused on various aspects of STM, thus results are presented by sensory modality instead of memory type. STM versus LTM contrasts and priming effects are also outlined.

##### *2.3.6.2.1 Visual memory*

Seven studies were in this group. The studies of Glass and Newman (2009), Christianson et al. (1996) and Dolan and Fullam (2005) also contained affective components. However, only non-affective memory data were considered in this section.

Dolan and Fullam (2005) observed a deficit in individuals with high psychopathy scores within the ASPD group compared with healthy controls (general public) on both long-term recognition and recall. However, the ASPD subgroups with and without psychopathy performed comparably. Bernstein et al. (2000) suggested a deficit in visuospatial STM (cued recall) but for RVF targets only. However, attentional processes may have mediated this as the recalled elements were not in focus during presentation. Glass and Newman (2009) adopted a similar paradigm but examined overall (non-lateralised) performance, failing to detect a deficit in psychopathy. Of the remaining studies, Newman et al. (1990) did not report memory data (focus was on passive avoidance). Ishikawa et al. (2001) did not indicate significant group differences but provided a composite score of their memory tasks including short and long-term components. As a consequence, it is not possible to

draw inferences in connection to either memory type. No further significant differences were reported.

A meta-analysis with strongest effects resulted in a small and non-significant pooled effect size estimate with no heterogeneity. Weakest effects also resulted in a small and non-significant pooled estimate, -0.02[-0.36,0.32], with comparable heterogeneity.

#### *2.3.6.2.2 Verbal memory*

Nine publications examined verbal memory in psychopathy (Bernstein et al., 2000; Glass & Newman, 2009; Hart et al., 1990; Jutai, Hare, & Connolly, 1987; Kiehl et al., 2001; Mercer et al., 2005; Pham et al., 2003; Raine & Venables, 1988; S. S. Smith et al., 1992). Of these, only S. S. Smith et al. investigated LTM. Mercer et al. (2005), Pham et al. (2003), Raine and Venables (1988) and S. S. Smith et al. (1992) did not report statistical comparisons. Hare and Jutai (1988) and Kiehl et al. (2001) employed two tasks involving verbal recognition but as the focus of these studies was not on memory the tasks were not challenging thereby ceiling effects were likely. No significant group differences were reported in any study.

A meta-analysis with strongest effects revealed a small and significant pooled effect size estimate with no heterogeneity. The failsafe *N* of -0.72 was below the critical value of 40. The funnel plot was not symmetrical either (Appendix D, Figure 12.11) and in conjunction with the failsafe *N* suggests likely publication bias. This is further supported by the observation that the significant pooled estimate was primarily due to the contribution of Mercer et al. (2005). By excluding this study, the pooled effect size estimate became small and did not reach significance, -0.11[-0.42,0.19], with comparable heterogeneity. With weakest effects, there was a small and non-significant pooled effect estimate, 0.10[-0.08,0.28], with substantial heterogeneity.

#### *2.3.6.2.3 STM and LTM*

A meta-analysis was also conducted stratifying data according to STM and LTM. Strongest effects resulted in small pooled effect size estimates significant for STM only with no heterogeneity. Once again, the major contributor was the study of

Mercer et al. (2005) and publication bias was likely with a failsafe *N* of -3.0 below the critical value of 40 and an asymmetrical funnel plot (Appendix D, Figure 12.11). Exclusion of Mercer et al. resulted in a small and non-significant pooled estimate,-0.05[-0.36,0.25], with comparable heterogeneity.

Weakest effects resulted in non-significant pooled estimates. For STM, it was small,-0.11[-0.29,0.07], with moderate heterogeneity. On the other hand, the pooled estimate for LTM was small to medium in favour of the antisocial group but only approached significance,0.34[-0.03,0.71],*P*=0.07, with substantial heterogeneity.

#### *2.3.6.2.4 WM*

The two studies examining WM were by Pham et al. (2003) and S. S. Smith et al. (1992). Mercer et al. (2005) also used this task but supplied a composite score from the STM and WM variations, thus representing STM more heavily. Pham et al. did not report statistical comparisons whereas S. S. Smith et al. did not observe a significant group difference. A meta-analysis produced a small and non-significant pooled effect size estimate with no heterogeneity.

#### *2.3.6.2.5 Implicit memory: priming*

Priming is considered a form of implicit memory (Gazzaniga et al., 2009; Strauss et al., 2006). Three publications reported on priming in their tasks (Table 2.16). There were 59 individuals with psychopathy and 63 without. Two studies (Drugge, 1998; Harpur, 1993) involved processes in which distractor stimuli became targets in the subsequent trials (negative priming) within Stroop colour-word and flanker-type paradigms. Brinkley et al. (2005) employed semantic priming to lexical decisions. The only significant effect was observed by Drugge (1998) who presented evidence supporting greater negative priming among individuals with psychopathy and high PCL-R factor 2 scorers in two different tasks. However, the effect in relation to factor 2 was less reliable in the second task as the result became significant in a one-tailed test only.

Table 2.16. *Studies which examined priming effects in psychopathy*

| Reference | Task | Task features | Primary outcomes | Results |
|---|---|---|---|---|
| Brinkley 2005 - Exp. 1 | Lexical decision | Semantic priming<br>Two prime-target delays | Response latency | ns |
| Drugge 1998 | Modified colour-word Stroop with negative priming | Baseline: Non-letter strings | Interference | ns |
| | | Negative priming | Response latency | ns |
| | | | Priming effect | Greater for individuals with psychopathy & high factor 2 scorers |
| | Modified flanker: Word naming with priming | | Accuracy | ns |
| | | | Correct response latency | ns |
| | | | Priming effect | Greater for individuals with psychopathy & high factor 2 scorers |
| Harpur 1991 - Exp. 3 | Modified colour-word Stroop with additional habituation and negative priming conditions | Negative priming | Correct response latency | ns |
| | | Habituation | Accuracy (interference condition) | ns |
| - Exp. 4 | Flanker-type with negative priming | Negative or no priming | Response latency | ns |
| | | | Accuracy | ns[a] |

| | | | | |
|---|---|---|---|---|
| - Exp. 5 | Flanker-type with negative priming | Negative or no priming | Response latency | ns |
| | | Variable spatial proximity of targets and distractors | Accuracy | ns |

*Note*. ns=Not significant.

[a]Significant Group x Condition interaction for accuracy but distributions were heavily skewed and result is likely to be due to outlier data – author disputed the result of ANOVA.

#### *2.3.6.2.6 Overall meta-analysis for psychopathy*

Strongest effects resulted in a small yet significant pooled effect size estimate in favour of the control group, with no heterogeneity. Data from Mercer et al. (2005) influenced the result the most as the pooled effect size estimate decreased and was no longer significant when this study was excluded,-0.20[-0.44,0.05], with comparable heterogeneity. Additionally, the failsafe *N* of 6.40 (for the original analysis sample) was very much below the critical value of 50. In conjunction with an asymmetrical funnel plot (Appendix D, Figure 12.12), it suggests likely publication bias.

Weakest effects resulted in a small and non-significant pooled effect size estimate in the opposite direction,0.15[-0.18,0.49] with substantial heterogeneity. When the data of Mercer et al. (2005) were excluded, heterogeneity was no longer significant and the small pooled effect size estimate approached significance, 0.26[-0.02,0.48], in favour of the antisocial group. Stratification according to study quality did not reveal any subgroup differences.

#### 2.3.6.3 Summary

##### *2.3.6.3.1 ASPD*

An overall meta-analysis highlighted a memory deficit in ASPD but strongest effects involved STM processes and publication bias was detected. When meta-analyses examined STM and LTM separately, some evidence suggested impairment in ASPD during short-term visual recognition but with publications bias whereas a deficit in LTM was not supported. However, there was evidence from individual studies suggesting that individuals with ASPD performed worse than controls in tasks involving STM (shorter delays) and visual LTM. Sampling bias and presence of psychopathy confounded these results whereas WM did not appear impaired in ASPD.

##### *2.3.6.3.2 Psychopathy*

Overall, there was some evidence in support of a memory deficit. However, this was not robust as there was a likelihood of publication bias also involving the contribution by Mercer et al. (2005). The same pattern occurred for STM while indications of impairments in LTM in one study were confounded with ASPD. There was limited evidence indicating deficit in visual memory whereas no reliable evidence in verbal memory emerged. WM did not appear impaired either.

### 2.3.7 Attention

In total, three publications on ASPD included 81 individuals with the diagnosis and 77 controls. A further 30 publications and dissertations examined psychopathy and included at least 607 individuals with psychopathy and 696 controls. One of the studies on psychopathy also examined DPD. Sustained, selective, divided, complex attention and reaction time paradigms were employed.

#### 2.3.7.1 Sustained attention

There were two studies on ASPD including 47 individuals with the diagnosis and 45 without (Barkataki et al., 2005; Swann et al., 2009). Ten studies on sustained attention in connection to psychopathy included at least 192 individuals with psychopathy and 212 without (Howard & McCullagh, 2007; Jutai et al., 1987; Kiehl, Bates, Laurens, Hare, & Liddle, 2006; Kiehl, Hare, Liddle, & McDonald, 1999; Kosson, 1996, 1998; Llanes & Kosson, 2006; Mills, 1995; Pham et al., 2003; Raine & Venables, 1988). An overview of studies is presented in Table 2.17.

Table 2.17. *Studies which examined sustained attention in ASPD and psychopathy*

| Reference | Task | Special features | Modality | Primary outcomes | Result favoured |
|---|---|---|---|---|---|
| ***ASPD*** | | | | | |
| Barkataki 2005 | CPT - Identical pairs | | Visual (numerals) | Errors | ns |
| | | | | Target/non-target discrimination | ns |
| | Adult Memory and Information Processing Battery (cancellation) | | Visual (numerals) | Adjusted score | Control |
| | | | | Accuracy | ns |
| | | | | Motor speed score | ns |
| Swann 2009 | CPT - Identical pairs | | Visual (numerals) | Correct detections | ns |
| | | | | Commission errors | ns |
| | | | | Correct responses/Commission errors ratio | ns |
| | | | | Correct response latency | ns |
| | | | | Commission error response latency | ns |
| | | | | Target/non-target discrimination | ns |
| | | | | Response bias | ns |
| ***Psychopathy*** | | | | | |

| | | | | | |
|---|---|---|---|---|---|
| Howard 2007 | CPT - oddball | Affective picture interlaced as distractors | Visual | Response latency | ns |
| | | | | Omission Errors | Favours control in high valence/low arousal and low valence/high arousal conditions |
| | | | | Commission Errors | ns |
| Jutai 1987 | CPT - oddball | | Auditory (verbal) | Accuracy | ns |
| | | | | Commission errors | ns |
| | Game | | Visual | Accuracy | ns |
| Kiehl, Bates 2006 | CPT - oddball | Two distractor types: novel & standard | Auditory | Correct responses (%) | ns |
| | | | | Response latency | ns |
| | | | | Commission errors to novel stimuli | ns |
| | | | | Commission errors to standard stimuli | ns |
| Kiehl, Hare, Liddle 1999 | CPT - oddball | | Visual | Response latency | ns |
| | | | | Accuracy | ns |
| | | | | Commission errors | ns |

| | | | | | |
|---|---|---|---|---|---|
| Kosson 1996 | Target discrimination | Consonants, numbers or mixed stimuli | Visual: consonants, numbers or mixed characters | Accuracy | ns |
| | | | | Commission errors | ns |
| | | | | Response latency | ns |
| | | | Auditory: ascending, constant, or mixed pitch tones | Accuracy | ns |
| | | | | Commission errors | ns |
| | | | | Response latency | ns |
| Kosson 1998 | Target discrimination | | Visual: consonants, numbers or mixed characters | Accuracy | ns |
| | | | | Response latency | ns |
| Llanes 2006 | Target discrimination: green, blue or mixed stimuli | LHA condition | Visual: green, red or mixed-colour characters | Accuracy | ns |
| | | | | Response latency | ns |
| | | EA condition | | Accuracy | ns |
| | | | | Response latency | Control |
| Mills 1995 | Letter cancellation | | Visual (verbal) | Omission errors | ns |
| | Symbol cancellation | | Visual | Omission errors | ns |

| | | | | |
|---|---|---|---|---|
| Pham 2003 | Letter cancellation | Visual (verbal) | Total items read in 20s | ns |
| | | | Omission errors | n/a |
| | | | Commission errors | ns |
| | | | Errors (%) | Control |
| | | | Performance variation | Control |
| Raine 1988 | CPT - oddball | Visual (numerals) | Accuracy | ns |
| | | | Omission errors | ns |
| | | | Commission errors | ns |
| | | | Response latency (for hits only) | ns |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; CPT=Continuous Performance Task; LHA/EA=Left hemisphere/equal activation; n/a=Not available; ns=Not significant.

#### *2.3.7.1.1 ASPD*

Barkataki et al. (2005) recruited the ASPD group from forensic psychiatric facilities and the control group from the general public, thus criminality may have been a confounder. Swann et al. (2009) recruited all groups from the general public. Neither study observed significant performance differences between the groups on CPTs. Barkataki et al. suggested that the antisocial group performed worse than the control group on a cancellation paradigm.

A meta-analysis with strongest effects yielded a significant medium to large pooled effect size estimate with non-significant heterogeneity in favour of controls. The failsafe *N* of 4.95 did not exceed the critical value of 20, suggesting possible publication bias. In addition, weakest effects resulted in a non-significant small pooled effect size estimate (in the opposite direction), -0.36[-0.78, 0.06], with no heterogeneity.

#### *2.3.7.1.2 Psychopathy*

The majority of studies adopted a CPT/oddball paradigm but cancellation and target discrimination tasks were also employed. Llanes and Kosson (2006) included LHA and equal activation (EA) conditions using a target frequency manipulation in each visual field. Responding was also lateralised for LVF and RVF targets. Only three studies reported significant differences between the groups. Howard and McCullagh (2007) suggested that controls performed better than the antisocial group during high emotional valence/low arousal and low valence/high arousal trials only, but this could be an effect of the affective manipulation (as discussed in the relevant section earlier). Pham et al. (2003) presented evidence showing that controls committed fewer errors overall and demonstrated more consistent performance than individuals with psychopathy. However, since these results originated from a cancellation test, performance was also dependent on visual processing and response inhibition. Finally, Llanes and Kosson (2006) highlighted that controls responded faster during EA but there was a trend for greater accuracy among individuals with psychopathy. The authors interpreted this result as a probable speed-accuracy trade-off effect rather than a group difference in cognitive function.

Meta-analysis with strongest effects resulted in a significant small to medium pooled effect size estimate with no heterogeneity. The failsafe *N* of 23.55 was below the critical value of 40. In addition, there was some asymmetry in the funnel plot (Appendix D, Figure 12.13) suggesting presence of publication bias. Weakest effects resulted in a small and non-significant pooled effect size estimate in the opposite direction, -0.25[-0.51,0.01], with non-significant heterogeneity. Stratification by study quality did not indicate significant study subgroup differences.

### 2.3.7.2 Selective Attention

#### *2.3.7.2.1 Stroop (1935) paradigms*

Studies using the Stroop paradigms were summarised earlier in the context of response inhibition and effective performance (Table 2.6). Only one of the eleven publication investigated ASPD while the remaining studies examined psychopathy. Accuracy/errors, response latencies and interference were discussed. Facilitation from peripheral stimuli is an additional outcome measure relevant to selective attention.

The additional data emerged from colour-word, box, and semantic Stroop paradigms. Two studies included conditions of facilitation in a colour-word Stroop paradigm (Brinkley et al., 2005; Schmitt, 2000). Brinkley et al. (2005) reported fewer errors in individuals with psychopathy when colour stimuli (colour-word) were congruent (facilitation condition). They also indicated that the LA group with psychopathy experienced more facilitation and appeared to have made fewer errors than controls in those trials (significant interaction but no post hoc analysis was reported). Regarding the box Stroop paradigm, neither Hiatt et al. (2004) nor Schmitt (2000) reported significant group differences in facilitation. Furthermore, the only significant group difference in the colour/colour-related word trials (semantic Stroop) was reported by Brinkley et al. (2005) where controls made fewer errors during facilitation (congruent colour-word) trials (significant interaction but no post hoc analysis was reported).

Although semantic Stroop data are confounded with semantic processing, semantic trial data were included in the meta-analysis of facilitation data in connection with Caucasian samples from Brinkley et al. (2005)/Schmitt (2000) because no other Stroop task data were available. The result of the meta-analysis was a small and non-significant pooled effect size estimate with moderate heterogeneity. Results varied between Caucasian (Brinkley et al., 2005/Schmitt, 2000) and African-American participants (Schmitt, 2000), with Caucasian controls exhibiting less facilitation (and thus stronger selective attention) than their counterparts with psychopathy.

#### *2.3.7.2.2 Non-Stroop paradigms*

Other selective attention tasks involved conflicting stimuli with the aim of disrupting task performance and a dichotic listening task (Table 2.7). Conflict resolution tasks were discussed in the section on effective performance. Of those studies, only Zeier et al. (2009) examined facilitation from peripheral information but did not suggest a significant performance difference between groups. No group differences were observed in accuracy during the single-target dichotic listening task with interfering distractors employed by Mills (1995). Additional data to extend the previous meta-analysis were not available.

#### 2.3.7.3 Divided attention paradigms

Such paradigms featured in six publications, all on psychopathy, (Hiatt et al., 2002; Jutai et al., 1987; Kosson, 1996, 1998; Llanes & Kosson, 2006; Suchy & Kosson, 2005), and included 119 individuals with psychopathy and 162 without from prison populations. An overview of studies is presented in Table 2.18.

Five studies reported significant group differences, two of which in favour of psychopathy. Hiatt et al. (2002) observed that individuals with psychopathy performed better in recognising emotional targets presented in the right ear only whereas there were no differences for non-emotional targets. Participants with psychopathy were more accurate but slower in the EA condition of Llanes and Kosson (2006).

On the other hand, four studies reported results in favour of the control groups. Suchy and Kosson (2005) concluded that controls committed fewer false alarms and responded faster in the LHA condition only. Controls also exhibited fewer false alarms than individuals with psychopathy in the auditory task of Kosson (1996) and both tasks of Kosson (1998). In addition, controls were more accurate for RVF targets only in the LHA condition of Llanes and Kosson (2006). As highlighted above controls were also less accurate but faster than individuals with psychopathy in the EA condition which may reflect a speed-accuracy trade-off.

For the purpose of meta-analysis, greater accuracy represented good performance. Data from the non-emotional condition of Hiatt et al. (2002) were preferred to avoid confounding. Furthermore, only right visual file data from the EA condition of Llanes and Kosson (2006) were available. This was the condition which favoured the group with psychopathy whereas data for LHA where controls performed better were not available for meta-analysis. With strongest effects, the resulting medium pooled effect size estimate was significant and heterogeneity was moderate. The failsafe *N* of 13.44 was below the critical value of 30, therefore indicating possible publication bias. In addition, weakest effects resulted in a small and non-significant pooled effect size estimate, 0.04[-0.65,0.74), with considerable heterogeneity.

Table 2.18. *Studies which examined divided attention in psychopathy*

| Reference | Task | Modality | Special features | Primary outcomes | Result favoured |
|---|---|---|---|---|---|
| Hiatt 2002 | Dichotic Listening Task | Auditory | Non-emotional targets | Accuracy | ns |
| | | | | Commission errors | ns |
| | | | | Laterality | ns |
| | | | Emotional targets: happiness, sadness, anger | Accuracy | ns overall |
| | | | | | Antisocial for right-ear targets |
| | | | | Commission errors | ns |
| | | | | Laterality | ns |
| Jutai 1987 | CPT - oddball | Auditory (verbal) | | Accuracy | ns |
| | | | | Commission errors | ns |
| | Game | Visual | | Accuracy | ns |
| | | | (For all tasks in Kosson, 1996) | | |
| Kosson 1996 | Primary task: target discrimination | Visual | Early & late SOAs | Accuracy | ns |
| | | | Significance condition: visual targets were twice as relevant as auditory targets | Commission errors | ns |
| | | | Frequency condition: visual tasks were twice as frequent as auditory task events | Response latency | ns |

| | | | | | |
|---|---|---|---|---|---|
| | Secondary task: target discrimination | Auditory | | Accuracy | ns |
| | | | | Commission errors | Control |
| | | | | Response latency | ns |
| | | | (All tasks) | | |
| Kosson 1998 | Primary task: target discrimination | Visual (letters) | Relatively focused attention condition: Primary and secondary task - one stimulus set with 67% and one with 36% targets respectively | Accuracy | ns overall, control on improvement from relatively focused to equally divided attention |
| | | | Equally divided attention condition: same number of targets and distractors | Commission errors | Control |
| | | | Early & late SOAs | Response latency | ns |
| | Secondary task: target discrimination | | | Accuracy | Control overall; control in relatively focused attention with LHA; antisocial on improvement from relatively focused to equally divided attention |
| | | | | Commission errors | Control |
| | | | | Response latency | ns |
| | | | (All tasks) | | |
| Llanes 2006 | Dual: target discrimination | Visual | LHA condition: 89% RVF, 41% LVF targets | Accuracy | Control for RVF targets |
| | | | | Response latency | ns |

| | | | | | |
|---|---|---|---|---|---|
| | | | EA condition: Targets & distractors presented in equal frequency in either visual field | Accuracy | Antisocial for RVF targets |
| | | | | Response late ncy | Control (LVF) |
| Suchy 2005 | Dichotic Listening Task | Auditory | LHA: 67% targets in right ear & 67% distractors in left ear | Accuracy | ns |
| | | | RHA: 67% targets in left ear & 67% distractors in right ear | Commission errors | Control (LHA only) |
| | | | | Response latency | Control (LHA only) |

*Note.* Highlighted outcomes contributed to meta-analyses; CPT=Continuous Performance Task; SOA=Stimulus onset asynchrony; LHA/RHA/EA=Left/right hemisphere/equal activation; RVF/LVF=Right/left visual field; ns=Not significant.

#### 2.3.7.4 Complex attention

An overview of the studies is presented in Table 2.19. There was only one study investigating ASPD (Stevens et al., 2003) which failed to find a significant effect. Six studies on psychopathy (Hart et al., 1990; Jozef & da Silva, 1999; Mercer et al., 2005; Pham et al., 2003; Raine & Venables, 1988; S. S. Smith et al., 1992) included at least 255 individuals with psychopathy and at least 299 without, all from prison settings. Statistical comparisons were not reported in all studies and only two highlighted significant group differences. Jozef and da Silva (1999) indicated that fewer individuals with psychopathy showed impaired performance on the TMT (Parts A & B combined) compared to controls while S. S. Smith et al. (1992) concluded that controls performed better than individuals with psychopathy in Part B of the test only.

Table 2.19. *Studies which employed complex attention paradigms including the TMT and Digit Symbol*

| Reference | Task | Primary outcomes | Result Favoured |
|---|---|---|---|
| ***ASPD*** | | | |
| Stevens 2003 | TMT A | Completion time | n/a |
| | TMT B | Completion time | ns |
| ***Psychopathy*** | | | |
| Hart 1990 | TMT A | Completion time | ns |
| | TMT B | Completion time | ns |
| Jozef 1999 | Digit symbol (WAIS) | Performance score | n/a |
| | TMT A&B | | Antisocial |
| | | Completion time part B-A | Antisocial |
| Mercer 2005 | Digit symbol (WAIS-R) | Performance score | n/a |
| | TMT A | Completion time | n/a |
| | TMT B | Completion time | n/a |
| Pham 2003 | Digit symbol (WAIS) | Performance score | n/a |
| | TMT A | Completion time | ns |
| | | Errors | ns |
| | TMT B | Completion time | ns |
| | | Errors | ns |
| Raine 1988 | Digit symbol (WAIS-R) | Performance score | n/a |
| Smith 1992 | TMT A | Completion time | ns |
| | TMT B | Completion time | Control (LA) |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; TMT=Trail Making Test; WAIS/-R=Wechsler Intelligence Scale/-Revised; LA=Low-anxious; n/a=Not available; ns=Not significant.

An overall meta-analysis of strongest effects with sensitivity analysis for TMT, Parts A and B resulted in a small pooled effect size estimate for the former and a medium pooled estimate for the latter neither of which reached significance, with considerable heterogeneity. Weakest effects resulted in small and non-significant pooled effect size estimates for both Part A and B, -0.09[-0.26,0.09], and 0.03[-

0.30,0.36], respectively. Heterogeneity was not significant for Part A and was moderate for Part B.

#### 2.3.7.5 Reaction time

No study was dedicated to investigating reaction time per se. Although many studies in this review measured response latencies, these were not suitable for the evaluation of reaction time as participant responses were not aimed to be as quick as possible. However, one of the identified publications (Forth & Hare, 1989) explored electroencephalic waves during a task involving what seemed to be valid measurement of reaction time as faster responses maximised winnings and minimised losses. Participants were presented with two tones (6-second interval) and had to respond to the second one on every occasion. The first tone provided information on whether the subsequent response outcome would be a reward, punishment or neither. No significant group differences were observed.

### 2.3.7.6 Meta-analysis on attention

#### *2.3.7.6.1 ASPD*

The Stroop task effects from Barkataki et al. (2005) were the only additional data to the previous meta-analysis on sustained attention with CPT and cancellation paradigms. However, the Stroop-related results lay within the range of strongest and weakest effects used in the previous meta-analysis.

#### *2.3.7.6.2 Psychopathy*

Several attention tasks involved other cognitive processes (e.g. executive, visuo-motor, etc). Minimising confounding from these non-attentional processes was achieved by selecting tasks and outcomes more relevant to attention when multiple sets of data were available from the same samples (e.g. TMT A was preferred over TMT B, Stroop errors over interference or facilitation).

Strongest effects resulted in a significant small to medium pooled effect size estimate in favour of controls, with moderate heterogeneity. The failsafe *N* of 138.48 exceeded the critical value of 100 but with some asymmetry in the funnel plot (Appendix D, Figure 12.13) thus suggesting possible publication bias. Significant differences between different study quality groups were observed. Studies of high and low quality contributed smaller pooled estimates overall. However, IQ and study quality score were not significantly correlated with effect sizes, *rho*=-0.33,*n*=15, and *rho*=0.06,*n*=20, respectively.

Weakest effects resulted in a small and non-significant pooled estimate, -0.17[-0.46,0.12], with substantial heterogeneity. Studies with high quality ratings yielded a significant small to medium pooled effect size estimate in favour of individuals with psychopathy, -0.31[-0.56,-0.06], with no heterogeneity. Studies with a medium and low quality rating did not yield a significant pooled effect size estimate.

#### 2.3.7.7 Summary

##### *2.3.7.7.1 ASPD*

Studies examined sustained and complex attention. Except sustained attention, meta-analyses did not yield significant pooled estimates. Some evidence of ASPD deficit appeared in sustained attention with use of a cancellation task but this was not supported with CPTs while the evidence provided by the relevant meta-analysis was not robust.

##### *2.3.7.7.2 Psychopathy*

Sustained, selective, divided and complex attention processes and reaction time were investigated. There was some evidence of an overall impairment in attention for psychopathy but studies of high and low quality contributed smaller effects. However, data in the opposite direction from studies of higher quality rating also supported better performance in psychopathy compared to controls. No group differences emerged in reaction time (one study) and evidence did not support a deficit in complex attention processes. Results in other attentional operations varied.

Some evidence indicated a deficit in sustained attention for psychopathy. This was limited by confounding with affective and visual processing or speed-accuracy trade-offs. A meta-analysis produced a significant pooled estimate in favour of controls with strongest effects with possible publication bias but weakest effects marginally favoured individuals with psychopathy.

The evidence was inconclusive for selective attention and meta-analyses did not support impairment in psychopathy. Tasks employing selective attention processes included Stroop and Flanker paradigms which also implicate effective performance processes. Results from response latency, interference and error data in these paradigms were inconclusive. Facilitation in these tasks was now relevant to selective attention but relevant significant results were conflicting between the colour-work and semantic Stroop. No further group differences were highlighted.

Finally, some contradictions existed in divided attention operations. One study indicated that the performance of individuals with psychopathy was enhanced for right-sided targets (some confounding with emotional processing was present) while

in other studies the evidence suggested impairment in these individuals in left hemisphere performance and during dual-task paradigms. Nevertheless, a meta-analysis supported a deficit in psychopathy but with strongest effects only and possible publication bias.

In sum, there was some evidence to support an overall impairment in attention in psychopathy but effects from higher quality studies were contradictory. Difficulties were observed in sustained and divided attention processes but possible biases were identified and some conflicting findings for the latter were highlighted. The review of research on selective attention was less conclusive but a deficit was not generally supported. Evidence did not support impairment in either reaction time or complex attention processes.

### 2.3.8 Language

Overall, five studies examined ASPD including at least 249 individuals with the diagnosis and 283 without. A total of 35 publications and dissertations investigated language in psychopathy including at least 460 individuals with psychopathy and 713 controls.

#### 2.3.8.1 Verbal expression

In terms of verbal expression, six publications investigated verbal fluency, eight examined vocabulary, six explored discourse and one focused on writing. Of these, two studies involved ASPD including 94 individuals with the diagnosis and 172 controls. A further 17 publications or dissertations examined psychopathy with at least 437 individuals with psychopathy and 683 controls. Tasks of verbal fluency were discussed within the section on productivity (executive functions) earlier.

There were eight studies on vocabulary, all on psychopathy, which included at least 341 individuals with psychopathy and 587 without. An overview is presented in Table 2.20. Four studies did not indicate any significant group differences whereas the remaining ones did not report any statistical comparisons.

Table 2.20. *Studies which examined vocabulary in psychopathy*

| Reference | Task | Result favoured |
|---|---|---|
| Drugge 1998 | Vocabulary (SILS) | n/a |
| Gillstrom 1995 | Vocabulary (WAIS-R) | ns |
| Hart 1990 | Vocabulary (WAIS-R) | ns |
| Johansson 2005 | Synonyms (Dureman-Sälde) | ns |
| Kosson 1998 | Vocabulary (SILS) | ns |
| Mercer 2005 | Vocabulary (WAIS-R) | n/a |
| Pham 2003 | Vocabulary (WAIS) | n/a |
| Raine 1988 | Vocabulary (WAIS-R) | n/a |

*Note.* Highlighted tests contributed to meta-analyses; SILS=Shipley Institute of Living Scale; WAIS/-R=Wechsler Adult Intelligence Scale/-Revised; n/a=Not available; ns=Not significant.

With the available data, a meta-analysis revealed a small and non-significant pooled effect size estimate with relatively inconsequential heterogeneity. The main source of heterogeneity appeared to be Mercer et al. (2005) with an effect in the opposite direction to the other studies. A random-effects model was employed in order to moderate bias from that study by assigning a smaller weight. When removing the study altogether, heterogeneity decreased to minimal levels and the pooled effect size estimate was not significant,-0.07[-0.35, 0.20]. Study quality ratings not seem to be associated with effect sizes (visual inspection).

#### *2.3.8.1.1 Discourse and writing*

There was one study on ASPD (Gawda, 2008b) discussed earlier in the context on affective processes. For psychopathy, six studies on discourse (oral narratives) investigated features of speech produced as a semi-structured narrative (Brinkley, Bernstein, & Newman, 1999; Brinkley, Newman et al., 1999; Klaver et al., 2007; Lee, Klaver, & Hart, 2008; Louth et al., 1998; Williamson, 1991). An overview is presented in Table 2.21. Studies included at least 59 individuals with psychopathy and 79 without, all from prison settings.

The studies on syntax examined various aspects of coherence and cohesion. Of these, Brinkley, Bernstein and Newman (1999, Caucasian sample only), Lee et al. (2008) and Williamson (1991) presented some evidence that the narratives of the control group were overall more coherent (closed to open plot units ratio) and had greater cohesion (greater total cohesion in the non-affective condition and fewer incompetent references in the affective condition) compared to those by individuals with psychopathy.

Table 2.21. *Studies which examined discourse and writing in ASPD and psychopathy*

| Reference | Format | Feature | Task | Special features | Primary outcomes | Result favoured |
|---|---|---|---|---|---|---|
| ***ASPD*** | | | | | | |
| Gawda 2008b | Written | Content | Semi-structured narrative generation | Romantic narrative | Actors | Higher in ASPD |
| | | | | | Actor traits | Higher in ASPD |
| | | | | | Strong emotions | Higher in ASPD |
| | | | | | Length | Higher in ASPD |
| | | | | | Actions | Higher in ASPD |
| | | | | | Presumptions | Higher in ASPD |
| | | | | | Wishes | Higher in ASPD |
| | | | | | Self-concentrating referents | Higher in ASPD |
| ***Psychopathy*** | | | | | | |
| Brinkley, Bernstein 1999 | Oral | Syntax | Semi-structured narrative generation Non-personal events | Aided or unaided | Closed units (adjusted for opened units) | Control (Caucasian sample only) |
| | | | | | Opened units | ns |
| Brinkley, Newman 1999 | Oral | Syntax | Semi-structured narrative generation Personal events | Anger & fear-related stories | Cohesion | ns |
| | | | | | Words | ns |
| | | | | | Clauses | ns |
| | | | | | Incompetent references | ns |

| | | | | | | |
|---|---|---|---|---|---|---|
| Klaver 2007 | Oral | Physical features of speech | Semi-structured narrative generation | True & deceptive crime accounts | Pauses | ns |
| | | | Personal events | | Shifts | ns |
| | | | | | Speech rate | ns |
| | | | | | Hesitations | ns |
| | | | | | Speech errors | ns |
| Lee 2008 | Oral | Syntax | Semi-structured narrative generation | True & deceptive crime accounts | Coherence | Control |
| | | Content | Personal events | | Overall | ns |
| | | | | | Spontaneous reproduction | ns |
| | | | | | Appropriate detail | Higher for individuals with psychopathy during deception |
| | | | | | Contextual embedding | ns |
| | | | | | Descriptions of interactions | ns |
| | | | | | Reproductions of conversation | ns |
| | | | | | Unexpected complications | ns |
| | | | | | Unusual details | ns |
| | | | | | Peripheral details | ns |
| | | | | | Accurately reported details misunderstood | ns |
| | | | | | Related external associations | ns |
| | | | | | Accounts of subjective mental state | ns |
| | | | | | Attribution of another's mental state | ns |
| | | | | | Details characteristic of a particular act | ns |

| | | | | | | |
|---|---|---|---|---|---|---|
| | | | | | Spontaneous corrections | Lower for individuals with psychopathy during truth but higher during deception |
| | | | | | Expressing insecurities | ns |
| | | | | | Admitting lack of memory | ns |
| | | | | | Providing reasons for lack of memory | ns |
| | | | | | Raising doubts about one's own testimony | ns |
| | | | | | Self-deprecation | ns |
| Louth 1998 | Oral | Quietness | Semi-structured narrative generation<br><br>Personal events | Positive & negative experiences<br><br>Baseline neutral recordings | Mean amplitude (quietness) | Control: higher and more variable amplitude for negative emotion |
| Williamson 1991 | Oral | Syntax | Semi-structured narrative generation<br><br>Personal events | Anger & personal difficulty-related stories | Lexical cohesion | ns |
| | | | | | Referential cohesion | ns |
| | | | | | Conjunctive cohesion | ns |
| | | | | | Total cohesion | Control (difficulty only) |
| | | | | | Incompetent references | Control (anger only) |
| | | | | | Closed/open units ratio | Control |
| | | | | | Open units | ns |
| | | | | | Closed units | ns |
| | | | | | Words | ns |
| | | | | | Clauses | ns |

| | | | |
|---|---|---|---|
| | | Mean utterance length | ns |
| | Thought disorder | Total Thought Disorder | Control |
| | | Poverty of Speech | n/a |
| | | Poverty of Content | n/a |
| | | Pressure of Speech | n/a |
| | | Tangentiality | n/a |
| | | Derailment | n/a |
| | | Illogicality | n/a |
| | | Incoherence | n/a |
| | | Distractible Speech | n/a |
| | | Circumstantiality | n/a |
| | | Loss of Goal | n/a |
| | | Positive Thought Disorder | n/a |
| | | Negative Thought Disorder | n/a |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; n/a=Not available; ns=Not significant.

Regarding content and thought disorder, Lee et al. (2008) included an extensive list of variables on content but the only significant group differences they observed indicated that individuals with psychopathy included more appropriate detail and spontaneous corrections than controls during deception while they made fewer spontaneous corrections when describing a true incident (the interaction for spontaneous corrections was not significant and only showed a trend, *P*=0.05, therefore the evidence from pairwise comparisons is questionable). Williamson (1991), on the other hand, examined various aspects of thought disorder and although she indicated greater impairment in individuals with psychopathy overall, group differences on individual aspects of thought disorder were not significant.

Finally, Klaver et al. (2007) did not find any significant group differences in physical features of speech. On the other hand, Louth et al. (1998) reported that individuals with psychopathy generally spoke more softly and showed less variation in amplitude between negative and neutral topics.

A meta-analysis was conducted for syntax in psychopathy. Strongest effects resulted in a large and significant pooled effect size estimate with considerable heterogeneity. The failsafe *N* of 38.98 was above the critical value of 35, but the funnel plot did not appear symmetrical (Appendix D, Figure 12.14), thus some publication bias was possible. Weakest effects resulted in a medium and non-significant pooled effect size estimate,-0.48[-1.49,0.53], with comparable heterogeneity.

#### *2.3.8.1.2 Meta-analysis on verbal expression*

Only data indicating good performance on verbal expression for psychopathy were sufficient for a meta-analysis. Strongest effects resulted in a significant medium pooled effect estimate with substantial heterogeneity. The failsafe *N* of 94.81 was above the critical cut-off of 65. Visual inspection of the funnel plot suggested asymmetry primarily on account of Brinkley, Bernstein et al. (1999), thus there may be a degree of publication bias (Appendix D, Figure 12.14). Study quality was not correlated with effect size, *rho*=0.33,*n*=11, and stratification according to study quality did not reveal any significant differences between study subgroups. The high level of heterogeneity appeared mostly due to data from Brinkley, Bernstein et al. (1999) with

LA Caucasian participants. When these were removed, the pooled estimate decreased but remained significant,-0.30[-0.52,-0.09],*P*<0.01, while heterogeneity was no longer significant. Weakest effects resulted in a small and non-significant pooled estimate,-0.07[-0.37, 0.23], with comparable heterogeneity for the entire sample of studies.

#### 2.3.8.2 Non-verbal expression

Two studies were identified (Gillstrom & Hare, 1988; Klaver et al., 2007), the latter distinguishing between true and false story-telling (Table 2.22). The studies included 17 individuals with psychopathy and 48 without. Gillstrom and Hare (1988), focused on hand movements during parts of the PCL interview and suggested that individuals with psychopathy made more rapid movements (beats, after controlling for number of words spoken) than controls. They did not find significant group differences in gestures reflecting the content of speech or non-speech-related gestures (e.g. touching of body, object manipulation, or postural changes). Klaver et al. (2007) indicated that individuals with psychopathy increased their head movements during deception whereas this was not observed in controls.

Table 2.22. *Studies which examined non-verbal expression in psychopathy*

| Reference | Function | Task | Primary outcomes | Result |
|---|---|---|---|---|
| Gillstrom 1988 | Hand gestures | PCL interview | Iconic language gestures | ns |
| | | | Beat language gestures | Higher rates in individuals with psychopathy |
| | | | Non-language gestures | ns |
| | | | Laterality | ns |
| Klaver 2007 | Non-verbal movements | Semi-structured narrative generation | Blinks | ns |
| | | | Head movements | Higher rates during deception than in truth for individuals with psychopathy only |
| | | | Self-manipulations | ns |
| | | | Smiles | ns |
| | | | Illustrators | ns |
| | | | Hand movements | ns |
| | | | Arm movements | ns |

| | | |
|---|---|---|
| | Foot & leg movements | ns |

*Note.* PCL=Psychopathy Checklist; ns=Not significant.

### 2.3.8.3 Academic skills

Nine studies examined reading in psychopathy (K. Blair, Morton, Leonard, & Blair, 2006; K. Blair, Newman et al., 2006; K. Blair, Richell et al., 2006; Drugge, 1998; D. S. Goldstein, 1998; Hart et al., 1990; Hervé et al., 2003; Mills, 1995; Swogger, 2006) and one examined writing in ASPD (Gawda, 2008a). An overview is presented in Table 2.23.

Table 2.23. *Studies which examined academic language skills in ASPD and psychopathy*

| Reference | Task | Primary outcomes | Result | |
|---|---|---|---|---|
| ***Reading*** | | | | |
| Blair, Morton 2006 | NART | Performance score | ns | |
| Blair, Newman 2006 | NART | Performance score | ns | |
| Blair, Richell 2006 | NART | Performance score | ns | |
| Drugge 1998 | Word naming with priming | Accuracy | ns | |
| | | Correct response latency | ns | |
| Goldstein 1998 | WRAT-2 | Performance score | ns | |
| Hart 1990 | WRAT-2 | Performance score | ns | |
| Hervé 2003 | WRAT-R | Performance score | ns | |
| Mills 1995 | WRAT-R | Performance score | ns | |
| | Word naming | Accuracy | ns | |
| | | Response latency | ns | |
| | | Laterality | ns | |
| Swogger 2006 | WRAT-R | Performance score | n/a | |
| ***Writing*** | | | ASPD vs: | |
| | | | *Non-ASPD offender* | *Healthy control* |
| Gawda 2008a | Dictated | Regular impulse | ns | lower in |

| | | | |
|---|---|---|---|
| transcription | | | healthy control |
| | Open shape 'a' | higher in non-ASPD | ns |
| | Circle in 'i, j' | Ns | lower in healthy control |
| | Sinusoidal line | higher in non-ASPD | ns |
| | Cut off finals of letters | higher in non-ASPD | ns |
| | Arcade form of 'm' | higher in non-ASPD | ns |
| | Arcade form of 'n' | higher in non-ASPD | ns |
| | Angular form of 'm' | ns | ns |
| | Angular form of 'n' | ns | ns |
| | Big pressure | ns | lower in healthy control |
| | Tremblings | ns | lower in healthy control |
| | Ataxies | ns | lower in healthy control |
| | Initial as hook-like | ns | lower in healthy control |
| | Words going down | ns | lower in healthy control |
| | Loops in ovals | ns | lower in healthy control |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; NART=National Adult Reading Test; WRAT= Wide Range Achievement Test; n/a=Not available; ns=Not significant.

#### *2.3.8.3.1 Reading*

Studies included at least 175 individuals with psychopathy and 165 without. The three studies by K. Blair and colleagues (K. Blair, J. Morton et al., 2006; K. Blair, Newman et al., 2006; K. Blair, Richell et al., 2006) had overlapping samples. Only reading scores of the National Adult Reading Test (NART) are discussed here whereas occasions where this test was used to estimate verbal IQ (VIQ) are outlined in the section on intelligence later. The word naming task employed by Mills (1995) nvolved three-letter words which had been examined by the participants prior to the f the experiment. No study reported significant group differences and Swogger (2006) did not report any statistical comparisons on the Wide Range Achievement Test

(WRAT). A meta-analysis with available data, produced a small and non-significant pooled effect size estimate with no heterogeneity.

#### *2.3.8.3.2 Writing*

Gawda (2008a) examined various graphical features of writing (from dictation) in relation to ASPD in a Polish sample (Table 2.23). Although there were some differences between the groups with and without ASPD (forensic and healthy control) these were not consistent. Significant differences between the forensic groups with and without ASPD did not extend to differences between the group with ASPD and the healthy control group. Similarly, where differences were observed between the group with ASPD and healthy controls, the forensic groups performed similarly compared to each other.

### 2.3.8.4 Semantic processing

Affective semantic processing was discussed in the context on affective processes and focus here is on studies of semantic processing per se. There was one publication on ASPD (Lorenz & Newman, 2002c), one on both ASPD and psychopathy (Kosson et al., 2006) and thirteen on psychopathy only (K. Blair, Richell et al., 2006; Brinkley et al., 2005; Hare & Jutai, 1988; Kiehl, Hare, McDonald et al., 1999; Kiehl et al., 2004; Lorenz & Newman, 2002a; Marshall, 1996; Schmitt, 2000; Suchy & Kosson, 2006; Williamson et al., 1991). An overview of studies is presented in Table 2.24.

Table 2.24. *Studies which examined semantic processing in ASPD and psychopathy*

| Reference | Task & special features | Special features | Primary outcomes | Result favoured |
|---|---|---|---|---|
| ***ASPD*** | | | | |
| Lorenz & Newman 2002c | Lexical decision | | Response latency | ns |
| | | | Accuracy | ns |
| ***ASPD & psychopathy*** | | | | |
| Kosson 2006 | Lexical decision | Verbal: semantic (words) | | |
| ***Psychopathy*** | | | | |
| Blair, Richell 2006 | Concrete discrimination | Semantic priming | Response latency | ns |
| | | | Errors | ns |
| Brinkley 2005 - Exp. 1 | Lexical decision | Semantic priming | Response latency | ns |
| | | Two prime-target delays | | |
| - Exp. 2 | Semantic Stroop | Baseline: Letter strings | Interference (CRW) | ns (test on LA groups only) |
| | | Congruent & incongruent stimulus associations | | |
| | | | Facilitation (CRW trials) | ns |
| | | | Errors (CRW interference trials) | ns |
| | | | Errors (CRW facilitation trials) | Control[a,b] |
| Hare 1988 | Concrete discrimination | | Response latency | ns |
| | | | Errors | ns |
| | | | Laterality | ns |
| | Abstract discrimination | | Response latency | ns |

| | | | | |
|---|---|---|---|---|
| | | | Errors | Control groups over individuals with psychopathy in RVF targets only |
| | | | Laterality | ns |
| Kiehl, Hare, McDonald 1999 | Lexical decision | | Response latency | ns |
| | | | Accuracy | ns |
| | Concrete vs. abstract discrimination | | Response latency | ns |
| | | | Accuracy | ns |
| Kiehl, Laurens 2006 | Sentence comprehension | Semantically congruent & incongruent stimuli | Errors | ns |
| Kiehl 2004 | Lexican decision | Concrete & abstract stimuli | Correct response latency | Controls |
| | | | Accuracy | Control (pseudowords only) |
| Lorenz & Newman 2002a | Lexical decision | | Frequency facilitation | ns overall, LA control (right hand only) |
| | | | Response latency | ns |
| | | | Accuracy | ns |
| Lorenz & Newman 2002b | Lexical decision | | Frequency facilitation | ns |
| | | | Response latency | n/a |

| | | | | |
|---|---|---|---|---|
| Marshall 1996 | Lexical decision | | Response latency | ns |
| | | | Accuracy | |
| Schmitt 2000 - African American sample only | Semantic Stroop | Baseline: Letter strings | Interference (CRW) | ns (test on LA groups only) |
| | | Congruent & incongruent stimulus associations | | |
| | | | Facilitation (CRW trials) | ns (test on LA groups only) |
| | | | Errors (interference trials) | ns |
| | | | Errors (facilitation trials) | ns |
| Suchy 2006 | Concrete discrimination | High & low cognitive demand | Response latency | ns |
| | | | Errors | Control (high demand only) |
| Williamson, Harpur 1991 | Lexical decision | | Correct response latency | ns |
| | | | Accuracy | ns |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; CRW=Colour-related word; LA=Low-anxious; RVF=Right visual field; n/a=Not available; ns=Not significant.

[a] from Schmitt (2000) - Exp. 3; [b] interaction with no post hoc comparisons.

#### *2.3.8.4.1 ASPD*

There were at least 155 offenders with a diagnosis of ASPD and 104 controls. Lorenz and Newman (2002c) did not observe any significant group differences in lexical decision while Kosson et al. (2006) did not report any statistical comparisons.

#### *2.3.8.4.2 Psychopathy*

There were thirteen studies including at least 136 individuals with psychopathy and 200 without. All samples were recruited from prison settings except Kiehl et al.'s (2004) control group and Hare and Jutai's (1988) second control group both of which originated from the general public. Various studies were discussed in previous sections on affective processing and Stroop tasks. Additional studies included those of Kiehl, Laurens et al. (2006) and Suchy and Kosson (2006).

There was some evidence of semantic deficit. Of the lexical decision studies, Kosson et al. (2006) did not report results for the semantic stimuli separately. The only studies highlighting significant differences between individuals with psychopathy and controls were those by Kiehl et al. (2004) and Lorenz and Newman (2002a). The former observed that controls were faster overall when responding correctly and were more accurate in recognising pseudo-words. The latter suggested that the semantic processing of the LA control group benefitted by higher word frequency on right-handed trials but not on left-handed trials. This facilitation was not observed in the group with psychopathy.

Of the concrete or abstract discrimination tasks, significant differences were observed by Hare and Jutai (1988) who reported that individuals with psychopathy performed worse than either control group (offenders and healthy controls) on abstract discrimination of RVF targets only. Further, Suchy and Kosson (2006) reported that individuals with psychopathy were less accurate in concrete discrimination during higher cognitive demand conditions only.

The last category of tasks in semantic processing involved the semantic Stroop paradigm. There was a significant interaction according to which individuals with psychopathy were less accurate than controls in facilitation trials, but this was not

confirmed by post hoc comparisons (Brinkley et al., 2005). There were no further significant group differences.

Error rates and response latency data were available for meta-analysis in some studies. Strongest effects resulted in a large and significant pooled effect size estimate, with considerable heterogeneity. The failsafe *N* of 69.62 was above the critical value of 40 suggesting some robustness against publication bias but the funnel plot was not symmetrical (Figure 12.15). Heterogeneity appeared mostly due to the contribution of Suchy and Kosson (2006). Removal of this study resulted in a medium and significant pooled effect size estimate, 0.53[0.22,0.84],$P<0.001$, with no heterogeneity. Weakest effects resulted in a medium and non-significant pooled estimate (in the opposite direction, -0.44[-1.17, 0.28], with considerable heterogeneity. The primary contributor to this heterogeneity appeared to be Lorenz and Newman (2002a), removal of which resulted in a small and non-significant pooled effect size estimate, -0.03[-0.33,0.27], with no heterogeneity.

#### 2.3.8.5 Knowledge acquisition and retention

The Information test was employed in four studies – one on ASPD (Stevens et al., 2003) and three on psychopathy (Mercer et al., 2005; Pham et al., 2003; Raine & Venables, 1988). All studies employed the task from the WAIS-R except Pham et al. (2003) who utilised the WAIS. The studies on psychopathy included 173 offenders with psychopathy and 219 without. Stevens et al. (2003) did not find a significant difference between groups while the remaining publications did not report any statistical comparisons. A meta-analysis with available data resulted in a significant medium pooled effect size estimate (random effects to moderate the contribution of Mercer et al., 2005 as a possible outlier) with moderate heterogeneity. The failsafe *N* of 10.5 was below the critical value of 20. Mercer et al. (2005) has often been an outlier therefore the pooled estimate is likely to be subject to publication bias.

#### 2.3.8.6 Meta-analysis on language functions

Sufficient data to further the meta-analysis on verbal expression earlier were available for psychopathy only. Strongest effects produced a significant medium to large pooled effect size estimate in favour of controls, with substantial heterogeneity. The failsafe *N* of 266.35 exceeded the critical value of 85. Heterogeneity and asymmetry in the funnel plot (Appendix D, Figure 12.15) appeared primarily due to the contribution of Brinkley, Bernstein et al. (1999) and Suchy and Kosson (2006). Removal of these sets of data resulted in a smaller, yet significant pooled estimate, 0.35[0.18,0.52],$P<0.001$, with non-significant heterogeneity. With weakest effects, the pooled estimate was small and did not reach significance, 0.04[-0.19,0.27], with moderate heterogeneity. Overall, study quality was not correlated with effect size, $rho=-0.27$,$n=13$, and stratification according to quality did not reveal any subgroups differences.

#### 2.3.8.7 Summary

##### *2.3.8.7.1 ASPD*

Research in language in ASPD focused on various areas including verbal expression, academic language skills, semantic processing and knowledge. Some but limited evidence of different performance in individuals with ASPD was present in verbal expression (discourse but not fluency) and features of writing, highlighting possible anomalies in ASPD. The former effect was confounded with affective processes while the latter was inconsistent with inherent contradictions. No deficits in semantic processing or knowledge were observed.

##### *2.3.8.7.2 Psychopathy*

Relatively robust evidence of overall language function impairment resulted from a meta-analysis of strongest effects but not weakest effects. Examining language functions more closely revealed some evidence supporting a deficit in verbal and non-verbal expression. Individuals with psychopathy also demonstrated impaired semantic processing across various domains. In addition, there was evidence suggesting reduced general knowledge (Information) in psychopathy but this may be subject to

publication bias as well as being influenced primarily by the outlier contribution of Mercer et al. (2005). Individuals with psychopathy and controls did not appear different in academic language skills (reading).

Although deficits in expressive functions were highlighted, they were more closely associated with syntax and thought disorder rather than verbal fluency and vocabulary where no group differences were observed. Furthermore, individuals with psychopathy performed differently compared to controls in areas including features of their writing, speech content between giving a truthful and deceptive account of events and gestural language. Individuals with psychopathy also spoke more softly and showed less variation in voice amplitude than controls during a speech about negative compared to neutral topics. Although these tasks do not reveal any deficits, they highlight potential anomalies in the communication of individuals with psychopathy.

### 2.3.9 Perception

#### 2.3.9.1 Visual perception

One publication investigated ASPD (Dolan & Park, 2002, visual recognition) while the remaining nine focused on psychopathy (D. S. Goldstein, 1998; Hart et al., 1990; Kosson, 1996, 1998; Kosson, Miller, Byrnes, & Leveroni, 2007; Lopez, Kosson, Weissman, & Banich, 2007; Mills, 1995; Pham et al., 2003; Suchy & Kosson, 2006). Details are shown in Table 2.25. Studies included at least 203 individuals with psychopathy and 215 without, all recruited from prison settings.

Table 2.25. *Studies which examined visual perception in ASPD and psychopathy*

| Reference | Function | Task | Stimuli | Primary outcomes | Result favoured |
|---|---|---|---|---|---|
| ***ASPD*** | | | | | |
| Dolan 2002 | Visual recognition (figure & design) | Matching to Sample | Patterns | Accuracy | ns |
| | | | | Response latency | ns |
| ***Psychopathy*** | | | | | |
| Goldstein 1998 | Visual recognition (angular) | Judgment of Line Orientation | Angled lines | Accuracy | ns |
| Hart 1990 | Visual organisation | Hooper's Visual Organisation | Fragmented objects | Accuracy | ns |
| Kosson 1996 | Visual recognition (figure) | Target discrimination | Letters & numerals) | Accuracy | ns |
| | | | | Commission errors | ns |
| | | | | Response latency | ns |
| Kosson 1998 | Visual recognition (figure) | Target discrimination | Letters & numerals | Accuracy | ns |
| | | | | Response latency | ns |
| Kosson 2007 | Visual recognition (figure) | Matching | Global-local letters | Response latency | Control (for local targets during local bias) |

| | | | | | |
|---|---|---|---|---|---|
| Lopez 2007 | Visual recognition (figure) | Matching | Global-local letters (high & low cognitive demand) | Accuracy | Control (right handed responses & local across-hemisphere trials, both during high cognitive demand) |
| | | | | Response latency | Control (for local stimuli overall & for trials of low cognitive demand) |
| Mills 1995 | Visual inattention | Letter cancellation<br>Symbol cancellation | Letters<br>Symbols | Omission errors | ns<br>ns |
| Pham 2003 | Visual inattention | Letter cancellation | Letters | Total items read in 20s | ns |
| | | | | Omission errors | n/a |
| | | | | Commission errors | ns |
| | | | | Errors (%) | Control |
| | | | | Performance variation | Control |
| Suchy 2006 | Visual recognition (figure & design) | Matching | Patterns | Response latency | ns |
| | | | | Errors | ns |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; n/a=Not available; ns=Not significant.

#### *2.3.9.1.1 Visual inattention/scanning*

Two publications employed cancellation tasks (Mills, 1995; Pham et al., 2003) which included over 30 individuals with psychopathy and 30 without. These tests also implicate operations of sustained attention and response inhibition to some extent (Lezak et al., 2004). Only Pham et al. (2003) observed a deficit, in psychopathy.

#### *2.3.9.1.2 Visual recognition and organisation*

Of six publications in this group, only one examined visual recognition in ASPD (Dolan & Park, 2002) with the DMS task from the CANTAB (Cambridge Cognition, 2006; Fray et al., 1996; Sahakian & Owen, 1992). The simultaneous presentation condition only was of interest here, involving a matching-to-sample paradigm. No differences between the groups were observed. No significant group differences were reported by Hart et al. (1990) on visual organisation in psychopathy either.

The studies on visual recognition in psychopathy included 163 individuals with psychopathy and 170 without with a variety of tasks. Tasks using global and local stimuli (larger letters made up of smaller letters) were employed by two studies (Kosson et al., 2007; Lopez et al., 2007) which were the only ones reporting significant effects. Kosson et al. (2007) observed that controls were faster than individuals with psychopathy in identifying local stimuli during local bias only. Lopez et al. (2007) also reported findings where controls performed more accurately than individuals with psychopathy during higher cognitive demand for right hand responses and for local across-hemisphere trials. Controls were also faster than individuals with psychopathy when responding to local stimuli overall and during less cognitive demand specifically.

#### *2.3.9.1.4 Meta-analysis*

Strongest effects resulted in a significant small to medium pooled effect size estimate with non-significant heterogeneity. The failsafe *N* of 8.46 was below the critical value of 30, indicating possible publication bias. Weakest effects resulted in a

small and non-significant pooled effect size estimate, 0.14[-0.12,0.39], with no heterogeneity.

#### 2.3.9.2 Auditory and olfactory perception

Four publications investigated auditory perception in psychopathy (Hiatt et al., 2002; Kosson, 1996; Mills, 1995; Suchy & Kosson, 2005) and included at least 68 individuals with psychopathy and 78 without, all recruited from prison settings. All studies examined auditory discrimination (ability to distinguish between different sounds) but dichotic listening implicated attention processes. An overview is provided in Table 2.26. Only Suchy and Kosson (2005) reported a significant effect where controls outperformed individuals with psychopathy (on errors and response latency) during LHA only. Only one study examined olfaction (Lapierre et al., 1995). Controls performed better than individuals with psychopathy during smell identification but there were no differences during odour detection.

Table 2.26. *Studies which examined auditory and olfactory perception in psychopathy*

| Reference | Task | Stimuli | Primary outcomes | Result favoured |
|---|---|---|---|---|
| ***Audition*** | | | | |
| Hiatt 2002 | Dichotic Listening Task | Phonemes | Accuracy<br>Commission errors<br>Laterality | ns<br>ns<br>ns |
| Kosson 1996 | Target discrimination | Tones | Accuracy<br>Commission errors<br>Response latency | ns<br>ns<br>ns |
| Mills 1995 | Dichotic listening tone identification | Tones | Accuracy | ns |
| Suchy 2005 | Dichotic Listening Task | Tones | Accuracy<br>Commission errors<br>Response latency | ns<br>Control (LHA only)<br>Control (LHA only) |
| ***Olfaction*** | | | | |
| Lapierre 1995 | Odour Detection Test | Odour | Accuracy | ns |

| | | | |
|---|---|---|---|
| | Modular Smell Identification Test | Odours | Control |

*Note.* Highlighted outcomes contributed to meta-analyses; LHA=Left hemisphere activation; ns=Not significant.

#### 2.3.9.4 Summary

There was only one study on ASPD reporting no group differences on visual perception. On the other hand, studies on psychopathy investigated vision, auditory perception and olfaction. Although the number of studies was limited, some evidence suggested that individuals with psychopathy may be impaired in visual perception, auditory discrimination, and olfaction. Visual processes were more extensively examined compared to other modalities. In individual studies, there was some but limited evidence supporting a deficit in visual attention/search (confounded with sustained attention) and visual recognition, but not visual organisation. A local-letter disadvantage was also observed in psychopathy, also associated with right-hand responses on one occasion, suggesting a possible impairment in left-hemisphere function. A meta-analysis on visual perception supported an overall deficit with strongest effects only.

### 2.3.10 Interhemispheric integration

Interhemispheric connection and handedness were examined in five publications (Hiatt, 2005; Hiatt & Newman, 2007; Lopez et al., 2007; Mayer & Kosson, 2000; Raine et al., 2003). The samples of Raine et al. (2003) were recruited from the general public and the group with personality disorder met criteria for both ASPD and psychopathy. The remaining studies included participants from prison populations. At least 206 individuals with psychopathy and 180 without took part. An overview of studies is presented in Table 2.27.

Table 2.27. *Studies which examined interhemispheric integration in ASPD & psychopathy*

| Reference | Task | Stimuli | Lateral features | Primary outcomes | Result favoured |
|---|---|---|---|---|---|
| ***ASPD & psychopathy*** | | | | | |
| Raine 2003 | Consonant-vowel-consonant | Visual/verbal | Bilateral & unilateral presentations | Interhemispheric integration | Greater in antisocial |
| | Letter matching | Visual/verbal | Between & within visual field presentation | | Greater in antisocial |
| ***Psychopathy*** | | | | | |
| Hiatt 2005 - Exp. 2 | Banich's letter name-identity | Visual/verbal | Intra & interhemispheric trials | Accuracy | ns |
| | | | Left vs. right hemisphere decisions | Response latency | ns |
| | | | Within-LVF vs. within-RVF match performance | IHA-accuracy | ns |
| | | | | IHA-response latency | ns |
| - Exp. 3 | Banich's letter name-identity with colour match/mismatch | Visual/verbal | Intra- & interhemispheric trials | Accuracy | ns |
| | | | Left vs. right hemisphere decisions | Response latency | ns |
| | | | Within-LVF vs. within-RVF match performance | IHA-accuracy | ns |
| | | | | IHA-response latency | ns overall, interactions for first 1/2 of trials[a] |

| | | | | | |
|---|---|---|---|---|---|
| Hiatt 2007 | Poffenberger paradigm | Visual | Intra- & interhemispheric trials (uncrossed-crossed response hand-stimulus presentation respectively) | Accuracy | n/a |
| | | | | Response latency | ns |
| | | | | Crossed-uncrossed diff. | Overall: greater in antisocial |
| | | | | | Left hand: greater in LA antisocial only |
| | | | | | Right hand: greater in antisocial |
| Lopez 2007 | Banich's letter name-identity with global/local stimuli | Visual/verbal | Intra- and interhemispheric trials | Accuracy | Control (right handed complex & local complex across trials only) |
| | | | More & less complex conditions Left vs. right hand performance | Response latency | Control (local overall & less complex local trials only) |
| Mayer 2000 | Handedness measure | | | Characterisation | Antisocial group showed greater mixed-handedness |

*Note.* ASPD=Antisocial Personality Disorder; RVF/LVF=Right/left visual field; IHA=Interhemispheric advantage; LA=Low-anxious; n/a=Not available; ns=Not significant.

[a] smaller IHA for individuals with psychopathy vs. controls on difficult trials (letter match/colour mismatch, and a larger IHA for individuals with psychopathy vs controls on easier trials (letter & colour match); smaller IHA for individuals with psychopathy vs. controls for right hemisphere-decision trials, and larger IHA for individuals with psychopathy vs. controls for left hemisphere-decision trials.

The most common paradigm in these studies was Banich's letter-name identity task in which participants were presented with two uppercase letter stimuli above fixation in each visual field and had to indicate whether there was a match with a single lowercase letter in the left or RVF below the fixation point. The paradigm was used in this form in the second experiment of Hiatt (2005). In the third experiment participants had to also indicate whether there was colour match between otherwise matching stimuli. In Lopez et al. (2007), the same paradigm involved global-local letter stimuli. Interaction effects were observed for the first half of trials only in Hiatt's studies: (i) Controls showed greater interhemispheric advantage (IHA) during more challenging trials but this was reversed during easier trials and (ii) there was larger IHA for controls when the lowercase stimulus was presented to the right hemisphere (right hemisphere-decision) while the same group showed a smaller IHA during left hemisphere-decision trials. Both effects disappeared once participants had practiced the procedure sufficiently. Lopez et al. reported significant differences indicating superior performance by controls in some instances during right-handed responses to local stimuli.

There were three other paradigms. Poffenberger's paradigm involving presentation of stimuli either ipsilaterally or contralaterally to the response hand was used by Hiatt and Newman (2007). Raine et al. (2003) employed the consonant-vowel-consonant task where participants identified non-sense trigrams presented either unilaterally or bilaterally and a matching task with uppercase and a lowercase letter stimuli presented either within or between-visual fields. Finally, Mayer and Kosson (2000) examined handedness.

Findings were conflicting. Raine et al. (2003) reported greater interhemispheric integration among individuals with psychopathy in comparison to controls and Mayer and Kosson (2000) reported that a greater proportion of individuals with psychopathy exhibited mixed handedness than controls. Hiatt and Newman (2007), on the other hand, presented evidence according to which individuals with psychopathy showed less interhemispheric integration, especially for right-handed responses.

In sum, the evidence was generally inconclusive. ASPD and psychopathy were associated with greater interhemispheric integration overall, during less challenging paradigms, for left-hemisphere decision trials and for right handed responses to local stimuli. On the other hand, more complex interhemispheric integration paradigms/conditions elicited better performance by controls. There was a greater proportion of mixed-handedness among individuals with psychopathy compared to controls. Conflicting studies were of same quality but sampling and task difficulty varied, possibly resulting in the observed contradictions.

### 2.3.11 Construction and visuospatial functions

All studies focused on psychopathy. An overview is provided in Table 2.28. Regarding mental orientation, tere were 42 individuals with psychopathy and 42 without, recruited from prisons. No impairments were highlighted.

There were eight studies on construction including at least 346 individuals with psychopathy and 581 without (Gillstrom, 1995; Hart et al., 1990; Johansson & Kerr, 2005; Jozef & da Silva, 1999; Mercer et al., 2005; Pham et al., 2003; Raine & Venables, 1988; S. S. Smith et al., 1992). The only significant group differences highlighted better performance on the Block Design by individuals with psychopathy on one occasion (Jozef & da Silva, 1999) and worse performance by LA individuals with psychopathy on another (S. S. Smith et al., 1992). Three studies did not report statistical comparisons. Meta-analysis of strongest effects yielded a small and non-significant pooled effect size estimate, with substantial heterogeneity. Weakest effects showed no overall effect, 0[-0.49,0.49], with considerable heterogeneity.

Table 2.28. *Studies which investigated construction and visuospatial operations in psychopathy where outcomes was the test score*

| Reference | Task | Result favoured |
|---|---|---|
| ***Construction*** | | |
| Gillstrom 1995 | Block Design (WAIS-R) | ns |
| Hart 1990 | Block Design (WAIS-R) | ns |
| Johansson 2005 | Block (Dureman-Sälde) | ns |
| Jozef 1999 | Block Design (WAIS) | Antisocial |
| Mercer 2005 | Block Design (WAIS-R) | n/a |
| | Object Assembly (WAIS-R) | n/a |
| Pham 2003 | Block Design (WAIS) | n/a |
| | Object Assembly (WAIS) | n/a |
| Raine 1988 | Block Design (WAIS-R) | n/a |
| | Object Assembly (WAIS-R) | n/a |
| Smith 1992 | Block Design (WAIS-R) | LA control |

| | | |
|---|---|---|
| ***Mental orientation*** | | |
| Lapierre 1995 | Mental rotation | ns |
| Mills 1995 | Mental rotation | ns |

*Note.* Highlighted tests contributed to meta-analyses; WAIS/-R=Wechsler Adult Intelligence Scale/-Revised; LA=Low-anxious; n/a=Not available; ns=Not significant.

### 2.3.12 Neurological soft signs (NSS) and motor skills

#### 2.3.12.1 NSS

Two studies examining NSS were identified, one focusing on ASPD (Lindberg et al., 2004) and another on psychopathy (Assadi et al., 2007). An overview of these studies is presented in Table 2.29. While there were no significant group differences between individuals with and without psychopathy, the ASPD group showed an overall impairment and more frequent abnormalities on blunt/sharp discrimination, tapping rhythm and dysdiadochokinesia (alternating movement sequences).

#### 2.3.12.2 Motor skills

Two studies examined motor performance (manual dexterity and motor cortex functions), one focusing on ASPD (Stevens et al., 2003) and the other on psychopathy (S. S. Smith et al., 1992). The studies employed tasks where participants had to reproduce finger tapping patterns. No significant group differences were reported (Table 2.29).

Table 2.29. *Studies which examined NSS and motor skills in ASPD and psychopathy*

| Reference | Task | Primary outcomes | Result favoured |
|---|---|---|---|
| ***NSS*** | | | |
| Assadi 2007 (Psychopathy) | Neurological evaluation scale | Overall score | ns |
| | | Sensory integration | ns |
| | | Motor coordination | ns |
| | | Complex motor acts | ns |
| | | Miscellaneous | ns |
| Lindberg 2004 (ASPD) | NSS assessment | Overall score | Control |
| | | Palmomental test | ns |
| | | Snout reflex | ns |
| | | Suck reflex | ns |
| | | Gaze impersistence | ns |
| | | Ocular vergence | ns |
| | | Nystagmus | ns |
| | | Oral apraxia | ns |
| | | Motor perseveration in spoken | ns |

| | | | |
|---|---|---|---|
| | | commands | |
| | | Grasp reflex | ns |
| | | Graphesthesia | ns |
| | | Simultaneous bilateral tactile extinction | ns |
| | | Imaginary acts | ns |
| | | Astereognosis | ns |
| | | Two objects test | ns |
| | | Blunt/sharp discrimination | Control |
| | | Tapping rhythm | Control |
| | | Dysdiadochokinesia | Control |
| | | Blink reflex | ns |
| | | Complex motor acts | ns |
| ***Motor skills*** | | | |
| Smith 1992 | Finger tapping (Halstead-Reitan) | Taps | ns |
| Stevens 2003 | Luria Motor Task 22 | Taps | ns |
| | | Errors | ns |
| | Luria Motor Task 23 | Taps | ns |
| | | Errors | ns |

*Note.* Highlighted outcomes contributed to meta-analyses; NSS=Neurological soft signs; ASPD=Antisocial Personality Disorder; ns=Not significant.

### 2.3.13 Intelligence

An overview of identified studies which reported data on intelligence in relation to ASPD, DPD and psychopathy can be seen in Table 2.30. Of these, only one study (Johansson & Kerr, 2005) aimed at examining intelligence per se.

Table 2.30. *Studies which examined intelligence in ASPD, DPD and psychopathy*

| Reference | IQ-relevant inclusion criteria | IQ assessment scale | IQ type | Result favoured |
|---|---|---|---|---|
| ***ASPD*** | | | | |
| Barkataki 2005 | Groups matched on age, SES, reading ability & years in education | WAIS-III | FSIQ | ns |
| | | | PIQ | ns |
| | | | VIQ | ns |
| | | NART | VIQ | |
| Barkataki 2008 | Groups matched on age, SES, reading ability & years in education | WAIS-III | FSIQ | n/a |
| Dolan 2002 | IQ-matched groups | NART | VIQ | ns |
| Gawda 2008a | | WAIS-R | FSIQ | ns |
| Gawda 2008b | | WAIS-R | FSIQ | ns |
| | | | PIQ | ns |
| | | | VIQ | ns |
| Kumari 2006 | | WAIS-III<br>NART | FSIQ<br>VIQ | ns<br>ns |
| Kumari 2005 | | NART | VIQ | ns |
| Kumari 2009 | Premorbid IQ-matched groups | NART | VIQ | ns |
| Lorenz & Newman 2002c | IQ≥70 & at least 4$^{th}$ grade level in achievement tests | SILS-R | FSIQ | ns |
| Raine 2000 | | WAIS-R (Vocabulary, Arithmetic, Digit Span, Block Design, Digit Symbol) | FSIQ | ns |

| | | | | |
|---|---|---|---|---|
| Stevens 2003 | | WAIS-R | PIQ | ns |
| | | | VIQ | ns |
| Völlm 2010 | IQ≥85 | Quick test | VIQ | ns |
| ***ASPD & psychopathy*** | | | | |
| Dolan 2004 | IQ-matched groups | NART | VIQ | ns |
| Dolan 2005 | IQ-matched groups | NART | VIQ | ns |
| Raine 2003 | | WAIS-R (Vocabulary, Arithmetic, Digit Span, Block Design, Digit Symbol) | FSIQ | ns |
| | | WAIS-R (Digit Symbol, Block Design) | PIQ | Control |
| | | WAIS-R (Vocabulary, Arithmetic, Digit Span) | VIQ | ns |
| ***DPD & psychopathy*** | | | | |
| Müller 2008/ Weber 2004 | IQ≥85 | n/a | n/a | n/a |
| Dolan 2006 | IQ-matched groups | NART | VIQ | ns |
| ***Psychopathy*** | | | | |
| Arnett 1993 | Above 4th grade level in reading | SILS-R | FSIQ | ns |
| Arnett 1997 - Exp. 1 | Above 4th grade level in reading | SILS-R | FSIQ | ns |
| - Exp. 2 | Above 4th grade level in reading | SILS-R | FSIQ | Control |
| Bagley 2009 | IQ≥70 | SILS-R | FSIQ | ns |
| Bernstein 2000 | Above borderline intelligence & above 4th grade level in reading & maths | SILS-R | FSIQ | n/a |
| Blair 1996 | IQ-matched groups | Not stated | IQ | n/a |
| Blair 1995 | | WAIS | FSIQ | ns |
| Blair, Sellars | IQ-matched groups | Not stated | IQ | n/a |

| | | | | |
|---|---|---|---|---|
| 1995 | | | | |
| Blair, Jones 1995 | IQ-matched groups | Not stated | IQ | n/a |
| Brinkley, Bernstein 1999 | Above 4th grade level in reading & maths | SILS-R | FSIQ | ns |
| Brinkley, Newman 1999 | Above 4th grade level in reading & maths | SILS-R | FSIQ | ns |
| Brinkley 2005 - Exp. 1 | Above 4th grade level in reading & maths | SILS-R | FSIQ | ns |
| - Exp. 2 | | | | Control |
| Cima 2010 | | Not stated | IQ | ns |
| Craig 2009 | Recruitment of participants with 'normal intelligence' | Not stated | FSIQ | n/a |
| Drugge 1998 | | SILS & WAIS | FSIQ | n/a |
| Dvorak-Bertsch 2007 | IQ≥70 | SILS-R | FSIQ | n/a |
| Gacono 1990 | | SILS-R | FSIQ | ns |
| Gacono 1991 | IQ>80 | SILS-R & WAIS-R | FSIQ | ns |
| Gacono 1992 | IQ>80 | SILS-R, WAIS-R & Quick test | FSIQ | ns |
| Gillstrom 1995 | IQ≥80 | WAIS-R (Vocabulary & Block Design) | FSIQ | ns |
| Glass 2006 | IQ≥70 | SILS-R | FSIQ | ns |
| Glass 2009 | IQ≥70 | SILS-R | FSIQ | ns |
| Goldstein 1998 | IQ≥80 | SILS-R | FSIQ | ns |
| Hare 1988 | | Not stated | IQ | ns |
| Hart 1990 - Sample 2 | | WAIS-R (Vocabulary & Block Design) | FSIQ | n/a |
| Herpertz 2001 | IQ≥85 | | FSIQ | ns |

| | | | | |
|---|---|---|---|---|
| Hiatt 2005 - Exp. 3 | Above borderline intelligence & above 4th grade level in achievement tests | SILS-R | FSIQ | ns |
| Hiatt 2002 | Above borderline intelligence & above 4th grade level in achievement tests | SILS-R | FSIQ | ns |
| Hiatt 2007 | IQ≥70 & above 4th grade level in reading & maths | SILS-R | FSIQ | ns |
| Hiatt 2004 - Exp. 1<br>- Exp. 2<br>- Exp. 3 | IQ≥70 & above 4th grade level in reading & maths | SILS-R | FSIQ | ns |
| Howard 2007 | IQ-matched groups | WAIS-R (Picture Completion, Object Assembly, Block Design, Digit Symbol) | PIQ | n/a |
| Ishikawa 2001 | | WAIS-R (Vocabulary, Arithmetic, Digit Span, Block Design, Digit Symbol) | FSIQ | ns |
| Johansson 2005 | | Synonyms-Reasoning-Block (Dureman-Sälde) | FSIQ | ns |
| Kiehl, Bates 2006 | | NART<br>Quick test | VIQ | ns |
| Kiehl, Laurens 2006 | | NART<br>Quick test | VIQ | ns |
| Kiehl 2001 | Matched healthy control participants | NART<br>Quick test | VIQ<br>VIQ | ns<br>ns |
| Kiehl 2004 | Matched healthy control participants | NART | VIQ | ns |

| | | | | |
|---|---|---|---|---|
| | | Quick test | | |
| Kosson 1996 | IQ at least average & at least 4th grade level in reading | SILS-R | FSIQ | ns |
| Kosson 2007 | Ability to read English | SILS-R | FSIQ | ns |
| Kosson 2002 | IQ≥70 & at least 4th grade level in reading | SILS-R | FSIQ | ns |
| Llanes 2006 | IQ≥70 | SILS-R | FSIQ | ns |
| Lopez 2007 | IQ≥70 | SILS-R | FSIQ | ns |
| Lorenz & Newman 2002b | IQ≥70 & at least 4th grade level in achievement tests | SILS-R | FSIQ | ns |
| Lorenz & Newman 2002a | IQ≥70 & at least 4th grade level in achievement tests | SILS-R | FSIQ | ns |
| Lösel 2004 | | WAIS (Information, Similarities, Picture completion, Block design) | FSIQ | n/a |
| Louth 1998 | | WAIS-R (Verbal subtests) | VIQ | n/a |
| Mayer 2000 | Basic literacy requirement | SILS-R | FSIQ | n/a |
| Mayer 2006 | IQ>75 | SILS-R | FSIQ | ns |
| Mercer 2005 | | WAIS-R | FSIQ | n/a |
| Mills 1995 - Exp. 1 | | WAIS-R (Block Design & Vocabulary) | FSIQ | ns |
| - Exp.2 | | WAIS-R (Block Design & Vocabulary) | FSIQ | ns |
| Newman 1992 | IQ≥75 & at least 4th grade level in reading | SILS-R | FSIQ | ns |

| | | | | |
|---|---|---|---|---|
| Newman 1990 - Study 1 | Above borderline/lower intelligence & at least 5th grade level in reading | SILS | FSIQ | ns |
| - Study 2 | Above borderline/lower intelligence & at least 5th grade level in reading | SILS | FSIQ | ns |
| - Study 3 | Above borderline/lower intelligence & at least 5th grade level in reading | SILS | FSIQ | ns |
| Newman 1987 | Above borderline/lower intelligence | SILS | FSIQ | ns |
| Newman 1997 - Caucasian sample | At least 4th grade level in reading & maths | SILS-R | FSIQ | LA psychopathy & HA controls |
| - African-American sample | At least 4th grade level in reading & maths | SILS-R | FSIQ | LA psychopathy & HA controls |
| Patterson 1990 - Exp. 1 | At least 5th grade level in achievement tests | SILS | FSIQ | ns |
| - Exp. 3 | At least 5th grade level in achievement tests | SILS | FSIQ | ns |
| Pham 2000 | | WAIS | FSIQ | ns |
| Pham 2003 | | WAIS | FSIQ | ns |
| Raine 2004 | | WAIS-R (Vocabulary, Arithmetic, Digit Span, Block Design, Digit Symbol) | FSIQ | ns |
| | | WAIS-R (Digit Symbol, Block Design) | PIQ | n/a |
| | | WAIS-R (Vocabulary, Arithmetic, Digit Span) | VIQ | n/a |
| Raine 1988 | | WAIS | | |

| | | | | |
|---|---|---|---|---|
| Reveillere 2003 | No intellectual disability | WAIS | FSIQ | ns |
| Schmitt 2000 - African-American sample - Study 1 | At least 4th grade level in reading & maths | SILS-R | FSIQ | ns |
| - Study 2 | At least 4th grade level in reading & maths | SILS-R | FSIQ | ns |
| - Study 3 | At least 4th grade level in reading & maths | SILS-R | FSIQ | ns |
| - Study 4 | At least 4th grade level in reading & maths | SILS-R | FSIQ | ns |
| Smith 1999 | At least average intelligence & groups matched on age & SES | NART<br>Quick test | Pre-morbid IQ<br>VIQ | ns<br>ns |
| Smith 1992 | Above borderline/lower intelligence & at least 5th grade level in achievement tests | SILS-R | FSIQ | ns |
| Snowden 2004/ Gray 2003 | | NART | VIQ | ns |
| Suchy 2005 - LHA<br>- RHA | IQ≥80 | SILS-R | FSIQ | ns |
| Suchy 2006 | IQ≥80 | SILS-R | FSIQ | ns |
| Swogger 2006 | IQ≥70 | SILS-R | FSIQ | ns |
| Yang 2005 | | WAIS-R (Vocabulary, Arithmetic, Digit Span, Block Design, Digit Symbol) | FSIQ | ns |
| Zeier 2009 | IQ≥70 | SILS-R | FSIQ | Control |

*Note.* Highlighted outcomes contributed to meta-analyses; ASPD=Antisocial Personality Disorder; DPD=Dissocial Personality Disorder; SES=Socio-economic status; WAIS/-R=Wechsler Intelligence Scale/-Revised; FSIQ/VIQ/PIQ=Full-scale/verbal/performance IQ;

NART=National Adult Reading Test; SILS/-R=Shipley Institute of Living Scale/-Revised; LA/HA=Low/high-anxious; n/a=Not available; ns=Not significant.

#### 2.3.13.1 ASPD

There were fifteen publications including at least 393 individuals with a diagnosis of ASPD and 370 individuals without. Sample sources varied between forensic inpatients, prisoners, university or hospital staff and the general public. The only studies which recruited both the antisocial and at least one comparison group from the same populations were Gawda (2008, 2008a), Lorenz and Newman (2002c), Raine et al. (2000, 2003) and Stevens et al. (2003). Dolan and Park (2002) and Dolan and Fullam (2004, 2005) recruited IQ-matched groups. No significant group differences were reported on either full-scale IQ (FSIQ) or VIQ. Only Raine et al. (2003) observed significantly lower performance IQ (PIQ) among individuals with ASPD and psychopathy scores compared to controls, with both samples from the general population. Using available data from studies with non-IQ-matched groups during recruitment, a meta-analysis revealed a small and non-significant pooled mean difference estimates with substantial heterogeneity. Studies associated with higher quality tended to favour the control group (visual inspection).

#### 2.3.13.2 DPD

There were only two studies in connection with DPD (Dolan & Fullam, 2006; Müller et al., 2008; Weber et al., 2004), the antisocial participants of which also met criteria for psychopathy. Studies included 59 individuals with DPD and 61 without. Müller et al. (2008)/Weber et al. (2004) did not report any statistical comparisons whereas the groups of Dolan and Fullam (2006) were matched on IQ.

#### 2.3.13.3 Psychopathy

There were 75 publications including at least 931 individuals with psychopathy and 1364 without. An overview of studies is presented in Table 2.30. Samples originated mostly from prison settings but some studies recruited from sources including forensic inpatients, university or hospital staff and the general public. Of these, only Craig et al et al. (2009), Herpertz et al. (2001) and Kiehl et al. (2004) did

not compare groups from the same population. Nine publications involved IQ-matched groups at recruitment and 31 had set a minimum IQ threshold for participation. A FSIQ deficit in psychopathy was reported by three studies only (Arnett et al., 1997; Brinkley et al., 2005; Zeier et al., 2009). Newman et al. (1997) observed a significant interaction according to which LA controls and HA individuals with psychopathy showed a possible deficit but no post hoc comparisons were reported. Regarding PIQ, Raine et al. (2003) found a deficit in individuals with both psychopathy and ASPD. Raine and Venables (1988) did not examine overall IQ and instead reported higher scores in individuals with psychopathy on a 'parietal' index (Block Design & Object Assembly subtests from the WAIS). Thirteen studies did not report any statistical comparisons.

A meta-analysis was conducted but studies with IQ-matched groups at recruitment were not included. With data from unsuccessful individuals with psychopathy from Ishikawa et al. (2001), there was a small yet highly significant pooled mean difference estimate for FSIQ in favour of the control group, with moderate heterogeneity. The failsafe *N* of 70.02 was below the critical value of 95 and the funnel plot was asymmetrical (Appendix D, Figure 12.16). However, because studies did not generally intend to examine IQ, publication bias was not likely to be strong. Results were comparable with data from successful individuals with psychopathy from Ishikawa et al. When those studies which incorporated a lowest IQ cut-off selection criterion (Craig et al., 2009; Herpertz et al., 2001; Reveillere et al., 2003; Zeier et al., 2009) were not taken into account (samples in each of the remaining studies originated from the same populations), the mean difference estimate remained small and significant, -3.29[-5.56,-1.03],$P<0.01$, with moderate heterogeneity. Overall, age and study quality were not significantly correlated with effect sizes, *rho*=0.18,*n*=13, and *rho*=-0.04,*n*=17 respectively. Stratification according to quality did not reveal significant subgroup differences (visual inspection).

Sufficient data were also available for a meta-analysis on VIQ (NART data from Kiehl, Bates et al., 2006), resulting in a very small and non-significant mean difference estimate, with no heterogeneity. Results were comparable for Quick Test data from Kiehl, Bates et al. (2006) in sensitivity analysis. None of the contributing

studies contained a minimum IQ selection criterion and comparison samples in each study were from the same population.

#### 2.3.13.4 Summary

There was no reliable evidence of difference in intelligence scores between ASPD and comparison groups. The only significant effect suggested higher PIQ in controls but presence of psychopathy confounded the result. Regarding psychopathy per se, the evidence supporting a deficit was very weak in individual studies. However, a meta-analysis revealed a small but significant and reliable deficit in psychopathy for FSIQ but not for VIQ whereas a meta-analysis was not possible for PIQ. Studies on DPD did not report statistical comparisons or the groups were IQ-matched. Although the studies in this review originated through a systematic search on neuropsychological function including intelligence, it is likely that studies in other areas of research may have measured intellectual function in order to describe their samples. These were not possible to locate and include here, therefore the present results may be unrepresentative of the published data.

## 2.4 Grand summary and interim discussion

A systematic review of research on neuropsychological functions in males meeting criteria for antisocial personality was conducted in order to summarise the findings of the multitude of studies in the field thereby facilitating the generation of specific hypotheses for this project. The antisocial personality was operationalised as ASPD, DPD, and psychopathy and studies used clinician-administered methods in identifying individuals with this personality type. The majority of the included publications examined psychopathy with 115 reports. This was followed by ASPD with 21 publications and then by DPD with two publications. Five of the identified studies investigated both psychopathy and ASPD while the two studies on DPD also examined psychopathy. As the number of publications was large, meta-analyses were helpful in summarising findings. An overview of these results is presented in Tables 2.31-36.

### 2.4.1 Summary of findings

Studies examined a range of functions in populations with an antisocial personality operationalised as ASPD, DPD, or psychopathy, and revealed a broad spectrum of deficits. These were located mostly in executive, abstraction, and affective functions. Results also indicated possible difficulties in cognitive flexibility (attentional set-shifting), memory, attention, perception, NSS, and intelligence, but evidence for these was weaker. Some language anomalies were also observed, more reliably in psychopathy than ASPD.

Most studies examined executive functions and indicated impairment in both ASPD and psychopathy. This was pronounced for motor regulation, particularly in response inhibition tasks, in both operationalisations. Individuals with ASPD also appeared consistently impaired in planning. On the other hand, individuals with psychopathy seemed to experience difficulties with cognitive flexibility and particularly during attentional set shifting (robust result) with additional impairment observed in response reversal and complex decision-making (less consistent). The evidence was less consistent for planning with deficits likely in problem-solving type tasks such as the ToL. In addition, there was evidence that some executive deficits

may not generalise to individuals with antisocial personality without a history of offending.

Apart from executive functions, there was also substantial research on abstraction (e.g. concept formation, reasoning, etc.), affect, and social cognition. In the former, both ASPD and psychopathy showed deficits in verbal concept formation but in psychopathy impairments appeared more extensive with evidence suggesting additional difficulties in visual concept formation and, although less consistently, in mathematical procedures and semantic abstraction.

Regarding affective processes and social cognition, ASPD, DPD, and psychopathy were associated with deficits in affect recognition. In psychopathy this deficit was particularly present in semantic affect recognition as well as recognition of happiness, disgust, surprise, and perhaps sadness but not fear. In addition, the language of antisocial individuals (either ASPD or psychopathy) was affected differently to controls when the content was emotional but this was confounded with language functions in ASPD. However, studies on psychopathy suggested a broader emotional deficit spanning across affective operations also including processing, memory, and language, but evidence was less consistent than for recognition. Both ASPD and psychopathy were associated with impairments in social cognition in some studies (more consistent in ASPD than psychopathy) but evidence was limited overall.

Memory and attention were also examined. Evidence of impairment in either ASPD or psychopathy was weak, although there was some consistency in highlighting a possible STM (visual recognition) deficit in ASPD. Findings tenuously suggested sustained attention difficulties in ASPD and psychopathy and possible impairments in divided attention in psychopathy. An overall deficit in attention may be present in psychopathy but, generally, evidence was weak and inconsistent.

A breadth of language functions was also investigated. Some studies suggested anomalies in the verbal expression and physical features of writing of individuals with ASPD compared to controls but evidence was weak and inconsistent overall. In psychopathy, some impairment in language functions was detected, particularly in syntax and semantic processing, and perhaps in general knowledge (outlier bias possible). Studies also observed anomalies in psychopathy in relation to a

range of language tasks involving non-verbal expression, writing, speech content (truth vs. deception), and voice amplitude.

Except for intelligence, the remaining cognitive functions were covered less extensively. Nevertheless, evidence indicated a possible impairment in perception in psychopathy (particularly visual perception but this was confounded with sustained attention) while a study on NSS in ASPD found some deficits associated with the diagnosis. Regarding interhemispheric connection, one study in ASPD suggested greater integration in individuals with the diagnosis compared to controls but findings in psychopathy were not consistent. Finally, although results did not suggest lower IQ in ASPD compared to controls, a meta-analysis on FSIQ revealed lower scores in psychopathy. Although IQ data originated from studies examining other cognitive functions, therefore publication bias was less likely (see below), these studies may not be representative of the published IQ data potentially in studies outside the focus of the present review.

### 2.4.2 Differences and commonalities between operationalisations

Although cognitive deficits were highlighted in a range of functions, an intriguing observation was that findings varied between different definitions of the antisocial personality. Some overlap existed, particularly in executive functions (motor regulation), affect recognition, and verbal concept formation but beyond that, ASPD, DPD, and psychopathy showed different patterns across cognitive domains. Those diagnosed with psychopathy emerged as more extensively impaired, particularly in affective operations, but this may well reflect the larger number of available studies – and therefore greater power – compared to ASPD or DPD. One likely explanation of the discrepancies in cognitive impairment is that the three operationalisations represent different configurations of symptoms (Hare, 1991, 2003; Hare et al., 1991; Hodgins, 2007) and even different populations (Coid et al., 2009; Fazel & Danesh, 2002; Hare, 2003; Hare et al., 2000; Hart & Hare, 1989), therefore results may suggest that each could be attributed to a different cognitive profile. On the other hand, the cognitive commonalities between operationalisations potentially underline those neuropsychological functions – and thus cerebral areas – which might

be important in understanding the antisocial personality. For motor regulation, those may be the ventromedial and orbitofrontal cortices (D. L. Clark et al., 2010; Lezak et al, 2004) and affect recognition may be linked to the amygdala (D. L. Clark et al., 2010; Kolb & Whishaw, 2009). Since these areas are closely inter-connected (D. L. Clark et al., 2010), an impaired interaction between them is also a possibility.

### 2.4.3 Heterogeneity

Apart from differences between operationalisations, heterogeneity was also observed within both ASPD and psychopathy in some cognitive domains (e.g. affect and intelligence for ASPD or executive functions for psychopathy) and within specific operations (e.g. set shifting for ASPD or planning, productivity for psychopathy). This often remained even after removal of outliers. The contribution of factors such as age, education, and study quality to heterogeneity was examined via meta-regression and stratification. Of these, only stratification by study quality explained some heterogeneity. This occured for attention and executive functions in psychopathy where studies with high and low quality ratings contributed more modest effects than studies with a medium quality rating. It is possible that better control in higher quality studies yielded more valid results while presence of error in lower quality studies could have moderated their results.

However, perhaps the strongest contributor to heterogeneity within operationalisations is the diversity within the populations which ASPD and psychopathy represent (Blackburn, 2009; De Brito & Hodgins, 2009). Level of anxiety, emotional stability, and degree of substance abuse are some of the factors which have been shown to differentiate between sub-types of psychopathy (Alterman et al., 1998; Blackburn, 2009; Brinkley, Newman, Widiger, & Lynam, 2004; Hicks, Markon, Patrick, Krueger, & Newman, 2004; Hicks & Patrick, 2006; Skeem, Johansson, Andershed, Kerr, & Louden, 2007). Conversely, substance abuse, depression, anxiety, high levels of psychopathy, and overlap with other personality disorders are all factors contributing to heterogeneity within ASPD (L. A. Clark, 2007; Compton, Conway, Stinson, Colliver, & Grant, 2005; De Brito & Hodgins, 2009; Goodwin & Hamilton, 2003; Grant, Stinson, Dawson, Chou, & Ruan, 2005; Robins et

al., 1991; Tyrer et al., 2007; Westen & Arkowitz-Westen, 1998; Widiger et al., 1996). Therefore, heterogeneity within cognitive functions for the different operationalisations is likely to reflect the inherent diversity characterising these populations (e.g. comorbidity, sub-types, etc.) information on which was not generally available in the studies.

### 2.4.4 Publication bias and outliers

Another important factor when interpreting results from systematic reviews and meta-analyses is publication bias, assessed here with the use of funnel plots and the failsafe *N*. Funnel plots provide information on the symmetry of the distribution of effect sizes but small study samples encountered throughout the present review limited its reliability. However, when a sufficient number of studies was available, it proved particularly helpful in identifying outliers. Possible bias when including such deviating results in meta-analyses was controlled via sensitivity analyses in which these effects were removed and via use of random effects models to moderate the weight assigned to them. Curiously, the results by Mercer et al. (2005) often appeared as outliers with effect sizes larger than what other studies found. The reason for this was that the study reported larger group differences than other studies, perhaps reflecting bias in the data.

When pooling study effect sizes in a meta-analysis, the failsafe *N* represents the required number of additional studies with a null effect in order for the pooled estimate to become non-significant. In the present review, use of the failsafe *N* suggested some robust findings in psychopathy, including executive functions (self-regulation, cognitive flexibility, and complex decision-making), affective operations (overall), attention (overall), and language (verbal expression, syntax, and semantic processing). The small number of studies in ASPD implied that publication bias was possible in all results. However, there is a caveat in employing the failsafe *N* in the present review. This is because the cut-off value above which it reflects a robust result operates on the principle that studies reporting null results are less likely to be published. As a large number of studies in the present review examined several cognitive functions (Appendix C, Table 11.1), it is likely that many may were

published because the reported some significant results. In these cases, non-significant findings were also included in the publication; therefore use of the failsafe *N* in these circumstances may have overestimated publication bias. Perhaps the most characteristic example of this was the case of FSIQ. It was reported in a large number of studies but was the primary focus of one publication only (Johansson & Kerr, 2005). The remaining studies reported IQ mainly to demonstrate that their groups were intellectually comparable. Publication bias was therefore likely to be very limited in the findings concerning FSIQ but the failsafe *N* suggested otherwise.

### 2.4.5 Comparison to previous reviews

Five reviews/meta-analyses similar to this work have been published in recent years, focusing on executive functions and facial affect recognition in antisocial behaviour. Morgan and Lilienfeld (2000) examined executive functions and the review was repeated by Ogilvie et al. in 2011. Marsh and Blair (2008), Wilson and colleague (2011), and Dawel et al. (2012) examined affect recognition. Both meta-analyses on executive functions suggested poorer executive performance in antisocial populations but evidence in the present review provided only partial support for this. There are several possible reasons for this difference. Focus in earlier reviews was on antisocial behaviour generally rather than antisocial personality and studies had examined heterogeneous antisocial samples identified not only on the basis of presence of antisocial personality but also of externalising disorders, criminality, violence, delinquency, various personality disorders (including DSM-III), and so forth. Studies using self-report measures of psychopathy to identify samples were also included.

Apart from sampling, another reason for differences between the present and previous findings on executive functions might be that these were more loosely defined previously. The relevant reviews included in their analysis tests like the PAL, DMS, Complex Figure Test, verbal learning and object classification, which primarily involved other, not executive, functions such as memory or abstraction (see Lezak et al., 2004 and Strauss et al., 2006, for test classifications), therefore contaminating results. In addition, even though earlier reviews were more general, the present

review included three additional papers on ASPD and 29 on psychopathy. As a result, the present findings are more representative of the literature on executive deficits in the antisocial personality. In conclusion, although previous reviews suggested that extensive executive deficits may be associated with antisocial behaviour generally, current findings indicate that executive impairments in the antisocial personality may be more specific to motor regulation and perhaps planning and cognitive flexibility.

Regarding affect recognition, Marsh and Blair (2008) and Wilson et al. (2011) also highlighted deficits in recognising emotions in facial stimuli, particularly for fear and sadness. Dawel et al. (2012) pooled adult and younger samples to higlight impairment in overall emotion recognition as well as individual emotions whilst including correlational data. In adults, however, only happiness and surprise recognition emerged as impaired. Though the present review provided some support for a deficit in affect recognition, the evidence on fear recognition did not suggest impairment and the support was weak for facial recognition alone. The conclusions on sadness recognition were also tenuous. The results from the present review were more in line with those of Dawel et al. (adult samples only) but apart from happiness and surprise, they highlighted additional possible impairments in disgust and sadness recognition.

Possible reasons for the discrepancies between prior reviews and present results may reflect differences in the included studies/samples and in the affective focus. Marsh and Blair considered antisocial behaviour generally (e.g. defined by aggression, offending, conduct disorder, fronto-temporal dementia, etc.) rather than limit inclusion to psychiatric diagnosis or PCL/-R criteria for antisocial personality. Wilson et al. (2011) and Dawel et al. (2012) also included antisocial populations on the basis of self-reported measures as well as non-adult individuals. Regarding affective focus, prior reviews examined emotion recognition rather than affect recognition more broadly (e.g. positive and negative affect) as in the present methodology.

As results from the adult samples of Dawel et al. (2012) were more in line with the present review, most discrepancies appear due to different inclusion criteria for studies. The additional evidence of potential deficits in disgust and sadness recognition may have arisen due to methodological differences as the present review

included sensitivity analyses using multiple outcome measures compared to Dawel et al. (2012) who may have missed these less robust effects. In light of these results, it appears that affect recognition may reflect a common feature between antisocial behaviour and personality though impairments seem more focal in the latter. However, further research is required in adult samples and ASPD as the number of studies in the present review was focused primarily on psychopathy and was relatively small, particularly for disgust and fear.

### 2.4.6 Neuropsychological theories revisited

Having highlighted cognitive impairments in the antisocial personality, of which motor regulation, affect recognition, and (verbal) concept formation were observed consistently, an attempt to evaluate the various neuropsychological theories discussed earlier is possible. A full evaluation is not feasible, however, as most models make additional predictions on non-cognitive functions which were not examined in this review, for example autonomic arousal and conditioning.

With focus on the evidence examined in the present review, Table 2.37 shows most theories to have a rather specific cognitive focus while none is able to explain all of the identified cognitive deficits in the antisocial personality, including those which are most consistent. The majority of the theories appear able to explain impaired motor regulation but do not seem to capture other anomalies, for example in language and concept formation. On the other hand, whereas the Left Hemisphere Activation hypothesis involves language functions better than other models, it does not account for key deficits in motor regulation or affect recognition.

The IES appears to be the only theory which captures deficits in both motor regulation and affect recognition (and other possible affective deficits), as it focuses on operations involving the joint work of the OFC (including the ventromedial area) and amygdala. Though recent evidence suggests that the amygdala is implicated in processing of fear as well as other emotions (Fitzgerald, Angstadt, Jelsone, Nathan, & Phan, 2006; Fusar-Poli et al., 2009), development of the model has relied considerably on evidence from fear recognition deficits in antisocial individuals (R. Blair et al., 2005) which was not supported by findings in adults with antisocial personality in the

present review or in the review by Dawel et al. (2012). Furthermore, the IES does not seem able to explain impairment in concept formation. In spite of a variety of theoretical models attempting to explain the antisocial personality, none seems able to reflect the range of cognitive deficits observed in this population. This highlights the need for further research as well as better integration of the evidence within the theories.

### 2.4.7 Causality and substance abuse

A limitation in the studies featuring in the present review is that their design was cross-sectional therefore only describing a correlation between antisocial personality and neurocognitive impairments rather than a causal relationship. The effect of substance abuse (e.g. alcohol, cannabis), which has been consistently associated with neurotoxicity and cognitive impairment (Lezak et al., 2004; Strauss et al., 2006), is an important factor to take into consideration here. Substance abuse is highly comorbid with antisocial personality (De Brito & Hodgins, 2009; Hare, 2003), early onset has been reported to predict greater frequency of externalising personality disorders including the antisocial type (Bakken, Landheim, & Vaglum, 2004) and evidence suggests it may mediate the relationship between childhood conduct disorder and adult antisocial behaviour (Khalifa, Duggan, Howard, & Lumsden, 2012). Interestingly, adolescent exposure to alcohol has been linked with impairments in those neural substrates involved in affective self-regulation, potentially fostering violent personality disordered pathology (Howard, 2009). Although it was not possible to examine this effect in the present review, it is plausible that a history of substance abuse in the examined samples may contribute to the observed cognitive impairments, particularly in affect and motor regulation.

### 2.4.8 Implications for treatment

Presence of neuropsychological deficits in the antisocial personality has several implications for treatment. Preferred psychological interventions for this personality type are cognitive and/or behavioural-based in nature and based on social learning theory (NICE, 2009), consistent with treatment approaches in personality

disorders generally (Alwin et al., 2006). As a result, these interventions involve a range of tasks such as planning, learning to identify and inhibit inappropriate responses, generating and appraising alternative options (set-shifting, reasoning), processing different concepts, ability to recognise and appraise emotions and social cues, communicating, learning new skills, and so forth (Alwin et al., 2006; Ledley, Marx, & Heimberg, 2010; NICE, 2009; Padesky, 1993; Westbrook, Kennerly, & Kirk, 2011). It follows that any impairment in relevant cognitive abilities is likely to impede progress. Furthermore, as many treatments for antisocial personality were developed in other areas of mental health (Howard & Howells, 2010), failing to tailor them to the specific needs of this population, is likely to further limit treatment effectiveness.

The notion that cognitive functioning is related to treatment progress is not new in mental health, however. Indeed, there is evidence to suggest that cognitive difficulties are associated with poorer outcomes in a number of clinical populations and treatments. These include memory impeding overall treatment in bipolar disorder (Torres et al., 2010), verbal memory and complex attention predicting poorer outcomes in depression (Story, Potter, Attix, Welsh-Bohmer, & Steffens, 2008), and risk-taking predicting worse outcome in computer-assisted cognitive-behavioural therapy for individuals with substance abuse (Carroll et al., 2011). In schizophrenia, which is also characterised by a range of neuropsychological deficits (Fullam & Dolan, 2008; Lewandowski et al., 2011), impairments in cognitive flexibility appear to impede problem solving therapy (Üçok et al., 2006), cognitive impairment seems to impact on adherence to medication (Spiekermann et al., 2011), and language and memory deficits have predicted poorer treatment outcomes (deVille, Baker, Lewin, Bucci, & Loughland, 2011; Mueser, Bellack, Douglas, & Wade, 1991). More akin to the antisocial personality, offenders with pronounced executive deficits showed higher drop-out rates and more disruptive behaviour in treatment in a study by Fishbein et al. (2009).

While the above findings support a negative relationship between impairment and treatment outcomes, it is important to note that there is some incongruence as the opposite has also been observed on some occasions. For example, deVille et al. (2011) also found that better performance in visuospatial/constructional abilities

predicted chronicity in schizophrenia while risky behaviours, which often reflect a deficit in decision-making (Bechara, Damasio, Tranel, & Damasio, 2005; Gazzaniga et al., 2009), have been associated with stronger treatment-seeking attitudes in individuals with anxiety disorder when measured via self-report (Lorian & Grisham, 2011).

Overall, however, the evidence seems to suggest a plausible link between cognitive abilities and treatment outcomes in clinical populations, with poorer performance predicting worse treatment progress. Regarding the antisocial personality, although the extant literature has highlighted a range of cognitive difficulties, no research has yet investigated its impact on treatment. In light of current poor treatment outcomes, exploring this relationship may prove clinically useful in identifying new ways to meet the needs of individuals with antisocial personality.

### 2.4.9 Conclusions

The present review examined and summarised what seemed to be extensive and complex evidence on cognitive functions in individuals with antisocial personality, furthering current understanding. Findings highlighted specific cognitive difficulties in this population and commonalities between different operationalisations involving motor regulation, affect recognition, and (verbal) concept formation. Additional impairments may be present in a range of other functions but evidence was less consistent. Whereas findings provided some support for current neuropsychological models of the antisocial personality, none was able to explain the observations in isolation which indicates a need for greater theoretical integration. Several factors limit conclusions in the present review including a lack of overlap between operationalisations, unexplained heterogeneity, and challenges in evaluating publication bias. Furthermore, as most samples originated from offender settings, it is unclear whether the identified cognitive difficulties would generalise to individuals with antisocial personality but without a recorded history of offending. Although there is considerable work on neuropsychological functions in the antisocial personality to date, much remains to be understood and further research is required to

both clarify current findings and examine the implications of neuropsychological impairment in this population, particularly in relation to treatment.

Table 2.31. *Summary of meta-analyses on neuropsychological findings in ASPD*

| Cognitive functions | *n*[a] | Total samples | | SMD (>0=favours controls) | Heterogeneity | Bias | |
|---|---|---|---|---|---|---|---|
| | | ASPD | Control | | | Failsafe *N* | Funnel plot |
| 1. Executive | 5 | 125-126 | 115 | 0.09 - 0.40** | ns | <CV | n/a |
| a. Planning | 4 | 73 | 65 | 0.52*** - 0.58*** | ns | <CV | n/a |
| b. Self-regulation | 5 | 126 | 114 | 0.21 - 0.48*** | ns | <CV | Some asymmetry |
| i. Cognitive flexibility | As attentional set shifting below | | | | | | |
| Attentional set shifting | 3 | 75 | 64 | 0.38 - 0.45 | Substantial | n/a | n/a |
| ii. Motor regulation | 5 | 125 | 114 | 0.28* - 0.41** | ns | <CV | Some asymmetry |
| Response inhibition | 4 | 91 | 82 | 0.30† - 0.42** | ns | <CV | n/a |
| Go/NoGo | 3 | 67 | 59 | 0.38 - 0.50** | Substantial - ns | <CV | n/a |
| 2. Abstraction | 3 | 63 | 64 | 0.23 | ns | n/a | n/a |
| a. Concept formation | 3 | 63 | 64 | 0.04 - 0.51** | ns | <CV | n/a |
| i. Verbal | 2 | 51 | 52 | 0.52** | ns | <CV | n/a |
| ii. Visual: Sorting & shifting | See attentional set shifting above | | | | | | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 3. Affect & social cognition | | | | | | | |
| a. Affective operations | 3 | 99 | 73 | 0.42 - 0.44 | Substantial | n/a | n/a |
| i. Affect recognition | 2 | 73 | 37 | 0.68** - 0.69** | ns | <CV | n/a |
| b. Social cognition | | | | | | | |
| i. Theory of Mind | 2 | 74 | 40 | (-0.19) - 1.16*** | ns | <CV | n/a |
| 4. Memory | 4 | 108-109 | 97 | 0.08 - 0.30* | ns - moderate | <CV | n/a |
| a. STM | 4 | 108-109 | 97 | 0.12 - 0.30 | ns - moderate | n/a | n/a |
| Verbal recall | 2 | 48 | 47 | 0.01 | ns | n/a | n/a |
| Visual recognition | 3 | 74 | 65 | 0.17 - 0.44* | ns - substantial | <CV | n/a |
| b. LTM | 2 | 41 | 35 | 0.27 - 0.38 | ns | n/a | n/a |
| 5. Attention | | | | | | | |
| a. Sustained | 2 | 44 | 45 | (-0.36) - 0.63*** | ns | <CV | n/a |
| 6. Intelligence | | | | (Mean difference) | | | |
| a. FSIQ | 3 | 95 | 89 | 1.89 | Substantial | n/a | n/a |
| b. VIQ | 4 | 123 | 112 | 1.56 - 2.20 | Substantial | n/a | n/a |
| c. PIQ | 4 | 123 | 112 | 3.73 | Substantial | n/a | n/a |

*Note.* ASPD=Antisocial Personality Disorder; SMD=Standardised mean difference; CV=Critical value; ns=Not significant; n/a=Not applicable; S/LTM=Short/long-term memory; FSIQ/VIQ/PIQ=Full scale/verbal/performance IQ.

***P<0.001; **P<0.01; *P<0.05.

[a] Sample of effect sizes.

† Marginally significant.

Table 2.32. *Summary of meta-analyses on executive functions in psychopathy*

| Executive functions | *n*[a] | Total samples | | SMD (>0=favours controls) | Heterogeneity | Publications bias & outliers | |
|---|---|---|---|---|---|---|---|
| | | Psychopathy | Control | | | Failsafe *N* | Funnel plot |
| Overall | 21 | 554 | 603 | 0.04 - 0.50**(0.28** without outliers) | Substantial - considerable (moderate without outliers) | >CV | Asymmetrical: 3 outliers |
| 1. Planning | 4 | 85 | 80 | 0.19 - 0.65 | Considerable | n/a | n/a |
| 2. Self-regulation | 20 | 538-543 | 585-596 | 0.09 - 0.52**(0.29* without outliers) | Substantial - considerable (ns without outliers) | >CV | Asymmetrical: 3 outliers |
| a. Productivity | 6 | 261-271 | 301-314 | 0.09 - 0.13 | Substantial | n/a | n/a |
| b. Cognitive flexibility | 12 | 418 | 485 | 0.13 - 0.68*** | Considerable | >CV | Asymmetrical |
| i. Attentional set shifting | 7 | 292 | 363 | 0.26* - 0.59*(inc. removal of outlier) | ns - considerable | >CV | Outlier: Mercer et al. (2005) |
| ii. Decision-making | 5 | 113 | 106 | 0.35 - 0.69 | Substantial - considerable | n/a | n/a |
| Complex: | 4 | 66 | 68 | 0.61 - 1.07*** | Substantial - ns | >CV | n/a |
| iii. Response reversal | 2 | 33 | 31 | 1.01 - 1.39* | Substantial - considerable | <CV | n/a |
| iv. Other | 6 | 232 | 274 | 0.03 - 0.20 | Considerable | n/a | n/a |
| c. Motor regulation | | | | | | | |
| i. Response inhibition | 14 | 315 | 286 | 0.21† - 0.35* | Moderate - substantial | <CV | Some symmetry-asymmetrical |
| Go/NoGo | 10 | 253 | 228 | 0.33 | Considerable | n/a | n/a |
| SST | 5 | 75 | 72 | -0.05 | ns | | |
| Stroop errors | 3 | 66 | 70 | 0.20 - 0.49*(Traditional | Moderate - ns | <CV | n/a |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | Stroop tasks only) | | | |
| 3. Effective performance | | | | | | | |
| Accuracy/errors | 10 | 284 | 336 | 0.11 - 0.14 | Substantial | n/a | n/a |
| Response latency: | | | | | | | |
| -Experimental | 7 | 124 | 153 | 0.41† - 0.44† | Substantial | n/a | n/a |
| -Baseline | 7 | 124 | 177 | 0.41† - 0.45† | Substantial | n/a | n/a |
| Interference | 6 | 104 | 127 | (-0.05) - 0.14 | ns | n/a | n/a |
| a. Stroop | | | | | | | |
| i. Response latency | | | | | | | |
| Experimental | 4 | 78 | 80 | 0.64** | ns | <CV | n/a |
| Baseline | 4 | 78 | 80 | 0.62** | ns | <CV | n/a |
| ii. Interference | 4 | 68 | 72 | 0.27 | ns | n/a | n/a |
| b. Non-Stroop | | | | | | | |
| i. Response latency | | | | | | | |
| Experimental | 3 | 46 | 73 | 0.15 | Considerable | n/a | n/a |
| Baseline | 3 | 46 | 97 | 0.19 | Considerable | n/a | n/a |
| ii. Accuracy | 6 | 88 | 95 | 0.02 | ns | n/a | n/a |
| iii. Interference | 6 | 88 | 95 | -0.44** | ns | <CV | Some asymmetry |

*Note*. SMD=Standardised mean difference; CV=Critical value; ns=Not significant; n/a=Not applicable.

***P<0.001; **P<0.01; *P<0.05.

[a] Sample of effect sizes.

† Marginally significant.

Table 2.33. *Summary of meta-analyses on abstraction in psychopathy*

| Abstraction | *n*[a] | Total samples | | SMD (>0=favours controls) | Heterogeneity | Publications bias & outliers | |
|---|---|---|---|---|---|---|---|
| | | Psychopathy | Control | | | Failsafe *N* | Funnel plot |
| Overall | 12 | 388 | 467 | 0 - 0.47*(0.34** without outlier) | ns - considerable (moderated without outlier) | n/a | Asymmetrical: 1 outlier |
| 1. Concept formation | 11 | 380 | 459 | 0 - 0.40 (inc. removal of outlier) | ns - considerable (moderate without outlier) | n/a | n/a |
| a. Verbal | 4 | 208 | 263 | 0.41** - 0.47*** | ns | <CV | n/a |
| b. Visual | 11 | 380 | 459 | 0.13 - 0.33 | Considerable | n/a | n/a |
| i. Sorting & shifting | See attentional set shifting above | | | | | | |
| ii. Short Category Test | 3 | 54 | 60 | 0.02 | ns | n/a | n/a |
| iii. Raven's matrices | 2 | 71 | 65 | 0.27 | ns | n/a | n/a |
| iv. Shipley Abstraction | 2 | 48 | 65 | 0.20 | ns | n/a | n/a |
| 2. Reasoning | | | | | | | |
| a. Verbal | 3 | 173 | 215 | -0.30 | Substantial | n/a | n/a |
| b. Visual | | | | | | | |
| i. Picture Completion | 2 | 35 | 46 | 0.29 | ns | n/a | n/a |
| ii. Picture Arrangement | 2 | 161 | 205 | 0.05 | ns | n/a | n/a |
| 3. Mathematical procedures | 2 | 161 | 205 | 0.43*** | ns | <CV | n/a |

*Note*. SMD=Standardised mean difference; CV=Critical value; ns=Not significant; n/a=Not applicable.

***P<0.001; **P<0.01; *P<0.05.

[a] Sample of effect sizes.

† Marginally significant.

Table 2.34. *Summary of meta-analyses on affect and social cognition in psychopathy*

| Affect & social cognition | *n*[a] | Total samples | | SMD (>0=favours controls) | Heterogeneity | Publications bias & outliers | |
|---|---|---|---|---|---|---|---|
| | | Psychopathy | Control | | | Failsafe *N* | Funnel plot |
| 1. Affective operations | 9 | 273 | 365 | 0.08 - 0.50*** | ns | >CV | Relatively symmetrical |
| a. Affective processing | 5 | 114 | 116 | 0.23 - 0.64*** | Moderate - ns | <CV | Asymmetrical |
| b. Affect recognition | 6 | 161 | 197 | 0.09 - 0.42*** | ns | <CV | Asymmetrical |
| i. Verbal | 4 | 87 | 97 | 0.04 - 0.51*** | ns | <CV | |
| Semantic | 3 | 66 | 63 | 0.36* - 0.62*** | ns | <CV | n/a |
| Prosodic | 2 | 55 | 68 | 0.02 - 0.36† | Considerable - ns | n/a | n/a |
| ii. Visual | 5 | 148 | 186 | 0.02 - 0.41* | Moderate | <CV | Asymmetrical |
| iii. Individual emotions | | | | | | | |
| Happiness | 6 | 176 | 191 | 0.28** - 0.37*** | ns | <CV | Some asymmetry |
| Sadness | 6 | 176 | 191 | 0.11 - 0.29** | ns | <CV | Some asymmetry |
| Anger | 5 | 159 | 174 | (-0.03) - 0.06 | ns | n/a | n/a |
| Disgust | 3 | 75 | 79 | 0.29† - 0.35* | ns | <CV | n/a |
| Surprise | 4 | 109 | 113 | 0.31* - 0.40** | ns | <CV | n/a |
| Fear | 4 | 125 | 140 | 0.25(-0.06 without outlier) - 0.31 | Substantial - moderate (ns without outlier) | n/a | n/a |
| c. Affect & memory | 3 | 118 | 185 | 0.13 - 0.38** | ns | <CV | n/a |
| 2. Social cognition | | | | | | | |
| a. Theory of Mind | 3 | 83 | 111 | (-0.26) - 0.05 | ns | n/a | n/a |

*Note*. SMD=Standardised mean difference; CV=Critical value; ns=Not significant; n/a=Not applicable.

***P<0.001; **P<0.01; *P<0.05.

[a] Sample of effect sizes.

† Marginally significant.

Table 2.35. *Summary of meta-analyses on memory and attention in psychopathy*

| Memory & attention | *n*[a] | Total samples | | SMD (>0=favours controls) | Heterogeneity | Publications bias & outliers | |
|---|---|---|---|---|---|---|---|
| | | Psychopathy | Control | | | Failsafe *N* | Funnel plot |
| 1. Memory | 8 | 262-265 | 325 | -0.15 (-0.26† without outlier) - 0.24**(0.20 without outlier) | Substantial - ns | <CV | Asymmetrical |
| a. Visual | 5 | 64 | 75 | (-0.02) - 0.15 | ns | n/a | n/a |
| b. Verbal | 6 | 228 | 272 | 0.10 - 0.22*(0.11 without outlier) | Substantial - ns | <CV | Asymmetrical |
| c. STM | 6 | 228 | 272 | 0.11 - 0.19*(0.05 without outlier) | ns - moderate | <CV | Asymmetrical |
| d. LTM | 3 | 58 | 59 | (-0.34†) - 0 | Substantial - ns | n/a | n/a |
| e. WM | 3 | 55 | 50 | 0.25 | ns | n/a | n/a |
| 5. Attention | 19 | 449-459 | 531-544 | (-0.17) - 0.30** | Moderate - substantial | >CV | Some asymmetry |
| a. Sustained | 6 | 110 | 130 | (-0.25) - 0.50*** | ns | <CV | Some asymmetry |
| b. Selective | | | | | | | |
| i. Stroop facilitation | 4 | 54 | 59 | 0.28 | Moderate | n/a | n/a |
| See above for Stroop errors, response latency & interference | | | | | | | |
| ii. Non-Stroop | See above | | | | | | |
| c. Divided | 4 | 88 | 129 | 0.04 - 0.50* | Considerable - moderate | <CV | n/a |
| d. Complex | 5 | 220-230 | 264-277 | (-0.02) - 0.51 | Considerable | n/a | n/a |

*Note*. SMD=Standardised mean difference; CV=Critical value; ns=Not significant; n/a=Not applicable; S/LT/WM=Short/long-term/working memory.

***P<0.001; **P<0.01; *P<0.05.

[a] Sample of effect sizes.

† Marginally significant.

Table 2.36. *Summary of meta-analyses on language, perception, construction, and intelligence in psychopathy*

| Cognitive functions | *n*[a] | Total samples | | SMD (>0=favours controls) | Heterogeneity | Publications bias & outliers | |
|---|---|---|---|---|---|---|---|
| | | Psychopathy | Control | | | Failsafe *N* | Funnel plot |
| 1. Language | 15 | 408 | 465 | 0.04 - 0.61***(0.35*** without outliers) | Moderate - substantial (ns without outliers) | >CV | Some asymmetry: 2 outliers |
| a. Verbal expression | 11 | 332 | 391 | 0.07 - 0.42*(0.30* without outliers) | Substantial | >CV | Asymmetrical |
| i. Fluency | See Executive functions: Productivity above | | | | | | |
| ii. Vocabulary | 5 | 236 | 295 | 0.11 | Ns | n/a | n/a |
| iii. Discourse: syntax | 5 | 44 | 79 | 0.48 - 1.18* | Considerable | >CV | Asymmetrical |
| b. Academic skills: reading | 2 | 56 | 60 | 0.26 | ns | n/a | n/a |
| c. Semantic processing | 6 | 106 | 107 | (-0.44; -0.03 without outlier) - 0.91*(0.53*** without outlier) | Considerable (ns without outliers) | >CV | Asymmetrical: 1 outlier |
| d. Knowledge | 2 | 161 | 205 | 0.49* | ns | <CV | n/a |
| 2. Perception: visual | 4 | 112 | 119 | 0.14 - 0.34* | ns | <CV | n/a |
| 3. Construction | 5 | 226 | 277 | 0 - 0.09 | Considerable - substantial | n/a | n/a |
| 4. Intelligence | | | | | | | |
| a. FSIQ | 13 | 402 | 468 | 2.55** - 3.29** | Moderate | <CV | Asymmetrical |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | (without studies adopting IQ recruitment cut-offs) | | | |
| b. VIQ | 5 | 79 | 115 | 0.58 - 0.59 | ns | n/a | n/a |

*Note*. SMD=Standardised mean difference; CV=Critical value; ns=Not significant; n/a=Not applicable; FSIQ/VIQ=Full-scale/verbal IQ.

***P<0.001; **P<0.01; *P<0.05.

[a] Sample of effect sizes.

† Marginally significant.

Table 2.37. *An evaluation of theories of the antisocial personality against current findings*

| Theory | May explain | May not explain |
|---|---|---|
| BIS/BAS (J. A. Gray, 1987) & Fear dysfunction (Lykken, 1995) | Impaired motor regulation. | Generalised affect recognition, other affective deficits, impairment in concept formation, language anomalies, or other possible executive impairments. |
| Response modulation (Patterson & Newman, 1993) | Impaired motor regulation. | As above. In addition, it predicts impaired attentional processes (primarily selective attention) for which evidence in this review was not robust. |
| Frontal lobe dysfunction (Raine, 2002) | Impaired motor regulation and other executive functions, perhaps concept formation deficit, and language anomalies. | Affective deficits or broader language anomalies. Frontal deficits (executive functions) did not appear as extensive as the theory might suggest. |
| Somatic Marker Hypothesis (Damasio, 1994) | Impaired motor regulation. | Generalised affect recognition, other affective deficits, impairment in concept formation (verbal), language anomalies, or other possible executive impairments. |
| LHA (Kosson, 1998) | Language anomalies and impairment in verbal concept formation. | Affective and executive deficits (including motor regulation). |
| VIM (R. Blair et al., 2005) | Some affect recognition deficits. | Impaired executive functions (including motor regulation), concept formation, or language anomalies. In addition, the evidence suggesting impaired sadness or fear recognition was not robust. |
| IES (R. Blair et al., 2005) | Impaired motor regulation, affect recognition, and other possible affective deficits. | Lack of impairment in fear recognition. Deficits in concept formation, any language anomalies, and other possible executive deficits. |

*Note*. LHA=Left Hemisphere Activation; VIM=Violence Inhibition Mechanism; IES=Integrated Emotion Systems.

# 3 AIMS AND HYPOTHESES

The systematic literature review indicated that various neurocognitive deficits have been consistently associated with the antisocial personality across operationalisations. These include motor regulation, affect recognition, and verbal concept formation. Beside these functions, however, ASPD and psychopathy did not appear to share the same neurocognitive profile. Cognitive flexibility (attentional set-shifting, response reversal, and complex decision-making) appeared consistently impaired in psychopathy but evidence was less consistent for ASPD (supported only with use of the CANTAB), while planning appeared impaired in ASPD but evidence was less conclusive for psychopathy. There was less clarity regarding other functions but some evidence indicated potential impairment in sustained attention, visual STM (but not verbal or WM) in ASPD but not psychopathy. Furthermore, verbal expression and visual perception may be impaired in psychopathy but evidence did not support this in ASPD. Although the literature suggests that ASPD and psychopathy exhibit different neuropsychological difficulties, no study has yet attempted to examine them in parallel. Therefore, further research is required in order to (i) clarify the presence of neurocognitive deficits in the antisocial personality beyond motor regulation, affect recognition, and verbal concept formation and (ii) investigate the differences in impairment between ASPD and psychopathy. The first aim of this project's empirical part was to address these issues.

The importance of investigating the effects of cognitive deficits on the course of treatment for individuals with antisocial personality was also emphasised. As current therapeutic interventions for this population are cognitive-behavioural based and involve a range of tasks closely related to specific neuropsychological functions, neurocognitive impairment is likely to impede progress in the treatment for people with antisocial personality. Investigating this effect formed the second aim of this project's empirical part.

### 3.1 Cognitive deficits in the antisocial personality

In examining cognitive deficits, focus was on areas where impairments were observed consistently but ASPD and psychopathy differed (planning and general cognitive flexibility) and where literature was less clear in supporting difficulties (e.g. sustained attention, attentional set-shifting, STM, verbal functions, and visual perception). In order to demarcate cognitive impairments and detect potentially mild deficits in these functions, accurate, sensitive, and cognitively focused measurement was necessary. The CANTAB was selected to meet these criteria, addressing the majority of the above functions while it lacks coverage of verbal expression only. Furthermore, the CANTAB is able to evaluate motor regulation (though with some confounding with affect recognition), further enabling confirmation or otherwise of one of the robust findings of the systematic review.

The hypotheses regarding cognitive impairment in ASPD and psychopathy were as follows:

#### 3.1.1 ASPD

1. Individuals with ASPD will demonstrate deficits primarily in (a) motor regulation, (b) planning, and (c) cognitive flexibility (response reversal, attentional set-shifting, & decision-making) and potentially in (d) sustained attention and (e) visual STM, using the CANTAB for testing these functions.
2. Individuals with ASPD will not show impairments in (a) verbal memory, (b) WM, and (c) visual perception, using the CANTAB for testing these functions.

#### 3.1.2 Psychopathy

1. Individuals with psychopathy will demonstrate deficits primarily in (a) motor regulation and (b) cognitive flexibility (response reversal, attentional set-shifting, & complex decision-making) and potentially in (c) planning and (d) visual perception, using the CANTAB for testing these functions.

2. Individuals with psychopathy will not show impairments in (a) sustained attention and (b) memory (visual, verbal, and WM), using the CANTAB for testing these functions.

### 3.2 Cognitive abilities and the course of treatment for antisocial personality

The second aim of the project was to assess the effect of impaired cognitive performance on progress in treatment for individuals with antisocial personality. Because this is an unexamined area, hypotheses were derived on the basis of those cognitive deficits expected in this population as outlined above while taking into account their relevance to the thinking processes involved in cognitive behavioural-based therapies which is the primary and preferred treatment modality for individuals with antisocial personality (NICE, 2009). Such processes often involve planning, learning to identify and inhibit inappropriate responses, generating and evaluating alternative options (set-shifting, reasoning), processing different concepts, ability to recognise and appraise emotions and social cues, communicating, learning new skills, and so forth (Ledley et al., 2010; Padesky, 1993; Westbrook et al., 2011). Therefore, the neurocognitive difficulties anticipated in ASPD and psychopathy (e.g. in motor regulation, cognitive flexibility, planning, sustained attention, STM, and even visual perception) would imply a reduced ability to benefit from cognitive-behavioural treatments over time resulting in poorer outcomes.

As many of the recommended psychological interventions for the antisocial personality also aim at imparting skills so that individuals are able to plan better, develop alternative behaviours, or act less impulsively (NICE, 2009), neuropsychological deficits in executive functions such as planning, cognitive flexibility, and self-regulation could prove considerable barriers to treatment. Furthermore, although functions like attention, memory and even perception may not appear as directly related to the main aims of treatment for the antisocial personality, they mediate learning and skills acquisition (Gazzaniga et al., 2009; Kolb & Whishaw, 2009; Martin, 2006; Rosenzweig et al., 2007). Therefore, impairment in these operations is also likely to impede treatment progress. On the other hand, unimpaired functions in ASPD and psychopathy should not affect treatment progress in these

populations. Translating these relationships into specific predictions regarding performance on CANTAB tasks and progress in treatment resulted in the following hypotheses for ASPD and psychopathy:

### 3.2.1 ASPD

1. Impairments in (a) motor regulation, (b) planning, and (c) cognitive flexibility and potentially in (d) sustained attention and (e) visual STM will independently predict negative progress in treatment in individuals with ASPD.
2. Performance in (a) verbal memory, (b) WM, and (c) visual perception as assessed by the CANTAB will not predict progress in treatment in individuals with ASPD.

### 3.2.2 Psychopathy

1. Impairments in (a) motor regulation and (b) cognitive flexibility and potentially in (c) planning and (d) visual perception will independently predict negative progress in treatment in individuals with psychopathy.
2. Performance in (a) sustained attention and (b) memory (visual, verbal, and WM) as assessed by the CANTAB will not predict progress in treatment in individuals with psychopathy.

# 4 DEVELOPMENT OF THE PROGRESS RATING SCHEDULE (PRS)

## 4.1 Introduction

This study (1) examined the neuropsychological performance of psychiatric patients with antisocial personality (ASPD or psychopathy) in comparison to peers with other personality difficulties and healthy controls; and (2) explored the relationship between cognitive ability and progress in treatment. However, to measure progress in treatment, it was necessary to develop a new instrument, the PRS.

Offenders with personality disorders in treatment often present with complex needs and there is uncertainty as what might reflect progress in treatment. Furthermore, many of the current interventions and an evaluation of their impact have been developed in other clinical areas of mental health and thus are not tailored to difficulties characterising personality disordered offenders (Howard & Howells, 2010). For instance, the instrument currently widely adopted in the UK to measure clinical outcome in forensic healthcare – the Health of the Nation Outcome Scales (HoNOS)-Secure Services (Dickens, Sugarman, & Walker, 2007) – does not appear sensitive to measuring change in personality disorder as it was devised for those with mental illness (e.g. psychosis). What complicates matters further is that as the treatment of personality disorder is multidisciplinary, it is unlikely that there will be agreement between different professionals for a particular patient – unless it is anchored to standardised criteria. For example, what different disciplines or individuals regard as good engagement, better behaviour or an improved mental state is likely to vary considerably. Hence, there is a need to operationalise what constitutes progress in treatment to ensure that members of the multidisciplinary team will agree.

These concerns and difficulties regarding measuring progress in treatment in personality disorders prompted the development of the Progress Rating Schedule (PRS) for offenders with personality disorder in this project. The aim was to derive an instrument conceptualising and operationalising progress in treatment as it is described within current clinical practice. This may not necessarily map onto specific symptoms from the diagnostic nomenclature, which are generally enduring (and therefore not suitable for assessing short or mid-term change) while their conceptualisation has been

both evolving and inconsistent over the years (APA, 1980, 1987, 1994, 2000; Hare, 1980, 1991, 2003; World Health Organisation, 1990). Furthermore, the PRS takes account of input from clinicians from a number of disciplines and therefore may offer a more consistent and systematic method of reporting progress than other approaches currently available.

## 4.2 Method

### 4.2.1 The Setting

The project was undertaken at the Personality Disorder Service (PDS) at Arnold Lodge Regional Secure Unit in the East Midlands region of England. The service comprises of 24 beds across two wards: Cannock and Ridgeway. Cannock admits individuals transferred primarily from prisons on a voluntary basis (Section 47/49 of the Mental Health Act, 1983) and delivers a 2-year treatment programme. Ridgeway accepts patients for longer-term stay most of whom were admitted to high secure hospitals under Section 37/41 of the 1983 Mental Health Act prior to their transfer to medium secure conditions.

Criteria for admission to the PDS include residence in the catchment area, possession of adequate intellectual functioning for the treatment programme, sufficient duration of remaining sentence and absence of psychosis and bipolar disorder. Generally, patients with a PCL-R>25 or an index offence of sexual nature are not admitted, as the treatment programme is not tailored to the specific needs of these individuals. However, since Ridgeway ward became operational in 2008 such patients are also being admitted. Furthermore, admission requires the patient to agree to a strict regimen so that persistent disengagement, rule breaking, or a major disruption leads to a premature discharge. As a consequence, only a quarter of the patients complete treatment at their first admission (McCarthy & Duggan, 2010). Treatment aims primarily at reducing reoffending by teaching skills so that individuals no longer continue to act so impulsively. It follows the national guidelines (NICE, 2009) comprising of mainly group-based cognitive-behavioural interventions (e.g. social problem solving, anger management, arson treatment). Consistent with forensic mental health practice in the UK, the PDS holds routine CPA meetings at which each professional discipline is expected to produce a report on the progress of the patient. These meetings are usually held at six-monthly intervals for each patient.

The PDS has strong links with prisons and therefore offers the benefit of providing a more representative context for the study of the antisocial personality than settings of low or high security alone as well as greater assessment and treatment

control than a prison. A disadvantage of the project's setting, however, is that it involved a single location thereby limiting generalisation of findings.

### 4.2.2 Participants

Participants were psychiatric patients (with and without antisocial personality). All were residents at the PDS of Arnold Lodge, the majority of whom were transferred from prison, comprising 81% of the sample. Participants were predominantly Caucasian (94.1%), with a further 4.7% and 1.2% of Mixed and Black backgrounds respectively. Between February 1999 and July 2011, 236 patients were referred to the unit of whom 134 were admitted (at the rate of just under one patient per month). Mean age at first admission was 30.75 years (*SD*=8.64, Range: 18.6 – 58.4) and mean IQ was 88.29 (*SD*=13.72, Range: 62 – 139). Data were collected on 102 of those patients whose admissions were consecutive. Data were not collected if consent was declined. Informed consent was obtained prior to or on admission following a briefing of the study and discussion of procedures if requested. Administration of the International Personality Disorder Examination (IPDE) determined the ASPD/Non-ASPD groups whereas administration of the PCL-R determined the groups with/without psychopathy. Diagnostic and screening interviews and assessments took place during pre-admission meetings.

### 4.2.3 Materials

A range of instruments were used for diagnosis, identification of psychopathy, measurement of confounders, and neuropsychological performance. The clinical setting supported the rigorous application of patient diagnostic assessments whereas comparable screening instruments and measures were selected to assess the healthy control group.

#### 4.2.3.1 Axis I and II psychopathology

All patients were assessed for Axis I and personality disorders, psychopathy and overall intelligence by suitably qualified clinicians as part of their routine pre-admission assessment. Axis I psychopathology was assessed by using the Schedule for

Affective Disorders and Schizophrenia-Lifetime diagnosis (SADS-L; Spitzer & Endicott, 1978) until 2004. The SADS-L was then replaced by the Structured Clinical Interview for DMS-IV Disorders-Axis I: Clinical version (SCID-I:CV; First, Spitzer, Gibbon, & Williams, 2002) as the Axis I diagnostic instrument. Previous research on the SADS-L and SCID found inter-rater reliability ranging between moderate to very high (Kappa and intraclass correlations [ICCs]: 0.75-0.85 and above). Both instruments have also shown moderate test-retest reliability in a number of studies (R. Rogers, 2001): for the SADS-L, Kappa has ranged between 0.57-0.73 with professional raters for periods up to six months whereas a mean Kappa of 0.61 has been reported for the SCID for periods up to two weeks. In addition, both instruments have shown good predictive, concurrent, construct, convergent and discriminant validity (R. Rogers, 2001).

The DSM module of the IPDE (Loranger, 1999) is a widely-adopted assessment of personality disorder and the interview (without third-party information) is used to assess presence of personality disorder for all patients on the PDS. It comprises of 99 items organised in six categories: work, self, interpersonal relations, affect, reality testing and impulse control. R. Rogers (2001) observed that dimensional ratings on personality disorders in this semi-structured clinical interview were associated with good inter-rater reliability (ICCs: 0.85-0.94) in various cultural settings but this was found to be moderate for categorical diagnoses (median Kappa: 0.70). Similarly, temporal stability over and average 6 months was reported to be high for dimensional ratings (median ICC=0.79) whereas it was found to be low-moderate for categorical diagnoses (median Kappa=0.48).

#### 4.2.3.2 The PCL-R (Hare, 2003)

This instrument evaluates the degree of psychopathy and is based on an earlier research scale. It contains 20 items scored from 0-2 based on file review and semi-structured interviews. Higher scores indicate greater presence of psychopathy and the European cut-off score for suggesting presence of psychopathy is 25 (Hare, 2003). The instrument is generally associated with relatively high inter-rater reliability (ICCs: 0.78-0.93) and internal consistency (*alpha*: 0.81-0.85) and moderate temporal stability

(ICCs: 0.43-0.60) over a period of two years (Hare, 2003; R. Rogers, 2001). The PCL-R has been extensively validated in a variety of criminological and secure hospital settings demonstrating good concurrent, construct and predictive validity and moderate convergent validity (Hare, 2003; R. Rogers, 2001).

#### 4.2.3.3 WAIS-Third Edition (WAIS-III; Wechsler, 1997)

In addition to the previous materials, the WAIS-Third Edition (WAIS-III; Wechsler, 1997) was used to measure and control for intelligence in this section. It is the most widely used neuropsychological battery employed to estimate age-graded scores of overall intelligence based on performance on 11 qualitatively different sub-tests. The scale has been extensively researched showing good validity and has good sensitivity with ability to capture mild and moderate impairment (Lezak et al., 2004).

#### 4.2.3.4 Defense Style Questionnaire (DSQ)

This questionnaire reflects overall adjustment by measuring three dysfunctional and one adaptive defence styles. High item-total correlations and factor analysis supported the instrument's internal consistency (Bond, Gardner, Christian, & Sigal, 1983) but this has not been re-examined more recently. Defence style immaturity has been associated with personality disorder severity (Sammallahti, Aalberg, & Pentinsaari, 1994) while use of a range of dysfunctional defence mechanisms has been shown to reflect personality disorder pathology (J. G. Johnson, Bornstein, & Krukonis, 1992; Mulder, Joyce, Sullivan, Bulik, & Carter, 1999; Zanarini, Weingeroff, & Frankenburg, 2009). More crucially, however, the DSQ has been able to capture progress in treatment in samples with personality disorders (Bond & Perry, 2004) thereby supporting its appropriateness in validating the PRS as a change measure in this population. PRS scores should reflect positive change in defence styles as measured by the DSQ.

#### 4.2.3.5 Social Problem-Solving Inventory-Revised (SPSI-R)

Unlike the DSQ, the SPSI-R is rather specific in its focus as it examines social problem solving attitudes and skills according to a specific model (D'Zurilla et al.

2002). The internal consistency of the instrument and its subscales has ranged from acceptable to good (*alphas*: 0.65-0.94). Social problem solving has been identified as an area of deficiency in personality disorder (McMurran, 2009), particularly antisocial and borderline, and a specific therapeutic intervention has been developed based on D'Zurrilla's model to address these difficulties (Huband, McMurran, Evans, & Duggan, 2007; McMurran, Egan, & Duggan, 2005). As the social problem-solving intervention forms a core component of the treatment programme provided at the PDS, examining the relationship between PRS and SPSI-R scores in the sample provided an additional avenue for assessing the validity of the new instrument. PRS scores should reflect a positive change in problem solving attitudes and skills as measured by the SPSI-R.

### 4.2.4 Instrument development

#### 4.2.4.1 Items

In developing the PRS, the aim was to capture a conceptualisation of progress in treatment within current clinical practice. To this end, qualitative methodology (Willig, 2008) was employed to identify and operationalise what clinicians routinely use to describe as progress in treatment, in a naturalistic setting (PDS). Within this inductive framework, thematic analysis, which aims to identify "repeated patterns of meaning" in the data (Braun & Clark, 2006, p. 86), was applied on a random sample of seven archived CPA minutes. The CPA minutes summarised treatment review reports from a range of professional disciplines and a discussion of the clinical team regarding the progress of a patient. The relevant sections of the data were coded by two researchers systematically and comprehensively across the dataset collating associated references. These emerging themes were then examined in relation to each other and organised within clusters reflecting those concepts in the data which were relevant to the research question. The researchers, whose background was in nursing, psychiatry, and psychology, undertook this process independently but discrepancies in the identified themes at the end of the process were resolved via discussions. The resulting initial inventory of progress items was then reviewed by the same individuals (independently once again) against a further set of seven anonymised reports, in a

procedure akin to theoretical sampling in grounded theory methodology (Willig, 2008). Further discrepancies in the results were also resolved via discussions, providing the final list of components for the PRS including specifics of their definition, scope, and scoring.

#### *4.2.4.1.1 Methodological reflections*

Thematic analysis can accommodate a range of epistemological approaches (Braun & Clarke, 2006). In this project, it was employed in a manner that focused on the research question with the aim of deriving suitable items for the PRS based on progress accounts available in a naturalistic setting. As such, it reflects an empiricist approach while supporting ecologically valid results. However, the process of identifying and organising themes involves judgment (Braun & Clarke, 2006) with the recognition that this is likely influenced by the researchers' background (theoretical, professional, epistemological, etc.). Furthermore, using minutes from meetings as data source and resolving discrepancies in the researchers' themes via discussions implied jointly constructed meanings. Nevertheless, these features formed part of the process towards identifying a collection of items representative of how progress is understood in current clinical practice rather than investigating phenomena of social construction. Therefore, the project's approach to developing the PRS lies closer to critical realism with hermeneutic aspects rather than relativism.

### 4.2.4.2 Refinement

Minutes from 29 treatment review meetings for 12 randomly selected patients were then rated independently by three individuals representing the disciplines of psychiatry, nursing, and psychology in a process which sought to improve face and content validity (McBurney & White, 2007) as well as allowing the evaluation of the instrument's inter-rater reliability. This process was repeated by two of the raters for a further 21 treatment review meetings for another 8 randomly selected patients from the same service. For the patients in this sample, length of stay on the PDS ranged from 10.3 to 125 (mean 60.7) weeks with treatment review meetings held at least twice for each patient. Weaker inter-rater agreement at this stage was considered as

indicator of greater ambiguity in items and was used to guide further item revisions to improve face validity. Using the amended criteria, PRS scores were blindly revised by each of the three raters and inter-rater reliability was reassessed.

#### 4.2.4.3 Clinician validation

Consistent with the practice of participant validation to verify findings in qualitative research (Willig, 2008), the PRS in its final format was trialled by the clinical teams on the PDS at six CPA meetings. It was rated at the end of each meeting following a detailed discussion of the patient's progress. Feedback and comments on the instrument's utility and validity were invited.

### 4.2.5 Psychometric properties

#### 4.2.5.1 Data

Available first, second, and final treatment review reports were scored for 101 patients for a total of 232 review meetings. Outliers were addressed and the dataset was screened for normality, linearity, multicollinearity, and singularity prior to analyses (Pallant, 2005; Tabachnick & Fidell, 2007), as discussed below.

#### 4.2.5.2 Internal consistency

The internal consistency (Sim & Wright, 2000) of Part A of the PRS, which contains items rated on a scale (see below for details on the format of PRS), was examined using item analyses.

#### 4.2.5.3 Criterion validity

Concurrent validity was evaluated by examining the relationships between PRS scores and the subscales of two psychometric instruments: the DSQ (Bond & Wesley, 1996) and the SPSI-R (D'Zurilla, Nezu, & Maydeu-Olivares, 2002). Both psychometric instruments formed part of a standard battery administered prior to each CPA meeting and were selected on the basis of reflecting personality disorder pathology.

Predictive validity was evaluated by examining PRS scores over time and their relation with psychopathy to assess the instruments ability to provide a clinically meaningful picture of patient progress. Although in the past psychopathy was considered untreatable, evidence suggests that it may not be so (Skeem, Monahan, & Mulvey, 2002) while general prognosis can vary depending on which dimension is under scrutiny (Hare, 2003). As far as the PDS cohort is concerned, however, McCarthy and Duggan (2010) have highlighted generally conservative completion rates and higher psychopathy scores in non-completers. In addition, higher psychopathy has predicted poorer criminological and psychosocial outcomes following discharge (McCarthy et al., 2012). Therefore, the PRS ought to reflect both variation between patient progress over time and smaller gains in PDS patients with psychopathy thus supporting the clinical utility of the instrument in predicting progress in treatment meaningfully and in line with existing clinical observations.

### 4.2.6 Statistical data analysis

#### 4.2.6.1 Data screening and assumptions

Assumption violations were reported only when detected. Data screening was conducted as discussed by Pallant (2005) and Tabachnick and Fidell (2007):

a. ***Outliers*:** Univariate and multivariate (detected via Mahalanobis distance) outliers were set at *alpha* level 0.001 (Tabachnick & Fidell, 2007) and exclusion from analyses was considered. Care was exercised not to exclude possible outliers when high standard deviation values resulted from non-normal distributions, so as not to render samples unrepresentative.
b. ***Normality*:** Assessed via histogram inspection, K-S test, and skewness and kurtosis (significant at *alpha* level 0.001). ANOVA techniques are considered robust against violations of normality (Pallant, 2005; Tabachnick & Fidell, 2007).
c. ***Linearity*:** Evaluated via examination of bivariate scatter plots.
d. ***Variance assumptions (heterogeneity)*:** Box's M (*alpha* level 0.001) and Levene's test. Pillai's Trace was selected in inferential statistics as more robust

to variance assumption violations (Tabachnick & Fidell, 2007). ANOVA techniques are generally robust to such violations, provided the samples are of reasonably similar size (1.5 ratio) (Pallant, 2005; Tabachnick & Fidell, 2007).

e. ***Multicollinearity and singularity*:** Two indicators were examined – bivariate correlations with $r>0.07$ and tolerance approaching zero (in the range of 0.1) or condition index exceeding 30 coupled with variance proportions greater than 0.5 for at least two different variables (Belsey, Kuh, & Welsch, 1980; Tabachnick & Fidell, 2007). For repeated measures, tolerance below 0.001 may indicate potentially problematic multicollinearity (Tabachnick & Fidell, 2007).

f. ***Missing data in Multilevel Modelling (MLM)*:** Missing data can be tolerated well in MLM particularly when either missing completely at random (MCAR, Little's test is not significant) or missing at random (MAR) (H. Goldstein, 2003; Rasbash, Steele, Browne, & Goldstein, 2009; Tabachnick & Fidell, 2007). The latter may be assumed when Little's test is significant but data are missing in an expected patter (Tabachnick & Fidell, 2007). For example, the presence of more missing PRS scores at later time-points was expected in this project as more PDS patients were discharged earlier in their treatment.

#### 4.2.6.2 Refinement

The instrument's refinement involved examination of inter-rater reliability. This was explored via the intraclass correlation coefficient (ICC) using a two-way random effects model (individual measures, absolute agreement definition) with 95% confidence intervals (McGraw & Wong, 1996). ICCs were interpreted according to Shrout (1998) as follows: virtually no agreement (0-0.10), slight agreement (0.11-0.40), fair agreement (0.41-0.60), moderate agreement (0.61-0.80), and substantial agreement (0.81-1.00).

#### 4.2.6.3 Psychometric properties

##### *4.2.6.3.1 Internal consistency*

These included item analyses involving principal components analysis (PCA; Pallant, 2005; Tabachnick & Fidell, 2007) to explore whether the scale of Part A

consisted of separable components and Cronbach's *alpha* coefficients (Pallant, 2005) to examine the overall internal consistency of the scale. Analyses were conducted for the first, second, and final treatment reports. Following screening for PCA, components were extracted using scree test, eigenvalues (>1.0), and item loadings (Pallant, 2005; Tabachnick & Fidell, 2007). Cronbach's *alpha* coefficients of 0.7 and above suggested acceptable internal consistency (Pallant, 2005).

#### *4.2.6.3.2 Criterion validity*

MLM (H. Goldstein, 2003; Rasbash et al., 2009; Tabachnick & Fidell, 2007) was selected to examine whether PRS scores were associated with psychometrics (DSQ & SPSI-R subscales) and days since admission (growth trajectories) at first, second, and final treatment reviews (Level 1) clustered within patients (Level 2). Models were developed separately for each predictor (individual psychometric subscales, time since admission, psychopathy) with the intercepts-only model, which contains no predictors, as baseline. Pseudo-$R^2$ was used to estimate the change in residual variance between two models (Raudenbush & Bryk, 2002; Singer & Willett, 2003). This quantified how much of the variation in PRS scores within and between patients was explained by predictors added at each stage. The pseudo-$R^2$ was computed as percentage of $((\sigma_2^2-\sigma_1^2)/(\sigma_1^2))$ where $\sigma_1^2$ and $\sigma_2^2$ represented the variance in the first and second comparison models respectively – this formula may produce negative values which are considered not interpretable (Raudenbush & Bryk, 2002; Singer & Willett, 2003).

For the analyses on validity, it was necessary to compute PRS total scores at each treatment review, by summing up the ratings of constituent items. As all item ratings were required for this, missing data were substituted for one missing constituent item. Missing data substitution differed between PRS Parts A and B because the former reflected a unitary factor whereas the latter did not (see below for details on the structure and psychometric properties of the PRS). For Part A, the missing item was replaced by the mean score of the available items of the same patient. For Part B, the missing item was replaced by the mean for that item across all patients. This process resulted in the total of available PRS scores to increase from

161 to 180 for Part A and from 202 to 228 for Part B. Because DSQ and SPSI-R data were not available for all these scores, analyses including the two psychometric instruments involved 174 scores for 96 patients on Part A and 173 scores for 95 patients on Part B.

#### 4.2.6.4 Software and statistical significance

MLM was conducted using MLwiN software, v.2.24 (Rasbash, Charlton, Browne, Healy, & Cameron, 2011). SPSS software, v.17.0 (SPSS Inc, 2009) was employed for all other analyses with an *alpha* level set at 0.05 for all statistical tests unless otherwise specified.

## 4.3 Results

### 4.3.1 Sample characteristics

Of the admitted patients, 32 did not consent to take part in the study or were discharged prior to the CANTAB assessment (76% response rate). MANOVA suggested that they were comparable to the 102 participants on age, $F(1,100)=1.89$, IQ, $F(1,100)=0.28$, total PCL-R score, $F(1,100)=0.44$, and number of personality disorders, $F(1,100)=3.13$. Of the participating patients, 17% were still undertaking treatment at the PDS at the time of this project, 25% had completed treatment during their initial admission and had been discharged whereas the remainder were discharged prematurely due to non-engagement (24%), violence (13%), management issues (6%), left against advice (5%) and other reasons. Approximately a quarter of the discharged patients were readmitted and 16% of those went on to complete treatment. An overview of samplecharacteristics is presented in Table 5.2.

### 4.3.2 Development of the PRS

#### 4.3.2.1 Items and structure

The preliminary list of items included behavioural, psychological, and social areas of functioning. Any routine records (e.g. drug screens), standardised evidence of progress (e.g. critical incident reports & anger logs), and other tangible evidence (CPA

reports) were incorporated. Following piloting and refinement, the resulting instrument consisted of 11 items organised in two parts. The PRS is presented in the Appendix including a list of items, their scope, and guidelines on rating.

Part A comprises of items intrinsic to treatment which reflect overall adjustment within the programme. Items include Engagement with the therapeutic programme (e.g. attendance, homework compliance), Behaviour (e.g. incidents of aggression, rule breaking), Mental state (e.g. Axis I symptoms, self-harm), Interactions with peers and non-staff individuals or other members of the public excluding family/friends (e.g. positive & appropriate contact), Interactions with staff (e.g. seeking support, therapeutic relationships), and Insight (e.g. accepting responsibility, recognising need for treatment). All items in Part A are measured on a Likert-type scale from 0-3 with 0 reflecting poor and 3 very good performance while rating is fully operationalised. For example, for the item *Interactions with peers and non-staff individuals*, a rating of 'poor' reflects serious concerns (i.e. clear indications of inappropriateness in interactions with at least one individual), whereas a rating of 'reasonable' reflects either limited interactions without significant concerns, or interactions with the majority that were problematic but less severe than 'poor'. Positive interactions with the majority with minimal concerns and difficulties with some peers are rated as 'good' whereas positive interactions with the majority with no concerns with (almost) all peers are rated as 'very good'.

Part B consists of heterogeneous items extrinsic to adjustment in treatment but nevertheless representing progress. These items may depend on external factors or agencies as well as individual patient circumstances. Items in Part B include supportive relationships (outside the health service, e.g. family, evidenced by visits, regular contact, etc.), risk/violence (actuarial: Historical Clinical Risk – 20 [HCR-20]; Webster, Douglas, Eaves, & Hart, 1997), employment (e.g. work placement within or outside the service such as shop assistant, further/higher education), leave status (escorted or unescorted), and final outcome (positive or negative based on reason for discharge & placement following discharge). Except risk/violence, which is rated as low/medium/high based on normative data, other items are rated as either present or

absent. It was not always clear whether an outside relationship was supportive leading to the additional rating of "maybe" for that item only.

It was possible to identify other progress items (e.g. various psychometrics, records of critical behavioural incidents) but these were not included in the PRS as they may be specific to the PDS of Arnold Lodge. Instead, a customisable Part C is included so that services wishing to adopt the measure may incorporate any progress items they use locally and consider central to patient progress.

#### 4.3.2.2 Refinement

Initial ICCs for the assessment of inter-rater agreement are shown in

Table 4.1. There was complete overall agreement on Final outcome. Furthermore, agreement was substantial on Engagement and Behaviour and moderate on Mental state, Interactions with peers and staff, and Insight. Fair agreement was achieved on Supportive relationship while there was only slight agreement on Employment attributed to lack of clarity on the initial scoring instructions. As Part B consists of dichotomous or categorical variables, even small disagreements result in low ICCs. Escorted and unescorted leave were not rater-dependent and were excluded from this analysis. Although some items showed good inter-rater reliability implying similar interpretations between raters, the content validity of other items such as interactions with staff, insight, and employment could be improved.

Table 4.1. *Initial inter-rater agreement for PRS items*

| | ICCs | | | |
|---|---|---|---|---|
| | **Overall (*n*=29)** | **1-2 (*n*=29)** | **1-3 (*n*=50)** | **2-3 (*n*=29)** |
| ***Part A:*** | | | | |
| **Engagement** | 0.82 | 0.73 | 0.86 | 0.98 |
| **Behaviour** | 0.81 | 0.75 | 0.89 | 0.93 |
| **Mental state** | 0.65 | 0.48 | 0.77 | 0.87 |
| **Interact-Peers** | 0.68 | 0.50 | 0.88 | 0.89 |
| **Interact-Staff** | 0.61 | 0.45 | 0.87 | 0.76 |
| **Insight** | 0.63 | 0.47 | 0.84 | 0.83 |
| | | | | |
| ***Part B:*** | | | | |
| **Supportive rel.** | 0.48 | 0.27 [a] | 0.65 | 0.65 |
| **Employment** | 0.37 | -0.05 [a] | 0.57 | CA |
| **Outcome** | CA | 0.76 | 0.89 | CA |

*Note*. ICC=Intraclass correlation; PRS=Progress Rating Schedule; CA=Complete Agreement; 1=NH; 2=BV; 3=MB; Raters 1 and 3 scored the PRS on *n*=50 treatment reports whereas rater 2 did so for *n*=29 reports; All results significant at *P*<0.05 unless stated otherwise.

[a] not significant

The ensuing consensus revisions of the PRS items and scope resulted in clarification as to which activities constitute Employment, introducing pro-rating for Engagement, further specifying incidents relevant to Behaviour, further operationalising and clarifying scoring of Mental State, elaborating the description of good/poor interaction with peers and staff, and further qualifying what could be considered relevant to Insight from materials in the examined reports. This process resulted in overall increase of inter-rater agreement for all items, as indicated by the final ICCs (

Table **4**.**2**). There was moderate agreement for mental state, interactions with staff, and supportive relationship whereas for the remaining items agreement was substantial or complete.

Table 4.2. *Final inter-rater agreement for PRS items*

| | ICCs | | | |
|---|---|---|---|---|
| | **Overall (*n*=29)** | **1-2 (*n*=29)** | **1-3 (*n*=50)** | **2-3 (*n*=29)** |
| ***Part A:*** | | | | |
| **Engagement** | 0.91 | 0.86 | 0.94 | 0.97 |
| **Behaviour** | 0.92 | 0.86 | 0.96 | 0.98 |
| **Mental state** | 0.69 | 0.51 | 0.85 | 0.89 |
| **Interact-Peers** | 0.81 | 0.72 | 0.92 | 0.95 |
| **Interact-Staff** | 0.63 | 0.44 | 0.89 | 0.79 |
| **Insight** | 0.71 | 0.55 | 0.85 | 0.90 |
| | | | | |
| ***Part B:*** | | | | |
| **Supportive rel.** | 0.66 | 0.52 | 0.77 | 0.74 |
| **Employment** | ---------- CA ---------- | | | |
| **Outcome** | ---------- CA ---------- | | | |

*Note*. ICC=Intraclass correlation; PRS=Progress Rating Schedule; CA=Complete Agreement; 1=NH; 2=BV; 3=MB; Raters 1 and 3 scored the PRS on *n*=50 treatment reports whereas rater 2 did so for *n*=29 reports; All results were significant at *P*<0.05.

#### 4.3.2.3 Clinician validation

On average, the PRS took approximately five minutes to complete. Feedback highlighted that the PRS made a relevant contribution. One medical professional made a specific suggestion of shifting the threshold for rating Behaviour as either 'poor' or 'reasonable' somewhat higher.

### 4.3.3 Psychometric properties

#### 4.3.3.1 Internal consistency

PCA and Cronbach's *alpha* coefficients suggested a reliable scale. Screening of suitability for PCA, revealed that the available sample *n* was below the required minimum (=60) for Time 3 (Pallant, 2005). In addition, Behaviour showed questionable linearity at Time 1 and overall positive skewness. Although these factors may have degraded analyses, results were comparable between time-points and the scale appeared to reflect a unitary factor explaining 51.2%-63.6% of variance.

Cronbach's *alpha* coefficients suggested a reliable scale (0.77-0.87). Although corrected item-total correlations were above 0.3 for all items at all time points, a value as low as 0.35 was computed for Mental State during Time 1. The alpha coefficients increased by removing Mental State for Times 1 and 2 and Behaviour for Time 3 but these changes were small or negligible (0.003-0.02). Therefore, all items appear to fit consistently within the scale.

#### 4.3.3.2 Criterion validity

##### *4.3.3.2.1 Concurrent validity*

PRS Part A and psychometric scores appeared normally distributed but Part B scores – many of which depending on the stage of treatment – were not and varied between time points. No univariate outliers were detected for DSQ scores but one was removed for SPSI-total (1st time-point). There was also one outlier Part B score which was retained as distribution was positively skewed and the score did not appear clinically unrealistic (it was smaller than the mean of Part B scores at the final time point). There were no multivariate outliers.

Part A and B covariance was significant across patients (Level 2), $\sigma_{A/B}^2$=0.84, $Chi^2$=7.71, $df$=1, $P$<0.01, which reflects a small to medium correlation, estimated $\rho$=0.33. A single multivariate model examining Parts A and B simultaneously was adopted. In the intercepts-only model, which contains no predictors and thus functions as baseline, Level 2 variance was significant for Part A only. ICCs for Part A suggested that 53% and 54% of the variance in DSQ and SPSI-R scores respectively were attributable to individual differences between patients (Table 4.3) thereby justifying use of MLM.

Table 4.3. *Sample size of DSQ and SPSI-R datasets and Level 2 (between-patient) residual variance of baseline model*

| Scale | Patient *n* | Scores | Part A | | Part B |
|---|---|---|---|---|---|
| | | | $\sigma_j^2$(*SE*) | ICC | $\sigma_j^2$(*SE*) |
| **DSQ** | 96 | 174 | 6.24(16.94)*** | 0.53 | 0.08(0.04) |
| **SPSI-R** | 95 | 173 | 6.31(18.52)*** | 0.54 | 0.10(0.96) |

*Note.* DSQ=Defence Style Questionnaire; SPSI-R=Social Problem Solving Inventory-Revised; ICC=Intraclass correlation; $\sigma_j^2$=Level-2 (between-patient) variance.
***$P$<0.001; **$P$<0.01; *$P$<0.05.

As Table 4.4 shows, the model improved significantly compared to the baseline (as indicated by a significant and positive $\Delta Chi^2$) when the DSQ subscales were added except for self-sacrificing. The model also improved significantly when SPSI-R subscales and total score were added except for positive orientation. Associations of Part A with most DSQ subscales were large (β) while associations with SPSI-R subscales were small.

Table 4.4. *Summary of results of multivariate MLM* for *PRS scores with the DSQ and SPSI-R as fixed predictors*

| Scale | $\Delta Chi^2$ | Part A | | | Part B | |
|---|---|---|---|---|---|---|
| | | **β(*SE*)** | $R_j^2$ | $R_i^2$ | **β(*SE*)** | $R_i^2$ |
| **DSQ** | | | | | | |
| Maladaptive | 12.2** | -0.73(0.20)*** | 14% | 4% | -0.11(0.07) | 4% |
| Image-distorting | 17.6*** | -0.73(0.18)*** | 16% | 5% | -0.16(0.06)** | 5% |
| Self-sacrificing | 0.64 | 0.15(0.2) | 1% | nil | 0.03(0.07) | nil |
| Adaptive | 8.84* | 0.53(0.18)* | 5% | 6% | 0.07(0.06) | 2% |
| **SPSI-R** | | | | | | |
| Positive orientation | 5.35 | 0.09(0.05) | 5% | 1% | 0.03(0.02) | 3% |
| Negative orientation | 11.0** | -0.08(0.03)*** | 4% | 8% | -0.01(0.01) | 3% |
| Rational problem solving | 7.24* | 0.03(0.01)* | 7% | 1% | 0.01(0.004)* | 4% |
| Impulsivity & carelessness | 8.55* | -0.06(0.02)** | 8% | 4% | -0.01(0.01) | 2% |
| Avoidance | 6.34* | -0.09(0.02)* | nil | 6% | -0.02(0.01) | 3% |
| Total | 10.3** | 0.18(0.06)** | 6% | 5% | 0.04(0.02)* | 4% |

*Note.* MLM=Multilevel Modelling; PRS=Progress Rating Schedule; DSQ=Defence Style Questionnaire; SPSI-R=Social Problem Solving Inventory-Revised; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi^2$=-2log Likelihood change compared to intercepts only model, $\Delta df$=2; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the intercepts-only model. As Level 2 variance for Part B was not significantly different from 0, estimating $R_j^2$ was meaningless.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

Part B showed fewer and smaller significant relationships with the two psychometric instruments. The only DSQ subscale significantly associated with PRS Part B was Image-distorting. Furthermore, although Part B scores were significantly associated with the total SPSI-R score, it showed a significant relationship with one SPSI-R subscale only: rational problem solving. Declaring the DSQ and SPSI-R subscales random predictors did not improve the model further, supporting the validity of these results in light of between-patient differences.

These findings support the concurrent validity of Part A as an indicator of overall adjustment in treatment for personality disorders. Although Parts A and B

covaried significantly, the latter (including items such as leave, employment, etc.) showed smaller and weaker correlations with relevant psychometrics. This suggests that the two parts may represent different aspects of progress.

#### *4.3.3.2.2 Predictive validity*

The distribution of time since admission (days) was positively skewed. Logarithmic transformation resulted in a near-normal distribution but did not alter the results of the analysis hence untransformed data were preferred for ease of interpretation.

The covariance of Parts A/B was again significant thus multivariate MLM was employed. Graphical exploration of the data (Figure 4.1) revealed variability between individual patient trajectories, especially for Part A. Part B scores showed an overall increase over time, as patients begin to achieve leave and employment.

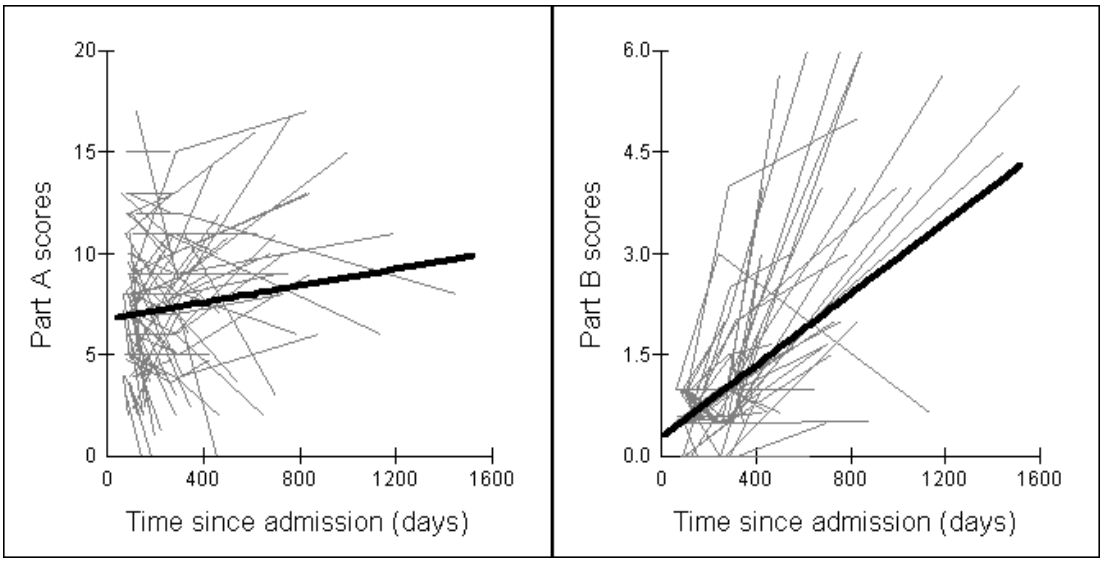



*Figure 4.1.* Patient trajectories of scores on Parts A and B of the PRS as a function of time since admission with highlighted grand mean (estimated in separate multilevel models for Parts A & B)

In the intercepts-only model, Level 2 variance was significant for Part A, $\sigma_j^2$=6.35, $Chi^2$=18.25, $df$=1, $P$<0.001, but not for Part B, $\sigma_j^2$=0.07, $Chi^2$=0.35, $df$ =1.

ICCs suggested that 48% of variance in Part A scores was due to differences between patients, justifying use of MLM.

Time since admission was initially declared a fixed predictor and was significant for both PRS Parts. Convergence was achieved when time since admission was declared a random predictor for Part B only but not Part A or Parts A and B simultaneously and the model improved further. Results are summarised in Table 4.5.

Table 4.5. *Summary of results of multivariate MLM for PRS scores with time since admission as predictor*

| | | | | Part A | | Part B |
|---|---|---|---|---|---|---|
| **Model** | **$\Delta Chi^2(\Delta df)$** | **$R_j^2$** | **$R_i^2$** | **Time β*(SE)* [e-3]** | **$R_i^2$** | **Time β*(SE)* [e-3]** |
| **0** | 139.9(2)*** | 10% | nil | 2.05(0.82)* | 51% | 3.26(0.23)*** |
| **1-Best** | 113.3(3)*** | 1% | <0 | 1.99(0.82)* | 54% | 2.38(0.41)*** |

*Note.* MLM=Multilevel Modelling; PRS=Progress Raring Schedule; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous model, with the intercepts-only model being first; Highlighted terms were random at Level 2; The fixed parameter estimates were small as they represented rate of change per day.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

To establish further whether variability in PRS scores was clinically meaningful, the effect of psychopathy on patient PRS trajectories was examined. Data on psychopathy were missing for three patients leading to new model estimations but results were comparable to the previous analysis.

As shown in Table 4.6, the addition of psychopathy and its interaction with time as fixed predictors resulted in a significantly improved and final model. A graphical representation of this is shown in Figure 4.2. Individuals with psychopathy began on similar levels of adjustment on the ward as those without psychopathy (Part A) but had somewhat higher Part B scores. However, Part A scores of patients with psychopathy decreased over time while Part B scores for this group increased at a slower rate compared to patients without psychopathy. These observations are in line with previous findings on treatment completion at the PDS (McCarthy & Duggan,

2010) and provide additional evidence to suggest that change in PRS scores is likely to be clinically meaningful.

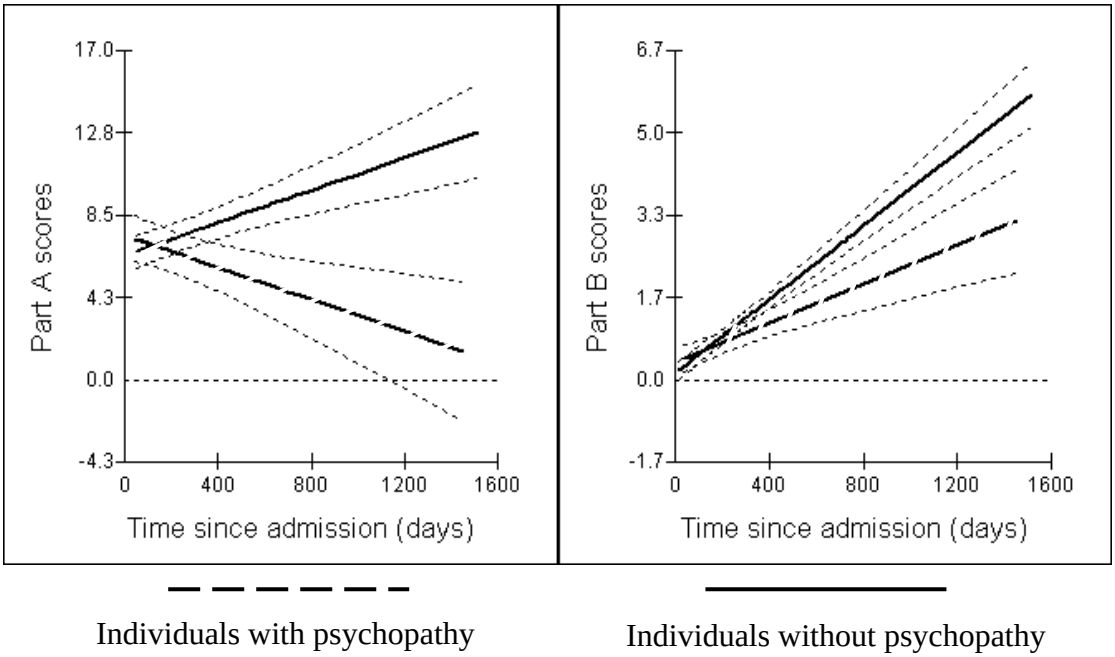



*Figure 4.2.* Average progress over time as measured by the Parts A and B of the Progress Rating Schedule (PRS) for patients with and without psychopathy with standard errors (95% confidence interval). The rate of change was significantly different between the two groups for both Parts A and B.

Table 4.6. *Summary of results of multivariate MLM for PRS scores with time since admission, psychopathy and their interaction as predictors*

| | | | | Part A | | | | Part B | | |
|---|---|---|---|---|---|---|---|---|---|---|
| **Model** | **$\Delta Chi^2(\Delta df)$** | **$R_j^2$** | **$R_i^2$** | **β*(SE)*** | | | **$R_i^2$** | **β*(SE)*** | | |
| | | | | **Time [e-3]** | **Psy** | **Psy x Time [e-3]** | | **Time [e-3]** | **Psy** | **Psy x Time [e-3]** |
| **0** | 138.5(2)*** | 9% | nil | 1.94(0.83)* | | | 52% | 3.29(0.23)*** | | |
| **1** | 107.5(3)*** | 2% | <0 | 1.92(0.83)* | | | 54% | 2.49(0.42)*** | | |
| **2** | 0.55(2) | 2% | nil | 1.91(0.84)* | -0.45(0.65) | | nil | 2.52(0.42)*** | -0.03(0.09) | |
| **3-Best** | 25.75(4)*** | <0 | 20% | 4.08(0.92)*** | 1.01(0.77) | -8.15(1.67)*** | 1% | 3.14(0.46)*** | 0.32(0.15)* | -2.49(0.94)** |
| **4** | nil(4) | nil | nil | 4.08(0.92)*** | 1.01(0.77) | -8.15(1.67)*** | nil | 3.14(0.46)*** | 0.32(0.15)* | -2.49(0.94)** |

*Note.* MLM=Multilevel Modelling; PRS=Progress Rating Schedule; Psy=Psychopathy; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous model, with the intercepts-only model being first; Highlighted terms were random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

### 4.3.4 Summary

The PRS was developed to measure progress in treatment for personality disorders in secure healthcare. Thematic analysis and refinement based on routine clinical records and multi-professional input resulted in an instrument with two main parts, A and B, followed by a supplementary, customisable section. Part A comprised six items intrinsic to treatment. Part B comprised five additional but miscellaneous progress items such as leave, employment, and risk. The scope and scoring of all items was operationalised.

Results provided some initial support for the instrument's inter-rater reliability while the scale forming Part A showed acceptable to good internal consistency. The PRS generally showed good validity as reflected in correlations with the DSQ and SPSI-R, measuring concepts relevant to change in personality disorder. Although both Parts A and B appeared sensitive to change in a clinically meaningful manner and co-varied significantly, each seemed to relate to different aspects of progress when related to psychometric instruments. Part A appeared to reflect 'process' change as evidenced by correlations with various defence styles and areas of social problem solving including both attitudes (e.g. negative problem orientation) and skills (rational problem solving). On the other hand, Part B scores were related to realistic appraisals (image-distorting defence style) and social problem solving skills more specifically but to a lesser extent compared with Part A.

# 5 COGNITIVE ABILITIES IN THE ANTISOCIAL PERSONALITY

This chapter relates to the method and results of the examination of cognitive difficulties in patients and healthy controls.

## 5.1 Method

### 5.1.1 Participants

As described earlier patients were recruited from the PDS at Arnold Lodge. Additionally, a sample of healthy controls comprising of Twenty male staff members from Arnold Lodge Regional Secure Unit was recruited via general email invitations and notices in ward communication books. Inclusion criteria for the latter group were: age at least 18 years and absence of a history of personality disorder, major mental illness, neurological conditions, traumatic brain injury or learning disability. Current substance use or psychotropic medication and a history of substance abuse were emphasised as necessary exclusions but ethical approval did not permit direct questioning regarding these. All individuals in this group were free from current or past Axis I or II diagnoses as assessed with the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998), the IPDE Screening Questionnaire and interview (Loranger, 1999), the latter was conducted only when the individual scored positively on the questionnaire. In addition, participants did not meet criteria for psychopathy according to the PCL:SV, and had an estimated IQ of at least 70 (Quick Test; Ammons & Ammons, 1962).

### 5.1.2 Materials

As mentioned in Chapter 4, patients were assessed usig a number of validated scales including SADS-L, SCID-I:CV, IPDE, PCL-R, WAIS-III, etc.

#### 5.1.2.2 Healthy controls

A battery of questionnaires and assessments was administered by MB with the purpose of screening participants of the healthy control group and collect socio-demographic data.

#### *5.1.2.2.1 Screening questionnaire and socio-demographic data (Appendix E)*

These questionnaires assessed inclusion criteria and collected data on age, years in education and handedness at an interview with participants.

#### *5.1.2.2.2 Axis I and II psychopathology*

Two instruments were used: the MINI (Lecrubier et al., 1997; Sheehan et al., 1998) and IPDE Screening Questionnaire (Loranger, 1999). The former is a short, structured interview schedule for the screening of DSM-IV Axis I mental disorders lasting approximately 15 minutes. The section on substance and alcohol abuse was excluded, as per ethical approval for this project. The instrument has been shown to have high inter-rater reliability (Kappa: 0.88-1.00) and acceptable to high test-retest reliability (Kappa: 0.76-0.93) and has been widely used to screen for mental disorders.

The IPDE Screening Questionnaire is a self-report tool used to screen for the presence of personality disorders and contains 77 items in a TRUE or FALSE response format. For those participants with a positive response to a minimum of three criteria for each personality disorder, the IPDE interview was employed to determine whether an individual truly had a personality disorder (this occurred for six control participants). MB had received suitable training for use of this instrument. Although the reliability and validity of the screening questionnaire remains under-researched (R. Rogers, 2001), it was selected in order to maintain diagnostic consistency between patients and healthy control participants in the assessment of personality disorders.

#### *5.1.2.2.3 PCL:SV (Hart et al., 1995)*

The PCL:SV is an abbreviated version of the PCL-R used to screen for psychopathy. It contains 12 items, each rated on a scale of 0-2. Scores are usually obtained via a semi-structured interview and file review. Only the former was administered here by MB, who had received suitable training. This was considered acceptable in a healthy control population. The PCL:SV has good internal consistency (*alpha*: 0.84), moderate/fair inter-rater reliability (Kappa: 0.48-0.51), and good test-retest reliability (*r*: 0.90-0.91). The checklist has also shown good

concurrent, convergent and discriminant validity in its ability to describe features of the antisocial personality (Hart, Cox, & Hare, 1995). The cut-off for psychopathy on the PCL-SV is 18.

#### *5.1.2.2.4 Quick Test (Ammons & Ammons, 1962)*

The Quick Test was used to assess IQ. It involves assignment of words to appropriate pictures. It is a rapid measure of intelligence that relies extensively on vocabulary. Although it may underestimate the mental ability of individuals, it has demonstrated high correlations with WAIS VIQ scores and good predictive validity for long-term outcomes in people with traumatic brain injury (Lezak et al., 2004).

### 5.1.2.3 Cognitive assessment: The CANTAB

The CANTAB, a standardised battery of neuropsychological tests,was employed to assess deficits in the antisocial personality in light of the range of ambiguities identified in the literature review,. The battery's computerisation and standardisation have the benefit of better accuracy of measurement compared to other batteries while the sensitivity and selective focus of its tests are advantages in focusing on and isolating specific cognitive operations (Strauss et al., 2006). Description of CANTAB tests and outcomes measures used in this study are provided in Table 5.1 alongside information on relevant neural substrates.

The battery's origins are in animal models and lesion studies and has been extensively validated and researched in over 600 peer-reviewed publications (Cambridge Cognition, 2006; Fray et al., 1996; Sahakian & Owen, 1992; Strauss et al., 2006). Administration takes place via a computer terminal and, although it can be applied to most age groups, there may be age and IQ effects (Strauss et al., 2006). Luciana (2003) reported adequate to high internal consistency for the battery (0.73 to 0.95, with children). In addition, many of its tests have been associated with adequate or high levels of test-retest reliability (e.g. Pattern Recognition Memory [PRM], Paired Associates Learning [PAL]-trials to success, IED-Extra-dimensional set shifting [EDS]), but others have shown marginal (e.g. PAL-first memory score, SOC, Spatial WM [SWM], Spatial Span [SSP]) and even low levels (Spatial Recognition Memory [SRM], DMS, SOC, IED-intra-dimensional set shifting) (Cambridge

Cognition, 2008; Strauss et al., 2006). Such discrepancies and lower levels of reliability have been explained by practice effects and loss of naïveté in repeat testing (Cambridge Cognition, 2008; Lowe & Rabbitt, 1998; Strauss et al., 2006). Nevertheless, tests associated with lower reliability are likely to require greater statistical power in order to detect small effects.

In terms of validity, the CANTAB has been extensively researched. Rabbit and Lowe (2000) presented factor analyses suggesting that relevant CANTAB tests describe memory, executive functions and dorsolateral operations (WM and problem solving). CANTAB tests appear to associate differentially with various cerebral regions in lesion, neurosurgical, and neurological patient studies and large bodies of research employed tests from the battery to outline neuropsychological impairments associated with various disorders including schizophrenia, dementia, ADHD and autism, among others (Strauss et al., 2006). Although the battery has been used extensively in a number of clinical conditions, this has not been the case for the antisocial personality. However, the study by Dolan and Park (2002) on patients with ASPD highlights its potential usefulness in detecting impairments in this population.

The CANTAB is likely to prove very useful in further clarifying the nature and extent of cognitive impairment in individuals with antisocial personality. Its detailed coverage of fronto-temporal operations provides a major benefit in investigating the complex executive functions as well as memory. Although the battery does not assess verbal operations, it shows a good coverage of the remaining functions of interest in this project. However, weaker reliability in some tests may lead to measurement error and loss of statistical power particularly in small samples.

Table 5.1. *List of CANTAB tests employed in the project*

| Test | Description | Outcome measures | Primary substrates |
|---|---|---|---|
| *Sustained attention* | | | |
| RVP | A continuous performance paradigm for *sustained attention* with a small WM component. It is also a measure of general performance. During presentation of a series of single-digit numerical stimuli, the person has to identify and respond to three different three-digit sequences (targets). | Total target hits | Parietal & frontal[a,b,c] |
| *Motor regulation* | | | |
| AGN | A traditional Go/NoGo paradigm targeting *response inhibition* with a reinforcement shift every two blocks. Participants are instructed to respond to targets only. Stimuli are either positive or negative words. Participants are required to respond to either positive or negative words in each block and these reversed every two blocks of stimuli.<br>This task involves affective cognitive functions thought to be associated with VMPFC function (Cambridge Cognition, 2006) which is considere a key area in inhibitory control (D. L. Clark et al., 2010). Although it is primarily used to assess affective bias, such emotional Go/NoGo tasks have been found to converge with non-emotional Go/NoGo tasks in terms of impaired inhibitory control (commission errors; Schulz et al., 2007), and this has also been suggested in psychopathy (Iria & Barbossa, 2009). At the same time, as VMPFC is considered important in the antisocial personality (R. Blair et al., 2005), affective stimuli in the GoNoGo may increase the sensitivity of this task in detecting difficulties in this population notwithstanding some limits to | 1. Commission errors<br>2. Response latency | VMPFC & lateral OFC[a,b,d] |

| | | | |
|---|---|---|---|
| | internal validity due to confounding with affective processing. | | |
| *Planning* | | | |
| SOC | Assesses *spatial planning* with contributions from *WM* in a ToL analogue paradigm. Participants move balls on the screen to achieve a target arrangement. There are 4 levels of increasing difficulty where problems require 2, 3, 4 or 5 moves to solution. | 1. Perfect solutions (problems solved in minimum number of moves)<br>2. Average number of moves to solution<br>3. Initial thinking time prior to commencing solution<br>4. Subsequent thinking time until solution | Prefrontal & DLPFC[a,b,c,d] |
| *Cognitive flexibility* | | | |
| IED | In this analogue of the WCST, participants are instructed to respond one of two sets of stimuli. They are required to work out which is the relevant dimension based on feedback. During the task, participants have to shift focus either within the same dimension of the set of stimuli (e.g. purple shapes) or to a previously irrelevant dimension (line drawings).<br>Among the initial seven stages, four involve *response reversal*. The eighth stage requires a *set shift* (EDS) which was followed by another reversal stage ($9^{th}$). The CANTAB output provides number of errors for each stage. Consistent with the hypotheses of this project, this task was used to examine reversal and EDS ability. However, because not all | 1. Total reversal errors (pre-EDS)<br>2. EDS errors | Fronto-striatal & DLPRF[a,b,c] |

| | | | |
|---|---|---|---|
| | participants completed the EDS, only the three reversal conditions prior to EDS were taken into account in calculating reversal errors. A similar approach had been adopted previously by Mitchell et al. (2002). | | |
| CGT | CGT assesses *risk-taking and decision-making*. Participants begin with 100 points during each block and were presented with a row containing a red and a blue section of boxes. The procedure involves selecting a colour based on which set of boxes they expect a yellow token to be hidden behind. Participants then place a bet on their choice. They are instructed to make as many points as possible and could win as well as lose points based on the result of the bet. The points balance is always shown on the screen when participants decide how much to bet at each trial.<br>The stake increment for each trial is either ascending (increasing bet) or descending (decreasing bet) and the order of these conditions was counterbalanced. The ratios of red to blue boxes reflects different conditions of betting odds and are 9:1, 8:2, 7:3, 6:4 & 5:5. | 1. Quality of decision-making (choosing the most likely outcome)<br>2. Risk-taking<br>3. Delay aversion (ascending vs. descending conditions) | OFC, VMPFC & Insular cortex[a,e,f] |
| *Memory* | | | |
| PAL | This is another form of delayed response procedure, which assesses new learning and *visuospatial STM* in a cued recall paradigm. After practice, the participant is required to remember the positions of patterns presented sequentially in stages containing of 3, 6 and 8 patterns. | 1. Completed stages<br>2. Total errors<br>3. Total errors adjusted for non-completed stages | Medial temporal[a,b,e] |
| DMS | This is a test of *visual STM*, a 4-choice pattern recognition memory | 1. Correct responses | Medial temporal |

| | | | |
|---|---|---|---|
| | paradigm. There are 4 different presentation conditions: simultaneous, immediate recognition and delayed recognition after 4 or 12 seconds. | (recognition)<br>2. Response latency | with some input from frontal areas[a,b,e] |
| SSP | SSP is a visuospatial analogue of the Digit Span test assessing *STM*. Participants are asked to reproduce increasingly longer sequences of flashing boxes on the screen. | Span length | Frontal & posterior temporal lobes[b,c,h] |
| VRM | Assesses *verbal memory* using recall (STM) and recognition conditions (STM & LTM). The patient is presented with 12 words. Long-term recognition is assessed after a 20-minute interval during which the DMS task is administered. | Correct responses (recall & recognition) | Temporal (left, anterior) & frontal lobes, left prefrontal cortex & hippocampus[c,g] † |
| SWM | A self-ordering task assessing *WM*. Stimuli are presented in 4 blocks of increasing number of stimuli (4, 6 and 8) following practice. | Total errors | DLPFC[a,b,d] |
| *Visual perception* | | | |
| MTS | A task involving visual search and (in)attention where participants are required to select a pattern among 2, 4, or 8 alternatives which matches the original sample both in colour and shape. | 1. Total errors<br>2. Correct and error reaction times | Frontal (visual search)[b] |

*Note.* CANTAB=Cambridge Neuropsychological Test Assessment Battery; AGN=Affective Go/NoGo; IED=Intra/Extra-Dimensional set shifting; EDS=Extra-dimensional set shifting; CGT=Cambridge Gambling Task; WCST=Wisconsin Card Sorting Test; SOC=Stockings of Cambridge; ToL=Tower of London; RVP=Rapid Visual Processing; PAL=Paired Associates Learning; DMS=Delayed Matching to Sample; SSP=Spatial Span; STM/LTM=Short/long-term memory; VRM=Verbal Recognition Memory; SWM=Spatial Working Memory; WM=Working memory; MTS=Matching to Sample Visual Search; VMPFC=Ventromedial prefrontal cortex; DLPFC=Dorsolateral prefrontal cortex; OFC=Orbitofrontal cortex.

[a]www.cambridgecognition.com; [b]Kolb & Whishaw (2009); [c]Lezak et al. (2004); [d]D. L. Clark et al. (2010); [e]L. Clark et al. (2008); [f]R. D. Rogers et al. (1999); [g]Strauss et al. (2006); [h]Gazzaniga et al. (2009).

†Evidence from verbal memory tests analogous to the VRM including the Auditory and California Verbal Learning Tests.

### 5.1.3 Apparatus

The CANTAB was administered on an IBM compatible computer terminal with Intel Pentium 4 processor (1.7GHz), 256 MB RAM, Windows XP operating system, fitted with a touch screen monitor and a press pad as appropriate (Cambridge Cognition, 2006).

### 5.1.4 Design

The research was quasi-experimental with group as between-subjects quasi-independent variable and CANTAB outcome measures as dependent variables. Some neuropsychological tests involved within-subjects factors resulting in a mixed design. The effects of a range of possible mediating variables (participant variables & sustained attention) were evaluated and controlled for where appropriate. Although the groups were matched on several participant variables, they may not be considered equivalent as random assignment from the same population was not possible. A consequence of this is that it was not possible to establish cause-and-effect relationships between the variables (McBurney & White, 2007).

### 5.1.5 Procedure

All assessments, interviews and neuropsychological tests were administered in a standardised manner and according to user manuals. The author was responsible for collecting all CANTAB data since April 2008 obtaining data on 49% of cases and all healthy controls. The remainder of 51% of cases had been collected by other PDS staff prior to April 2008.

#### 5.1.3.1 Patient group

CANTAB assessment took place in the first few weeks of admission in a quiet room on the hospital wards. Following two introductory tests for the battery, the main tests in Table 5.1 were administered over 5 sessions. Data collection was undertaken over a period of approximately 12 years beginning in 1999.

#### 5.1.3.2 Healthy controls

Participants were briefed on the purposes and procedures of the study and were given the opportunity to ask questions before they provided their informed consent. Data collection took place over four months in the beginning of 2011 and took place in two sessions beginning with screening and diagnostic interviews and ending with the CANTAB (including introductory tests). The IPDE interview was administered only where the screening had failed. No controls met criteria for any diagnosis. Participants in this group received a modest fee for their participation upon completion.

### 5.1.6 Statistical data analysis

#### 5.1.6.1 Additional data screening and assumptions

In addition to the assumptions mentioned in the previous chapter on the PRS, homogeneity of regression slopes was also tested as a critical assumption in ANCOVA. It assumes that a covariate affects each group similarly (Tabachnick & Fidell, 2007). A significant group x covariate interaction suggests violation of this assumption.

#### 5.1.6.2 Cognitive deficits

The first set of hypotheses involved identifying neuropsychological deficits in antisocial individuals compared to non-antisocial groups. Antisocial personality was operationalised using DSM (antisocial personality disorder) or PCL-R criteria (psychopathy) leading to two parallel sets of comparisons. Each set of comparisons involved three groups: ASPD versus non-ASPD versus healthy controls and psychopathy versus non-psychopathy versus healthy controls. A supplementary set of analyses (ASPD-only) were also conducted after removing those individuals with psychopathy from the group with ASPD, as different neurocognitive impairments were expected for these operationalisations. However, the converse was not possible for psychopathy due to small sample sizes. ANOVA statistical methods were employed (Pallant, 2005; Tabachnick & Fidell, 2007). Bonferroni post hoc comparisons were preferred in order to limit Type I error in light of a large number of analyses (Field, 2009). Although task condition effects were examined and reported for validity of the manipulations, they were not described in detail for economy purposes, as these were not relevant to the hypotheses. Where normality was violated, the Kruskal-Wallis non-parametric ANOVA equivalent test was also employed to confirm findings. Standard error of the mean was represented as error bars in figures.

##### 5.1.6.2.1 Confounders

These included participant variables (e.g. demographics), clinical variables (e.g. antidepressant and antipsychotic medication), history of SRD, and sustained attention as measured by the Rapid Visual Processing (RVP) test of the CANTAB.

Possible confounding variables were entered as covariates when groups performance means were significantly different and where the covariate also correlated significantly with cognitive performance. Parametric (Pearson's) and non-parametric (Spearman's) correlations were employed as appropriate (Pallant, 2005). Covariates were assessed using ANCOVA and MLM, the latter conducted when the critical assumption of homogeneity of regression slopes was violated (Tabachnick & Fidell, 2007). For MLM, participants were set as Level 1 variable and group as Level 2 variable with the covariate as Level 1 predictor. This allowed the intercept and slope of the covariate as predictor to vary (random effects) between groups.

#### 5.1.6.3 Software and statistical significance

SPSS software, v.17.0 (SPSS Inc, 2009) was employed for all analyses with an *alpha* level set at 0.05 for all statistical tests unless otherwise specified.

## 5.2 Results

### 5.2.1 Sample characteristics

Of the 102 patients who were assessed on the CANTAB, 17 were excluded. Reasons were IQ<70, history of major mental illness (psychosis & bipolar disorder) unrecognised at the time of admission, or serious traumatic brain injury (periods of unconsciousness exceeding 2 hours following head trauma). In the final patient sample, ASPD and psychopathy showed only a degree of overlap (Figure 5.1). Of patients with ASPD, 50% also met criteria for psychopathy whereas of those without ASPD, 10% met criteria for psychopathy. Conversely, of patients with psychopathy, 88% met criteria for ASPD whereas of those without psychopathy, 50% met criteria for ASPD.

Final group characteristics are presented in Table 5.2. PCL-R data were missing for three patients who could not be allocated to a psychopathy group as a result. Although the ASPD and non-ASPD groups were comparable in IQ, education and the number of personality disorders other than ASPD, differences existed in age, PCL-R, medication and history of substance-related disorders. Compared to the Non-ASPD group, patients with ASPD were younger, had higher PCL-R scores, and were more frequently diagnosed with SRD. Furthermore, they were prescribed antidepressants less frequently but received antipsychotics more often than patients without ASPD. On the other hand, the groups with and without psychopathy were comparable in all variables except, by definition, PCL-R scores and history of substance-related disorders. The healthy control group was matched to the patient groups on age, IQ and years in basic education but had completed more years in further education (college, vocational training, etc.). Because the Affective Go/NoGo (AGN), Verbal Recognition Memory (VRM) and Cambridge Gambling Task (CGT) tests from the CANTAB were introduced later in the PDS, samples were smaller for those tests (AGN & VRM: 38 ASPD vs. 16 non-ASPD & 22 individuals with psychopathy vs. 29 without; CGT: 11 ASPD vs. 6 non-ASPD & 8 individuals with psychopathy vs. 8 without).

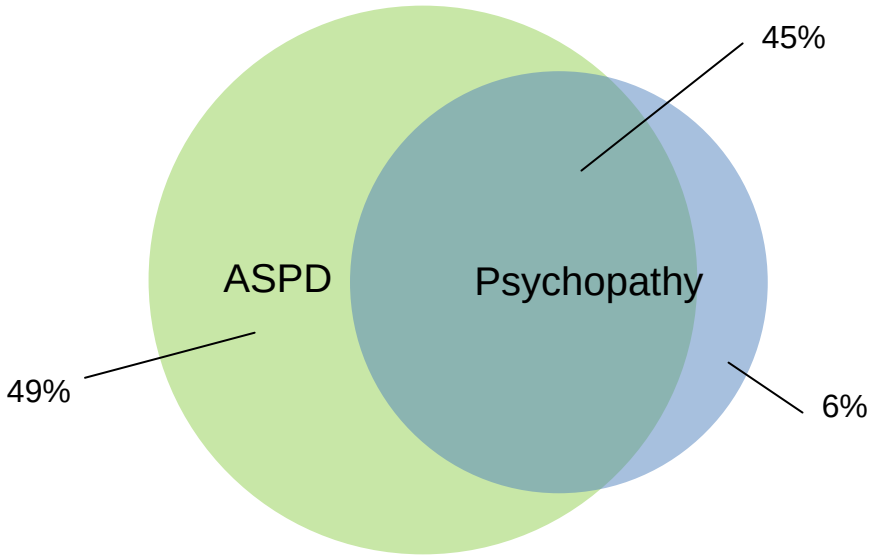



*Figure 5.1.* Venn diagram showing the degree of overlap between Antisocial Personality Disorder (ASPD) and psychopathy in the project's sample. Approximately half of the patients with ASPD also met criteria with psychopathy. Conversely, 88% of those with psychopathy, also met criteria for ASPD.

Excluding individuals with psychopathy from the ASPD group resulted in a group of 26 patients with ASPD only, which was used for supplementary comparisons against non-ASPD PDS patients and healthy control groups. In terms of participant characteristics, results on age, IQ, advanced education, PCL-R scores, number of additional personality disorders, and anti-depressant medication were comparable to the previous analysis with the larger ASPD group. However, healthy controls had received significantly more years of basic education than the smaller, ASPD-only group, $F(2,57)=3.9, P<0.05$, post hoc, $P<0.05$, but there were no longer significant differences in terms of prior SRD, $Chi^2=0.63$, anti-psychotic medication, $Chi^2=1.21$, and mood stabilisers, $Chi^2\approx 0$.

Table 5.2. *Sample characteristics for ASPD, psychopathy and healthy controls*

| Study groups | ASPD *M(SD)* | Non-ASPD *M(SD)* | Healthy control *M(SD)* | Psy *M(SD)* | Non-psy *M(SD)* | Differences ASPD | Psychopathy |
|---|---|---|---|---|---|---|---|
| *N* | 52 | 33 | 20 | 27 | 55 | | |
| Age (years) | 30.3(8.9) | 37.8(9.2) | 33.9(10.7) | 34.3(10.9) | 31.5(8.8) | **Non-ASPD>ASPD**, $F(2,72)=3.8$, $P<0.05$ | ns |
| IQ | 87.8(10.1) | 93.7(18.1) | 87.6(13.0) | 86.5(13.9) | 91.0(13.1) | ns | ns |
| Education (years) | | | | | | | |
| Basic | 9.8(2.0) | 10.1(2.4) | 11 | 9.7(2.5) | 9.9(2.0) | ns | ns |
| Advanced | 0.2(0.5) | 0.2(0.7) | 3.4(2.2) | 0.1(0.3) | 0.1(0.6) | **Control>ASPD & non-ASPD,** $F(2,102)=55.9$, $P<0.01$ | **Control>Psy & non-psy,** $F(2,99)=55.9$, $P<0.01$ |
| PCL-R | 24.0(4.9) | 15.9(6.4) | - | 28.0(2.7) | 17.7(5.2) | **ASPD>Non-ASPD** $F(1,80)=37.2$, $P<0.001$ | **Psy>Non-psy,** $F(1,80)=88.8$, $P<001$ |
| *N* of additional PDs | 1.7(1.2) | 1.7(1.1) | - | 2.6(1.4) | 2.2(1.2) | ns | ns |
| SRD | 55.1% | 27.1% | - | 30.8% | 51% | **ASPD>Non-ASPD** $Chi^2=4.2$, $P<0.05$ | **Non-psy> Psy,** $Chi^2=5.2$, $P<0.05$ |
| Medication | | | | | | | |
| Antidepressant | 14.8% | 19.3% | - | 9.4% | 25.9% | **Non-ASPD>ASPD,** $Chi^2=6.2$, $P<0.05$ | $Chi^2=0.8$, ns |
| Antipsychotic | 21.6% | 5.7% | - | 11.8% | 14.1% | **ASPD>Non-ASPD,** $Chi^2=4.4$, $P<0.05$ | $Chi^2=2.1$, ns |
| Mood stabiliser | 9.1% | 4.5% | - | 5.9% | 7.1% | $Chi^2=0.2$, ns | $Chi^2=0.9$, ns |

*Note.* ASPD=Antisocial Personality Disorder; PD=Personality Disorder; Psy=Psychopathy; PCL-R=Psychopathy Checklist-Revised; SRD=Substance-related disorder diagnosis; ns=not significant.

### 5.2.2 Cognitive deficits in the antisocial personality

#### 5.2.2.1 Sustained attention

Although the literature did not support sustained attention deficits in psychopathy, it suggested possible impairment in ASPD. It was hypothesised that the CANTAB would provide further evidence to support a deficit in ASPD but not psychopathy. The RVP test was used to examine sustained attention. Outcome measure was total number of hits.

##### *5.2.2.1.1 ASPD*

There were minor deviations from normality and K-S test was significant for the non-ASPD group. The Kruskal-Wallis test revealed a significant main effect of group for RVP total hits, $Chi^2$=16.2, *df*=2, *P*<0.001. This was confirmed by ANOVA, *F*(2,90)=9.85, *P*<0.001. Post hoc analysis indicated that both patient groups made fewer RVP hits than the healthy control group, *Ps*<0.01, but were not different compared to each other (Figure 5.2). Excluding individuals with psychopathy from the ASPD group yielded comparable results.

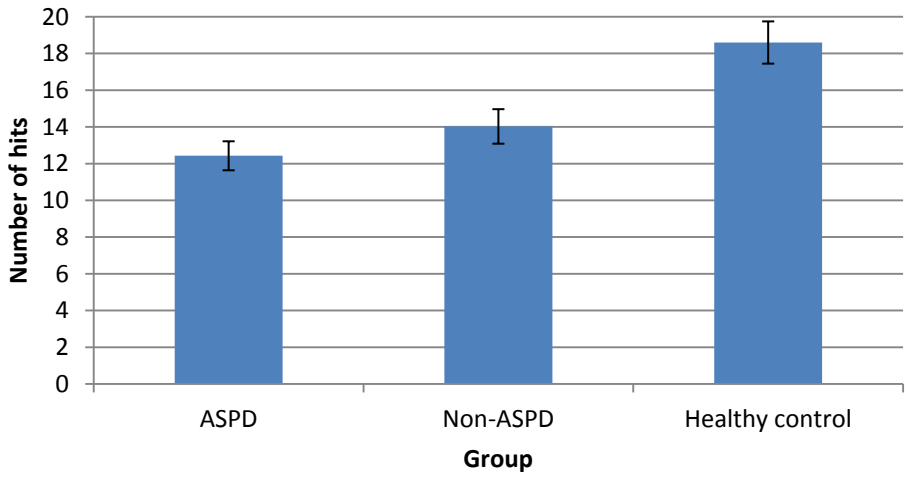



*Figure 5.2.* Mean total hits on the Rapid Visual Processing task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. Both patient groups performed significantly worse than controls but were comparable to each other.

#### 5.2.2.1.2 Psychopathy

There were minor deviations from normality but the K-S test was significant for the group with psychopathy only. Kruskal-Wallis tests revealed a significant main effect of group, $Chi^2$=17.2, *df*=2, *P*<0.001. This was confirmed by ANOVA, *F*(2,87)=10.65, *P*<0.001. Post hoc analysis indicated that both patient groups made fewer RVP hits than the healthy control group, *Ps*<0.01, but were not different compared to each other (Figure 5.3).

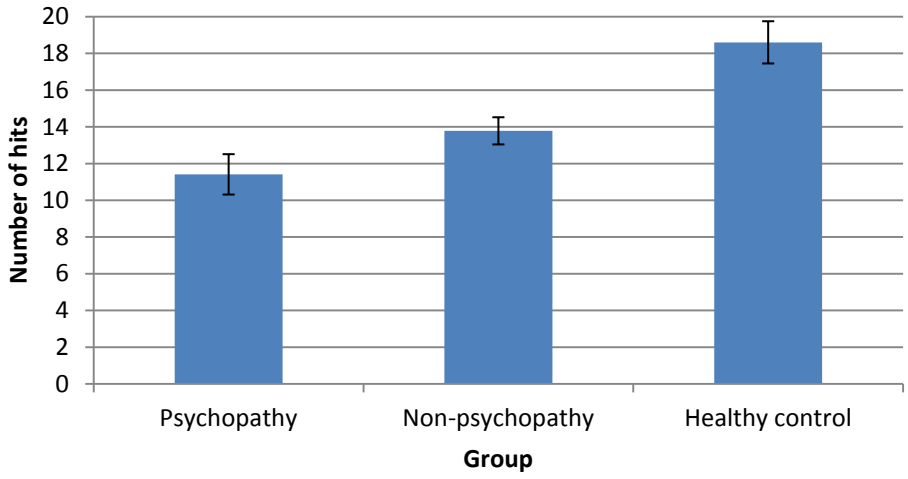



*Figure 5.3.* Mean total hits on the Rapid Visual Processing task for patients with and without psychopathy and healthy controls. Both patient groups performed significantly worse than controls but were comparable to each other.

#### 5.2.2.1.3 Summary of sustained attention

Results provided some support for the hypothesised deficit in sustained attention in the patient group when compared to healthy controls. Contrary to expectations, however, impaired sustained attention was also detected in psychopathy.

### 5.2.2.2 Motor regulation

#### *5.2.2.2.1 ASPD*

Two univariate outliers for AGN commission errors were removed from each of the ASPD and the healthy control groups. There were some deviations from normality in ASPD (skewness, kurtosis, K-S test). A Kruskal-Wallis test revealed a significant main effect of group, $Chi^2$=16.3, *df*=2, *P*<0.001. This was confirmed by ANOVA, *F*(2,67)=6.62, *P*<0.01. Post hoc analysis suggested that the ASPD group made significantly more commission errors than healthy controls, *P*<0.01, but there were no further group differences (Figure 5.4). The groups did not differ in response latency, *F*(2,71)=0.25.

Regarding the effect of attention, RVP hits were significantly correlated with AGN commissions, *r*=-0.32, *P*<0.05, and there was a significant group x covariate interaction, *F*(3,56)=4.20,*P*<0.01. Using MLM to control for RVP hits, did not result in a significantly improved model, with RVP hits entered either as fixed or random predictor, $\Delta Chi^2$=1.5, $\Delta df$=1 and 2 respectively. Further, excluding individuals with psychopathy from the ASPD group yielded comparable results.

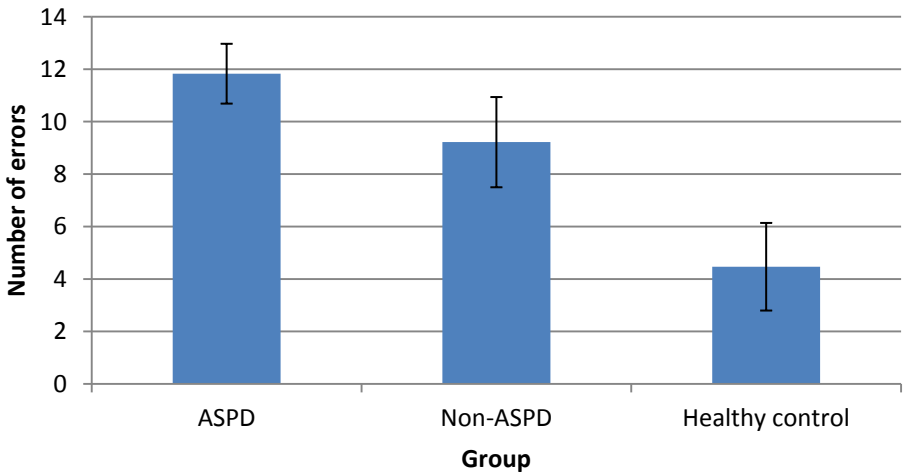



*Figure 5.4.* Commission errors on the Affective Go/NoGo task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. The ASPD group performed significantly worse than healthy controls only. There were no further significant groups differences.

#### 5.2.2.2.2 Psychopathy

One univariate outlier in commission errors was removed from each of the groups with and without psychopathy and two were removed from the healthy control group. There was some skewness in the group with psychopathy. ANOVA revealed a significant main effect for group, $F(2,64)=6.58$, $P<0.01$. Post hoc analysis suggested that both patient groups made significant more commission errors than the healthy control group, $Ps<0.05$, but were not significantly different compared to each other (Figure 5.5). The groups were not different in response latency, $F(2,68)=0.09$.

Regarding attention, RVP hits were correlated significantly with AGN errors and demonstrated a comparable interaction with group as for the ASPD analysis above. Using MLM to control for RVP hits did not result in a significantly improved model, with RVP hits entered either as fixed or random predictor, $\Delta Chi^2$=-9.65, $\Delta df$=1 and 2 respectively.

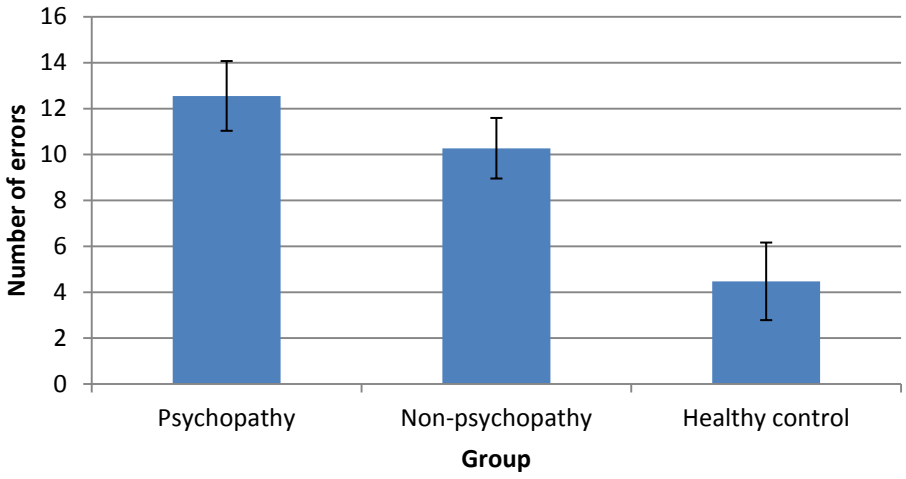



*Figure 5.5.* Commission errors on the Affective Go/NoGo task for patients with and without psychopathy and healthy controls. Both patient groups performed significantly worse than healthy controls but were comparable to each other.

#### 5.2.2.2.3 Summary of motor regulation

Results indicated that individuals with ASPD, psychopathy, and personality disorders other than psychopathy demonstrated deficits in motor regulation against

healthy controls. However, the differences did not reach significance when offenders with personality disorders other than ASPD were compared to controls.

### 5.2.2.3 Planning

#### *5.2.2.3.1 ASPD*

##### *5.2.2.3.1.1 Perfect solutions*

K-S test was significant for all groups. A Kruskal-Wallis test revealed a significant main effect of group, $Chi^2$=13.11, *df*=2, *P*<0.01, also supported by ANOVA, *F*(2,97)=7.49, *P*<0.01. Post hoc analysis suggested that both patient groups performed significantly worse than the healthy control group, *Ps*<0.05, but were not different compared to each other (Figure 5.6).

Regarding the effect of attention, RVP hits were significantly correlated with SOC perfect solutions, *rho*=0.26, *P*<0.05. There was also a significant group x covariate interaction, *F*(3,87)=2.98, *P*<0.05. Nevertheless, adding RVP hits as fixed and random predictor to the MLM did not result in significant improvements, $\Delta Chi^2$=0.44, $\Delta df$=1 and 2 respectively. Furthermore, excluding individuals with psychopathy from the ASPD group yielded comparable results.

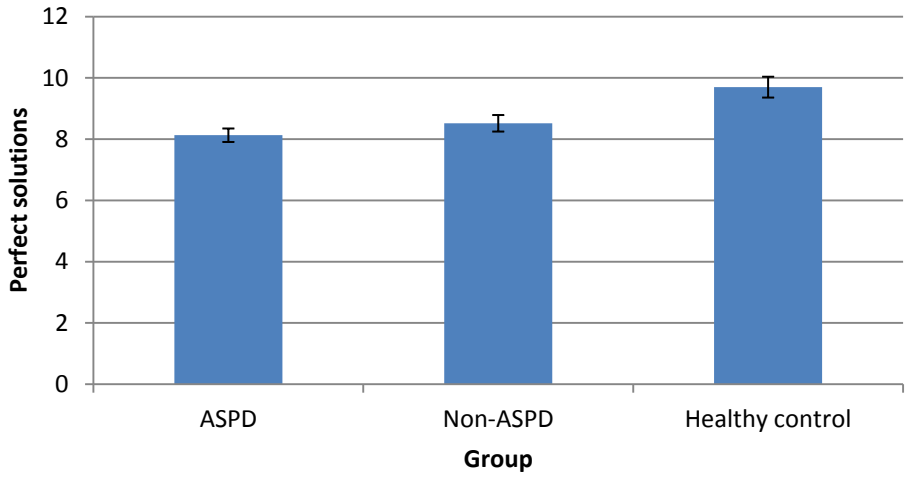



*Figure 5.6.* Perfect solutions on the Stockings of Cambridge task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. Both patient groups performed significantly worse than controls but were comparable to each other.

*5.2.2.3.1.2 Mean moves to solution*

One univariate outlier from the ASPD group and one multivariate outlier from each of the ASPD and non-ASPD groups were removed. Deviations from normality were present and pronounced for the baseline conditions (2 & 3-moves). A mixed ANOVA with problem difficulty as within-groups factor revealed a significant main effect of difficulty, *Trace*=0.97, $F(3,91)=1006.01$, $P<0.001$. There was also a significant main effect of group, $F(2,93)=4.96$,$P<0.01$, but no significant group x difficulty interaction, *Trace*=0.12, $F(6,184)=1.89$. Post hoc analyses indicated that the patient groups made more moves than the healthy control group, *Ps*<0.05, but were not different compared to each other (Figure 5.7).

Regarding attention, RVP hits were not significantly correlated with SOC moves to solution, *rhos*>-0.20. Further, excluding individuals with psychopathy from the ASPD group yielded comparable results.

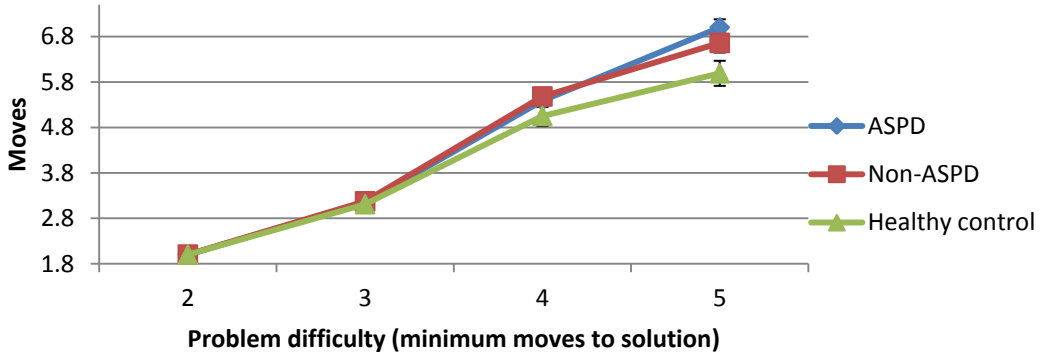



*Figure 5.7.* Number of move on the Stockings of Cambridge task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. Both patient groups performed significantly worse than controls overall but were comparable to each other.

*5.2.2.3.1.3 Initial thinking time*

One univariate outlier from the ASPD group and one multivariate outlier from the non-ASPD group were removed. There were deviations from normality in all conditions. Levene's test was significant for the final task condition only. Mixed ANOVA with problem difficulty as within-groups factor revealed a significant main effect of difficulty, *Trace*=0.67, $F(3,92)=63.21$, $P<0.001$. There was also a significant

main effect of group, $F(2,94)=4.96$, $P<0.001$, and a significant group x difficulty interaction, *Trace*=0.17, $F(4,186)=2.92$, $P<0.05$.

Post hoc analyses suggested that the ASPD group spent significantly less time thinking prior to initiating a solution compared to healthy controls, $P<0.001$, and marginally less time compared to the non-ASPD group, $P=0.05$. Unpacking the interaction revealed that there were no significant group differences for the 2-move problems, $F(2,96)=1.66$, but the ASPD group spent significantly less time planning the problems compared to the healthy control group for 3-move, $F(2,95)=5.16$, 4-move, $F(2,94)=6.87$, and 5-move problems, $F(2,96)=5.96$, *Ps*<0.01, post hoc, *Ps*<0.05 (Figure 5.8). There were no further significant differences.

Regarding attention, RVP hits were not significantly correlated with SOC initial thinking time, *rhos*<0.15. However, ASPD-only analyses did not provide comparable results though mixed ANOVA revealed significant main effects of group and difficulty and a significant interaction as before. This time, post hoc analyses suggested that the ASPD-only group spent significantly less time thinking prior to initiating a solution compared to either non-ASPD and healthy control groups, *Ps*<0.01, but individuals without ASPD and healthy controls performed comparably to each other. Unpacking the interaction yielded comparable results as before except a trend for the ASPD-only group to exhibit shorter initial thinking times compared to controls during 2-move problems, $F(2,72)=3.38$, $P<0.05$, post hoc, $P=0.52$, and that the ASPD-only group showed significantly shorter initial thinking times during 3-move problems compared to either comparison group, *Ps*<0.05.

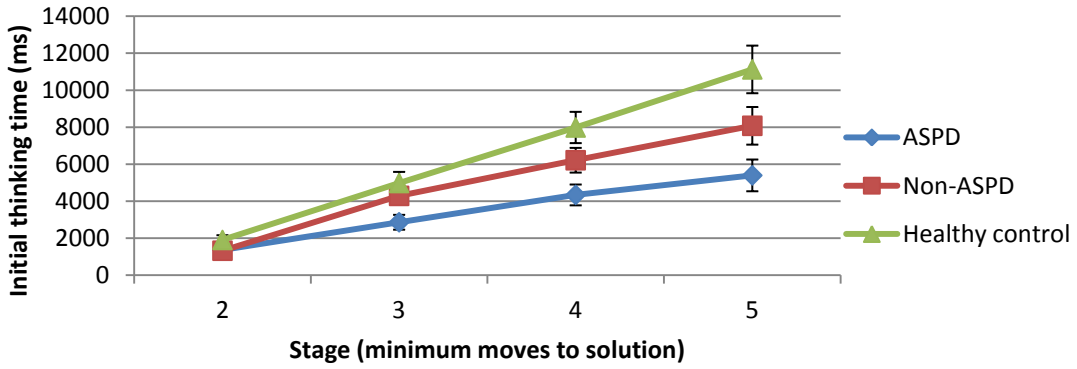



*Figure 5.8.* Initial thinking time on the Stockings of Cambridge task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy

controls. Both patient groups performed significantly worse than controls overall but were comparable to each other.

*5.2.2.3.1.4 Subsequent thinking time*

This measure resulted from subtracting movement time from total time and can contain relatively large measurement error. As a result, large deviations from the mean resulted in five univariate outliers in the ASPD group, three in the non-ASPD group whereas there were two multivariate outliers in the healthy control group. All outliers were removed. Deviations from normality were also present. Box's and Levene's test were significant for all except the final task condition.

Mixed ANOVA with problem difficulty as within-groups factor revealed a significant main effect of difficulty, *Trace*=0.46, $F(3,84)=23.33$, $P<0.001$. However, the main effect of group only approached significance, $F(2,86)=2.92$, $P=0.06$, whereas the group x difficulty interaction was not significant, *Trace*=0.03, $F(6,170)=0.44$ (Figure 5.9). Excluding individuals with psychopathy from the ASPD group yielded comparable results with the exception of the main effect of group which no longer approached significance, $F(2,66)=2.11$.

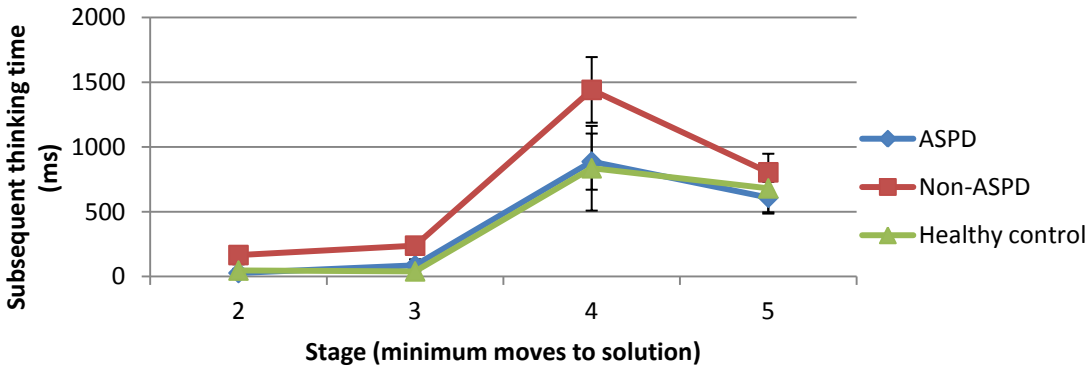



*Figure 5.9.* Subsequent thinking time on the Stockings of Cambridge task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. There were no significant differences.

#### *5.2.2.3.2 Psychopathy*

##### *5.2.2.3.2.1 Perfect solutions*

K-S test was significant for the group without psychopathy only. A Kruskal-Wallis test revealed a significant main effect of group, $Chi^2$=13.63, *df*=2, *P*<0.01, also supported by ANOVA, *F*(2,94)=7.8, *P*<0.01. Post hoc analysis suggested that both patient groups performed worse than the healthy control group, *Ps*<0.05, but were not different compared to each other (Figure 5.10).

Regarding attention, RVP hits were significantly correlated with SOC perfect solutions. There was also a significant group x covariate interaction, *F*(3,85)=4.06, *P*<0.05. Adding RVP hits as fixed predictor to the MLM did not result in a significantly improved model, $\Delta Chi^2$=0.96, $\Delta df$=1, whereas there was no convergence with RVP hits as random predictor.

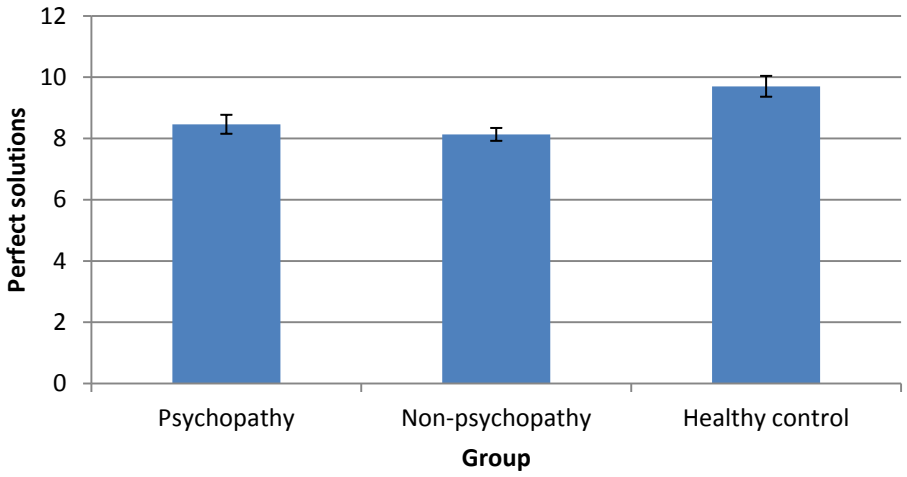



*Figure 5.10.* Perfect solutions on the Stockings of Cambridge task for patients with and without psychopathy and healthy controls. Both patient groups performed significantly worse than controls but were comparable to each other.

##### *5.2.2.3.2.2 Mean moves to solution*

One univariate outlier from the group with psychopathy and one multivariate outlier from each of the groups with and without psychopathy were removed. Deviations from normality were present especially for baseline conditions. All participants performed 2-moves at the 2-move problems.

A mixed ANOVA with difficulty as within-groups factor revealed a significant main effect of difficulty, *Trace*=0.97, $F(3,88)=912.18$, $P<0.001$. There was also a significant main effect of group, $F(2,90)=5.90$, $P<0.01$, and a significant group x difficulty interaction, *Trace*=0.17, $F(6,178)=2.79$, $P<0.05$.

Post hoc analyses indicated that the group without psychopathy made significantly more moves than the healthy control group, $P<0.01$. Unpacking the interaction suggested that there were no differences for 3-move problems, $F(2,90)=0.32$. The group without psychopathy performed significantly worse than the group with psychopathy only in 4-move problems, $F(2,92)=4.54$, $P<0.05$, while both patient groups performed significantly worse than the healthy control group for 5-move problems, $F(2,92)=4.39$, $P<0.05$, post hoc, $Ps<0.05$. There were no further significant group differences (Figure 5.11). Regarding the effect of attention, RVP hits were not correlated significantly with SOC mean moves to solution.

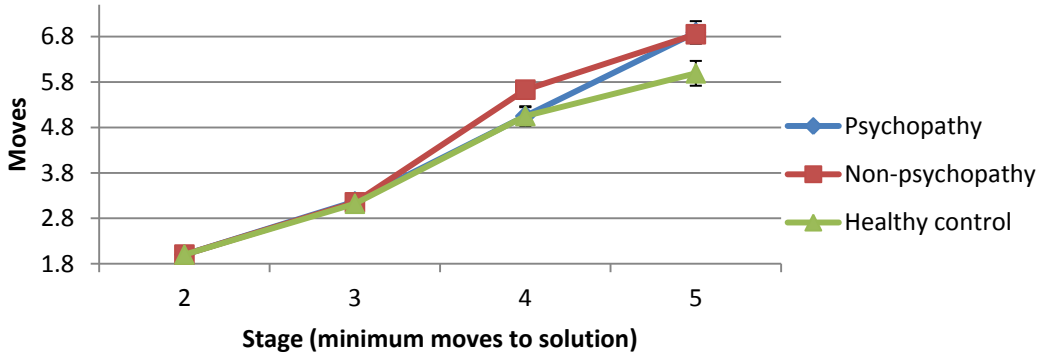



*Figure 5.11.* Number of move on the Stockings of Cambridge task for patients with and without psychopathy and healthy controls. Patients without psychopathy performed worse than controls overall and during 4 and 5-move problems. Patients with psychopathy performed worse than controls during 5-move problems only. Patients with and without psychopathy were otherwise comparable to each other.

*5.2.2.3.2.3 Initial thinking time*

One univariate outlier was removed from each of the groups with and without psychopathy. There were deviations from normality in all conditions. A Mixed ANOVA with difficulty as within-groups factor revealed a significant main effect of

group, *Trace*=0.62, *F*(3,90)=48.59, *P*<0.001. There was also a significant main effect of group, *F*(2,92)=5.65, *P*<0.01, but no significant group x difficulty interaction, *Trace*=0.09, *F*(6,182)=1.44.

Post hoc analyses suggested that the patient groups spent significantly less time thinking prior to initiating a solution than the healthy control group, *P*<0.05, but were not different compared to each other (Figure 5.12). Regarding the effect of attention, RVP hits were not correlated significantly with SOC initial thinking time, like before.

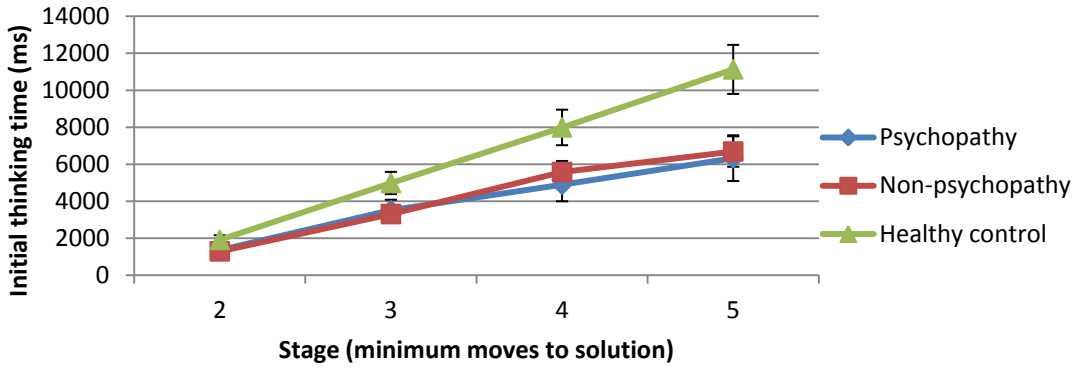



*Figure 5.12.* Initial thinking time on the Stockings of Cambridge task for patients with and without psychopathy and healthy controls. Both patient groups performed significantly worse than controls overall but were comparable to each other.

*5.2.2.3.2.4 Subsequent thinking time*

There were four univariate outliers in the ASPD group, two in the non-ASPD group and two multivariate outliers in the healthy control group, all of which were removed. Deviations from normality were present. Box's test and Levene's test for 3-move problems only were significant. A Mixed ANOVA with problem difficulty as within-groups factor revealed a significant main effect of difficulty, *Trace*=0.42, *F*(3,82)=20.07, *Ps*<0.001. There was neither a significant main effect of group, *F*(2,84)=0.84, nor a significant group x difficulty interaction, *Trace*=0.02, *F*(6,166)=0.31, (Figure 5.13).

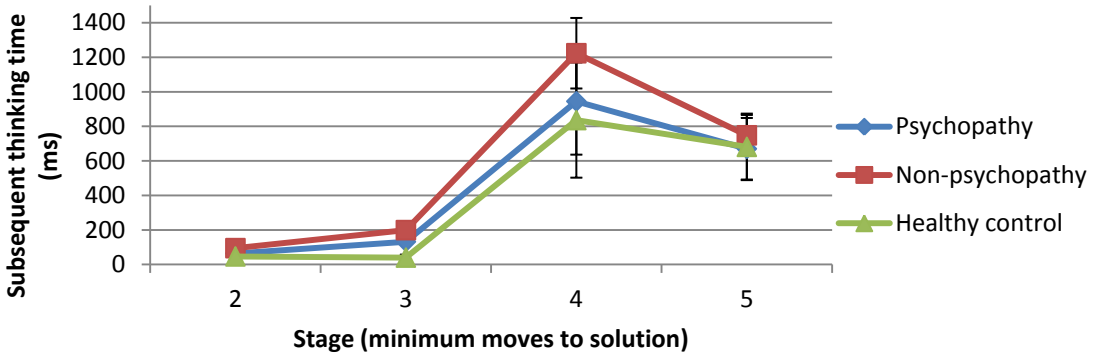



*Figure 5.13.* Subsequent thinking time on the Stockings of Cambridge task for patients with and without psychopathy and healthy controls. There were no significant differences.

#### *5.2.2.3.3 Summary of planning*

Results indicated that the patient group generally demonstrated a planning deficit compared to healthy controls. Although individuals with psychopathy exhibited shorter planning times overall compared to controls, the deficit in efficiency (number of moves) emerged during most challenging problems only. However, this was not as pronounced as in individuals without psychopathy who performed worse than controls in easier problems also.

### 5.2.2.4 Cognitive flexibility

#### *5.2.2.4.1 ASPD*

##### *5.2.2.4.1.1 Intra/Extra-Dimensional Set Shifting task*

Deviations from normality were present in all groups. Box's and Levene's tests were significant. After Bonferroni correction, Kruskal-Wallis tests revealed a significant main effect of group for reversal errors, $Chi^2$=9.94, *df*=2, *P*<0.01, but the main effect of group only approached significance for EDS errors, $Chi^2$=6.97, *df*=2, *P*=0.03. MANOVA suggested an overall main effect of group, *Trace*=0.18, *F*(4,188)=4.73, *P*<0.01, which was significant (after Bonferroni correction) for both reversal, *F*(2,94)=5.71, *P*<0.01, and EDS errors, *F*(2,94)=4.45, *P*<0.025. Post hoc comparisons indicated that the ASPD group performed significantly more reversal errors than non-ASPD patients and healthy controls and more EDS errors than healthy controls only, *Ps*<0.05 (Figure 5.14 and Figure 5.15). There were no further significant group differences. The possible demographic covariates between non-ASPD and ASPD groups (inc. total PCL-R score) were not correlated with reversal errors.

Regarding the effect of attention, RVP hits were correlated significantly with reversal errors, *rho*=-0.22, *P*<0.05. There was also a significant group x covariate interaction, *F*(3,86)=3.36, *P*<0.05. Using MLM, the model did not improve when RVP hits were added either as fixed or random predictor, $\Delta Chi^2$=0.55, $\Delta df$=1, and, $\Delta Chi^2$=-1.73, $\Delta df$=3 respectively. RVP hits was also correlated significantly with EDS errors, *rho*=-0.26, *P*<0.05. As the group x covariate interaction was not significant, *F*(3,86)=2.67, ANCOVA was conducted. RVP hits did not make a significant adjustment, *F*(1,86)=2.07. History of SRD, and prescription of antidepressant and antipsychotic medication were not significantly correlated with reversal errors, *rhos*=-0.04 to 0.22.

Regarding the ASPD-only group, Kruskal-Wallis tests revealed the opposite pattern of results where a main effect of group was significant for EDS errors, $Chi^2$=8.94, *df*=2, *P*<0.025, whereas the effect approached significance for reversal errors, $Chi^2$=7.25, *df*=2, *P*=0.027. Regarding reversal errors, the result may be a statistical artefact as it must be noted that the group with psychopathy also exhibited

this deficit (Figure 5.19). Conducting a MANOVA, however, yielded comparable results as before.

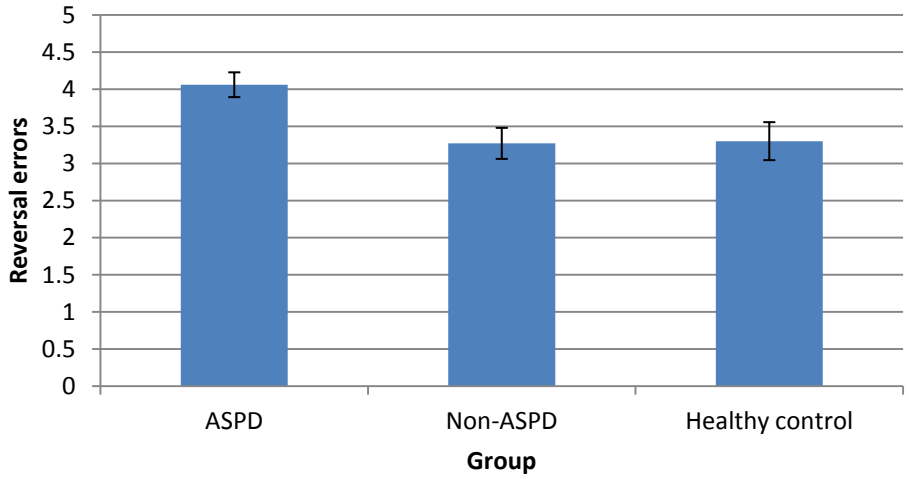



*Figure 5.14.* Reversal errors on the Intra/Extra-Dimensional Set Shifting task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. The ASPD group performed significantly worse than both the non-ASPD group and controls, which were comparable to each other.

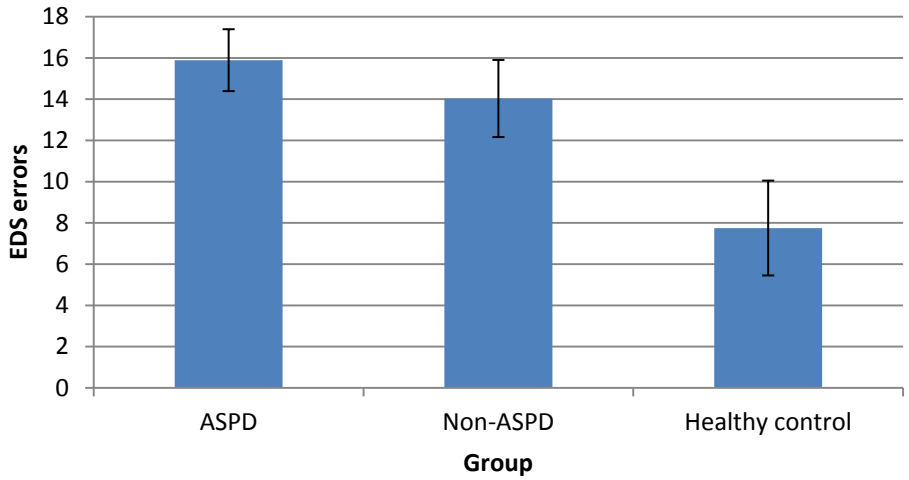



*Figure 5.15.* Extra-dimensional shift errors on the Intra/Extra-Dimensional Set Shifting task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. The ASPD group performed significantly worse than controls. There were no further significant groups differences.

*5.2.2.4.1.2 Cambridge Gambling Task*

5.2.2.4.1.2.1 Quality of decision-making

One univariate outlier was removed from the healthy control group and a multivariate outlier from the ASPD group. There were some deviations from normality, particularly for the healthy control group. Mixed ANOVA was conducted with increment (ascending/descending) and odds (9:1, 8:2, etc.) as within-groups factors. Levene's test was significant on several occasions. There was a significant main effect of increment, *Trace*=0.28, $F(1,31)=12.04$, and a significant main effect of odds, *Trace*=0.58, $F(3,29)=13.48$, *Ps*<0.01,but no significant increment x odds interaction, *Trace*=0.03, $F(3,29)=0.34$.

There were also a significant main effect of group, $F(2,31)=6.55$, *P*<0.01, a significant group x increment interaction, *Trace*=0.24, $F(2,31)=4.78$, *P*<0.05, and a significant group x odds interaction, *Trace*=0.55, $F(6,60)=3.82$, *P*<0.01. The group x increment x odds interaction was not significant, *Trace*=0.10, $F(6,60)=0.54$.

Post hoc analysis revealed that both patient groups showed significantly worse decision-making than healthy controls, *Ps*<0.05, but were not significantly different compared to each other. Both patient groups performed worse than controls in the ascending, $F(2,33)=8.07$, *P*<0.01 (post hoc, *Ps*<0.05), but not in the descending condition, $F(2,33)=3.60$, *P*<0.05 (but post hoc tests were not significant). Means are shown in Figure 5.16. ASPD participants performed worse than healthy controls in the 9:1 condition, $F(2,32)=4.91$, *P*<0.05, post hoc, *Ps*<0.05. There were no group differences in the 8:2 condition, $F(2,33)=1.82$. Both patient groups performed worse than the healthy control group in the 7:3 condition, $F(2,33)=9.93$, *P*<0.001,post hoc, *Ps*<0.01, whereas the main effect of group only approached significance in the 6:4 condition, $F(2,33)=3.25$, *P*=0.51 (Figure 5.16). There were no further significant group differences.

Regarding attention, RVP hits were not significantly correlated with CGT quality of decision-making, *rhos*<0.34. Furthermore, there were broadly comparable results for the ASPD-only group. Exceptions were that group x increment interaction no longer reached significance, *Trace*=0.12, $F(2,24)=1.66$, and that the ASPD-only group performed significantly worse than healthy controls in the 6:4 condition, $F(2,26)=4.78$, *P*<0.05, post hoc, *P*<0.05.

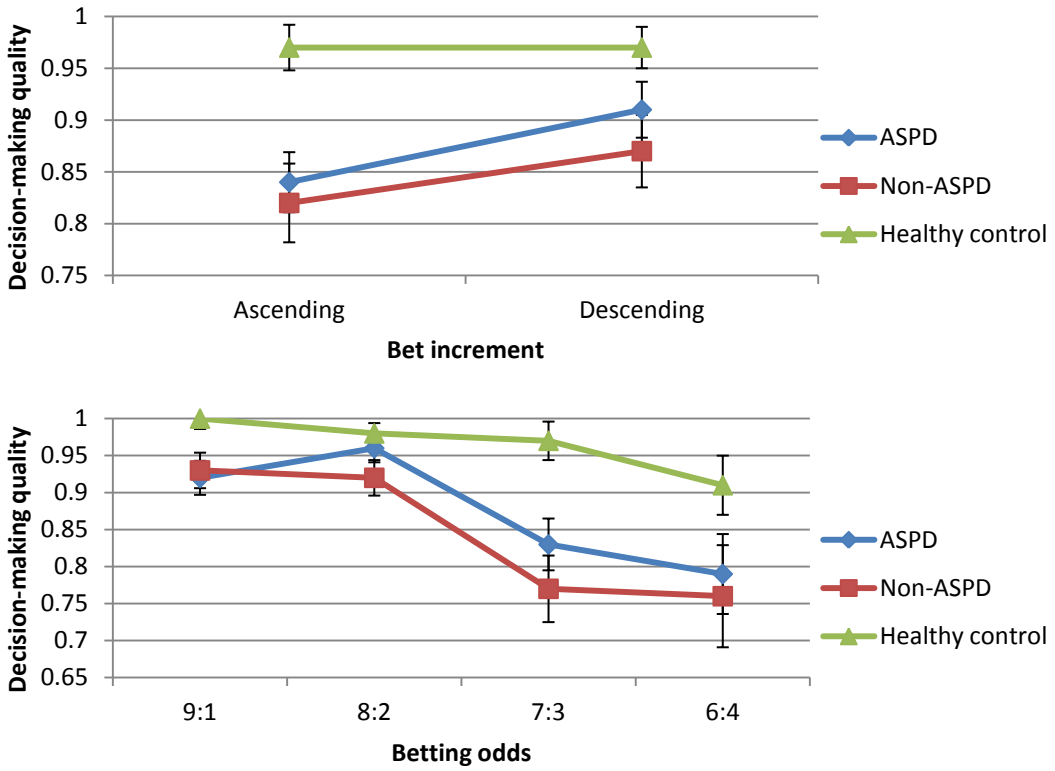



*Figure 5.16.* Quality of decision-making on the Cambridge Gambling Task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. Both patient group performed significantly worse than healthy controls overall, for ascending bets, and for 7:3 odds. The ASPD group performed significantly worse than controls at 9:1 odds. Patients groups were comparable to each other otherwise.

5.2.2.2.4.1.2.2 Risk-taking

One univariate outlier was removed from the healthy control group. There were also minor deviations from normality. Levene's test was significant for descending 9:1 and 8:2 conditions only. Mixed ANOVA with increment and odds as within-groups factors revealed a significant main effect of increment, *Trace*=0.49, $F(1,33)=31.46$, and a significant main effect of odds, *Trace*=0.63, $F(3,31)=17.82$, *Ps*<0.001, but no increment x odds interaction, *Trace*=0.03, $F(3,31)=0.34$.

The main effect of group approached significance, $F(2,33)=2.71$, $P=0.08$. There was no group x increment interaction, *Trace*=0.54, $F(2,33)=0.94$, group x odds

interaction, *Trace*=0.30, *F*(6,64)=1.91, or group x increment x odds interaction, *Trace*=0.20, *F*(6,64)=1.17, (Figure 5.17).

Regarding the ASPD-only group, results were comparable with the exception of the group x odds interaction which became significant, *Trace*=0.44, *F*(6,50)=2.33, *P*<0.05. Unpacking this interaction revealed that the ASPD-only group exhibited significantly more risk-taking compared to controls in the 6:4 condition only, *F*(2,27)=4.78, *P*<0.05, post hoc, *P*<0.05.

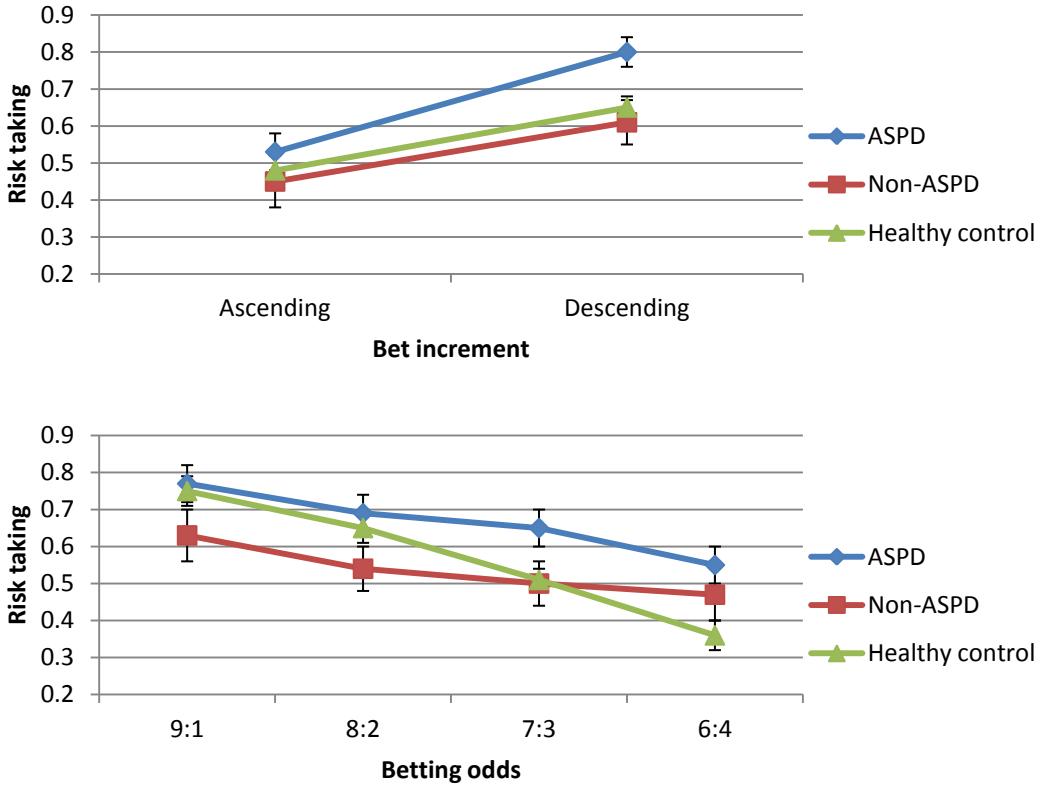



*Figure 5.17.* Risk-taking on the Cambridge Gambling Task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. There were no significant group differences.

5.2.2.4.1.2.3 Delay aversion

Mixed ANOVA with odds as within-groups factors was conducted. There was no significant main effect of odds, *Trace*=0.03, *F*(3,32)=0.37 No results involving group were significant including main effect, *F*(3,34)=1.09, and group x odds interaction, *Trace*=0.19, *F*(6,66)=1.18, (Figure 5.18). Supplementary analyses by

excluding individuals with psychopathy from the ASPD group yielded comparable results.

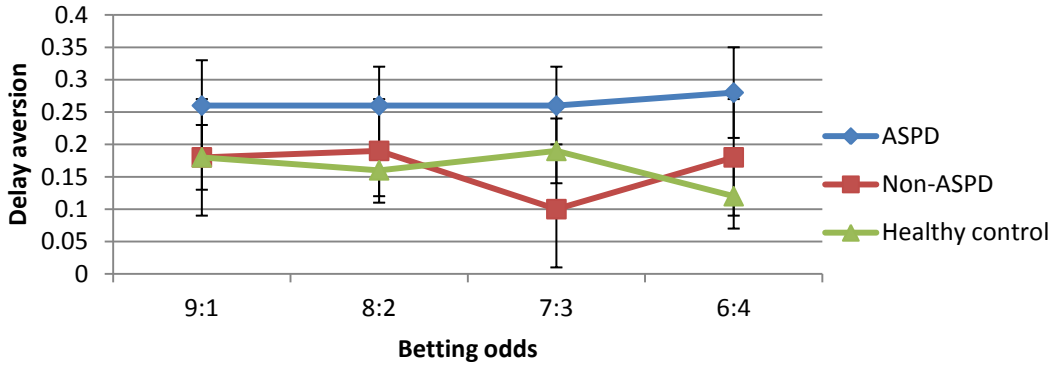



*Figure 5.18.* Delay aversion on the Cambridge Gambling Task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. There were no significant group differences.

In sum, results indicated deficits in cognitive flexibility in ASPD (decision-making [CGT], response reversal [IED], and attentional set-shifting [IED]), in line with the first hypothesis. Of the identified deficits, those in response reversal were not detected in offenders with other personality disorders when the groups were compared to controls. Furthermore, the impairments in ASPD during decision-making emerged in easier conditions than for individuals with other personality disorders.

#### *5.2.2.4.2 Psychopathy*

##### *5.2.2.4.2.1 Intra/Extra-Dimensional Set Shifting task*

Deviations from normality were present in all groups. Box's and Levene's tests were significant. After Bonferroni correction, Kruskal-Wallis tests revealed a significant main effect of group for EDS errors, $Chi^2$=9.97, *df*=2, *P*<0.01, but not a significant main effect of group for reversal errors, $Chi^2$=6.37, *df*=2, *P*>0.025 (Bonferroni correction). MANOVA suggested an overall main effect of group, *Trace*=0.19, *F*(4,182)=4.67, *P*<0.01, which was significant (after Bonferroni correction) for both reversal, *F*(2,91)=4.36, *P*<0.025, and EDS errors, *F*(2,91)=5.00, *P*<0.01. Post hoc comparisons indicated that patients with psychopathy showed significantly more reversal errors than healthy controls, whereas patients without

psychopathy showed significantly more EDS errors than healthy controls, *Ps*<0.05. The latter may be attributed to presence of ASPD, as removal of individuals with ASPD from the group without psychopathy resulted in a non-significant effect, $Chi^2$=5.26, *df*=2, and, *F*(2,68)=2.61. However, the groups are all confounded, making it difficult to to delineate the effects of different diagnoses, although it is evident that the patient group are on the whole impaired. There were no further significant group differences (Figure 5.19 and Figure 5.20). The possible demographic covariates between the groups with and without psychopathy were not correlated with reversal errors.

Regarding attention, there was a marginally significant correlation between RVP hits and reversal errors, *rho*=-0.21, *P*=0.05. There was also a significant group x covariate interaction, *F*(3,83)=2.78. Using MLM, the model did not improve for IED reversal errors when RVP hits were added either as fixed or random predictor, $\Delta Chi^2$=0.01, Δ*df*=1, and, $\Delta Chi^2$=-5.02, Δ*df*=2 respectively. RVP hits were also correlated significantly with EDS errors, *rho*=-0.26, *P*<0.05. As the group x covariate interaction was not significant, *F*(3,86)=2.67, ANCOVA was conducted. RVP hits did not result in a significant adjustment, *F*(1,86)=2.07. Regarding history of SRD, this was not correlated with IED reversal errors, *rho*=-0.04, *P*>0.05.

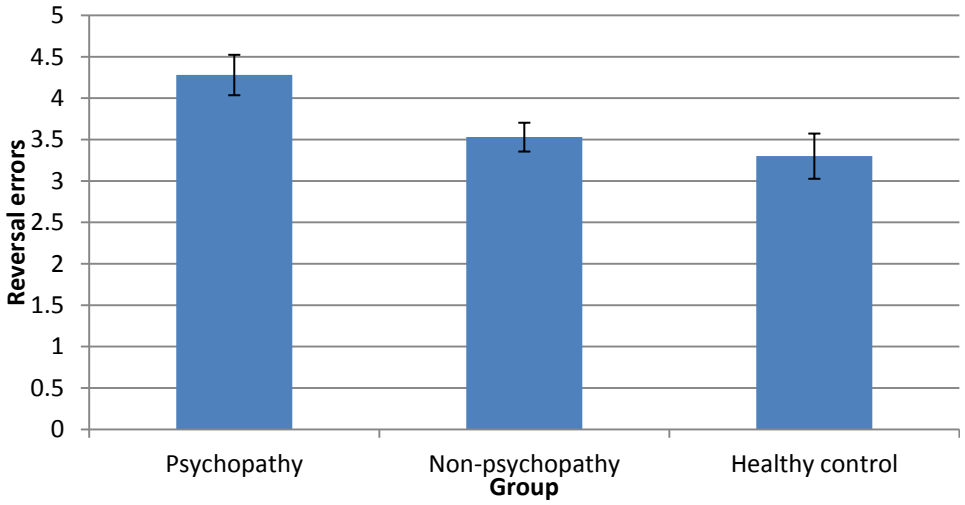



*Figure 5.19.* Reversal errors on the Intra/Extra-Dimensional Set Shifting task for patients with and without psychopathy and healthy controls. The group with psychopathy performed significantly worse than both the non-psychopathy group and controls, which were comparable to each other.

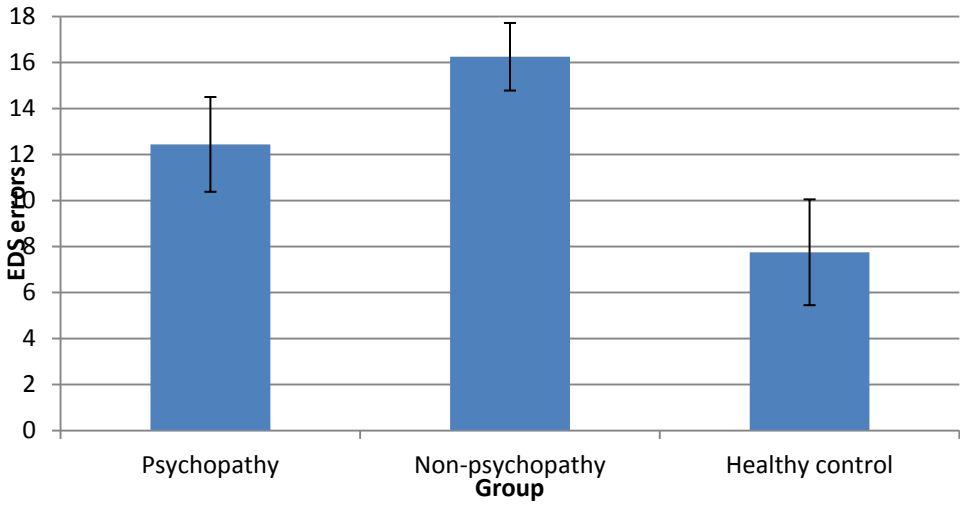



*Figure 5.20.* Extra-dimensional shift errors on the Intra/Extra-Dimensional Set Shifting task for patients with and without psychopathy and healthy controls. Patients without psychopathy (but not patients with psychopathy) performed significantly worse than controls. There were no further significant groups differences. The groups are all confounded, making it difficult to to delineate the effects of different diagnoses.

*5.2.2.4.2.2 Cambridge Gambling Task*

5.2.2.4.2.2.1 Quality of decision-making

A univariate outlier was removed from the healthy control group and a multivariate outlier was removed from the psychopathy group. There were some deviations from normality, particularly in the healthy control group. Levene's test was significant on several occasions. A mixed ANOVA with increment and odds as within-groups factors revealed a significant main effect of increment, *Trace*=0.23, $F(1,29)=8.53$, $P<0.01$, and odds, *Trace*=0.59, $F(3,27)=12.74$, $P<0.001$, but no significant increment x odds interaction, *Trace*=0.07, $F(3,27)=0.65$.

There was also a main effect of group, $F(2,29)=5.17$, $P<0.05$, a significant group x increment interaction, *Trace*=0.25, $F(2,29)=4.86$, $P<0.05$, and a significant group x odds interaction, *Trace*=0.44, $F(6,56)=2.60$, $P<0.05$. The group x increment x odds interaction was not significant, *Trace*=0.11, $F(6,56)=0.52$. Post hoc analysis

suggested that the group without psychopathy performed worse than the healthy control group, *P*<0.05, with no further significant differences.

Significant interactions indicated different decision-making patterns for the three groups. Both patient groups performed worse than the healthy control group in the ascending condition, *F*(2,32)=7.5, *P*<0.01, post hoc, *Ps*<0.05. In the descending condition, only the group without psychopathy performed worse than the healthy control group, *F*(2,32)=6.07, *P*<0.01, post hoc, *P*<0.01. There were no further significant differences (Figure 5.21).

In the 9:1 and 6:4 odds, patients without psychopathy performed significantly worse than the healthy control group, *F*(2,31)=4.96, and *F*(2,32)=4.62, *Ps*<0.05, post hoc, *P*<0.05. In 7:3 odds, both patient groups performed worse than controls, *F*(2,32)=8.87, *P*<0.01,post hoc, *Ps*<0.05. There were no further significant differences in these conditions and there was no main effect of group for 8:2 odds, *F*(2,32)=2.02, (Figure 5.21).

Regarding the effect of attention, RVP hits were not correlated significantly with CGT quality of decision-making, *rhos*<0.34.

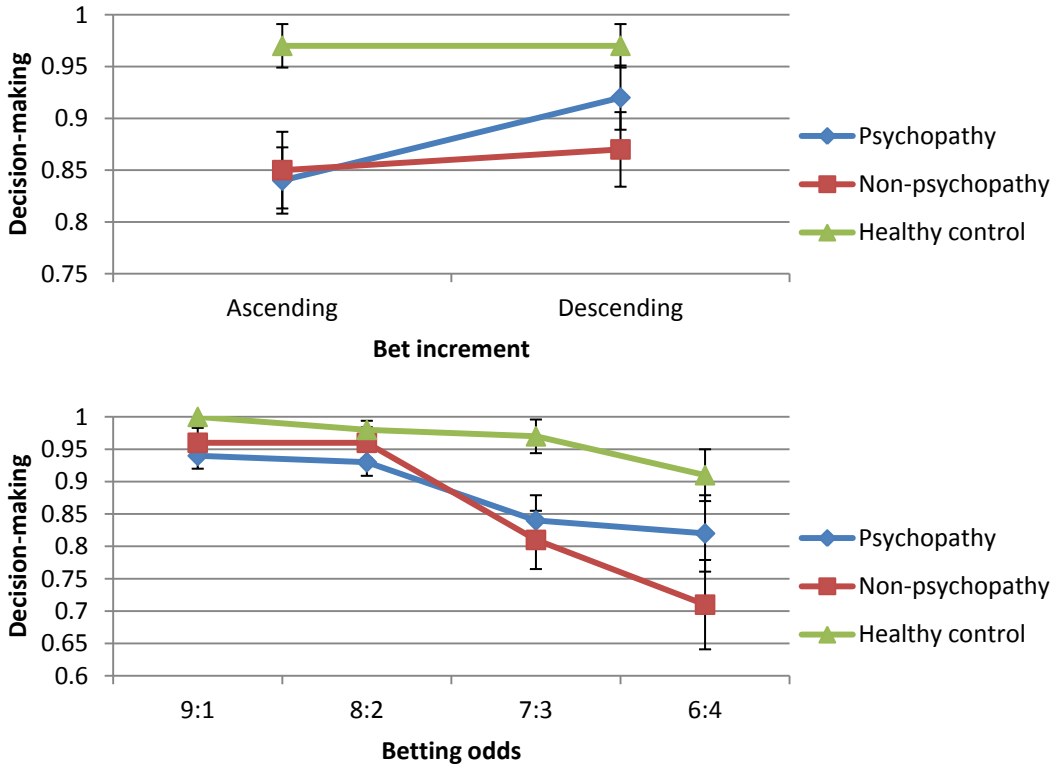


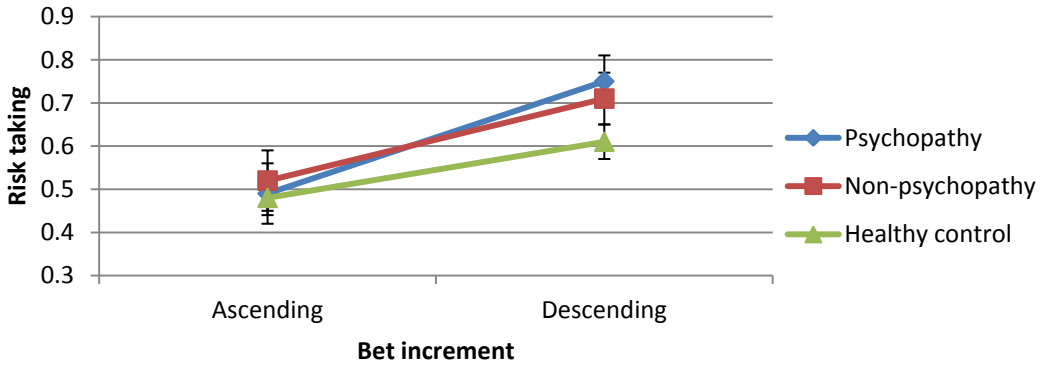

*Figure 5.21.* Quality of decision-making on the Cambridge Gambling Task for

patients with and without psychopathy and healthy controls. Patients without psychopathy performed significantly worse than controls overall, in both ascending and descending conditions, and in most betting odd conditions. Patients with psychopathy performed significantly worse than controls on the ascending condition and for 7:3 odds. Patient groups were comparable to each other otherwise.

5.2.2.4.2.2.2 Risk-taking

One univariate outlier was removed from the healthy control group. There were minor deviations from normality. Levene's test was significant for descending 9:1 and 8:2 conditions only. A mixed ANOVA with increment and odds as within-groups factors revealed a significant main effect of increment, *Trace*=0.52, $F(1,32)=34.14$, and odds, *Trace*=0.52, $F(1,32)=34.14$, $P<0.001$. The increment x odds interaction was not significant, *Trace*=0.30, $F(3,30)=1.84$.

There was no significant main effect of group, $F(2,32)=0.57$, group x increment interaction, *Trace*=0.04, $F(2,32)=0.63$, group x odds interaction, *Trace*=0.30, $F(6,62)=1.84$, or group x increment x odds interaction, *Trace*=0.17, $F(6,62)=0.95$, (Figure 5.22).



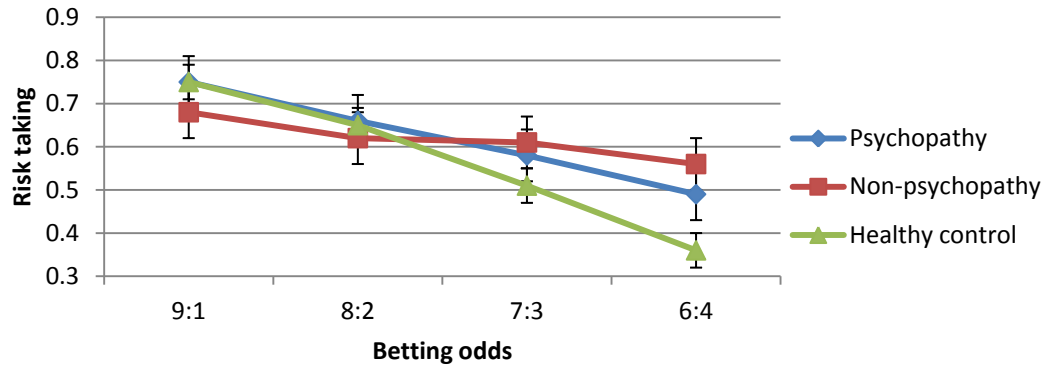



*Figure 5.22.* Risk-taking on the Cambridge Gambling Task for patients with and without psychopathy and healthy controls. There were no significant group differences.

5.2.2.4.2.2.3 Delay aversion

Mixed ANOVA with odds as within-groups factors revealed no significant main effect of odds, *Trace*=0.01, $F(3,31)=0.11$, main effect of group, $F(2,33)=0.77$, or group x odds interaction, *Trace*=0.19, $F(6,66)=1.18$, (Figure 5.23).

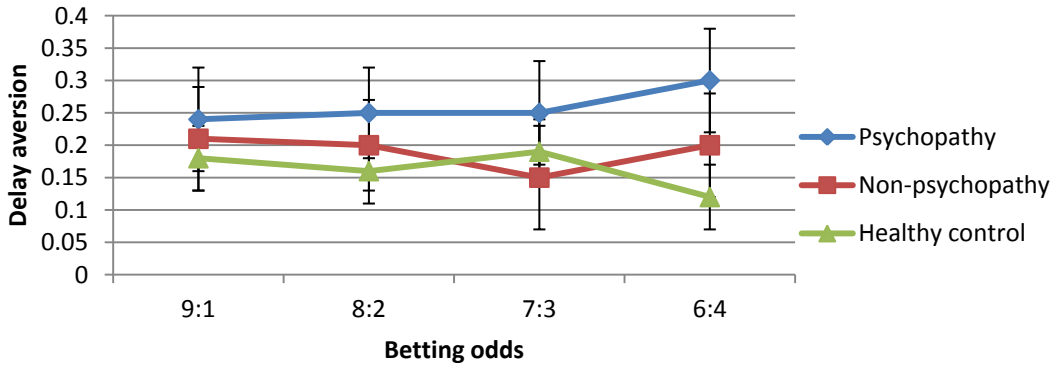



*Figure 5.23.* Delay aversion on the Cambridge Gambling Task for patients with and without psychopathy and healthy controls. There were no significant group differences

In sum, results suggested deficits in aspects of cognitive flexibility (response reversal [IED], decision-making [CGT]) in psychopathy thereby providing some support for the relevant hypothesis. However, no deficits were identified in attentional set-shifting (IED), contrary to expectations. Furthermore, the difficulties in response reversal were not observed in offenders with other personality disorders against

controls. Finally, individuals without psychopathy performed worse than controls on attentional set-shifting and decision-making compared to individuals with psychopathy. The former may be attributed to presence of ASPD in the group without psychopathy but the groups are all confounded, making it difficult to delineate the effects of different diagnoses.

### 5.2.2.5 Memory

#### *5.2.2.5.1 ASPD*

##### *5.2.2.5.1.1 Visual short-term memory*

5.2.2.5.1.1.1 Paired Associates Learning

Regarding completed stages, there were deviations from normality. A Kruskal-Wallis test did not reveal a significant main effect of group, $Chi^2$=2.23, *df*=2, (Figure 5.24). Supplementary analyses by excluding individuals with psychopathy from the ASPD group yielded comparable results.

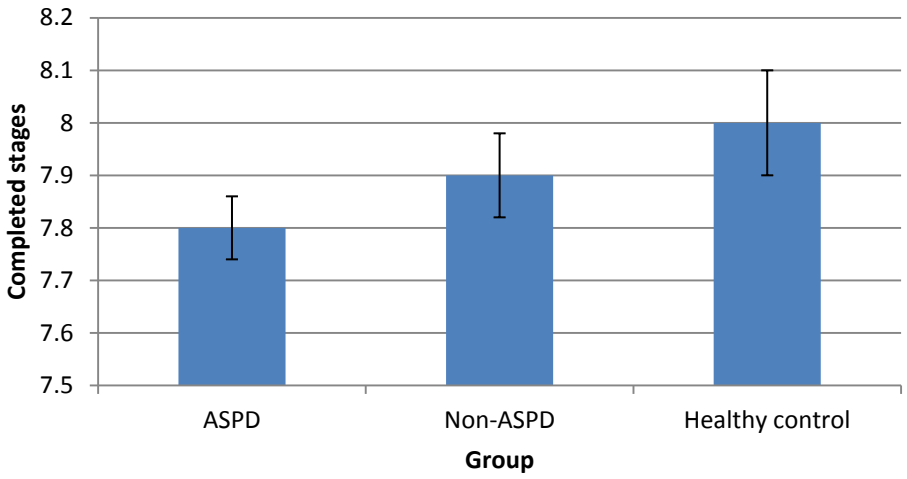



*Figure 5.24.* Number of completed stages on the Paired Associates Learning task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. Group differences did not reach significance.

Regarding errors, one univariate outlier was removed from the non-ASPD group and one multivariate outlier from each of the non-ASPD and ASPD groups. Deviations from normality were present. A mixed ANOVA with number of patterns as within-groups factor revealed a main effect of number of patterns, *Trace*=0.58, $F(4,86)=30.24$, $P<0.001$. There was not a significant main effect of group, $F(2,89)=0.90$, or group x difficulty interaction, *Trace*=0.04, $F(8,174)=0.43$ (Figure 5.25). Results were comparable using the adjusted number of errors. Supplementary analyses by excluding individuals with psychopathy from the ASPD group yielded comparable results.

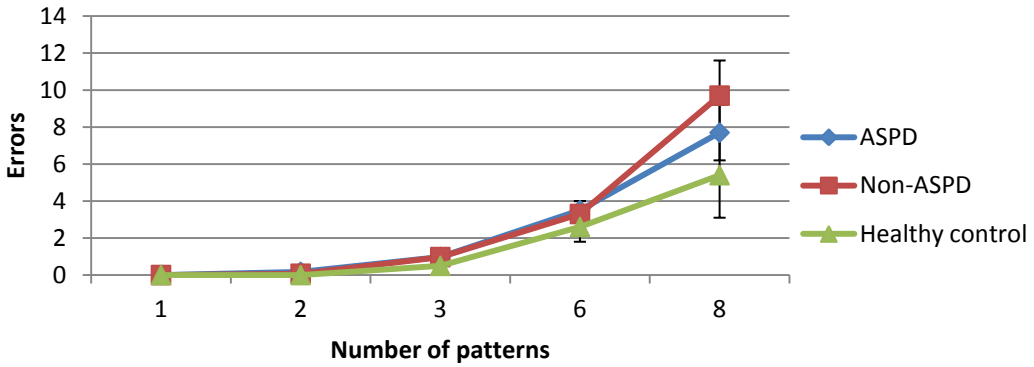



*Figure 5.25.* Number of errors on the Paired Associates Learning task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. There were no significant group differences.

5.2.2.5.1.1.2 Delayed Matching to Sample

Three univariate outliers from the non-ASPD and one from the ASPD group were removed. Minor deviations from normality were observed. Levene's test was significant for all except the immediate recognition condition. A mixed ANOVA with delay as within-groups factor revealed a significant main effect of delay, *Trace*=0.52, $F(3,96)=34.66$, $P<0.05$. There was also a significant main effect of group, $F(2,98)=8.63$, $P<0.001$, and a significant group x delay interaction, *Trace*=0.14, $F(6,194)=2.39$, $P<0.05$. The groups were comparable in reaction times, with a non-significant main effect, $F(2,97)=0.83$, and group x delay interaction, *Trace*=0.08, $F(6,192)=1.33$.

Post hoc analysis for correct responses indicated that both patient groups performed worse compared to the healthy control group overall, *Ps*<0.05. Unpacking the interaction revealed no significant group differences during simultaneous presentation, $F(2,98)=1.2$, but suggested that the ASPD group performed worse compared to the healthy control group during the immediate, $F(2,99)=4.34$, $P<0.05$ and 4-second delayed recognition, $F(2,99)=5.41$, $P<0.01$. Both patient groups performed significantly worse than the healthy control group in the 12-second delayed recognition, $F(2,99)=6.2$, $P<0.01$. For all post hoc tests *P* was <0.01, except for immediate recognition, $P<0.05$. There were no further significant group differences (Figure 5.26).

Regarding attention, RVP total hits were correlated significantly with overall number of correct responses, $r$=0.38, $P$<0.001, but there was a significant group x delay covariate interaction, $F(3,88)$=7.82, $P$<0.001. Using MLM to control for RVP total hits (random at Level 1-repeated measures), the model did not converge when the RVP hits was declared random at group level. However, its addition as a fixed predictor alongside group resulted in a significantly better model, $\Delta Chi^2$=8.84, $\Delta df$=1, $P$<0.01. Parameter estimates supported previous findings where both patient groups performed worse than the healthy control group, non-ASPD: *beta*=-0.53, *SE*=0.24, ASPD: *beta*=-0.60, *SE*=0.24, *Ps*<0.05. The patient groups performed comparably to each other, *beta*=0.07, *SE*=0.19.

Finally, excluding individuals with psychopathy from the ASPD group resulted in comparable results with the exception of the group x delay interaction which approached significance, *Trace*=0.16, $F(6,146)$=2.10, $P$=0.06.

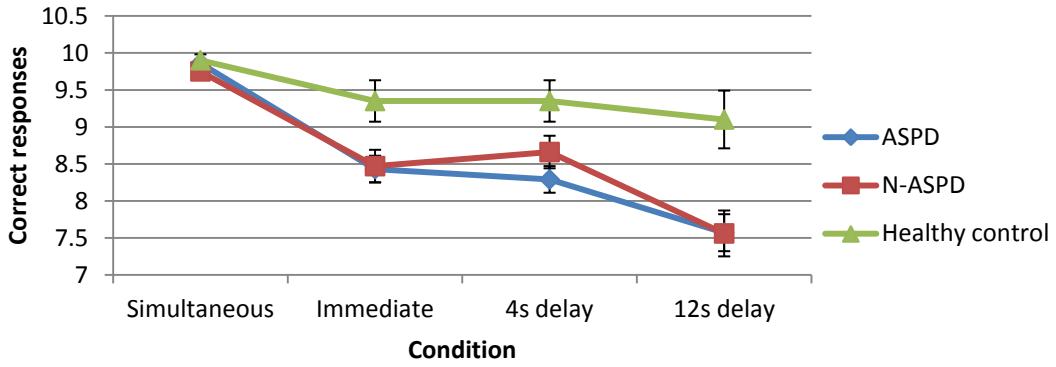



*Figure 5.26.* Number of correct responses on the Delayed Matching to Sample task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. Patients with ASPD performed worse than controls overall and in both immediate and delayed recognition. Patients without ASPD performed worse than controls overall and during recognition after the 12s delay only. There were no further significant group differences.

5.2.2.5.1.1.3 Spatial Span task

Deviations from normality were present and the K-S test was significant for all groups. A Kruskal-Wallis test revealed a significant main effect of group, $Chi^2$=6.9, $df$=2, $P$<0.05. This was confirmed by one-way ANOVA, $F(2,96)$=4.64, $P$<0.05. Post

hoc analysis suggested that both patient groups performed worse than the healthy control group, *Ps*<0.05, but were not different compared to each other (Figure 5.27).

Regarding attention, RVP total hits were correlated significantly with SSP span length, *rho*=0.27, *P*<0.05, and there was a significant group x covariate interaction, *F*(3,86)=3.76, *P*<0.05. Using MLM, adding RVP total hits to the model (fixed effects) resulted in a marginally significant improvement of the model, $\Delta Chi^2$=3.72, $\Delta df$=1, *P*=0.054, whilst the model did not converge when RVP hits were declared random at Level 2. Furthermore, excluding individuals with psychopathy from the ASPD group yielded comparable results.

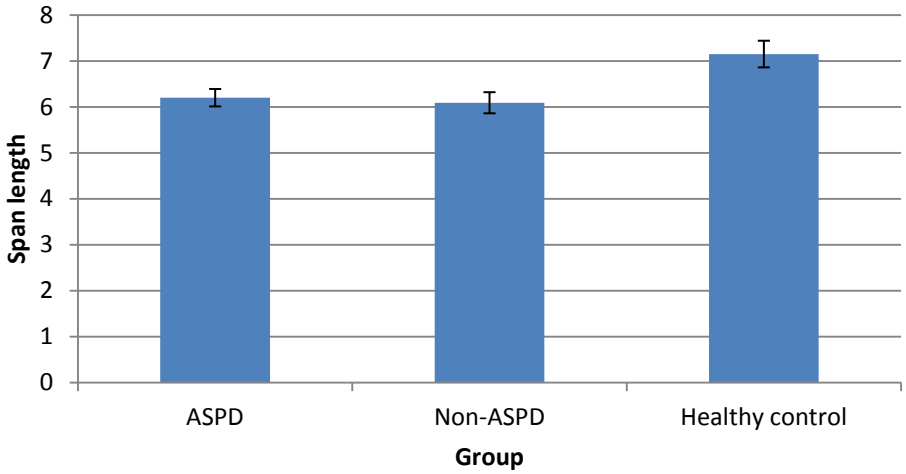



*Figure 5.27.* Span length on the Spatial Span task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. Both patient groups performed significantly worse than controls but were comparable to each other.

*5.2.2.5.1.2 Verbal memory*

Regarding correct recalls, some deviations from normality were present. A Kruskal-Wallis test did not reveal a significant main effect of group, $Chi^2$=1.77, *df*=2, (Figure 5.28). Results were comparable after excluding individuals with psychopathy from the ASPD group.

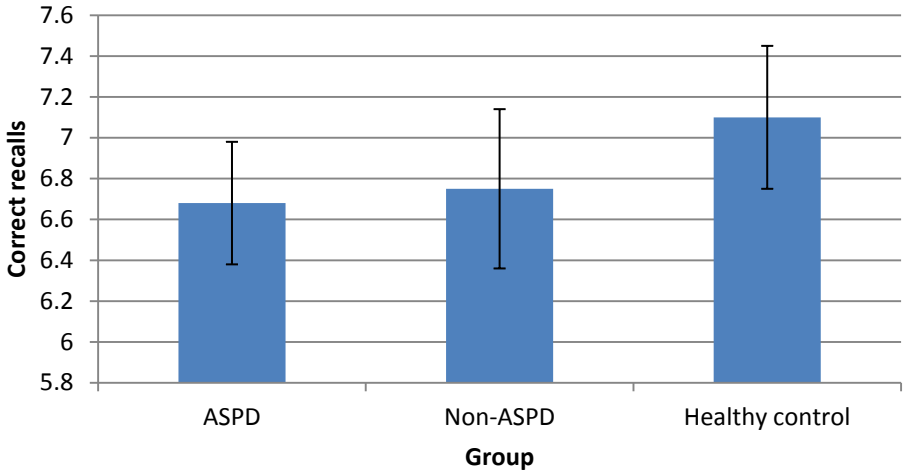



*Figure 5.28.* Number of correct recalls on the Verbal Recognition Memory task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. There were no significant group differences.

Regarding correct recognitions, one multivariate outlier was removed from the non-ASPD group. Deviations from normality were present. A mixed ANOVA with immediate/delayed recognition as within-groups factor did not reveal a significant main effect of task condition, *Trace*=0.01, $F(1,58)=0.04$, a main effect of group, $F(2,58)=0.13$, or group x condition interaction, *Trace*=0.03, $F(2,58)=0.77$, (Figure 5.29). Results were comparable after excluding individuals with psychopathy from the ASPD group.

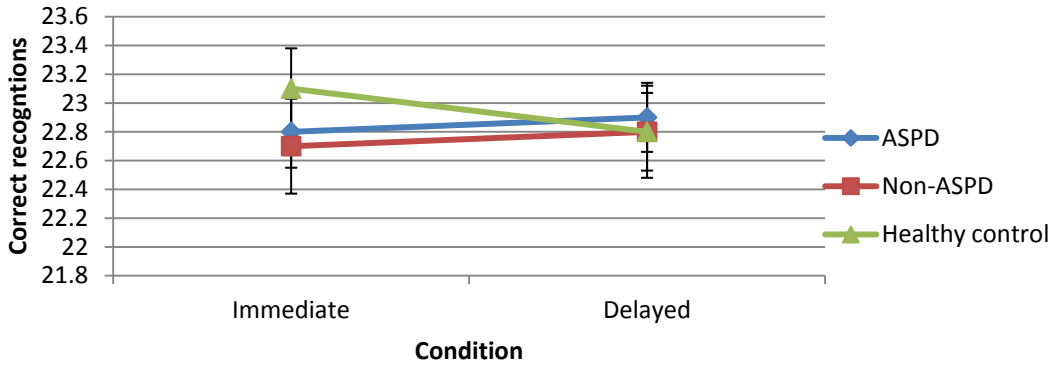



*Figure 5.29.* Number of correct recognitions on the Verbal Recognition Memory task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. There were no significant effects involving group.

*5.2.2.5.1.3 Working memory*

Two univariate outliers from the non-ASPD group and another three from the ASPD group were removed. Some deviations from normality were present. Box's and Levene's tests were significant for the 6-box condition. A mixed ANOVA with number of boxes (difficulty) as within-groups factor revealed a main effect of difficulty, *Trace*=0.63, $F(2,91)=77.52$, $P<0.001$. There was no significant main effect of group, $F(2,92)=2.71$, but there was a significant group x difficulty interaction, *Trace*=0.13, $F(4,184)=3.24$, $P<0.05$.

Unpacking the interaction revealed that there were no significant group differences for 4-box problems, $F(2,93)=0.09$, and that differences were marginal for 6-box, $F(2,96)=3$, $P=0.05$, and 8-box problems, $F(2,97)=3.09$, $P=0.05$. The significant interaction likely arose primarily due to these marginal effects for which post hoc analysis suggested that the ASPD group performed significantly worse than the healthy control group during 8-box stages only, $P<0.05$. There were no further significant differences (Figure 5.30).

Regarding the effect of attention, RVP total hits were significantly correlated with total SWM errors, *rho*=-0.29, $P<0.01$, but did not interact with group, $F(3,84)=2.31$, and did not emerge as a significant covariate in mixed ANCOVA, $F(1,84)=2.88$. Furthermore, excluding individuals with psychopathy from the ASPD group yielded comparable results except that the main effect of group became significant, $F(2,69)=3.27$, $P<0.05$. This indicated that the ASPD-only group performed worse than healthy controls, $P<0.05$, but there were no further significant differences.

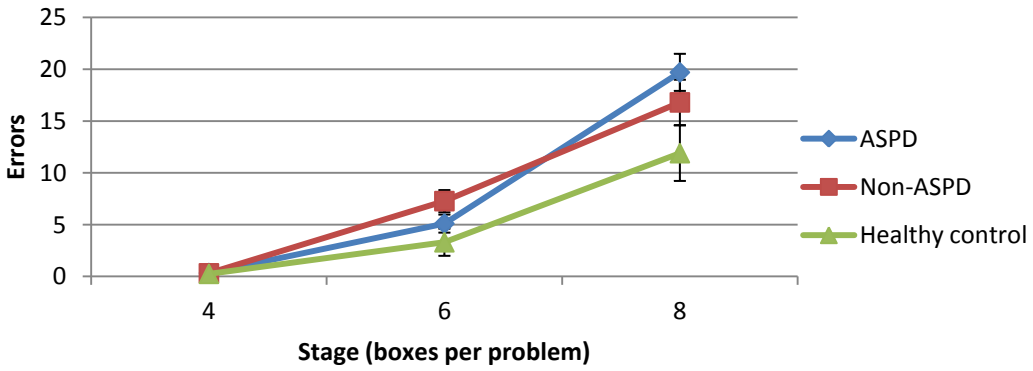



*Figure 5.30.* Number of errors on the Spatial Working Memory task for patients with

and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. A significant group x stage interaction indicated that patients with ASPD performed significantly worse than controls during 8-box stages. There were no further significant group differences.

In sum, results from ASPD provided some support for the hypothesised deficit in visual STM (DMS & SSP). Offenders with other personality disorders also exhibited these deficits compared to controls. In addition, the impairment of individuals with ASPD during the DMS emerged in the easier conditions of the task, a pattern that was not observed in offenders with other personality disorders. However, no impairment was detected in visual STM in ASPD during cued recall/learning (PAL), contrary to expectations. Regarding verbal memory and WM, although no deficits were identified in the former as hypothesised there was an impairment in WM, contrary to expectations. The latter was not detected in offenders with other personality disorders when the groups were compared to controls.

#### *5.2.2.5.2 Psychopathy*

##### *5.2.2.5.2.1 Visual short-term memory*

###### 5.2.2.5.2.1.1 Paired Associates Learning

Regarding completed stages, there were deviations from normality. A Kruskal-Wallis test did not reveal a significant main effect of group, $Chi^2$=2.32, *df*=2, (Figure 5.31).

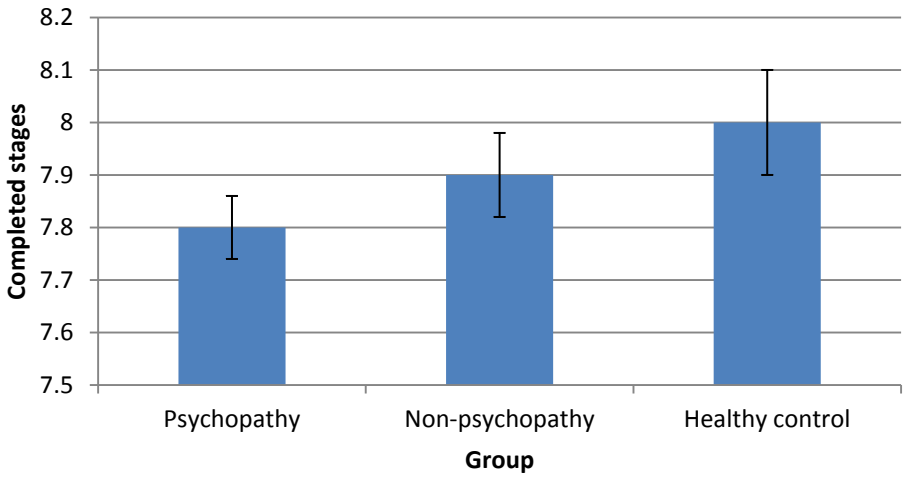



*Figure 5.31.* Number of completed stages on the Paired Associates Learning task for patients with and without psychopathy and healthy controls. There were no significant group differences.

Regarding errors, one multivariate outlier was removed from each of the groups with and without psychopathy. Deviations from normality were observed. Levene's test was significant for the 3- and 6-pattern stages. A mixed ANOVA with number of patterns as within-groups factor revealed a significant main effect of number of patters, *Trace*=0.57, $F(4,84)=27.79$, $P<0.001$. There was no significant main effect of group, $F(2,87)=1.02$, and no group x difficulty interaction, *Trace*=0.06, $F(8,170)=0.65$ (Figure 5.32). Results were comparable using the adjusted number of errors.

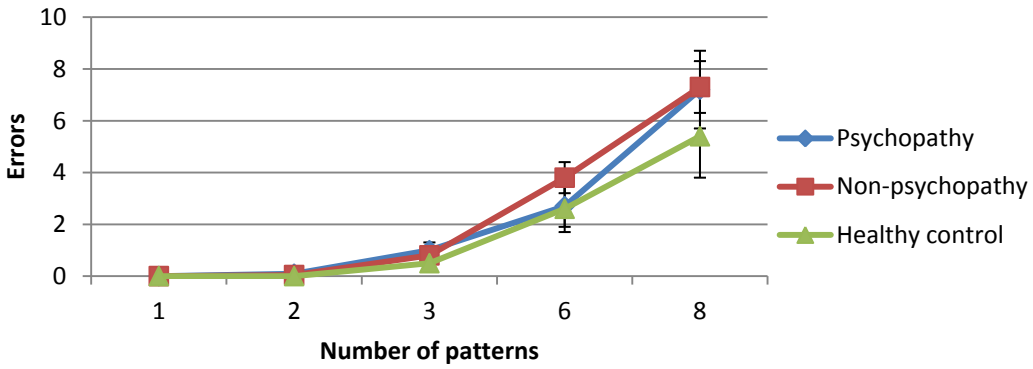



*Figure 5.32.* Number of errors on the Paired Associates Learning task for patients

with and without psychopathy and healthy controls. There were no significant group differences.

5.2.2.5.2.1.2 Delayed Matching to Sample task

Two univariate outliers from each of the groups with and without psychopathy were removed. Minor deviations from normality were observed. Levene's test was significant for all except the immediate recognition condition. A mixed ANOVA with delay as within-groups condition revealed a significant main effect of delay, *Trace*=0.54, $F(3,93)=35.90$, $P<0.001$. There was also a significant main effect of group, $F(2,95)=8.83$, $P<0.001$, and a significant group x condition interaction, *Trace*=0.17, $F(6,188)=2.39$, $P<0.01$.

Post hoc analysis suggested that both patient groups performed significantly worse than the healthy control group overall. Unpacking the interaction revealed no significant group differences during simultaneous presentation, $F(2,95)=1.71$. The group without psychopathy performed worse compared to the healthy control group during immediate recognition, $F(2,96)=4.72$, $P<0.05$. The group with psychopathy performed significantly worse than the healthy control group during the 4-second delayed recognition, $F(2,96)=5.93$, $P<0.01$. Finally, both patient groups performed significantly worse than the healthy control group in the 12-second delayed recognition, $F(2,96)=6.86$, $P<0.01$, (Figure 5.33). For the significant post hoc comparisons $P$ was <0.01.

Regarding attention, RVP total hits were correlated significantly with DMS correct responses, similarly to the ASPD analysis. There was a significant group x covariate interaction, $F(3,85)=7.90$, $P<0.001$. Using MLM to control for RVP hits as before, resulted in a significantly better model, $\Delta Chi^2=48.13$, $\Delta df=5$, $P<0.001$, with RVP hits random at task condition and group levels. Parameter estimates suggested that only the group without psychopathy performed significantly worse than the healthy control group, *beta*=-0.51, *SE*=0.20, $P<0.05$, but no further significant group differences were detected.

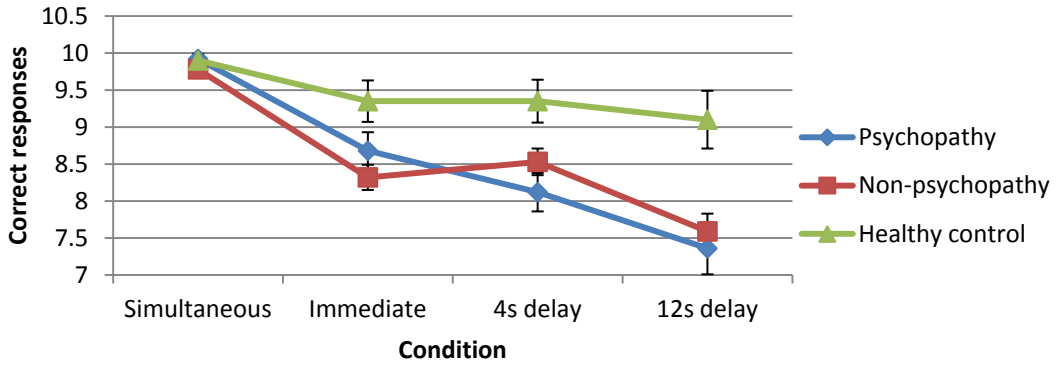



*Figure 5.33.* Number of correct responses on the Delayed Matching to Sample task for patients with and without psychopathy and healthy controls. Patients with psychopathy performed worse than controls overall and in both 4s and 12s recognition. Patients without psychopathy performed worse than controls overall also and during immediate and 12s delayed recognition. There were no further significant group differences.

5.2.2.5.2.1.3 Spatial Span task

Deviations from normality were observed and the K-S test was significant for all groups. A Kruskal-Wallis test revealed a significant main effect of group, $Chi^2$=9.44, *df*=2, *P*<0.01. This was confirmed by one-way ANOVA, *F*(2,93)=6.10, *P*<0.01. Post hoc analysis suggested that only the group without psychopathy performed significantly worse than the healthy control group, *P*<0.01, but there were no further differences (Figure 5.34). The effect remained following exclusion of individuals with ASPD from the group without psychopathy, $Chi^2$=7.55, *df*=2, , and, *F*(2,71)=4.68, *Ps*<0.05.

Regarding attention, the correlation between RVP total hits and SSP span length was significant, *rho*=0.27, *P*<0.05, as was a group x covariate interaction, *F*(3,83)=4.98, *P*<0.01. Using MLM, adding RVP total hits to the model (fixed effects) resulted in a significant improvement, $\Delta Chi^2$=4.42, $\Delta df$=1, *P*<0.05. However, the model did not converge when RVP hits were declared random at Level 2. The group without psychopathy performed worse than both controls, *beta*=-0.93, *SE*=0.36, *P*<0.01, and the group with psychopathy, *beta*=-0.83, *SE*=0.33, *P*<0.01. There were no further significant differences.

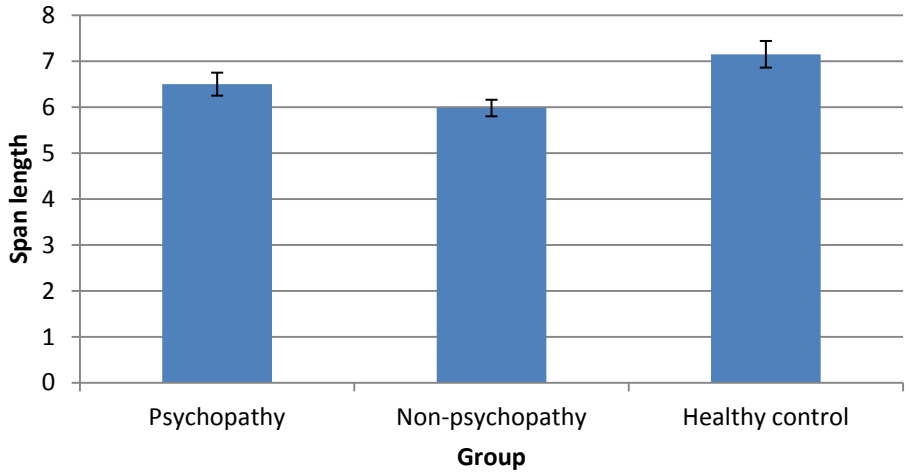



*Figure 5.34.* Span length on the Spatial Span task for patients with and without psychopathy and healthy controls. Patients without psychopathy performed significantly worse than controls but there were no further significant differences.

*5.2.2.5.2.2 Verbal Memory*

Regarding correct recalls, some deviations from normality were observed. A Kruskal-Wallis test did not reveal a significant main effect of group, $Chi^2$=1.85, *df*=2, (Figure 5.35).

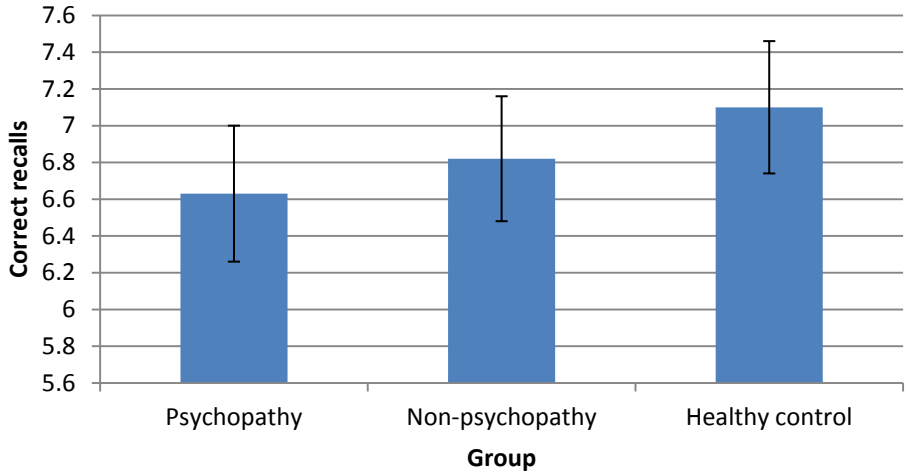



*Figure 5.35.* Number of correct recalls on the Verbal Recognition Memory task for patients with and without psychopathy and healthy controls. There were no significant group differences**.**

Regarding correct recognitions, one multivariate outlier was removed from the group without psychopathy. Deviations from normality were present. Levene's test was significant for the immediate condition. A mixed ANOVA with immediate/ delayed recognition as within-groups factor did not reveal a main effect of task condition, *Trace*=0.01, $F(1,56)=0.56$, a main effect of group, $F(2,56)=1.07$, or a group x condition interaction, *Trace*=0.01, $F(2,56)=0.33$, (Figure 5.36).

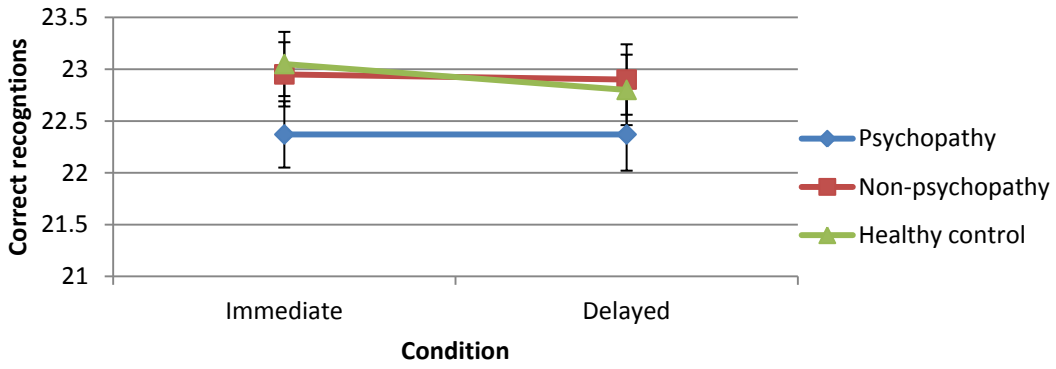



*Figure 5.36.* Number of correct recognitions on the Verbal Recognition Memory task for patients with and without psychopathy and healthy controls. There were no significant group differences.

*5.2.2.5.2.3 Working memory*

Three univariate outliers from the group without psychopathy and two from the group with psychopathy were removed. Some deviations from normality were present. Box's test and Levene's test for the 4- and 6-box conditions were significant. A mixed ANOVA revealed a significant main effect of difficulty, *Trace*=0.62, $F(2,88)=70.56$, $P<0.001$. There was also a significant main effect of group, $F(2,89)=3.2$, $P<0.05$, but no significant group x difficulty interaction, *Trace*=0.08, $F(4,178)=1.77$. Post hoc analyses indicated that the group without psychopathy performed significantly worse than the healthy control group only, $P<0.05$, but there were no further significant group differences (Figure 5.37). The apparent deficit in the group without psychopathy may be attributed to presence of ASPD, as removal of those individuals resulted in a non-significant effect, $F(2,67)=1.72$.

Regarding attention, RVP total hits were correlated significantly with SWM errors, as before. There was a significant group x covariate interaction, $F(3,81)=2.93$,

$P<0.05$. Using MLM, adding RVP hits alongside group membership (no convergence with RVP hits as random predictor at group level), resulted in a significantly better model, $\Delta Chi^2=4.03$, $\Delta df=1$, $P<0.05$, indicating no significant group differences. .

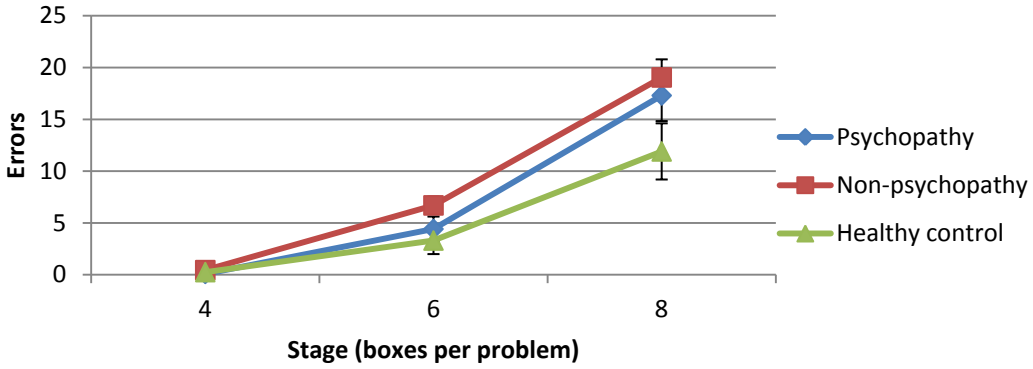



*Figure 5.37.* Number of errors on the Spatial Working Memory task for patients with and without psychopathy and healthy controls. Patients without psychopathy performed significantly worse than controls overall. There were no further significant group differences.

#### *5.2.2.5.3 Summary of memory*

No deficits were observed in psychopathy in visual STM (PAL, DMS, SSP) after controlling for sustained attention, verbal memory (VRM), and WM (SWM), in line with expectations. However, offenders without psychopathy exhibited impairments in visual STM (SSP) and WM (SWM) compared to controls. Although the latter may be attributed to presence of ASPD in the group, the groups are confounded and therefore it is difficult to delineate the effects of different diagnoses.

### 5.2.2.6 Visual perception

#### *5.2.2.6.1 ASPD*

Two univariate outliers were removed from the non-ASPD and three from the ASPD group. The K-S test was significant for all groups. Levene's test was also significant. A Kruskal-Wallis test revealed a significant main effect of group, $Chi^2$=8.85, *df*=2, *P*<0.05. This was confirmed by one-way ANOVA, *F*(2,92)=4.78,*P*<0.05. Post hoc analysis indicated that the ASPD group performed worse than the healthy control group, *P*<0.01, whereas there were no further significant group differences (Figure 5.38). The groups were comparable in both correct and error reaction times, using Kruskal-Wallis tests, $Chi^2$=2.51, *df*=2, and $Chi^2$=0.71, *df*=2, respectively.

Regarding attention, RVP hits were not correlated significantly with MTS correct responses, *rho*=0.16. Furthermore, excluding individuals with psychopathy from the ASPD group yielded comparable results.

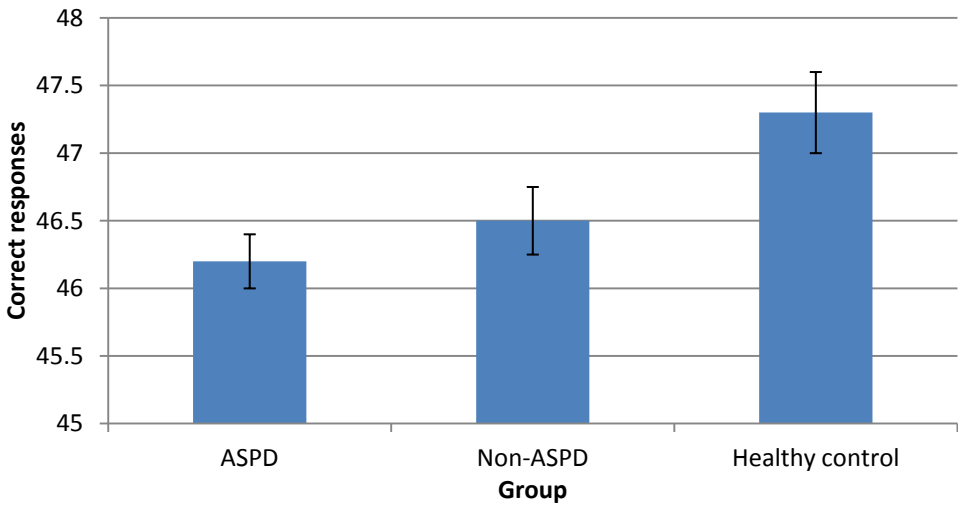



*Figure 5.38.* Number of correct responses on the Matching to Sample Visual Search task for patients with and without Antisocial Personality Disorder (ASPD and non-ASPD) and healthy controls. The ASPD group performed significantly worse than healthy controls. There were no further significant groups differences.

#### *5.2.2.6.2 Psychopathy*

One univariate outlier was removed from the group without psychopathy. The K-S test was significant for all groups. Levene's test was also significant. A Kruskal-Wallis test revealed a significant main effect of group, $Chi^2$=9.97,*df*=2, *P*<0.01. This was confirmed by one-way ANOVA, *F*(2,93)=4.86, *P*<0.05. Post hoc analysis indicated that individuals with psychopathy performed worse than healthy controls, *P*<0.01, but there were no further significant group differences (Figure 5.39). The groups were comparable in both correct and error reaction times using Kruskal-Wallis tests, $Chi^2$=2.46, *df*=2, and $Chi^2$=0.18, *df*=2, respectively. Regarding attention, RVP hits were not correlated with MTS correct responses, *rho*=0.16.

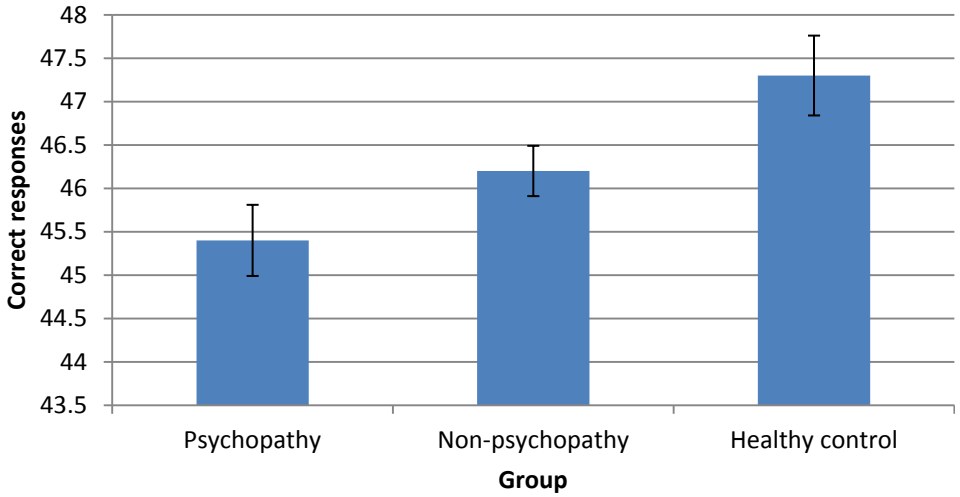



*Figure 5.39.* Number of correct responses on the Matching to Sample Visual Search task for patients with and without psychopathy and healthy controls. Patients with psychopathy performed significantly worse than healthy controls**.** There were no further significant groups differences.

#### *5.2.2.6.3 Summary of visual perception*

Results indicated a visual perception deficit in ASPD which was contrary to expectations. Furthermore, this was not detected in offenders with other personality disorders when the groups were compared to controls. Results also suggested a visual perception deficit in psychopathy compared to healthy control, as hypothesised. This was not observed in individuals without psychopathy.

#### 5.2.2.7 Overall summary of cognitive deficits in ASPD and psychopathy

##### *5.2.2.7.1 ASPD*

Results indicated a range of deficits in ASPD (Table 5.3). Individuals with ASPD performed worse than healthy controls on tasks of motor regulation, planning, and cognitive flexibility, thus supporting the first hypothesis. Individuals with ASPD also performed worse than controls in tasks of sustained attention and visual STM as hypothesised. Further results suggesting impairments in WM, and visual perception did not support the hypotheses whereas no deficit was identified in verbal memory, in line with expectations. Excluding individuals with psychopathy from the ASPD group yielded broadly comparable results with the exception of the deficit in reversal errors which became marginally significant using non-parametric testing.

Some of the identified deficits appeared present in ASPD but not offenders with other personality disorders. These were in tasks of motor regulation, response reversal, risk-taking during most ambiguous odds, WM, and visual perception, while the group with ASPD experienced greater difficulty during visual STM and decision-making tasks than individuals with other personality disorders when the groups were compared to healthy controls. However, in other functions, including planning, visual STM span, and attentional set-shifting, the deficits appeared present in the patient group as a whole.

##### *5.2.2.7.2 Psychopathy*

A range of deficits were also identified in psychopathy (Table 5.3). Individuals with psychopathy performed worse than healthy controls on tasks of motor regulation and some but not all of the examined aspects of cognitive flexibility. Thus, the first hypothesis was partly supported. Individuals with psychopathy also performed worse than controls in visual perception and planning as hypothesised. Although memory impairments were not observed in psychopathy, in line with the second hypothesis, deficits in sustained attention were detected which was contrary to expectations.

The observed deficits in response reversal and visual perception appeared present in psychopathy but not offenders without psychopathy. However, individuals with psychopathy showed similar difficulties in motor regulation and comparable

planning times to offenders without psychopathy against healthy controls. On the other hand, when compared to controls, individuals with psychopathy demonstrated fewer impairments than those without psychopathy who experienced additional difficulties in planning, attentional set-shifting, and visual STM.

Finally, offenders without psychopathy demonstrated deficits in attentional set-shifting, decision-making, visual STM span, and WM which were not present in offenders with psychopathy but it is possible that these may be attributable to a diagnosis of ASPD, particularly in the cases of attentional set-shifting and WM.

Table 5.3. *Summary of impairments on the CANTAB in the antisocial personality*

| **Function** | **ASPD** | **Psychopathy** |
|---|---|---|
| Motor regulation (AGN) | | |
| Cognitive flexibility: | | |
| - Response reversal (IED) | | |
| - Attentional set-shifting (IED) | | |
| - Decision-making (CGT) | Quality of decision-making | |
| | Risk-taking (6:4 odds)† | |
| Planning (SOC) | | Most challenging problems |
| Sustained attention (RVP) | | |
| Visual STM (PAL, DMS, SSP) | DMS & SSP | |
| Verbal memory (VRM) | | |
| WM (SWM) | | |
| Visual perception (MTS) | | |

*Note.* Shaded areas indicate a deficit in cognitive task performance with darker shading reflecting impairment in the antisocial personality not encountered in other personality disorders; ASPD=Antisocial Personality Disorder; AGN=Affective Go/NoGo; IED= Intra/Extra-Dimensional Set Shifting; CGT=Cambridge Gambling Task; SOC=Stocking of Cambridge; RVP=Rapid Visual Processing; STM=Short-term memory; PAL=Paired Associates Learning; DMS=Delayed Matching to Sample; SSP=Spatial Span; VRM=Verbal Recognition Memory; WM=Working memory; SWM=Spatial Working Memory; MTS=Matching to Sample Visual Search.

†=Emerged when individuals with psychopathy were removed from the ASPD group.

# 6 THE RELATIONSHIP BETWEEN NEUROPSYCHOLOGICAL DEFICITS AND PROGRESS IN TREATMENT

Following the examination of cognitive deficits in the antisocial personality, the second aim of the present project was to explore the relationship of cognitive ability to treatment progress in individuals with antisocial personality, once again operationalised as ASPD or psychopathy. Progress in treatment was measured with the PRS. Different deficits in ASPD and psychopathy led to dissimilar expectations regarding cognitive function and progress in treatment for each.

## 6.1 Method

Participants, materials, apparatus, and procedure were explained in previous chapters. Details on the design and data analysis are outlined below.

### 6.1.2 Design

The design was longitudinal correlational, as it examined the relationship of neuropsychological deficits with progress in treatment over time. The potential effect of intellectual functioning as a mediating variable was also evaluated and controlled for where possible. Once again, this design did not allow the establishment of cause-and-effect relationships (McBurney & White, 2007).

### 6.1.3 Statistical data analysis

Screening for assumptions was desrcibed in previous chapters. Multivariate MLM was conducted in order to investigate whether neuropsychological performance predicted progress in treatment in antisocial individuals. As antisocial personality was operationalised using DSM (antisocial personality disorder) or PCL-R criteria (psychopathy), analyses for each of these were conducted in parallel. The models had the same structure as the PRS analysis. However, because the purpose here was to examine the predictive effect of neuropsychological variables on the PRS scores and change over time, the baseline model consisted of the intercept and time since

admission as fixed predictor (Model 0). The subsequent analysis for each CANTAB test was conducted in the following manner:

1. ***Initial analysis:*** CANTAB outcome measures and their interactions were added successively to the baselines model as Level-2 predictors (first declared fixed and then random at each model). Although overall CANTAB outcome measures were preferred, where these were not available main analyses were conducted using data from the most difficult task condition with accompanying sensitivity analyses for less challenging ones. As these supplementary analyses were lengthy, details were reported when results were not comparable to the main findings for economy. For the same reason, where models with random effects converged, only the most parsimonious models – those with fewest degrees of freedom – were reported.
2. ***ASPD-only:*** The analysis on ASPD was repeated for a smaller ASPD group following exclusion of those individuals who also had high scores on psychopathy.
3. ***Controlling for IQ:*** This was achieved by successively adding the relevant terms (IQ, IQ x CANTAB, IQ x Time, IQ x CANTAB x Time) to the final model and examining improvements from the best model. It was anticipated that the small sample sizes may not permit correction for IQ in many cases, as convergence was likely to be difficult to achieve with the additional parameters (Tabachnick & Fidell, 2007) particularly where random effects were present.

#### 6.1.3.1 Software and statistical significance

MLM was conducted using MLwiN software, v.2.24 (Rasbash, Charlton, Browne, Healy, & Cameron, 2011). SPSS software, v.17.0 (SPSS Inc, 2009) was employed for all other analyses with an *alpha* level set at 0.05 for all statistical tests unless otherwise specified.

## 6.2 Results

Because the available sample sizes were not the same for all cognitive tests and outcome measures, it was often necessary to calculate the models' parameter estimates

and fit indices anew. Consistent with findings from the PRS validation above, Level 2 variance in PRS Part B scores was not significant on any occasion at the intercepts-only stage, therefore, the pseudo-$R^2$ was not calculated in these cases.

### 6.2.1 Sustained attention

It was hypothesised that impairments in sustained attention would predict negative progress in ASPD but would be unrelated to progress in treatment in psychopathy. As before, RVP hits were the outcome measure of interest.

#### 6.2.1.1 ASPD

A total of 43 patients contributed 89 measurements for Part A and 101 for Part B. RVP hits and RVP hits x time since admission interaction were added as predictors without leading to significant improvements (Table 6.1). RVP hits were not a significant predictor of PRS scores or their change over time. Results regarding the relationship of PRS scores to performance on the RVP task were comparable after excluding the individuals with psychopathy. Finally, the models did not improve significantly for either ASPD group, $\Delta Chi^2$=16.38 and 20.73, $\Delta df$=12, after controlling for IQ.

Table 6.1. *Summary of results of multivariate MLM for RVP hits as predictor of PRS scores and growth curves in ASPD*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β*(SE)* | | | $R_i^2$ | β*(SE)* | | |
| | | | | Time(*e-3*) | RVP | RVP x time(*e-3*) | | Time (*e-3*) | RVP | RVP x time(*e-3*) |
| **0** | 28.72(2)*** | <0 | 6% | -1.06(1.39) | | | 25% | 2.59(0.46)*** | | |
| **1-Best** | 57.31(2)*** | 1% | <0 | -0.87(1.41) | | | | 2.06(0.70)** | | |
| **2** | 0.18(2) | nil | nil | -0.86(1.39) | 0.04(0.11) | | nil | 2.06(0.70)** | 0.001(0.014) | |
| **3** | 2.56(4) | nil | nil | 0.61(3.94) | 0.07(0.13) | -0.14(0.34) | nil | -1.00(2.16) | -0.03(0.03) | 0.24(0.16) |
| **4** | -52.50(6)*** | <0 | 2% | 0.77(3.90) | 0.07(0.13) | -0.18(0.34) | <0 | 0.46(1.27) | -0.03(0.03) | 0.19(0.11) |

*Note.* MLM=Multilevel Modelling; RVP=Rapid Visual Processing; PRS=Progress Rating Schedule; ASPD=Antisocial Personality Disorder; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms: random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

#### 6.2.1.2 Psychopathy

The RVP task assessed sustained attention with total number of hits as outcome measure. A total of 22 patients contributed 47 measurements for Part A and 52 for Part B. RVP hits and RVP hits x time since admission interaction were added as predictors without significantly improving the model (Table 6.2). RVP hits were not a significant predictor of PRS scores or their change over time in psychopathy. Controlling for IQ did not lead to a significant improvement, $\Delta Chi^2$=-4.49, $\Delta df$=12.

#### 6.2.1.3 Summary of sustained attention

Performance on sustained attention was not related to progress in treatment in either ASPD or psychopathy. Although this was in line with expectations for the latter, the opposite had been hypothesised for ASPD.

Table 6.2. *Summary of results of multivariate MLM for RVP hits as predictor of PRS scores and growth curves in psychopathy*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β(*SE*) | | | $R_i^2$ | β(*SE*) | | |
| | | | | Time(*e-3*) | RVP | RVP x time(*e-3*) | | Time (*e-3*) | RVP | RVP x time(*e-3*) |
| **0** | 32.42(2)*** | <0 | 43% | -4.34(1.14)*** | | | 34% | 2.23(0.44)*** | | |
| **1** | nil(2) | nil | nil | -4.34(1.14)*** | | | nil | 2.23(0.44)*** | | |
| **2-Best** | 21.13(2)*** | 2% | <0 | -4.27(1.16)*** | | | 62% | 1.76(0.83)* | | |
| **3** | 0.72(2) | 3% | nil | -4.30(1.16)*** | -0.09(0.13) | | nil | 1.79(0.82)* | -0.01(0.02) | |
| **4** | 3.46(4) | 4% | 7% | -1.24(2.21) | 0.004(0.144) | -0.38(0.23) | nil | 2.69(1.82) | 0.004(0.029) | -0.08(0.15) |
| **5** | 3.46(5) | 4% | 7% | -1.24(2.21) | 0.004(0.144) | -0.38(0.23) | nil | 2.69(1.82) | 0.004(0.029) | -0.08(0.15) |
| **6** | 3.45(5) | 4% | 7% | -1.24(2.21) | 0.004(0.144) | -0.38(0.23) | nil | 2.69(1.82) | 0.004(0.029) | -0.08(0.15) |

*Note.* MLM=Multilevel Modelling; RVP=Rapid Visual Processing; PRS=Progress Rating Schedule; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms: random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

#### 6.2.4.2 Motor regulation

It was hypothesised that impairment in motor regulation would predict negative progress in treatment in ASPD and psychopathy. As before, AGN commission errors was the outcome measure of interest.

#### 6.2.2.1 ASPD

A total of 34 patients contributed 66 measurements for Part A and 35 patients supplied 75 measurements for Part B. The addition of AGN commission errors and their interaction with time since admission improved the model significantly but predicted PRS Part B scores only (Table 6.3). More AGN commission errors were associated with a higher initial Part B scores (intercept) but also predicted a faster decline of these scores over time in ASPD. Results regarding the relationship of PRS scores to performance on the AGN task were comparable for the ASPD-only analysis.

Controlling for IQ improved the models significantly for both the larger and the smaller ASPD sample, $\Delta Chi^2$=15.62 and 26.72 respectively, $\Delta df$=8, *Ps*<0.05. AGN commission errors no longer predicted PRS scores reliably in the larger ASPD sample. In the ASPD-only group, AGN commission errors showed a positive relationship with PRS Part B scores over time, *beta*=2.14*e*-3, *SE*=0.67*e*-3, *P*<0.05, which effect was attenuated for higher IQ scores (IQ x AGN x Time interaction), *beta*=-0.03*e*-3, *SE*=0.01*e*-3, *P*<0.001.

Table 6.3. *Summary of results of multivariate MLM for AGN commission errors as predictor of PRS scores and growth curves in ASPD*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β(*SE*) | | | $R_i^2$ | β(*SE*) | | |
| | | | | **Time(*e-3*)** | **AGN** | **AGN x time(*e-3*)** | | **Time (*e-3*)** | **AGN** | **AGN x time(*e-3*)** |
| **0** | 17.96(2)*** | <0 | 23% | -3.71(1.59)* | | | 14% | 2.14(0.57)*** | | |
| **1** | 0.29(2) | <0 | nil | -3.75(1.59)* | -0.009(0.079) | | nil | 2.10(0.57)*** | -0.008(0.015) | |
| **2-Best** | 11.25(4)** | <0 | 1% | -1.92(4.20) | 0.02(0.09) | -0.17(0.34) | 20% | 6.41(1.34)*** | 0.05(0.02)* | -0.39(0.11)*** |

*Note.* MLM=Multilevel Modelling; AGN=Affective Go/NoGo; PRS=Progress Rating Schedule; ASPD=Antisocial Personality Disorder; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first. ****P*<0.001; ***P*<0.01; **P*<0.05.

#### 6.2.2.2 Psychopathy

A total of 20 patients contributed 41 measurements for Part A and 46 for Part B. AGN commissions and their interaction with time since admission failed to improve the model significantly (Table 6.4). AGN commissions were not a significant predictor of PRS scores or their change over time in psychopathy. The model did not improve significantly following adjustment for the effect of IQ, $\Delta Chi^2$=-0.44, $\Delta df$=12.

#### 6.2.2.3 Summary of motor regulation

Results did not support the hypotheses for either ASPD or psychopathy overall as impairments in motor regulation did not predict progress in treatment in either. Impaired motor regulation was associated with higher initial but then declining PRS Part B scores over time in ASPD prior to controlling for IQ only.

Table 6.4. *Summary of results of multivariate MLM for AGN commission errors as predictor of PRS scores and growth curves in psychopathy*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β(*SE*) | | | $R_i^2$ | β(*SE*) | | |
| | | | | Time(*e-3*) | AGN | AGN x time(*e-3*) | | Time (*e-3*) | AGN | AGN x time(*e-3*) |
| **0** | 26.73(2)*** | <0 | 53% | -5.31(1.18)*** | | | 26% | 2.00(0.49)*** | | |
| **1** | nil(3) | nil | nil | -5.31(1.18)*** | | | nil | 2.00(0.49)*** | | |
| **2-Best** | 21.26(2)*** | 1% | <0 | -5.28(1.20)*** | | | 68% | 1.64(0.94) | | |
| **3** | 0.13(2) | nil | nil | -5.29(1.20)*** | -0.02(0.10) | | nil | 1.64(0.94) | -0.004(0.013) | |
| **4** | 0.70(4) | 1% | 1% | -8.26(4.72) | -0.05(0.11) | 0.26(0.40) | <0 | 2.36(2.16) | 0.005(0.028) | -0.06(0.17) |
| **5** | 0.70(5) | 1% | 1% | -8.26(4.72) | -0.05(0.11) | 0.26(0.40) | <0 | 2.36(2.16) | 0.005(0.028) | -0.06(0.17) |
| **6** | 0.70(5) | 1% | 1% | -8.26(4.72) | -0.05(0.11) | 0.26(0.40) | <0 | 2.36(2.16) | 0.005(0.028) | -0.06(0.17) |

*Note.* MLM=Multilevel Modelling; AGN=Affective Go/NoGo; PRS=Progress Rating Schedule; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms: random at Level 2.

****P*<0.001; ***P*<0.01; **P*<0.05.

### 6.2.3 Planning

It was hypothesised that impairments in planning would predict negative progress in treatment in ASPD and psychopathy. The SOC evaluated planning with mean moves to solution, initial and subsequent thinking times for 5-move problems as repeated outcome measures. Sensitivity analysis with these outcome measures for 4-move problems was also conducted.

#### 6.2.3.1 ASPD

There were slightly different datasets for each outcome measure due to outlier exclusions. A total of 45-47 patients contributed 88-93 measurements for PRS Part A and 101-107 for Part B. Only the addition of subsequent thinking time improved the model significantly which emerged as a significant predictor of Part B scores only (Table 6.5). No other SOC outcome measure or interaction with time led to significant improvements. Overall, higher subsequent thinking times were associated with lower overall PRS Part B scores in ASPD. However, performance on the SOC as measured by perfect solutions, number of moves to solution and initial thinking time did not predict PRS scores or their change over time in ASPD. Sensitivity analyses revealed comparable results for mean moves to solution and initial thinking time whereas results were not significant for subsequent thinking time. The best models for all SOC outcome measures were comparable for the ASPD-only analysis.

After controlling for IQ, the models involving perfect solutions improved significantly for the larger ASPD group, $\Delta Chi^2$=29.06, $\Delta df$=12, $P$<0.01, but not the ASPD-only group, $\Delta Chi^2$=17.18, $\Delta df$=12. This did not alter the results concerning the effect of perfect solutions, however. Furthermore, the models did not improve significantly for number of moves, $\Delta Chi^2$=6.7 and 10.18, $\Delta df$=12, initial thinking time (convergence was not achieved for the ASPD-only group), $\Delta Chi^2$=12.78, $\Delta df$=12. Finally, although the models improved for subsequent thinking time for both ASPD and ASPD-only groups, $\Delta Chi^2$=24.16 and 24.72 respectively, $\Delta df$=10, $P$<0.01, the effect of subsequent thinking time itself was no longer significant. Sensitivity analyses yielded comparable results.

Table 6.5. *Summary of results of multivariate MLM for SOC outcomes as predictor of PRS growth curves in ASPD*

| Model | $\Delta Chi^2(\Delta df)$ | Part A $R_i^2$ | Part A $R_i^2$ | Part A β*(SE)* Time(*e-3*) | Part A β*(SE)* SOC measure | Part A β*(SE)* SOC measure x time | Part B $R_i^2$ | Part B β*(SE)* Time (*e-3*) | Part B β*(SE)* SOC measure | Part B β*(SE)* SOC measure x time |
|---|---|---|---|---|---|---|---|---|---|---|
| **0** | 21.15(2)*** | <0 | 4% | -0.39(1.48) | | | 19% | 2.07(0.42)*** | | |
| **1-Best** | 49.65(2)*** | 4% | <0 | -0.14(1.51) | | | 55% | 1.71(0.67)* | | |
| Perfect solutions | | | | | | (e-3) | | | | (e-3) |
| **2** | 0.31(2) | 1% | nil | -0.08(1.51) | 0.16(0.28) | | nil | 1.71(0.67)* | 0.004(0.040) | |
| **3** | 0.31(4) | 1% | nil | 0.60(10.36) | 0.17(0.37) | -0.08(1.29) | nil | 1.68(3.89) | 0.004(0.080) | 0.004(0.481) |
| Mean moves to solution | | | | | | (e-3) | | | | (e-3) |
| **2** | 0.64(2) | 2% | nil | -0.17(1.51) | -0.20(0.33) | | nil | 1.71(0.67)* | 0.02(0.05) | |
| **3** | 3.49(4) | <0 | 3% | -5.89(8.89) | -0.36(0.42) | 0.85(1.33) | nil | 7.36(3.57)* | 0.14(0.09) | -0.80(0.50) |
| Initial thinking time | | | | | (e-4) | (e-7) | | | (e-4) | (e-7) |
| **0** | 23.51(2)*** | <0 | 5% | -0.72(1.47) | | | 19% | 2.12(0.42)*** | | |
| **1-Best** | 50.10(2)*** | 3% | <0 | -0.50(1.50) | | | 55% | 1.72(0.67)* | | |
| **2** | 5.12(2) | 14% | nil | -0.04(1.50) | 1.76(0.87)* | | nil | 1.72(0.67)* | -0.14(0.12) | |
| **3** | 6.60(4) | 15% | 1% | 1.64(2.26) | 2.64(1.14) | -4.04(3.41) | <0 | 1.53(1.03) | -0.19(0.23) | 0.32(1.30) |
| Subsequent thinking time | | | | | (e-4) | (e-6) | | | (e-4) | (e-6) |
| **0** | 22.61(2)*** | <0 | 9% | -1.53(1.51) | | | 18% | 2.20(0.46)*** | | |
| **2-Best** | 12.11(2)** | 9% | nil | -1.57(1.51) | 9.19(5.53) | | 1% | 2.23(0.45)*** | -2.97(1.01)** | |
| **3** | 5.98(2) | 1% | 1% | 0.10(1.97) | 14.12(6.79)* | -2.00(1.69) | 6% | 3.11(0.57)*** | 0.20(1.63) | 1.25(0.51)* |
| **4** | 7.12(3) | 10% | 8% | 0.60(2.03) | 14.38(6.67)* | -3.31(2.19) | 6% | 3.11(0.57)*** | 0.20(1.63) | 1.25(0.51)* |
| **5** | 5.98(4) | 1% | 1% | -1.03(1.97) | 14.12(6.79)* | -2.00(1.69) | 6% | 3.11(0.57)*** | 0.20(1.63) | 1.25(0.51)* |

*Note.* MLM=Multilevel Modelling; SOC=Stockings of Cambridge; PRS=Progress Rating Schedule; ASPD=Antisocial Personality Disorder; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms: random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

#### 6.2.3.2 Psychopathy

There were different datasets for each outcome measure due to outlier exclusions. A total of 23-24 patients contributed 47-49 measurements for PRS Part A and 52-54 for Part B. Time since admission improved the intercepts-only model significantly as a fixed predictor (baseline) in all cases (Table 6.6). Perfect solutions did not predict PRS scores or their change over time. However, results were more complex for mean moves to solution and thinking times.

Regarding mean moves to solution, the addition of the interaction between mean moves to solution with time since admission as a fixed predictor resulted in a significant improvement to the model. There was a positive association between mean moves to solution and PRS Part A and B scores over time for 5-move problems. On the other hand, sensitivity analysis with data for 4-move problems was contradictory. Here, mean moves to solution were associated with lower PRS Part A and B scores over time. The model including 4-move problems had slightly better fit than that with 5-move problems, $\Delta Chi^2$=3.02, with the same degrees of freedom.

In connection with initial thinking time, the model improved significantly with the addition of initial thinking time for 5-move problems and its interaction with time since admission as fixed predictors. Results suggested a positive association between initial thinking time and PRS Part B scores over time, possibly mediating the effect of time since admission. Sensitivity analysis supported this. However, there was also a positive association between initial thinking time for 4-move problems and starting Part A scores (intercept) and an additional negative association between initial thinking time for 4-move problems and starting Part B scores (intercepts).

Regarding subsequent thinking time, data from 5-move problems led to significant improvements to the model. Results indicated a negative association between subsequent thinking times and initial PRS Part B scores (intercept) but they showed a positive association with Part B scores over time, possibly mediating the effect of time since admission. Subsequent thinking time was not associated with PRS Part A scores. Sensitivity analysis did not replicate these results. Here, subsequent thinking time was not associated with Part B scores while it showed a negative association with Part A scores over time. However, the 5-move problem model provided a better fit for the data, $\Delta Chi^2$=12.32, $\Delta df$=2, $P$<0.01.

After controlling for IQ, there were no significant improvements for perfect solutions, $\Delta Chi^2$=12.78, $\Delta df$=12, mean moves to solution (5-move), $\Delta Chi^2$=5.81, initial thinking times (5-move), $\Delta Chi^2$=9.43, and subsequent thinking times, $\Delta Chi^2$=0.26, $\Delta df$=8. Results were comparable for 4-move problems.

#### 6.2.3.3 Summary of planning

Results did not support the hypotheses for either ASPD or psychopathy. Regarding the former, impairments in planning did not predict progress in treatment and longer thinking times were associated with lower overall Part B scores prior to controlling for IQ only.

In connection with psychopathy, planning was associated with change in PRS scores over time. Requiring more moves to solve less difficult problems predicted a decline in PRS scores whereas the opposite was observed for most difficult problems. In addition, thinking for longer prior to and during problem-solving was associated with higher Part B scores over time whereas longer thinking times during less difficult problems predicted a decline in Part A scores during admission. Apart from progress over time, results suggested that shorter planning times for less difficult problems also predicted higher initial Part A scores in this population. On the other hand, shorter planning times for less difficult problems and longer thinking times while carrying out solutions to the most difficult problems predicted lower initial Part B scores.

Table 6.6. *Summary of results of multivariate MLM for SOC outcomes as predictor of PRS growth curves in psychopathy*

| Model | $\Delta Chi^2(\Delta df)$ | Part A $R_j^2$ | Part A $R_i^2$ | Part A β(SE) Time(*e-3*) | Part A β(SE) SOC measure | Part A β(SE) SOC measure x time | Part B $R_i^2$ | Part B β(SE) Time (*e-3*) | Part B β(SE) SOC measure | Part B β(SE) SOC measure x time |
|---|---|---|---|---|---|---|---|---|---|---|
| Perfect solutions | | | | | | (e-3) | | | | (e-3) |
| **0-Best** | 31.16(2)*** | <0 | 41% | -4.24(1.24)*** | | | 29% | 1.79(0.35)*** | | |
| **1** | nil(1) | nil | nil | -4.24(1.24)*** | | | nil | 1.79(0.35)*** | | |
| **2** | 0.98(2) | nil | nil | -4.25(1.24)*** | -0.08(0.49) | | 1% | 1.77(0.35)*** | 0.07(0.07) | |
| **3** | 1.66(4) | nil | 1% | -10.45(15.26) | -0.21(0.56) | 0.72(1.78) | 4% | -1.48(3.99) | -0.001(0.108) | 0.38(0.46) |
| **4** | 1.66(5) | nil | 1% | -10.45(15.26) | -0.21(0.56) | 0.72(1.78) | 4% | -1.48(3.99) | -0.001(0.108) | 0.38(0.46) |
| **5** | 9.72(5) | 2% | <0 | -12.34(15.56) | -0.23(0.56) | 0.94(1.81) | 42% | -1.81(4.88) | 0.02(0.10) | 0.35(0.57) |
| Mean moves to solution | | | | | | (e-3) | | | | (e-3) |
| **0** | 25.55(2)*** | <0 | 40% | -4.05(1.27)** | | | 29% | 1.69(0.35)*** | | |
| **1** | nil(1) | nil | nil | -4.05(1.27)** | | | nil | 1.69(0.35)*** | | |
| 5-move problems | | | | | | | | | | |
| **2** | 0.70(2) | nil | nil | -4.04(1.27)** | -0.02(0.53) | | <0 | 1.71(0.35)*** | 0.07(0.09) | |
| **3-Best** | 9.95(4)* | <0 | 12% | -21.94(8.89)* | -0.58(0.59) | 2.56(1.27)* | 16% | -5.63(2.39)* | -0.18(0.12) | 1.05(0.34)** |
| **4** | nil(1) | nil | nil | -21.94(8.89)* | -0.58(0.59) | 2.56(1.27)* | nil | -5.63(2.39)* | -0.18(0.12) | 1.05(0.34)** |
| 4-move problems | | | | | | | | | | |
| **2** | 2.62(2) | 9% | nil | -3.87(1.26)** | -0.84(0.70) | | nil | 1.78(0.35)*** | -0.16(0.11) | |
| **3-Best** | 11.34(4)* | <0 | 30% | 23.04(9.67)* | 0.16(0.83) | -5.35(1.87)** | 9% | 7.98(2.80)** | 0.10(0.16) | -1.24(0.54)* |
| **4** | nil(1) | nil | nil | 23.04(9.67)* | 0.16(0.83) | -5.35(1.87)** | nil | 7.98(2.80)** | 0.10(0.16) | -1.24(0.54)* |
| Initial thinking time 5-move problems | | | | | (e-4) | (e-7) | | | (e-4) | (e-7) |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| **0** | 31.16(2)*** | <0 | 41% | -4.24(1.24)*** | | | 29% | 1.79(0.35)*** | | |
| **1** | nil(1) | nil | nil | -4.24(1.24)*** | | | nil | 1.79(0.35)*** | | |
| **2** | 4.51(2) | 23% | <0 | -4.27(1.24)*** | 2.25(1.01)* | | nil | 1.79(0.35)*** | 0.06(0.16) | |
| **3-Best** | 13.45(4)*** | 22% | 1% | -5.31(2.12)* | 1.95(1.12) | 1.10(1.90) | 23% | 0.31(0.55) | 0.35(0.20) | 0.16(0.05)** |
| **4** | nil(5) | nil | nil | -5.31(2.12)* | 1.95(1.12) | 1.10(1.90) | nil | 0.31(0.55) | 0.35(0.20) | 0.16(0.05)** |
| **5** | nil(5) | nil | nil | -5.31(2.12)* | 1.95(1.12) | 1.10(1.90) | nil | 0.31(0.55) | 0.35(0.20) | 0.16(0.05)** |
| | | | | | | | | | | |
| 4-move problems | | | | | (e-4) | (e-6) | | | (e-4) | (e-6) |
| **0** | 30.26(2)*** | <0 | 46% | -4.68(1.23)*** | | | 30% | 1.66(0.33)*** | | |
| **1** | nil(1) | nil | nil | -4.68(1.23)*** | | | nil | 1.66(0.33)*** | | |
| **2** | 8.76(2)* | 19% | nil | -4.76(1.23)*** | 3.62(1.81)* | | nil | 1.73(0.33)*** | -0.46(0.24) | |
| **3** | 8.09(2)* | 2% | 8% | -1.12(2.44) | -0.90(0.36)* | -0.62(0.37) | 11% | 0.77(0.64) | -0.90(0.36)* | 0.17(0.10) |
| **4** | nil(1) | nil | nil | -1.12(2.44) | -0.90(0.36)* | -0.62(0.37) | nil | 0.77(0.64) | -0.90(0.36)* | 0.17(0.10) |
| **5-Best** | 10.78(1)* | 2% | <0 | -1.22(2.50) | 5.03(1.99)* | -0.62(0.38) | 51% | -0.70(0.70) | -0.78(0.30)* | 0.29(0.14)* |
| | | | | | | | | | | |
| Subsequent thinking time | | | | | | | | | | |
| **0** | 30.26(2)*** | <0 | 46% | -4.68(1.23)*** | | | 30% | 1.66(0.33)*** | | |
| **1** | nil(1) | nil | nil | -4.68(1.23)*** | | | nil | 1.66(0.33)*** | | |
| | | | | | | | | | | |
| 5-move problems | | | | | (e-4) | (e-6) | | | (e-4) | (e-6) |
| **2** | 6.47(2)* | 24% | <0 | -4.31(1.25)*** | 17.78(7.83)* | | nil | 1.81(0.35)*** | -1.21(1.22) | |
| **3** | 13.78(2)*** | <0 | 2% | -5.67(2.52)* | 15.06(8.68) | 1.10(1.78) | 33% | -0.32(0.59) | -5.14(1.54)*** | 1.71(0.42)*** |
| **4** | nil(1) | nil | nil | -5.67(2.52)* | 15.06(8.68) | 1.10(1.78) | nil | -0.32(0.59) | -5.14(1.54)*** | 1.71(0.42)*** |
| **5-Best** | 7.68(1)** | 4% | <0 | -6.03(2.58)* | 14.91(8.65) | 1.31(1.82) | 31% | -0.47(0.58) | -4.44(1.45)** | 1.61(0.64)* |
| | | | | | | | | | | |
| 4-move problems | | | | | (e-4) | (e-6) | | | (e-4) | (e-6) |
| **2** | 2.82(2) | <0 | 1% | -4.22(1.24)*** | -1.08(4.01) | | 1% | 1.83(0.34)*** | -0.87(0.50) | |
| **3-Best** | 9.69(4)** | <0 | 23% | -2.64(1.27)* | 4.37(4.56) | -1.77(0.64)** | 1% | 1.90(0.40)*** | -0.63(0.87) | -0.08(0.22) |
| **4** | nil(1) | nil | nil | -2.64(1.27)* | 4.37(4.56) | -1.77(0.64)** | nil | 1.90(0.40)*** | -0.63(0.87) | -0.08(0.22) |
| **5** | 0.14(1) | nil | nil | -2.65(1.27)* | 4.30(4.55) | -1.75(0.65)** | 3% | 1.81(0.41)*** | -0.76(0.87) | 0.03(0.25) |

*Note.* MLM=Multilevel Modelling; SOC=Stockings of Cambridge; PRS=Progress Rating Schedule; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms: random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

### 6.2.4 Cognitive flexibility

It was hypothesised that impairments in cognitive flexibility (attentional set-shifting, response reversal, and decision-making) would be associated with negative progress in both ASPD and psychopathy. As before, two CANTAB tests assessed aspects of cognitive flexibility: IED (response reversal and attentional set-shifting) and CGT (complex decision-making) with the same outcome measures.

#### 6.2.4.1 ASPD

##### *6.2.4.1.1 Intra/Extra-Dimensional Set Shifting task*

There were slightly different datasets for each outcome measure due to outlier exclusions. A total of 46-48 patients contributed 94-97 measurements for PRS Part A and 107-111 for Part B. The model improved significantly when time since admission was declared random for Part B in both reversal and EDS errors. Neither IED outcome measures as single predictors nor their interaction with time since admission led to significant improvements (Table 6.7). Overall, performance on the IED did not predict PRS scores or their change over time in ASPD.

Regarding the ASPD-only analysis, there was a significantly improved Model 2 for reversal errors, $\Delta Chi^2$=15.95, $\Delta df$=2, $P$<0.001, suggesting a negative relationship between reversal errors and Part B scores overall, *beta* =-0.06, *SE*=0.03, $P$<0.05. Other results regarding the relationship between reversal and EDS errors with PRS scores were comparable to the previous analysis with the larger ASPD group.

After controlling for IQ, convergence was achieved without the 3-way interaction for the models involving reversal errors in the larger ASPD group. This occurred by further excluding the IQ x Time interaction for the ASPD-only group. Neither suggested significant improvements to the respective models, $\Delta Chi^2$=6.04 and 3.84, $\Delta df$=10 and 6. Regarding EDS errors, improvements were also not significant, $\Delta Chi^2$=-10 and -0.45, $\Delta df$=12 and 8, for the larger ASPD and ASPD-only group respectively.

Table 6.7. *Summary of results of multivariate MLM for IED outcomes as predictor of PRS growth curves in ASPD*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β(*SE*) | | | $R_i^2$ | β(*SE*) | | |
| | | | | Time(*e-3*) | IED | IED x time(*e-3*) | | Time (*e-3*) | IED | IED x time(*e-3*) |
| Reversal errors | | | | | | | | | | |
| **0** | 30.65(2)*** | <0 | 6% | -0.92(1.40) | | | 25% | 2.59(0.44)*** | | |
| **1-Best** | 63.46(2)*** | 2% | <0 | -0.71(1.41) | | | 59% | 1.99(0.69)** | | |
| **2** | 3.26(2) | 4% | nil | -0.62(1.42) | 0.33(0.31) | | nil | 1.92(0.69)** | -0.06(0.04) | |
| **3** | 1.14(4) | 4% | nil | -3.74(5.86) | 0.20(0.39) | 0.84(1.54) | nil | 2.85(2.58) | -0.03(0.09) | -0.24(0.64) |
| **4** | 1.14(5) | 4% | nil | -3.74(5.86) | 0.20(0.39) | 0.84(1.54) | nil | 2.85(2.58) | -0.03(0.09) | -0.24(0.64) |
| EDS errors | | | | | | | | | | |
| **0** | 30.13(2)*** | <0 | 6% | -0.96(1.43) | | | 25% | 2.61(0.45)*** | | |
| **1-Best** | 61.25(2)*** | 1% | <0 | -0.74(1.44) | | | 59% | 2.03(0.69)** | | |
| **2** | 2.33(2) | 1% | nil | -0.74(1.44) | 0.02(0.05) | | nil | 2.01(0.69)** | -0.01(0.01) | |
| **3** | 3.38(4) | <0 | 3% | -2.81(2.40) | -0.01(0.05) | 0.12(0.12) | nil | 1.71(1.33) | -0.01(0.01) | 0.02(0.06) |
| **4** | 5.59(5) | <0 | 25 % | -2.33(2.17) | 0.01(0.05) | -0.02(0.14) | <0 | 1.73(1.32) | -0.01(0.01) | 0.02(0.06) |
| **5** | 3.38(5) | <0 | 3% | -2.81(2.40) | -0.01(0.05) | 0.12(0.12) | nil | 1.71(1.33) | -0.01(0.01) | 0.02(0.06) |

*Note.* MLM=Multilevel Modelling; IED=Intra/Extra-Dimensional set shifting; EDS=Extra-dimensional shift; PRS=Progress Rating Schedule; ASPD=Antisocial Personality Disorder; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms: random at Level 2.

****P*<0.001; ***P*<0.01; **P*<0.05.

#### *6.2.4.1.2 Cambridge Gambling Task*

A total of 10 patients contributed 19 measurements for each PRS Part. Time since admission alone did not significantly improve the intercepts-only model but made a significant contribution in conjunction with CGT risk-taking. However, except risk-taking, the remaining CGT outcomes and their interaction with time since admission did not improve the model significantly. These included quality of decision-making and delay aversion. An overview of results can be seen in Table 6.8. Although higher CGT risk-taking was associated with lower initial PRS Part A scores (intercept), it showed a positive relationship with Part A scores over time. The remaining CGT outcomes were not significant predictors of PRS scores or their change over time in ASPD. Analyses for the ASPD-only group were not conducted as the sample size decreased to $n$=4 when individuals with psychopathy were removed.

Regarding the effect of IQ, convergence was achieved for decision-making only when the 3-way interaction term was omitted and no random effects were defined. Although the model improved significantly, $\Delta Chi^2$=38.84, $\Delta df$=10, $P$<0.001, decision-making did not become a significant predictor of PRS scores. Regarding risk-taking and delay aversion, the models also improved after controlling for IQ, $\Delta Chi^2$=36.46 and 30.77, $\Delta df$=8 and 12, respectively, $Ps$<0.001. Both outcomes became significant predictors of Part B scores. Risk-taking showed a positive relationship with Part B over time, *beta*=0.24, *SE*=0.06, but this was attenuated when IQ was higher, *beta*=-2.45e-3, *SE*=0.65e-3, *Ps*<0.001. Higher delay aversion predicted higher initial Part B scores, *beta*=23.66, *SE*=7.54, which was attenuated when IQ was also higher, *beta*=-0.30, *SE*=0.09, *Ps*<0.01. In addition, higher delay aversion predicted a faster decline of Part B scores over time, *beta*=-0.21, *SE*=0.05, again attenuated when IQ was higher, *beta*=2.66e-3, *SE*=0.69, *Ps*<0.001.

Table 6.8. *Summary of results of multivariate MLM for CGT outcomes as predictor of PRS scores and growth in ASPD*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β*(SE)* | | | $R_i^2$ | β*(SE)* | | |
| | | | | **Time(*e-3*)** | **CGT** | **CGT x time(*e-3*)** | | **Time (*e-3*)** | **CGT** | **CGT x time(*e-3*)** |
| **0** | 3.78(2) | <0 | 47% | -7.33(2.96)* | | | 18% | 0.66(0.54) | | |
| **1** | -2.84(5) | <0 | 35% | -5.92(3.13) | | | | -0.22(0.82) | | |
| Quality of decision-making | | | | | | | | | | |
| **2** | 7.51(4) | 11% | nil | -7.40(2.95)* | -15.15(14.29) | | nil | 0.64(0.53) | -2.15(1.73) | |
| **3** | 11.94(6) | 1% | 38% | 28.78(16.82) | -5.89(15.44) | -41.85(19.08)* | 2% | 1.71(3.85) | -1.86(1.00) | -1.24(4.36) |
| **4** | 11.94(7) | 1% | 38% | 28.78(16.82) | -5.89(15.44) | -41.85(19.08)* | 2% | 1.71(3.85) | -1.86(1.00) | -1.24(4.36) |
| **5** | 11.94(7) | 1% | 38% | 28.78(16.82) | -5.89(15.44) | -41.85(19.08)* | 2% | 1.71(3.85) | -1.86(1.00) | -1.24(4.36) |
| Risk-taking | | | | | | | | | | |
| **2** | 6.68(4) | <0 | 47% | -7.28(2.92)* | -26.78(16.58) | | 19% | 0.65(0.54) | 2.28(2.21) | |
| **3-Best** | 13.37(6)* | nil | 71% | -62.62(18.93)*** | -39.72(16.09)* | 87.09(29.81)** | 19% | 1.84(4.55) | 2.57(2.46) | -1.90(7.15) |
| **4** | nil(1) | nil | nil | -62.62(18.93)*** | -39.72(16.09)* | 87.09(29.81)** | nil | 1.84(4.55) | 2.57(2.46) | -1.90(7.15) |
| **5** | -6.16(2)* | nil | <0 | -63.98(19.43)*** | -40.19(16.17)* | 90.41(30.57)** | <0 | 3.27(6.51) | 3.07(2.63) | -5.35(10.13) |
| Delay aversion | | | | | | | | | | |
| **2** | 4.45(4) | <0 | 47% | -7.22(2.94)* | 6.94(6.99) | | 18% | 0.66(0.54) | -0.08(0.91) | |
| **3** | 8.11(6) | <0 | 66% | -12.49(4.11)** | 2.79(7.61) | 24.17(15.91) | 32% | 1.58(0.77)* | 0.68(1.05) | -4.33(3.02) |
| **4** | 8.11(7) | <0 | 66% | -12.49(4.11)** | 2.79(7.61) | 24.17(15.91) | 32% | 1.58(0.77)* | 0.68(1.05) | -4.33(3.02) |
| **5** | 8.11(7) | <0 | 66% | -12.49(4.11)** | 2.79(7.61) | 24.17(15.91) | 32% | 1.58(0.77)* | 0.68(1.05) | -4.33(3.02) |

*Note.* MLM=Multilevel Modelling; CGT=Cambridge Gambling Task; PRS=Progress Rating Schedule; ASPD=Antisocial Personality Disorder; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms: random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

#### 6.2.4.2 Psychopathy

##### *6.2.4.2.1 Intra/Extra-Dimensional Set Shifting task*

There were slightly different datasets for each outcome measure due to outlier exclusions. A total of 23-25 patients contributed 49-53 measurements for PRS Part A and 57 for Part B. In some instances, fixed parameters for factors involving EDS errors reached significance indicating a negative association between PRS scores and EDS errors. Nevertheless, this did not reflect an overall improvement in the models. In fact, neither IED outcome measures as single predictors nor their interaction with time since admission lead to significant improvements (Table 6.9). Overall, performance on the IED as measured by reversal and EDS errors did not predict PRS scores or their change over time in psychopathy. Furthermore, the model for reversal and EDS errors did not improve significantly following adjustment for the effect of IQ, $\Delta Chi^2$=-6.02 and -6.52 respectively, $\Delta df$=12.

Table 6.9. *Summary of results of multivariate MLM for IED outcomes as predictor of PRS growth curves in psychopathy*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β(*SE*) | | | $R_i^2$ | β(*SE*) | | |
| | | | | Time(*e-3*) | IED | IED x time(*e-3*) | | Time (*e-3*) | IED | IED x time(*e-3*) |
| Reversal errors | | | | | | | | | | |
| **0** | 32.54(2)*** | <0 | 41% | -4.26(1.18)*** | | | 33% | 2.23(0.42)*** | | |
| **1** | nil(3) | nil | nil | -4.26(1.18)*** | | | nil | 2.23(0.42)*** | | |
| **2-Best** | 23.73(2)*** | 2% | <0 | -4.17(1.20)*** | | | 62% | 1.69(0.81)* | | |
| **3** | 0.10(2) | nil | nil | -4.17(1.20)*** | -0.06(0.39) | | nil | 1.68(0.81)* | -0.01(0.05) | |
| **4** | 4.27(4) | <0 | 7% | -13.27(6.00)* | -0.27(0.44) | 2.31(1.51) | <0 | -3.10(2.94) | -0.17(0.11) | 1.20(0.71) |
| **5** | 4.27(5) | <0 | 7% | -13.27(6.00)* | -0.27(0.44) | 2.31(1.51) | <0 | -3.10(2.94) | -0.17(0.11) | 1.20(0.71) |
| **6** | 4.27(5) | <0 | 7% | -13.27(6.00)* | -0.27(0.44) | 2.31(1.51) | <0 | -3.10(2.94) | -0.17(0.11) | 1.20(0.71) |
| EDS errors | | | | | | | | | | |
| **0** | 31.02(2)*** | <0 | 4% | -4.29(1.21)*** | | | 33% | 2.24(0.44)*** | | |
| **1** | nil(3) | nil | nil | -4.29(1.21)*** | | | nil | 2.24(0.44)*** | | |
| **2-Best** | 20.89(2)*** | 2% | <0 | -4.22(1.23)*** | | | 62% | 1.75(0.82)* | | |
| **3** | 3.64(2) | 5% | 1% | -4.21(1.22)*** | 0.07(0.07) | | <0 | 1.75(0.83)* | -0.01(0.01) | |
| **4** | 5.72(4) | 5% | 2% | -3.60(1.87) | 0.08(0.08) | -0.04(0.10) | <0 | 0.51(1.19) | -0.03(0.01)* | 0.10(0.07) |
| **5** | 11.86(6) | 7% | 30% | -3.03(1.61) | 0.12(0.07) | -0.32(0.16)* | <0 | 0.20(1.30) | -0.03(0.01)* | 0.09(0.07) |
| **6** | 5.72(6) | 5% | 2% | -3.60(1.87) | 0.08(0.08) | -0.04(0.10) | <0 | 0.51(1.19) | -0.03(0.01)* | 0.10(0.07) |

*Note.* MLM=Multilevel Modelling; IED=Intra/Extra-Dimensional set shifting; EDS=Extra-dimensional shift; PRS=Progress Rating Schedule; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms: random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

#### *6.2.4.2.2 Cambridge Gambling Task*

A total of 7 patients contributed 16 measurements for each PRS Part. The multivariate model did not converge at intercepts-only. As a result, two separate model sets were defined for PRS Parts A and B for analysis but with the same Level 1 and 2 configuration.

CGT outcomes including quality of decision-making, risk-taking, and delay avoidance and their interactions with time were not significant predictors of either PRS Part A or B scores in the final models (Table 6.10 and Table 6.11). Overall, CGT performance was not a significant predictor of PRS scores or their change over time in psychopathy. The models did not improve significantly following adjustment for IQ (for Part B the model involving risk-taking converged only after omitting the 3-way interaction), $\Delta Chi^2$=1.48 to 10.95, $\Delta df$=6.

### 6.2.4.3 Summary of cognitive flexibility

Findings provided limited support for the hypothesis on ASPD and no support for the hypothesis on psychopathy. In connection with ASPD, although a relationship between PRS scores and attentional set-shifting [IED] was not detected, two aspects of decision-making (risk-taking & delay aversion) and response reversal were associated with PRS scores. Contrary to expectations, however, more risk-raking predicted lower initial but improving Part A scores over time (mediated by IQ) as well as positive change in Part B scores (but only after controlling for IQ). On the other hand, delay aversion was associated with higher initial but declining Part B scores over time after controlling for IQ, in line with the hypothesis. Impairments in response reversal predicted lower Part B scores overall. Regarding psychopathy, cognitive flexibility was not associated with progress in treatment, contrary to expectations.

Table 6.10. *Summary of results of MLM for CGT outcomes as predictor of PRS Part A growth curves in psychopathy*

| **Model** | **$\Delta Chi^2(\Delta df)$** | **$R_i^2$** | **β*(SE)*** | | |
|---|---|---|---|---|---|
| | | | **Time(*e-3*)** | **CGT** | **CGT x time(*e-3*)** |
| **0-Best** | 4.82(1)* | 46% | -3.82 (1.45)** | | |
| Quality of decision-making | | | | | |
| **1** | 0.73(1) | nil | -3.87(1.45)** | -11.09(12.77) | |
| **2** | 1.63(2) | 11% | 6.37(10.41) | -7.22(13.52) | -12.55(12.62) |
| Risk-taking | | | | | |
| **1** | 3.36(1) | nil | -4.17(1.45)** | -23.22(11.2)* | |
| **2** | 3.36(2) | nil | -3.96(5.68) | -23.09(11.78) | -0.43(11.39) |
| **3** | 4.79(3) | 56% | 2.22(9.71) | -21.73(11.47) | -15.42(19.16) |
| Delay aversion | | | | | |
| **1** | 0.04(1) | nil | -3.83(1.45)** | 1.64(8.10) | |
| **2** | 1.65(2) | 15% | -7.47(3.05)* | -1.43(8.34) | 9.43(7.17) |
| **3** | 1.65(3) | 15% | -7.47(3.05)* | -1.43(8.34) | 9.43(7.17) |

*Note.* MLM=Multilevel Modelling; CGT=Cambridge Gambling Task; PRS=Progress Rating Schedule; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_i^2$=Change in Level 1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms: random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

Table 6.11. *Summary of results of MLM for CGT outcomes as predictor of PRS Part B growth curves in psychopathy*

| **Model** | **$\Delta Chi^2(\Delta df)$** | **$R_i^2$** | **β*(SE)*** | | |
|---|---|---|---|---|---|
| | | | **Time (*e-3*)** | **CGT** | **CGT x time(*e-3*)** |
| **0** | 22.01(1)*** | 87% | 2.81(0.34)*** | | |
| **1-Best** | 5.95(1)** | ~100%[1] | 3.24(1.19)** | | |
| Quality of decision-making | | | | | |
| **2** | 3.55(1) | -[1] | 3.58(1.77)** | -1.84(0.90)* | |
| **3** | 4.40(2) | -[1] | 11.36(9.74) | -1.58(0.96) | -9.08(9.07) |
| Risk-taking | | | | | |
| **2** | nil(1) | -[1] | 3.19(1.19)** | 0.12(1.13) | |
| **3** | 2.16(2) | -[1] | -6.13(6.16) | -0.59(0.97) | 16.67(10.62) |
| **4** | 1.85(3) | -[1] | -2.24(4.77) | -0.56(0.99) | 9.99(8.92) |
| Delay aversion | | | | | |
| **2** | 1.28(1) | -[1] | 3.44(1.30)** | 0.73(0.56) | |
| **3** | 2.06(2) | -[1] | 5.02(2.16)* | 0.86(0.57) | -5.88(6.51) |

*Note.* MLM=Multilevel Modelling; CGT=Cambridge Gambling Task; PRS=Progress Rating Schedule; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_i^2$=Change in Level 1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

[1]The Level 2 variance of model 1 was not significantly different from 0.

Highlighted terms: random at Level 2.

****P*<0.001; ***P*<0.01; **P*<0.05.

### 6.2.5 Memory

Visual STM, verbal memory, and WM were examined. For ASPD, it was hypothesised that only impairments in visual STM would predict negative progress in treatment. For psychopathy, on the other hand, no memory sub-type was expected to predict progress. Visual STM was investigated using the PAL, DMS, and SSP tests. The VRM examined verbal memory and SWM assessed WM. The outcome measures of interest were the same as before.

#### 6.2.5.1 ASPD

##### *6.2.5.1.1 Visual short-term memory*

###### *6.2.5.1.1.1 Paired Associates Learning*

A total of 49 patients contributed 97 measurements for PRS Part A and 111 for Part B. The addition of PAL completed stages and their interaction with time since admission did not improve the model significantly. On the other hand, PAL errors led to a significant improvement but predicted PRS Part A scores only. The association was positive for initial Part A scores (intercept) but negative for their change over time (Table 6.12). Results were comparable using the adjusted number of errors.

Excluding individuals with psychopathy from the ASPD group yielded comparable results regarding the relationship of PRS scores to PAL completed stages and overall errors. In connection with adjusted errors, however, Model 2 was significantly improved, $\Delta Chi^2$=17.74, $\Delta df$=2, $P$<0.001, and became the best model for this PAL outcome measure indicating a negative relationship between overall adjusted errors and Part B scores only, *beta*=-3.74*e*-3, *SE*=1.83*e*-3, $P$<0.05. The remaining results were comparable to the previous analysis with the larger ASPD group.

After controlling for IQ, the model of completed stages improved significantly for the larger ASPD group but convergence was only achieved without the three-way interaction of completed stages with IQ and time since admission, $\Delta Chi^2$=27.61, $\Delta df$=10, $P$<0.01. Completing more stages on the PAL predicted lower initial Part B scores only, *beta*=-3.17, *SE*=0.94, $P$<0.001, but this effect was less pronounced for higher IQ scores, *beta*=0.04, *SE*=0.01, $P$<0.01. Convergence was not achieved for the ASPD-only group.

Regarding PAL errors, the model for the larger ASPD group did not converge. On the other hand, the model did not improve significantly for the ASPD-only group, $\Delta Chi^2$=13.65, $\Delta df$=8. Results were comparable for adjusted errors.

Table 6.12. *Summary of results of multivariate MLM for PAL outcomes as predictor of PRS growth curves in ASPD*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β(*SE*) | | | $R_i^2$ | β(*SE*) | | |
| | | | | **Time(*e-3*)** | **PAL measure** | **PAL measure x time(*e-3*)** | | **Time (*e-3*)** | **PAL measure** | **PAL measure x time(*e-3*)** |
| **0** | 29.63(2)*** | <0 | 7% | -1.20(1.40) | | | 23% | 2.50(0.43)*** | | |
| **1** | 68.11(2)*** | 1% | <0 | -0.99(1.42) | | | 61% | 1.74(0.69)* | | |
| Completed stages | | | | | | | | | | |
| **2** | 0.98(2) | 4% | nil | -1.03(1.43) | -0.89(0.90) | | nil | 1.73(0.69)* | 0.02(0.12) | |
| **3** | 5.11(4) | 2% | 4% | -25.88(17.78) | -1.61(1.02) | 3.27(2.34) | <0 | -14.19(9.98) | -0.25(0.20) | 2.05(1.28) |
| Overall errors | | | | | | | | | | |
| **2** | 3.77(2) | 3% | nil | -1.06(1.43) | 0.03(0.03) | | nil | 1.76(0.68)* | -0.007(0.004) | |
| **3** | 9.03(4) | 3% | 7% | 2.39(2.18) | 0.07(0.03)* | -0.14(0.07)* | nil | 2.49(0.91)** | 3e-5(661e-5) | -0.04(0.04) |
| **4** | 9.03(5) | 3% | 7% | 2.39(2.18) | 0.07(0.03)* | -0.14(0.07)* | nil | 2.49(0.91)** | 3e-5(661e-5) | -0.04(0.04) |
| **5-Best** | 11.3(5)* | 3% | 7% | 2.42(2.17) | 0.07(0.03)* | -0.14(0.07)* | 1% | 2.23(1.03)* | -3e-4(72e-4) | -0.04(0.06) |

*Note.* MLM=Multilevel Modelling; PAL=Paired Associates Learning; PRS=Progress Rating Schedule; ASPD=Antisocial Personality Disorder; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms-random at Level 2.

****P*<0.001; ***P*<0.01; **P*<0.05.

*6.2.5.1.1.2 Delayed Matching to Sample*

A total of 50 patients contributed 101 measurements for PRS Part A and 114 measurements for Part B. Neither overall correct responses nor their interaction with time since admission improved the model significantly. Details are presented in Table 6.13. Correct responses on the DMS were not a significant predictor of PRS scores or their change over time in ASPD. Furthermore, excluding individuals with psychopathy from the ASPD group yielded comparable results regarding the relationship of PRS scores to performance on the DMS task. Finally, the model did not converge when terms involving IQ were added.

Table 6.13. *Summary of results of multivariate MLM for DMS correct responses as predictor of PRS growth curves in ASPD*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β*(SE)* | | | $R_i^2$ | β*(SE)* | | |
| | | | | **Time(*e-3*)** | **DMS measure** | **DMS measure x time(*e-3*)** | | **Time (*e-3*)** | **DMS measure** | **DMS measure x time(*e-3*)** |
| **0** | 30.55(2)*** | <0 | 6% | -0.98(1.38) | | | 25% | 2.57(0.43)*** | | |
| **1-Best** | 63.5(2)*** | 1% | <0 | -0.78(1.39) | | | 58% | 2.09(0.69)** | | |
| **2** | 4.01(2) | 13% | <0 | -0.66(1.39) | 0.22(0.11) | | nil | 2.09(0.69)** | -0.02(0.01) | |
| **3** | 8.19(4) | 12% | <0 | -4.09(18.11) | 0.20(0.14) | 0.10(0.53) | <0 | 1.25(6.82) | -0.05(0.03) | 0.43(0.20) |

*Note.* MLM=Multilevel Modelling; DMS=Delayed Matching to Sample; PRS=Progress Rating Schedule; ASPD=Antisocial Personality Disorder; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms-random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

*6.2.5.1.1.3 Spatial Span task*

Outcome measure was spatial span. A total of 47 patients contributed 96 measurements for each PRS Part A and 110 measurements for Part B. Neither spatial span nor its interaction with time since admission improved the model significantly. Details are presented in Table 6.14. Spatial span was not a significant predictor of PRS scores or their change over time in ASPD.

Excluding individuals with psychopathy from the ASPD group resulted in a significantly improved Model 3, $\Delta Chi^2$=9.91, $\Delta df$=4, $P$<0.05, suggesting a negative adjustment to initial Part B scores by SSP span, *beta*=-0.22, *SE*=0.11, $P$<0.05, but a positive relationship over time (interaction term), *beta* =1.70*e*-3, *SE*=0.63*e*-3, $P$<0.01.

Controlling for IQ did not result in a significant improvement to the model for the larger ASPD group, $\Delta Chi^2$=15.25, $\Delta df$=12, while convergence was not achieved for the ASPD-only group.

Table 6.14. *Summary of results of multivariate MLM for SSP span as predictor of PRS growth curves in ASPD*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β*(SE)* | | | $R_i^2$ | β*(SE)* | | |
| | | | | **Time(*e-3*)** | **SSP span** | **SSP span x time(*e-3*)** | | **Time (*e-3*)** | **SSP span** | **SSP span x time(*e-3*)** |
| **0** | 30.78(2)*** | <0 | 6% | -0.96(1.39) | | | 25% | 2.60(0.44)*** | | |
| **1-Best** | 65.99(3)*** | 2% | <0 | -0.88(1.40) | | | 60% | 1.60(0.70)* | | |
| **2** | 1.41(2) | 7% | <0 | -0.80(1.40) | 0.37(0.35) | | nil | 1.67(0.70)* | 0.03(0.05) | |
| **3** | 8.62(4) | 7% | 1% | -5.74(8.74) | 0.37(0.43) | 0.81(1.44) | <0 | -9.29(3.83)* | -0.21(0.09)* | 1.76(0.60)** |
| **4** | -39.95(6)*** | nil | 3% | -6.76(8.70) | 0.34(0.44) | 0.94(1.43) | <0 | -9.46(2.46)*** | -0.25(0.10)* | 1.97(0.40)** |

*Note.* MLM=Multilevel Modelling; SSP=Spatial Span; PRS=Progress Rating Schedule; ASPD=Antisocial Personality Disorder; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms-random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

#### *6.2.5.1.2 Verbal memory*

There were different datasets for delayed recognition measures due to outlier exclusions. A total of 26-28 patients contributed 49-52 measurements for PRS Part A and 57-60 measurements for Part B. Both correct recalls and delayed recognitions improved the model significantly and both showed a negative association with initial PRS Part B scores (intercept). However, their association with change over time in Part B scores was positive (Table 6.15). Sensitivity analysis with immediate recognition replicated these findings.

Excluding individuals with psychopathy from the ASPD group resulted in mostly comparable findings regarding the relationship between VRM recall performance and PRS scores. However, the intercept of VRM recall for Part B was no longer significant in Models 3-4, *beta* =-0.17, *SE*=0.14, (Models 5-6 did not converge).

Regarding delayed recognition, although the improvements in model fit were comparable to those with the larger ASPD group, neither the intercept nor the interaction term in connection with VRM recognition were significant in the final Model 5, *beta* =-0.22, *SE*=0.28, and *beta* =0.61*e*-3, *SE*=1.62*e*-3, respectively (Model 6 did not converge). Sensitivity analysis with immediate recognition replicated these findings.

Controlling for IQ did not lead to a significant improvement for recall with either the larger or ASPD-only group (convergence for the latter was achieved when the 3-way interaction was omitted), $\Delta Chi^2$=8.57 and 9.58 respectively, $\Delta df$=8 and 6. There were comparable results for delayed recognition and sensitivity analysis with immediate recognition, although convergence for either ASPD group was only achieved without the 3-way and IQ x Time interactions.

Table 6.15. *Summary of results of multivariate MLM for VRM outcomes as predictor of PRS growth curves in ASPD*

| Model | $\Delta Chi^2(\Delta df)$ | Part A $R_j^2$ | Part A $R_i^2$ | Part A β(*SE*) Time(*e-3*) | Part A β(*SE*) VRM measure | Part A β(*SE*) VRM measure x time(*e-3*) | Part B $R_i^2$ | Part B β(*SE*) Time (*e-3*) | Part B β(*SE*) VRM measure | Part B β(*SE*) VRM measure x time(*e-3*) |
|---|---|---|---|---|---|---|---|---|---|---|
| Correct recalls | | | | | | | | | | |
| **0** | 14.83(2)*** | <0 | 33% | -4.72(1.73)** | | | 11% | 1.74(0.54)** | | |
| **1** | nil(1) | nil | nil | -4.72(1.73)** | | | nil | 1.74(0.54)** | | |
| **2** | 0.42(2) | 1% | nil | -4.73(1.73)** | -0.28(0.51) | | nil | 1.74(0.54)** | 0.03(0.09) | |
| **3** | 18.79(4)*** | nil | 2% | -5.34(8.93) | -0.34(0.56) | 0.08(1.44) | 34% | -9.40(2.39)*** | -0.29(0.11)** | 1.81(0.38)*** |
| **4** | nil(1) | nil | nil | -5.34(8.93) | -0.34(0.56) | 0.08(1.44) | nil | -9.40(2.39)*** | -0.29(0.11)** | 1.81(0.38)*** |
| **5-Best** | 18.53(1)*** | 1% | <0 | -3.68(9.00) | -0.32(0.56) | -0.18(1.45) | 39% | -8.94(4.01)* | -0.26(0.09)** | 1.58(0.60)** |
| **6** | -18.59(3)*** | <0 | 2% | -5.35(8.93) | -0.34(0.56) | 0.08(1.44) | <0 | -9.41(2.39)*** | -0.29(0.11)** | 1.81(0.38)** |
| Correct recognitions (delayed) | | | | | | | | | | |
| **0** | 14.40(2)*** | <0 | 33% | -4.68(1.75)** | | | 12% | 1.75(0.56)** | | |
| **1** | nil(3) | nil | nil | -4.68(1.75)** | | | nil | 1.75(0.56)** | | |
| **2** | 2.08(2) | 9% | 1% | -4.75(1.75)** | 0.82(0.56) | | nil | 1.76(0.56)** | 0.01(0.10) | |
| **3** | 28.97(4)*** | 8% | 1% | -9.58(45.91) | 0.75(0.63) | 0.21(2.00) | 48% | -65.28(11.10)*** | -0.47(0.12)*** | 2.93(0.49)*** |
| **4** | nil(1) | nil | nil | -9.58(45.91) | 0.75(0.63) | 0.21(2.00) | nil | -65.28(11.10)*** | -0.47(0.12)*** | 2.93(0.49)*** |
| **5-Best** | 13.10(1)*** | 1% | <0 | -4.92(46.27) | 0.77(0.63) | 0.01(0.20) | 29% | -64.38(18.88)*** | -0.45(0.12)*** | 2.85(0.82)*** |
| **6** | -17.15(3)*** | <0 | <0 | -5.34(46.03) | 0.77(0.63) | 0.03(2.01) | <0 | -60.06(11.83)*** | -0.44(0.12)*** | 2.71(0.52)*** |

*Note.* MLM=Multilevel Modelling; VRM=Verbal Recognition Memory; PRS=Progress Rating Schedule; ASPD=Antisocial Personality Disorder; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms-random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

#### *6.2.5.1.3 Working memory*

A total of 48 patients contributed 97 measurements for Part A and 111 measurements for Part B. The addition of SWM errors or their interaction with time did not lead to significant improvements. An overview of results can be seen in Table 6.16. SWM errors did not predict PRS scores or their change over time in ASPD reliably. Furthermore, the ASPD-only analysis yielded comparable results. Finally, controlling for IQ did not result in a significant improvement for either the larger ASPD or the ASPD-only group (convergence was achieved for the latter only when all interaction terms involving IQ were omitted), $\Delta Chi^2$=11.12 and 9.43, $\Delta df$=12 and 6.

Table 6.16. *Summary of results of multivariate MLM for SWM errors as predictor of PRS growth curves in ASPD*

| | | Part A | | | | | Part B | | |
|---|---|---|---|---|---|---|---|---|---|
| **Model** | **$\Delta Chi^2(\Delta df)$** | **$R_j^2$** | **$R_i^2$** | **β*(SE)*** | | | **$R_i^2$** | **β*(SE)*** | |
| | | | | **Time(*e-3*)** | **SWM errors** | **SWM errors x time(*e-5*)** | | **Time (*e-3*)** | **SWM errors** | **SWM errors x time(*e-5*)** |
| **0** | 30.72(2)*** | <0 | 6% | -0.95(1.38) | | | 25% | 2.58(0.44) | | |
| **1-Best** | 63.91(2)*** | 1% | <0 | -0.75(1.40) | | | 59% | 1.95(0.69)** | | |
| **2** | 0.35(2) | 1% | nil | -0.75(1.40) | -0.01(0.03) | | nil | 1.94(0.69)** | -0.001(0.004) | |
| **3** | 5.57(4) | 1% | nil | -1.36(3.46) | -0.02(0.03) | 0.02(0.11) | 1% | 4.92(1.37)*** | 0.01(0.01) | -0.11(0.05)* |
| **4** | 5.57(5) | 1% | nil | -1.36(3.46) | -0.02(0.03) | 0.02(0.11) | 1% | 4.92(1.37)*** | 0.01(0.01) | -0.11(0.05)* |

*Note.* MLM=Multilevel Modelling; SWM=Spatial Working Memory; PRS=Progress Rating Schedule; ASPD=Antisocial Personality Disorder; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms-random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

#### 6.2.5.2 Psychopathy

##### *6.2.5.2.1 Visual short-term memory*

###### *6.2.5.2.1.1 Paired Associates Learning*

A total of 25 patients contributed 51 measurements for PRS Part A and 56 measurements for Part B. Neither the addition of PAL outcomes measures nor their interaction with time since admission improved the model significantly (Table 6.17). Performance on the PAL was not a significant predictor of PRS scores or their change over time in psychopathy. Results were comparable using the adjusted number of errors. Furthermore, after controlling for IQ, there were no significant improvements for either completed stages or error, $\Delta Chi^2$=1.08 and 0.64, $\Delta df$=12, replicated by sensitivity analysis with adjusted errors.

Table 6.17. *Summary of results of multivariate MLM for PAL outcomes as predictor of PRS growth curves in psychopathy*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β(*SE*) | | | $R_i^2$ | β(*SE*) | | |
| | | | | Time(*e-3*) | PAL measure | PAL measure x time(*e-3*) | | Time (*e-3*) | PAL measure | PAL measure x time(*e-3*) |
| **0** | 32.09(2)*** | <0 | 46% | -4.61(1.14)*** | | | 32% | 2.12(0.41)*** | | |
| **1** | nil(1) | nil | nil | -4.61(1.14)*** | | | nil | 2.12(0.41)*** | | |
| **2-Best** | 28.18(2)*** | 2% | <0 | -4.56(1.16)*** | | | 68% | 1.20(0.80) | | |
| Completed stages | | | | | | | | | | |
| **3** | 1.81(2) | nil | nil | -4.56(1.16)*** | 0.54(1.54) | | nil | 1.18(0.81) | -0.29(0.21) | |
| **4** | 3.86(4) | <0 | 7% | -26.71(19.08) | -0.003(1.624) | 2.86(2.47) | nil | -13.03(14.44) | -0.51(0.30) | 1.83(1.85) |
| **5** | 3.86(5) | <0 | 7% | -26.71(19.08) | -0.003(1.624) | 2.86(2.47) | nil | -13.03(14.44) | -0.51(0.30) | 1.83(1.85) |
| **6** | 1.56(5) | <0 | 7% | -26.21(19.06) | 0.001(1.63) | 2.79(2.47) | <0 | -19.87(11.03) | -0.64(0.26)* | 2.74(1.42) |
| Overall errors | | | | | | | | | | |
| **3** | 0.69(2) | nil | nil | -4.57(1.16)*** | 0.01(0.04) | | nil | 1.26(0.79) | -0.04(0.05) | |
| **4** | 3.76(4) | <0 | 10% | -2.60(1.69) | 0.04(0.04) | -0.09(0.06) | <0 | 1.87(1.01) | 0.001(0.008) | -0.03(0.04) |
| **5** | 3.76(5) | <0 | 10% | -2.60(1.69) | 0.04(0.04) | -0.09(0.06) | <0 | 1.87(1.01) | 0.001(0.008) | -0.03(0.04) |

*Note.* MLM=Multilevel Modelling; PAL=Paired Associates Learning; PRS=Progress Rating Schedule; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms-random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

*6.2.5.2.1.2 Delayed Matching to Sample*

A total of 26 patients contributed 55 measurements for PRS Part A and 59 measurements for Part B. Overall DMS correct responses improved the model significantly and demonstrated a positive association with overall PRS Part A scores (intercept). Their interaction with time did not lead to significant improvements (Table 6.18). DMS correct responses predicted higher PRS Part A scores (intercepts) in psychopathy and this appeared stronger during immediate recognition and shorter retention delays. DMS correct responses were not a significant predictor of Part B scores or any change of PRS scores over time in psychopathy. Controlling for IQ did not result in a significant improvement to the model, $\Delta Chi^2$=3.04, $\Delta df$=10.

Table 6.18. *Summary of results of multivariate MLM for DMS correct responses as predictor of PRS growth curves in psychopathy*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β*(SE)* | | | $R_i^2$ | β*(SE)* | | |
| | | | | **Time(*e-3*)** | **DMS measure** | **DMS measure x time(*e-3*)** | | **Time (*e-3*)** | **DMS measure** | **DMS measure x time(*e-3*)** |
| **0** | 31.7(2)*** | <0 | 41% | -4.32(1.15)*** | | | 33% | 2.19(0.43)*** | | |
| **1** | nil(1) | nil | nil | -4.32(1.15)*** | | | nil | 2.19(0.43)*** | | |
| **2** | 22.62(2)*** | 2% | <0 | -4.23(1.17)*** | | | 62% | 1.85(0.83)* | | |
| **3-Best** | 6.12(2)* | 25% | 6% | -4.21(1.16)*** | 0.37(0.14)** | | nil | 1.87(0.83)* | -0.004(0.022) | |
| **4** | 1.26(2) | <0 | 7% | -8.48(19.97) | 0.35(0.17)* | 0.12(0.57) | <0 | -8.28(8.57) | -0.04(0.04) | 0.30(0.25) |
| **5** | 1.26(6) | <0 | 7% | -8.48(19.97) | 0.35(0.17)* | 0.12(0.57) | <0 | -8.28(8.57) | -0.04(0.04) | 0.30(0.25) |

*Note.* MLM=Multilevel Modelling; DMS=Delayed Matching to Sample; PRS=Progress Rating Schedule; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms-random at Level 2.

****P*<0.001; ***P*<0.01; **P*<0.05.

*6.2.5.2.1.3 Spatial Span task*

A total of 26 patients contributed 53 measurements for PRS Part A and 59 measurements for Part B. Neither spatial span nor its interaction with time since admission improved the model significantly (Table 6.19). Spatial span was not a significant predictor of PRS scores or their change over time in psychopathy. Furthermore, controlling for IQ did not result in an improved model, $\Delta Chi^2$=-2.21, $\Delta df$=12.

Table 6.19. *Summary of results of multivariate MLM for SSP span as predictor of PRS growth curves in psychopathy*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β*(SE)* | | | $R_i^2$ | β*(SE)* | | |
| | | | | **Time(*e-3*)** | **SSP span** | **SSP span x time(*e-3*)** | | **Time (*e-3*)** | **SSP span** | **SSP span x time(*e-3*)** |
| **0** | 28.16(2)*** | <0 | 43% | -4.77(1.26)*** | | | 25% | 1.99(0.44)*** | | |
| **1** | nil(1) | nil | nil | -4.77(1.26)*** | | | nil | 1.99(0.44)*** | | |
| **2-Best** | 26.77(2)*** | 2% | <0 | -4.71(1.28)*** | | | 62% | 1.35(0.84) | | |
| **3** | 0.93(2) | 4% | nil | -4.66(1.27)*** | 0.57(0.63) | | nil | 1.35(0.83) | 0.03(0.09) | |
| **4** | 2.20(4) | 2% | 4% | -11.96(8.56) | 0.36(0.68) | 1.13(1.33) | nil | -3.74(6.07) | -0.08(0.15) | 0.78(0.93) |
| **5** | 2.20(8) | 2% | 4% | -11.96(8.56) | 0.36(0.68) | 1.13(1.33) | nil | -3.74(6.07) | -0.08(0.15) | 0.78(0.93) |

*Note.* MLM=Multilevel Modelling; SSP=Spatial Span; PRS=Progress Rating Schedule; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms-random at Level 2.

****P*<0.001; ***P*<0.01; **P*<0.05.

#### *6.2.5.2.2 Verbal memory*

A total of 19 patients contributed 39 measurements for PRS Part A and 44 measurements for Part B. Correct recalls and delayed recognitions did not improve the model significantly and showed no association with PRS scores (Table 6.20). Sensitivity analysis with immediate recognition confirmed these findings. Correct VRM verbal recalls and recognitions were not significant predictors of PRS scores or their change over time. Furthermore, controlling for IQ did not result in an improved model for either recall or recognition, $\Delta Chi^2$=-2.53 and 14.74 respectively, $\Delta df$=12, which was replicated by sensitivity analysis.

Table 6.20. *Summary of results of multivariate MLM for VRM outcomes as predictor of PRS growth curves in psychopathy*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β(*SE*) | | | $R_i^2$ | β(*SE*) | | |
| | | | | Time(*e-3*) | VRM measure | VRM measure x time(*e-3*) | | Time (*e-3*) | VRM measure | VRM measure x time(*e-3*) |
| **0** | 29.46(2)*** | <0 | 50 % | -4.56(1.15)*** | | | 34% | 2.25(0.46)*** | | |
| **1** | nil(1) | nil | nil | -4.56(1.15)*** | | | nil | 2.25(0.46)*** | | |
| **2-Best** | 20.67(2)*** | 3% | <0 | -4.47(1.18)*** | | | 67% | 1.66(0.88) | | |
| Correct recalls | | | | | | | | | | |
| **3** | 1.31(2) | 2% | nil | -4.49(1.18)*** | 0.33(0.56) | | nil | 1.70(0.88) | -0.07(0.07) | |
| **4** | 2.74(4) | nil | 6% | -9.80(5.41) | 0.17(0.59) | 0.71(0.72) | 1% | -2.03(4.73) | -0.15(0.11) | 0.52(0.66) |
| **5** | 2.74(5) | nil | 6% | -9.80(5.41) | 0.17(0.59) | 0.71(0.72) | 1% | -2.03(4.73) | -0.15(0.11) | 0.52(0.66) |
| **6** | 2.74(5) | nil | 6% | -9.80(5.41) | 0.17(0.59) | 0.71(0.72) | 1% | -2.03(4.73) | -0.15(0.11) | 0.52(0.66) |
| Correct recognitions (delayed) | | | | | | | | | | |
| **3** | 4.93(2) | 24% | nil | -4.50(1.17)*** | 0.79(0.35)* | | <0 | 1.77(0.88)* | -0.06(0.05) | |
| **4** | 6.17(4) | 24% | nil | 1.50(22.55) | 0.82(0.39)* | -0.26(0.98) | <0 | -10.42(15.85) | -0.12(0.10) | 0.53(0.69) |
| **5** | 11.53(7) | 29% | nil | 5.48(33.65) | 0.89(0.38)* | -0.57(1.47) | 2% | -13.94(16.31) | -0.13(0.10) | 0.67(0.71) |

*Note.* MLM=Multilevel Modelling; VRM=Verbal Recognition Memory; PRS=Progress Rating Schedule; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms-random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

#### *6.2.5.2.3 Working memory*

A total of 26 patients contributed 54 measurements for Part A and 59 for Part B. Neither the addition of SWM errors nor their interaction with time since admission improved the model significantly (Table 6.21). SWM errors were not a significant predictor of PRS scores or their change over time in psychopathy. Furthermore, there was not a significant improvement to the model after controlling for IQ, $\Delta Chi^2$=10.4, $\Delta df$=12.

### 6.2.5.3 Summary of memory

Findings were mostly in line with expectations for both ASPD and psychopathy but there were exceptions. Regarding ASPD, results on visual STM and WM supported the hypotheses. WM was unrelated to progress in treatment and more errors during visual cued-recall/learning (PAL) predicted higher initial but then declining PRS scores (Parts A & B) over time (completing more stages showed the reverse effect on initial Part B scores after partially controlling for IQ). Furthermore, although better STM capacity (SSP) was associated with lower initial Part B scores, it predicted improvement over time, after patients high in psychopathy were excluded from the ASPD group. Contrary to expectations, however, performance on visual STM recognition (DMS) was unrelated to PRS scores and verbal memory predicted lower initial but improving Part B scores over time. The latter effect remained for verbal recall only when individuals with psychopathy were excluded from the ASPD group.

Regarding psychopathy, most results (visual STM [SSP & PAL], verbal, and WM) supported the hypothesis that memory would not predict progress in treatment. However, performance on the DMS showed a positive relationship with overall Part A scores.

Table 6.21. *Summary of results of multivariate MLM for SWM errors as predictor of PRS growth curves in psychopathy*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β(*SE*) | | | $R_i^2$ | β(*SE*) | | |
| | | | | Time(*e-3*) | SWM errors | SWM errors x time(*e-3*) | | Time (*e-3*) | SWM errors | SWM errors x time(*e-3*) |
| **0** | 32.88(2)*** | <0 | 41% | -4.22(1.16)*** | | | 32% | 2.22(0.42)*** | | |
| **1** | nil(1) | nil | nil | -4.22(1.16)*** | | | nil | 2.22(0.42)*** | | |
| **2-Best** | 25.07(2)*** | 2% | <0 | -4.13(1.18)*** | | | 62% | 1.60(0.81)* | | |
| **3** | 0.45(2) | 2% | nil | -4.13(1.18)*** | -0.03(0.04) | | nil | 1.59(0.81)* | 0.001(0.006) | |
| **4** | 3.05(4) | <0 | 7% | -0.38(3.47) | -0.003(0.049) | -0.15(0.13) | nil | 3.63(1.73)* | 0.01(0.01) | -0.09(0.07) |
| **5** | 3.05(5) | <0 | 7% | -0.38(3.47) | -0.003(0.049) | -0.15(0.13) | nil | 3.63(1.73)* | 0.01(0.01) | -0.09(0.07) |

*Note.* MLM=Multilevel Modelling; SWM=Spatial Working Memory; PRS=Progress Rating Schedule; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms-random at Level 2.

****P*<0.001; ***P*<0.01; **P*<0.05.

### 6.2.6 Visual perception

It was hypothesised that visual perception would not be associated with progress in treatment in ASPD but that impairments in this function would predict negative progress in treatment in psychopathy. The MTS task evaluated visual perception with total correct responses as outcome measure.

#### 6.2.6.1 ASPD

A total of 45 patients contributed 90 measurements for Part A and 103 for Part B. Neither MTS correct responses nor their interaction with time since admission were significant predictors of PRS scores (Table 6.22). For the ASPD-only analysis, convergence was achieved with time since admission declared random at Level 2 prior to adding components related to MTS performance. This led to a significant improvement, $\Delta Chi^2$=24.63, $\Delta df$=2, $P$<0.001. However, adding the components related to the MTS did not improve the model further. Finally, convergence was not achieved when the terms involving IQ were added.

#### 6.2.6.2 Psychopathy

A total of 25 patients contributed 52 measurements for Part A and 57 for Part B. Neither MTS correct responses not their interaction with time since admission were significant predictors of PRS scores (Table 6.23). While controlling for IQ, the model converged only after the 3-way interaction was omitted but there was no significant improvement, $\Delta Chi^2$=5.91, $\Delta df$=10.

#### 6.2.6.3 Summary of visual perception

Visual perception did not predict progress in treatment in either ASPD or psychopathy. This was as hypothesised for ASPD but contrary to expectations for psychopathy.

Table 6.22. *Summary of results of multivariate MLM for MTS correct responses as predictor of PRS scores and growth curves in ASPD*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β(*SE*) | | | $R_i^2$ | β(*SE*) | | |
| | | | | Time(*e-3*) | MTS | MTS x time(*e-3*) | | Time (*e-3*) | MTS | MTS x time(*e-3*) |
| **0** | 30.66(2)*** | <0 | 11% | -1.97(1.41) | | | 24% | 2.64(0.47)*** | | |
| **1** | nil(3) | nil | nil | -1.97(1.41) | | | nil | 2.64(0.47)*** | | |
| **2** | 1.21(2) | 1% | nil | -1.97(1.41) | 0.17(0.31) | | nil | 2.65(0.47)*** | 0.06(0.06) | |
| **3-Best** | 62.34(5)*** | 4% | <0 | -1.80(1.44) | 0.08(0.30) | | 59% | 1.53(0.73)* | -0.01(0.04) | |
| **4** | 1.32(2) | <0 | nil | 19.90(43.03) | 0.26(0.37) | -0.47(0.93) | <0 | -20.10(20.08) | -0.08(0.08) | 0.47(0.43) |

*Note.* MLM=Multilevel Modelling; MTS=Matching to Sample Visual Search; PRS=Progress Rating Schedule; ASPD=Antisocial Personality Disorder; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms: random at Level 2.

****P*<0.001; ***P*<0.01; **P*<0.05.

Table 6.23. *Summary of results of multivariate MLM for MTS correct responses as predictor of PRS scores and growth curves in psychopathy*

| Model | $\Delta Chi^2(\Delta df)$ | Part A | | | | | Part B | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $R_j^2$ | $R_i^2$ | β(*SE*) | | | $R_i^2$ | β(*SE*) | | |
| | | | | Time(*e-3*) | MTS | MTS x time(*e-3*) | | Time (*e-3*) | MTS | MTS x time(*e-3*) |
| **0** | 35.52(2)*** | <0 | 41% | -4.26(1.18)*** | | | 33% | 2.23(0.42)*** | | |
| **1** | nil(3) | nil | nil | -4.26(1.18)*** | | | nil | 2.23(0.42)*** | | |
| **2-Best** | 23.73(2)*** | 2% | <0 | -4.17(1.20)*** | | | 62% | 1.69(0.81)* | | |
| **3** | 1.26(2) | 4% | nil | -4.19(1.12)*** | 0.20(0.21) | | 1% | 1.59(0.83) | 0.02(0.03) | |
| **4** | 4.24(4) | 3% | 8% | 31.05(20.79) | 0.35(0.23) | -0.78(0.46) | 1% | 9.11(14.35) | 0.04(0.05) | -0.16(0.31) |
| **5** | 4.24(7) | 3% | 8% | 31.05(20.79) | 0.35(0.23) | -0.78(0.46) | 1% | 9.11(14.35) | 0.04(0.05) | -0.16(0.31) |
| **6** | 4.24(8) | 3% | 8% | 31.05(20.79) | 0.35(0.23) | -0.78(0.46) | 1% | 9.11(14.35) | 0.04(0.05) | -0.16(0.31) |

*Note.* MLM=Multilevel Modelling; MTS=Matching to Sample Visual Search; PRS=Progress Rating Schedule; ASPD=Antisocial Personality Disorder; β=Fixed parameter estimate; *SE*=Standard error; $\Delta Chi_1^2$=-2Log likelihood difference test compared to previous significantly improved model, with the intercepts-only model being first; $R_{j/i}^2$=Change in Level 2/1 residual variance compared to the previous significantly improved model, with the intercepts-only model being first.

Highlighted terms: random at Level 2.

***$P$<0.001; **$P$<0.01; *$P$<0.05.

### 6.2.7 Overall summary of cognitive performance and progress in treatment in ASPD and psychopathy

#### 6.2.7.1 ASPD

Results indicated that performance on some CANTAB tasks predicted progress in treatment in individuals with ASPD. A summary is provided in Table 6.24. Impairments in motor regulation and longer thinking times during problem solving predicted negative progress over time as measured by PRS Part B scores, in line with the first hypothesis. However, the effect of the former was reversed whereas the latter was no longer significant after controlling for IQ. Difficulties in cognitive flexibility also predicted progress in treatment, although not for attentional set-shifting. Although risk taking predicted improvement over time on both parts of the PRS contrary to expectations, decision-making and response reversal predicted lower Part B scores.

Difficulties in visual learning also predicted negative progress as measured by both parts of the PRS while impairment in STM capacity predicted decline in Part B scores only. These findings were consistent with the hypotheses. However, further analyses did not reveal an effect of sustained attention and other visual STM functions on PRS change over time, contrary to expectations.

The second hypothesis suggested that the remaining of the examined functions including verbal memory, WM, and visual perception would not predict progress in treatment. This proposition was partly supported the exception being verbal memory. which predicted decline in Part B scores only.

#### 6.2.7.2 Psychopathy

Results indicated that performance on the SOC only predicted progress in treatment in individuals with psychopathy (Table 6.24). Performance on motor regulation, cognitive flexibility, and visual perception were not related to PRS scores, contrary to the first hypothesis which received very limited support overall. During planning, impaired efficiency in less challenging problems predicted negative progress in treatment but the opposite pattern emerged for most challenging problems. In

addition, longer planning and thinking times predicted positive change in Part B scores over time whereas longer thinking times for less difficult solutions predicted negative change in Part A scores during admission. Results largely supported the second hypothesis as sustained attention and memory did not generally predict progress in treatment. However, good performance on one task of visual STM (DMS) showed a positive correlation with Part A scores overall.

Table 6.24. *Summary of relationships between performance on the CANTAB and progress in treatment in the antisocial personality*

| **Function** | **ASPD** | | **Psychopathy** | |
|---|---|---|---|---|
| | *PRS Part A* | *PRS Part B* | *PRS Part A* | *PRS Part B* |
| Motor regulation (AGN) | | †$^{\Delta}$ | | |
| Cognitive flexibility: | | | | |
| - Response reversal (IED) | | † | | |
| - Attentional set-shifting (IED) | | | | |
| - Decision-making (CGT) | Risk-taking | Risk-taking$^{\Delta}$ | | |
| | | Delay aversion$^{\Delta}$ | | |
| Planning (SOC) | | Longer thinking during most difficult problems | Efficiency & longer thinking: less difficult problems | Efficiency: less difficult problems |
| | | $\Delta$ | Efficiency: most difficult problems | Efficiency: most difficult problems; Longer planning & thinking |
| Sustained attention (RVP) | | | | |
| Visual STM (PAL, DMS, SSP) | | PAL: errors; SSP† | DMS | |
| Verbal memory (VRM) | | | | |
| WM (SWM) | | | | |
| Visual perception (MTS) | | | | |

*Note.* Red shading=Difficulties in these functions predicted deterioration over time; Green shading=Difficulties in these functions predicted improvement over time; ASPD=Antisocial Personality Disorder; PRS=Progress Rating Schedule; AGN=Affective Go/NoGo; IED= Intra/Extra-Dimensional Set Shifting; CGT=Cambridge Gambling Task; SOC=Stocking of Cambridge; RVP=Rapid Visual Processing; STM=Short-term memory; PAL=Paired Associates

Learning; DMS=Delayed Matching to Sample; SSP=Spatial Span; VRM=Verbal Recognition Memory; WM=Working memory; SWM=Spatial Working Memory; MTS=Matching to Sample Visual Search.

†=Emerged when individuals with psychopathy were removed from the ASPD group.

Δ=Emerged after controlling for IQ.

# 7 DISCUSSION

The present project investigated the neuropsychological deficits in the antisocial personality and their impact on treatment. The term antisocial personality refers here to those traits associated with impulsive behaviour and a pervasive disregard for the rights of others in pursuing personal goals. The concept has proven difficult to define and currently there are three mainstream operationalisations – ASPD, DPD, and psychopathy – showing only a degree of overlap in their conceptualisations. Those with antisocial personality pose many challenges to social systems and have proven to be difficult to treat. Nonetheless, current research into its neurobiological nature has shown promise leading to an increase in both its scientific and clinical understanding. However, although several neuropsychological theories of the concept have emerged over the years, a unified explanation remains elusive while findings from individual investigations often seem unclear and contradictory. In addition, no attempt has been made to use the tentative evidence of neurocognitive dysfunction to inform contemporary interventions and improve the poor outcomes they currently obtain. The present project therefore aimed to both clarify further both the extent of neurocognitive impairment in the antisocial personality and examine their relationship to progress in treatment.

The breadth of the existing neuropsychological research in the antisocial personality together with considerable variability in the findings highlighted the need to conduct a systematic review with meta-analyses to aid the generation of hypotheses. This comprised a major component of the thesis. Adopting a conservative approach, for example by focusing on rigorously conducted studies and examining pooled effect size margins, the review indicated consistent deficits in motor regulation, affect recognition, and (verbal) concept formation across operationalisations of the antisocial personality. Although with less consistency, the literature also supported impairments in planning, sustained attention, and visual STM in ASPD and in cognitive flexibility, verbal expression, and visual perception in psychopathy. The evidence was less conclusive regarding other neuropsychological functions.

Limitations in the literature included a major focus on psychopathy relative to ASPD and DPD, an over-representation of offender samples, and a lack of comparison

between the different operationalisations of the antisocial personality. These observations were helpful in directing the empirical part of this project. They also highlighted the need to utilise a method of measurement suitable for detecting potentially subtle deficits, particularly in those functions where the literature appeared less consistent.

Following on from the systematic literature review, the empirical part of this project compared individuals with antisocial personality (ASPD or psychopathy) to individuals with other personality disorders and healthy controls on a range of neuropsychological functions. Further, the relationship of neurocognitive impairments with progress in treatment in the antisocial samples was assessed. Participants with personality disorders were recruited from a specialist medium security personality disorder inpatient service while healthy controls consisted of ancillary staff members from the same setting. The CANTAB was selected for the neuropsychological assessment because of its focus on and sensitivity to a range of neurocognitive functions while acknowledging its limited coverage of verbal and affective functions. A special measure, the PRS, was developed within the service in order to provide a comprehensive assessment of patient progress in treatment for this project.

It was hypothesised that ASPD would show deficits primarily in motor regulation, planning, and cognitive flexibility with the potential of further deficits in sustained attention and visual STM whereas verbal memory, WM, and visual perception should not be impaired. On the other hand, psychopathy was expected to be associated with impairments primarily in motor regulation and cognitive flexibility with the potential of further impairments in planning and visual perception whereas sustained attention and memory were not expected to be impaired. Furthermore, it was hypothesised that impairments would independently predict negative progress in treatment. It was expected that this would be more pronounced for those functions more significantly impaired in the antisocial personality (i.e. motor regulation, planning, and cognitive flexibility for ASPD and motor regulation and cognitive flexibility in psychopathy).

### 7.1 Neurocognitive deficits in the antisocial personality

The empirical investigation generally supported the hypotheses regarding neurocognitive deficits. Compared with healthy controls, patients with ASPD showed hypothesised deficits in motor regulation, cognitive flexibility (response reversal, attentional set-shifting, and decision-making), planning, sustained attention, and memory. Of these, impairments in planning, visual STM span, and attentional set-shifting were observed in patients with other personality disorders also and therefore did not appear unique to ASPD.

On the other hand, findings in psychopathy supported the hypothesised deficits in motor regulation, cognitive flexibility (response reversal and decision-making), planning, and visual perception. The majority of these functions did not appear unique to psychopathy as were also impaired in the other personality disorder groups with the exception of response reversal and visual perception.

Results were broadly in line with the conclusions of the systematic literature review and provided further confirmatory evidence in areas where the literature had appeared less clear. The latter included deficits in cognitive flexibility, sustained attention, and visual STM in ASPD and planning and visual perception in psychopathy. Tests which had not supported these deficits in ASPD were the WCST for cognitive flexibility (Barkataki et al., 2005; Stevens et al., 2003), the CPT for sustained attention and visual STM (Barkataki et al., 2005; Swann et al., 2009), and the WMS (Barkataki et al., 2005), Digit Span (Stevens et al., 2003), and emotional memory task (Dolan & Fullam, 2005) for visual STM. Ceiling effects or considerable involvement of more than one cognitive function in these tasks (Epstein, Johnson, Varia, & Conners, 2001; Kaufman, McLean, & Reynolds, 1991; Lezak et al., 2004; Mountain & Snow, 1993; Strauss et al., 2006) may have been limiting factors in their measurement. On the other hand, the IED (cognitive flexibility), RVP (sustained attention), and DMS (visual STM) in this project had a narrower functional focus and provided a more detailed assessment (Cambridge Cognition, 2006; Strauss et al., 2006), e.g. by incorporating conditions of increasing difficulty. Findings also replicated and extended those of Dolan and Park (2002), as pronounced impairments were detected in planning (SOC) and visual STM (DMS). However, these may reflect sample differences, particularly additional personality disorders and a history of SRDs

in the present study, which are also related with neuropsychological impairments (Baldacchino, Balfour, Passetti, Humpris, & Matthews, 2012; Bazanis et al., 2002; Ersche & Sahakian, 2007; R. D. Rogers & Robbins, 2001). Overall, the present findings in conjunction with inconsistencies in prior literature, may indicate that cognitive flexibility, sustained attention, and visual STM reflect secondary or milder deficits in ASPD, in conjunction with robust impairment in motor regulation, planning, and visual perception.

Regarding psychopathy, the lack of clarity in the literature regarding deficits in planning and visual perception may have resulted from lack of specificity and measurement error as these impairments were supported in the present project. Regarding planning in particular, the degree to which the tests implicated working memory may have played a key role. Tests involving this function to a relatively small degree were the Porteus mazes and ToL (Daigneault, Braun, & Whitaker, 1992; Lezak et al., 2004; Phillips, Wynn, Gilhooly, Della Salla, & Logie, 1999), which detected a deficit in psychopathy (Lapierre et al., 1995; Pham et al., 2003). The SOC used in the present project, which provided further evidence of impairment in planning, falls into the same category (Cambridge Cognition, 2006). However, the Digit span backward test, which involves working memory to a relatively large degree (Lezak et al., 2004, Wechsler, 1981, 1997) had failed to reveal a deficit (Mercer et al., 2005; Pham et al., 2003; S.S. Smith et al., 1992). It follows that a planning deficit in psychopathy may exist irrespective of functional working memory.

In connection with visual perception in psychopathy, prior literature had examined a heterogeneous group of relevant operations but only studies involving matching (Kosson et al., 2007; Lopez et al., 2007) and one study on inattention (Pham et al., 2003) had suggested a deficit. The cancellation task in the latter was also confounded by attentional processes to a degree (Amieva, Lafont, Dartigues, & Fabrigoule, 1999; Lezak et al., 2004). On the other hand, the MTS task used in the present project was focused on visual recognition with a small element of attention and included conditions of increasing difficulty thereby potentially enhancing its sensitivity and thus revealing a deficit in psychopathy. Consequently, a subtler visual perception deficit, potentially specific to visual recognition/matching, may be present in psychopathy.

### 7.1.1 Unsupported hypotheses and unexpected deficits

Although most results were in line with expectations, some of the findings did not support the hypotheses. Regarding ASPD, impairments in WM and visual perception had not been expected but the hypotheses were based on a very limited literature and potentially lack of power. For instance, data for the two studies reporting no deficits in WM (Barkataki et al., 2005; Kumari et al., 2006) were derived from the same ASPD sample. Furthermore, the study on visual perception (recognition) featured a relatively simple task (simultaneous presentation of the DMS, Dolan & Park, 2002) thereby suggesting a ceiling effect. It follows that the unexpected deficits in WM and visual perception in ASPD as detected by the CANTAB may have once again been the result of more power (larger samples) and more sensitive measurement. However, they might also reflect Type I error, therefore, replication remains necessary.

Regarding psychopathy, the deficit in sustained attention was not expected whereas the hypothesised deficit in attentional set-shifting was not supported. Apart from Type I error, there might be other plausible explanations. The literature had not suggested a reliable deficit in sustained attention (strongest and weakest effects yielded opposite results). The oddball CPT tasks, which are primarily used to assess attention (Conners, 2000; Lezak et al., 2004), in particular, had failed to detect impairment (Howard & McCullagh, 2007; Jutai & Hare, 1987; Kiehl, Bates, et al., 2006; Kiehl, Hare, Liddle, et al., 1999; Raine & Venables, 1988) while tasks implicating visual perception, e.g. target discrimination and cancellation (Lezak et al., 2004), were the only ones indicating a deficit (Kosson, 1998; Llanes & Kosson, 2006; Mills, 1995; Pham et al., 2003). Although the RVP task used in this project operated on a CPT paradigm, it involved greater difficulty than the oddball paradigms in previous research (Strauss et al., 2006). As a result, it may be less susceptible to ceiling effects and therefore more able to detect a deficit. Although this might suggest a mild sustained attention deficit in psychopathy, it is also plausible that the high proportion of individuals with ASPD in the group may have led to this result.

Regarding set-shifting in psychopathy, the meta-analysis had indicated a small to medium but robust deficit but only two of the seven individual studies had observed this effect. Furthermore, use of the CANTAB in one study (Mitchell et al., 2002) had

also failed to observe a deficit. This was replicated in the present project but the sample of individuals with psychopathy was small. These considerations suggest that the ability to detect impairment in attentional set-shifting in psychopathy may be compromised by lack of power, especially as the potential effect is likely to be small.

### 7.1.2 Antisocial versus other personality disorders

Overall, the empirical investigation of this project added to the evidence suggesting a link between cognitive impairment and the antisocial personality in offender samples. Patients with other personality disorders seemed to perform somewhere between healthy controls and peers with antisocial personality on motor regulation, attentional set-shifting, working memory, and visual perception, but were not significantly different to either. Although further research should ascertain the level of additional impairment in these functions in antisocial individuals compared to other personality disorders these findings might suggest that the antisocial personality is associated with more pronounced deficits.

A further point of interest might concern the overlap between neuropsychological deficits in offenders with and without antisocial personality, which suggest maybe that some frontal and temporal impairments may underlie criminal activity more generally. This is also in line with the existing literature on offender populations. For example, neuropsychological and neurological deficits (e.g. executive, affective, frontal, and temporal) have been observed not only in individuals exhibiting antisocial behaviour/aggression (Barker et al., 2007; R. Blair, 2004, 2010; Marsh & Blair, 2008; Ogilvie et al., 2011; Wahlund & Kristiansson, 2009) and other personality disorders (Bazanis et al., 2002; Berlin, Rolls, & Iversen, 2005; Dolan, Anderson, & Deakin, 2001; Dolan, Deakin, Roberts, & Anderson, 2002; Völlm et al., 2007; Völlm et al., 2004) but also in individuals from other clinical forensic populations such as schizophrenia and bipolar disorder (Fullam & Dolan, 2008; Lewandowski, Cohen, & Ögnur, 2011). Consequently, although the antisocial personality might reflect more extensive or pronounced impairment in some clusters of neurocognitive function within offender populations, a range of other factors are also likely to determine its symptomatology.

### 7.1.3 ASPD versus psychopathy

The systematic review of prior literature highlighted some similarities between the two operationalisations, particularly in showing impaired executive function (motor regulation), affect recognition, and verbal concept formation, but the CANTAB results from this project suggested further commonalities between their neurocognitive profiles (Table 5.3). Individuals identified through both operationalisations showed deficits in motor regulation, cognitive flexibility (response reversal), planning, sustained attention, and visual perception. Interestingly, the deficits in motor regulation, response reversal, and visual perception were not identified in the other personality disorder groups, which might imply that these functions could play a key role in the development of the antisocial personality.

Caution is required in interpreting the above similarities between ASPD and psychopathy, however, as the two operationalisations formed different subgroups of the same patient cohort in this project as 88% of participants who met the criteria for psychopathy also met those for ASPD while this was 50% vice versa (Figure 5.1). In an attempt to control for this, the pattern of deficits in individuals with ASPD was inspected and remained largely unchanged when those individuals who also met criteria for psychopathy were removed from the group. However, it was not possible to examine the reverse due to sample size limitations; therefore, it is plausible that some of the results on psychopathy may have emerged because of their association with ASPD. As the groups were confounded to such a large extent, it is not possible to dissociate between the two operationalisations credibly.

Apart from similarities between the neurocognitive profiles of ASPD and psychopathy, it is important to note the discrepancies that were observed as these occurred in spite of sample overlaps. ASPD was associated with a wider range of fronto-temporal deficits than psychopathy in which impairment concentrated around frontal functions with no evidence of memory deficit. Even within frontal functions, however, individuals with psychopathy appeared somewhat less impaired in planning and showed fewer deficits in cognitive flexibility (i.e. impairment in response reversal decision-making but not attentional set-shifting) compared to ASPD. Since the ASPD diagnosis is more behaviourally orientated than psychopathy (APA, 2000; Hare, 2003), it may reflect more pronounced frontal dysfunction whereas psychopathy may

be a reflection of more specific affective deficits related to the amygdala (R. Blair e al., 2005; Dolan & Park, 2002). Furthermore, compared to psychopathy, ASPD populations are both more diverse and show more comorbidity with other disorders and particularly substance abuse (Blackburn, 2009; Coid et al., 2006; De Brito & Hodgins, 2009; Hare, 2003), the latter also considerably associated with neurocognitive deficits (Baldacchino et al., 2012; Bazanis et al., 2002; Ersche & Sahakian, 2007; R. D. Rogers & Robbins, 2001). Consequently, the wider fronto-temporal impairment observed in ASPD is also likely to reflect its higher heterogeneity compared to psychopathy.

Findings suggested that ASPD may show a wider range of fronto-temporal deficits compared to psychopathy. However, this does not necessarily suggest wider overall neuropsychological impairment as the present investigation was limited by the scope of the CANTAB which does not assess affective and language functions. According to the systematic literature review, psychopathy was associated with deficits in these functions more strongly than ASPD and it has been though that the former might be more circumscribed to the affective functions related to the amygdala (R. Blair et al., 2005; Dolan & Park, 2002). Although, the present results emerged from the first parallel examination of ASPD and psychopathy the limitations of the CANTAB in terms of affective and language functions highlight the need to extend the investigation to these operations also.

### 7.1.4 Offenders without psychopathy

An interesting observation, though not immediately relevant to the project's focus, was that offenders without psychopathy showed more impairment in some functions compared to controls than individuals with psychopathy. These deficits were in concept formation functions (attentional set-shifting & decision-making), visual STM span, and WM. Of these, impairments in attentional set shifting and WM may be attributed to presence of ASPD but the groups were confounded, making it difficult to demarcate the effects of different diagnoses while for several functions findings indicated imparements in the patient group as a whole.

### 7.1.5 Neuropsychological deficits and neurological substrates

The fronto-temporal deficits associated with ASPD and more localised frontal impairments in psychopathy may reflect structural and functional abnormalities in the underlying neural networks. Furthermore, less localised functions such as sustained attention and visual scanning might reflect a wider cerebral dysfunction. Neuroimaging studies in the antisocial personality are consistent with both these suggestions and particularly with abnormalities in fronto-temporal networks.

#### 7.1.5.1 ASPD

Although not extensive, the imaging research in ASPD has revealed a range of anomalies. Volumetric comparisons have shown whole brain and temporal lobe volume reductions, medial inferior and right sensory motor cortical thinning, and putamen volume increases in individuals with ASPD compared to controls (Barkataki, Kumari, Das, Taylor, & Sharma, 2006; R. Blair et al., 2005; Narayan et al., 2007). Grey matter reductions have been consistently associated with the disorder and appear to extend across the fronto-temporal network including prefrontal, fronto-polar, orbitofrontal, and anterior temporal cortices, the superior temporal sulcus, and insular areas (de Oliveira-Souza et al., 2008; Raine et al., 2000; Tiihonen et al., 2008). However, prefrontal reductions – particularly those in dorsolateral, orbitofrontal and medial prefrontal areas – may be attributed to alcohol abuse, as observed in a study of ASPD and alcohol-related diagnoses (Laakso et al., 2002). Subcortical connections also appear affected in ASPD as individuals with the disorder were found to have a corpus callosum with increased white matter volume and length but reduced thickness compared to healthy controls (Raine et al., 2003). These structural deficits appear consistent with neuropsychological impairments observed in this project, particularly in terms of executive and memory functions in relation to fronto-temporal networks and attentional processes in relation to wider cerebral function (D. L. Clark, Boutros, & Mendez, 2010; Gazzaniga et al., 2009; Kolb & Whishaw, 2009).

Functional magnetic resonance imaging (fMRI) studies on ASPD have examined mostly executive performance. Attenuated activation was observed during a working memory task in the left frontal gyrus, anterior cingulate, and precuneus among offenders diagnosed with ASPD versus healthy controls (Kumari et al., 2006).

There were similar findings regarding the striatum during punishment sensitivity conditions while thalamic hypoactivation was evident in both punishment sensitivity and response inhibition (Barkataki et al., 2008; Kumari et al., 2009). Furthermore, during response inhibition, individuals with ASPD demonstrated a pattern of prefrontal activation which was more bilateral and extended compared to individuals with borderline personality disorder (Völlm et al., 2004). They also showed impaired function in dorsolateral, orbitofrontal, and anterior cingulate cortices in the context of reward and punishment sensitivity (Völlm et al., 2007). These findings suggest that the executive deficits observed in ASPD are likely to reflect different neurobiological configurations in a range of frontal areas compared to controls. However, further research is required to explore this hypothesis in relation to other operations such as memory, attention, and visual perception, which were also impaired in ASPD.

#### 7.1.5.2 Psychopathy

Neuroimaging research in psychopathy has been much more extensive than ASPD with evidence suggesting a range of structural and functional anomalies particularly in limbic, paralimbic, and striatal areas. Structural imaging studies have associated psychopathy with prefrontal grey matter reductions (Yang et al., 2005), hippocampal asymmetries (Laakso et al., 2001; Raine et al., 2004), reduced volumes and deformations in the OFC and amygdala (Boccardi et al., 2011; Ermer, Cope, Nyalakanti, Calhoun, & Kiehl, 2012; Yang, Raine, Narr, Colletti, & Toga, 2009), anomalies in connective pathways (Craig et al., 2009), and atypical striatal morphology (Boccardi et al., 2013; Glenn, Raine, Yaralian, & Yang, 2010). These appear consistent with the executive deficits observed in psychopathy and particularly reward/punishment-based operations and motor control (e.g. decision-making, response inhibition, and response reversal) which are associated with these regions (Bryden, Burton, Kashtelyan, Barnett, & Roesch, 2012; D. L. Clark et al., 2010; Elliott, Agnew, & Deakin, 2008, 2010; Elliott & Deakin, 2005; Hampshire, Chaudhry, Owen, & Roberts, 2012; Horn, Dolan, Elliott, Deakin, & Woodruff, 2003; Klanker, Post, Joosten, Feenstra, & Denys, 2013; Zald & Andreotti, 2010). Since memory was not impaired in individuals with psychopathy, the role of the hippocampus in psychopathy, as a core memory area, might be related to its input in other limbic

regions, e.g. in providing contextual cues for conditioned responses (Maren, Phan, & Liberzon, 2013).

Functional imaging studies in psychopathy have recorded various frontotemporal, limbic, and paralimbic abnormalities. Their focus has been primarily on affective, moral, and conditioning rather than executive functions, in contrast to ASPD. Single photon emission computed tomography at rest highlighted associations between the interpersonal/affective features of psychopathy and reduced perfusion in frontotemporal circuitries (Soderstrom et al., 2002). Positron emission tomography during semantic and affective processing indicated that the interpersonal/affective features of psychopathy predicted abnormal activation in fronto-temporal and medial frontal cortical areas as well as parts of the caudate nuclei and the hippocampus (Intrator et al., 1997). Furthermore, neural differentiation in anterior temporal areas was absent in individuals with psychopathy compared to controls during semantic processing alone in fMRI (Kiehl et al., 2004). Further fMRI studies have indicated activation anomalies during emotional processing and memory in anterior and posterior cingulate areas, the amygdala, hippocampus, parahippocampal gyrus, ventral striatum, and the frontotemporal circuitry (Kiehl et al., 2001; Müller et al., 2008), which extended to the fusiform gyrus when facial stimuli were used (Deeley et al., 2006).

Cerebral functioning during moral reasoning and conditioning has also been the focus of fMRI research in psychopathy. Studies on moral reasoning suggested decreased amydgalar activation in individuals with higher psychopathy scores and abnormal activations in the broader moral network comprising medial prefrontal, posterior cingulate, and angular areas (Glenn, Raine, & Schug, 2009; Pujol et al., 2012), potentially extending to ventromedial prefrontal and temporal cortices (Harenski, Harenski, Shane, & Kiehl, 2010). In relation to conditioning, individuals with psychopathy showed differential patterns of activation in the amygdala and dorsolateral prefrontal cortex (Schneider et al., 2000) and reduced activation in the amygdala, orbitofrontal cortex, insula, and anterior cingulate cortex compared to healthy controls (Birbaumer et al., 2005; Veit et al., 2002).

The focus of functional neuroimaging research in psychopathy reflects the traditional view that this personality type reflects a moral/affective disturbance (Blackburn, 2009; R. Blair et al., 2005; Cleckley, 1976; Hare, 2003). However, the

systematic review of the neuropsychological research and the results of the present project also supported a range of executive deficits. Some of these may be attributed to dysfunction of limbic and paralimbic networks, e.g. decision-making, response inhibition, and response reversal (D. L. Clark et al., 2010; Elliott et al., 2008, 2010; Elliott & Deakin, 2005; Horn et al., 2003; Zald & Andreotti, 2010). However, the degree of executive dysfunction observed in individuals with psychopathy highlights the need to extend the neuroimaging research beyond affective and moral paradigms to other cognitive functions and frontal regions.

Overall, it appears that the present findings are consistent with findings from structural and functional imaging in ASPD and psychopathy. However, further research is required in order to examine the relationship between impaired functions and neurological substrates directly as well as investigating affective and language functions, which were not examined in this project due to the limited scope of the CANTAB.

### 7.1.6 Theoretical considerations

The present evidence provided mixed support for the neuropsychological theories of the antisocial personality although a comprehensive evaluation was not possible since the project did not include affective and language functions in its investigation. The BIS/BAS model (J. A. Gray, 1987) is consistent with deficits in motor regulation and perhaps response reversal on the premise of an imbalance between response inhibition and activation (Fowles, 1980; Lykken, 1995; Scerbo et al., 1990). However, it focuses on the role of fear and punishment in shaping behaviour (Levenston, Patrick, Bradley, & Lang, 2000; Lykken, 1995; Ogloff & Wong, 1990) and therefore does not account for the memory, attentional, planning, and perceptual deficits observed in this study.

The response modulation hypothesis (Newman, 1998; Patterson & Newman, 1993) is consistent with attentional and motor regulation impairments and potentially response reversal. However, it is not able to explain additional deficits in perception, memory, and planning, particularly in ASPD. Furthermore, as the theory predicts a deficit in the ability to shift the focus of attention to peripheral information in order to

adjust behaviour (Newman, 1998; Patterson & Newman, 1993), it precludes a primary deficit in attentional set-shifting at least in psychopathy which this model has been primarily based on. However, this was not supported in the present study.

The frontal lobe dysfunction/somatic marker hypothesis (R. Blair, Colledge, & Mitchell, 2001; Gorenstein, 1982; Moffitt, 1993; Raine, 2002) could explain the range of frontal deficits observed in both ASPD and psychopathy (though less so for the latter as it appeared less impaired). However, it does not account for the temporal deficits in ASPD. Furthermore, it is a general theory and, although it may be compatible with the notion of secondary attentional and perceptual deficits emerging from frontal dysfunction (Kolb & Whishaw, 2009; Lezak et al., 2004), it does not make specific predictions on the underlying mechanism.

Although the present project did not include affective/distress tasks, thereby not allowing the evaluation of the VIM model, there was some support for its successor, the IES. The OFC/amygdala circuitry plays a central role in motor regulation/inhibition, reversal learning, and decision-making (Bryden et al., 2012; D. L. Clark et al., 2010; Elliott et al., 2008, 2010; Elliott & Deakin, 2005; Hampshire et al., 2012; Horn et al., 2003; Klanker et al., 2013; Zald & Andreotti, 2010). It follows that deficits in these operations as observed in the present project might indicate dysfunction of this cerebral region. This provides some support for the IES in considering the OFC/amygdala circuitry central in the development of the antisocial personality (R. Blair et al., 2005). However, the theory does not consider other cerebral areas playing a key role in such neuropsychological operations, namely the anterior cingulate and medial prefrontal cortices (Etkin, Egner, & Kalisch, 2011). Impairment in the anterior cingulate is, however, consistent with difficulties in attentional set-shifting (Bissonette, Powell, & Roesch, 2013; D. L. Clark et al., 2010) which was evident in ASPD and in a milder form in psychopathy. Therefore, the model remains incomplete and requires further development. Furthermore, it does not explain planning deficits, particularly as these seemed unrelated to working memory impairment and therefore OFC input (Barbey, Koenigs, & Grafman, 2011), and does not encompass deficits in visual perception and memory.

It appears that each theory is able to explain some but not all of the observed deficits in the antisocial personality. This highlights the need for greater theoretical integration towards a framework which encompasses the complex neuropsychological

profile that seems to characterise this personality type more fully. However, the lack of consensus in defining the antisocial personality is likely to continue impeding this process.

#### 7.1.6.1 Operational definitions of the antisocial personality

The present findings – particularly the discrepancies between ASPD and psychopathy – highlight the deficiencies of current operational definitions in capturing the construct of the antisocial personality. This indicates the need for greater definitional integration guided more by emerging neuropsychological and other evidence rather than clinical opinion and theorising, as was the case for the three mainstream operationalisations ASPD, DPD, and Hare's psychopathy (APA, 2000; Hare, 2003; World Health Organisation, 1990). However, with the DSM-V having shifted its focus towards the interpersonal/affective traits of the syndrome and the ICD-11 having removed this diagnosis entirely, the debate on how to operationalise this personality type and personality disorders more generally remains inconclusive (Duggan & Howard, in press).

Notwithstanding the divergence between the diagnostic nomenclatures, there is a recent conceptualisation of psychopathy which attempts to refocus the operationalisation of psychopathy and its assessment on three core components: disinhibition, boldness, and meanness (Patrick et al., 2009, 2012). Although it requires further research, this model is promising, as it is not only consistent with the mainstream operationalisations of the antisocial personality in representing both its interpersonal and behavioural features but has also been developed based on the neuropsychological evidence (Patrick, Drislane, & Strickland, 2012; Patrick, Fowles, & Krueger, 2009). Continuing to develop the constructs of the antisocial personality drawing on the emerging evidence in order to both capture the key features of this population better and increase convergence appears instrumental in addressing some of the barriers facing current research.

## 7.2 Progress in treatment and the effect of neuropsychological impairment

The effect of neuropsychological impairment on progress in treatment in the antisocial personality has remained an unexamined area to date notwithstanding a breadth of research and theorizing in the neurological/cognitive function of this population together with a rationale on the possible effects of neuropsychological impairment on treatment progress. Thus, the second aim of this project was to provide evidence on this relationship. The absence of a measure for the evaluation of progress in treatment in forensic populations with personality disorders indicated the need to develop a suitable instrument.

### 7.2.1 The Progress Rating Schedule

The instrument was developed systematically using a qualitative ("bottom-up") methodology (Braun & Clarke, 2006; Willig, 2008) to measure the progress in treatment of offenders with personality disorder based on multi-professional clinical input. In its final format, it contained two main parts and a third, supplementary, and customisable section. Part A comprised items intrinsic to the treatment process while Part B consisted of progress items such as leave, employment, and risk. The scope and scoring of all items was operationalised. The resulting instrument standardises the evaluation of progress in treatment, stemming from and utilising routine clinical practice. Consequently, it combines the strengths of structured measurement and clinical judgment which, alongside its brevity, make it viable for use in clinical settings.

#### 7.2.1.1 Psychometric properties and clinical utility

The PRS was associated with good inter-rater reliability and the scale forming Part A showed acceptable to good internal consistency. In addition, the instrument generally showed good concurrent validity and sensitivity of different subtypes of personality disorder in forensic settings while progress over time appeared consistent with prior observations in the patient cohort.

Inter-rater reliability is an important property of the PRS as it is intended for use by different professionals and therefore it ought to provide an accurate point of reference in clinical discussions. Intra-class correlations indicated a good level of

inter-rater agreement between different disciplines thereby supporting the validity of the operationalisations and thus the instrument's application by different members of the multidisciplinary team. However, some discrepancies in interpretation were present between raters and pairs of raters, particularly in connection with mental state and insight. In consequence, improving the operationalisation of the scale may be beneficial, especially for these items, while consensual scoring between members of the multidisciplinary team may enhance validity. Nevertheless, the intra-class correlations suggested better inter-rater agreement for the PRS items compared to the commonly used HoNOS-Secure (Dickens et al., 2007) and were comparable to widely used risk assessments in the field such as the HCR-20 (Douglas & Reeves, 2010) and the Violence Risk Scale (Wong & Olver, 2010).

Although less important than inter-rater reliability, acceptable to good internal consistency for Part A was helpful in demonstrating the PRS' ability to capture progress holistically. In combination with good concurrent validity, as evidenced by correlations with the DSQ and SPSI-R, Part A appears to measure the process of change in treatment where engagement, behaviour, interpersonal relationships, mental state, and insight complement each other as facets of underlying personality disorder pathology. This is consistent with the current understanding of personality disorder as having both a personal and interpersonal dimension (Alwin et al., 2006; NICE, 2009) and extends beyond the assortment of behavioural and interpersonal psychometric methods of assessing progress in this population (Duggan, 2004). Furthermore, correlations with the DSQ suggested that the PRS may be sensitive to psychiatric symptomatology and relevant change in personality disorders (Bond & Perry, 2004) while its association with the SPSI-R associated it directly with the treatment aims and content (Huband et al., 2007; McMurran et al., 2005). In conjunction with significant variance within the relatively short periods of its administration (up to 6 months), Part A may be well suited for the assessment of relevant change with the added benefit of higher temporal sensitivity compared to instruments based on diagnosis (e.g. IPDE), which requires that personality disorder traits are enduring (Alwin et al., 2006; APA, 2000).

Part B was significantly correlated with Part A but this was small to medium only. Internal consistency was not considered relevant for Part B, since it consisted of inherently heterogeneous indicators of progress/achievements from the outset. Its

components of leave, building external supportive relationships, completing education, achieving employment, and completing treatment within the current level of security appear to represent aspects of progress through the forensic care pathway (McMurran et al, 2009). However, the relatively small correlation with Part A and fewer as well as smaller correlations with the psychometric measures suggest that the two sections may reflect different aspects of change. This implies that intrinsic progress in treatment may not be equated with systemic progress and therefore Part A and B seem to complement each other in assessing outcome within the PRS.

The examination of PRS score trajectories over time suggested that progress within the programme (Part A) varied significantly between patients but progress through the forensic pathway (Part B) appeared more uniform. In conjunction with the evidence on concurrent validity, these results support the instrument's ability to provide a credible means of quantifying patient progress in forensic personality disorder settings. This was further corroborated by considering high-psychopathy scoring patients admitted to the PDS who have shown both conservative completion rates and poorer post-discharge outcomes (McCarthy & Duggan, 2010; McCarthy et al., 2012). Though this may not generalise to all treatment settings (D'Silva, Duggan, & McCarthy, 2004), the PRS appeared able to qualify these observations on the service by demonstrating that treatment non-completion in this patient group was consistent with poorer benefits from the treatment programme. This was evidenced both by reduction in functioning within the ward (potentially explaining the premature discharge) and by slower progress through the forensic pathway. These findings are consistent with observations of a negative relationship between psychopathy and violence risk change during treatment (Olver, Lewis, & Wong, 2013) and also with evidence of increased institutional challenge and complex treatment needs in individuals with psychopathy (Guy, Edens, Anthony, & Douglas, 2005; Wong, in press). Consequently, the results support the usefulness of the PRS in treatment evaluation alongside its potential as a means of identifying less responsive patient groups thereby directing treatment development.

Finally, the supplementary part of the PRS was included for any particularly relevant assessments or records (e.g. psychometrics) that may be used locally. Its contents will vary from setting to setting and will depend on the judgment of clinicians involved. At the PDS of Arnold Lodge, for example, this section included a list of

psychometrics and frequency of violent incidents. Although this third section will undoubtedly add heterogeneity to the PRS, its inclusion is important in enhancing the schedule's flexibility and therefore its ability to meet diverse service needs.

#### 7.2.1.2 Limitations and future directions

Although the present study provided some initial evidence to support the reliability, validity, and clinical utility of the PRS, there were limitations and further work in all of these areas is required. Demonstrating good inter-rater reliability is important and future research should extend the current investigation. Final ICCs in this project resulted from revising the scoring of items once the relatively ambiguous ones had been identified and refined guided by the levels of inter-rater agreement. Furthermore, the same individuals involved with the refinement of the instrument also undertook this re-scoring, which may have introduced bias thereby inflating ICC estimations. It follows that it is important to examine the instrument's inter-rater reliability with different raters as well as additional disciplines.

An important limitation concerns risk assessment scores (HCR-20 at the PDS). These were assigned to Part B of the PRS but insufficient data did not permit their inclusion in calculating total scores thereby limiting conclusions on validity and clinical utility. As treatment of criminogenic needs and measurement of risk are important components of forensic healthcare, e.g. in the risk-need-responsivity model (Andrews, Bonta, & Wormith, 2011; Heilbrun, Yasuhara, & Shah, 2010; NICE, 2009), it is important that future research extends the present examination by including risk scores.

A further limitation of the PRS exists with regard to content validity. As it was developed based on clinical records, the conceptualisation of progress in treatment reflected clinician rather than patient views. In terms of treatment aims, these views can be very discrepant (Huband, Evans, Duggan, & Khan, 2012) while incongruence between self-report, peer-report, and clinician assessments are also well-documented (Milton et al., 2005; Perry, 1992; Zimmerman, 1994). Consequently, further research is required to examine the extent to which the PRS represents patient perspectives also.

Replication of all findings on reliability and validity in larger and different samples is necessary in order to strengthen current observations. In addition, extending the validation of the instrument to its association with other psychometrics and outcome variables relevant to personality disorders will be germane to establishing its utility. It will also be important to demonstrate the predictive validity of the PRS in connection with long-term psycho-social outcomes and reoffending. These are key outcomes in the field and remain a cause for concern in the treatment of personality disorders (Coid et al., 2007; Davies et al., 2007; McCarthy & Duggan, 2010; Ministry of Justice, 2011; NICE, 2009), therefore, they are important elements in supporting the clinical utility of the PRS as an instrument for evaluating relevant treatment progress.

### 7.2.2 The relationship between neurocognitive deficits and treatment progress

The present findings provided mixed support for the hypotheses and the evidence indicating a relationship between neuropsychological difficulties and progress in treatment was generally limited. The majority of effects were observed in offenders with ASPD where some deficits predicted negative progress in PRS Part B scores only. Although these results suggested that cognitive impairments might not predict progress within the treatment programme (PRS Part A), executive and memory deficits may signal slower progress within the forensic care pathway (Part B). However, controlling for IQ suggested that the effects of executive functions (motor regulation and planning in particular) may be due to the influence of intellectual functioning in these operations rather than executive deficits per se (Ardila, Pineda, & Rosselli, 2000; Arffa, 2007).

Contrary to expectations, not all neuropsychological deficits appeared relevant to treatment progress in ASPD, since sustained attention and attentional set-shifting were not related to any PRS scores. Furthermore, unimpaired memory functions (verbal memory and visual learning in particular) predicted better progress. Although deficits in the latter have predicted poorer progress in several populations including bipolar disorder (Torres et al., 2010), schizophrenia (deVille et al., 2011; Mueser et al., 1991), and depression (Story et al., 2008), they did not appear impaired in ASPD in the present project and were, therefore, not expected to predict progress. However, neuropsychological theory suggests that memory functions play a key role in the process of learning and development (Gazzaniga et al., 2009; Kolb & Whishaw, 2009; Martin, 2006). As treatment programmes for antisocial personality aim at imparting skills (NICE, 2009), it follows that it is plausible that verbal memory and visual learning continue to play an important part in the learning process of treatment even in the absence of impairment.

A surprising effect was the positive relationship between risk-taking and progress in treatment in ASPD, which might appear counterintuitive and was contradictory to prior research, for example in the field of substance abuse (Carroll et al., 2011). However, it is possible that this reflected benefits derived from a treatment programme designed to reduce impulsivity in patients or could indicate a facilitative effect of risk-taking during treatment thereby resulting in positive change. This notion

is not new as it has been suggested that risk-taking may play an instrumental role in supporting behavioural and interpersonal experimentation leading to adaptive change in therapy (Yalom & Leszcs, 2005). Research on this hypothesis is very limited but Lorian and Grisham (2011) observed a positive relationship between self-reported risk-taking and seeking treatment in individuals with anxiety disorders. A similar effect might be possible in other clinical populations including individuals with antisocial personality, thus highlighting an area for future research.

Whereas several functions showed significant relationships with aspects of progress in treatment in ASPD, only planning and visual STM predicted progress in psychopathy. Thus, the hypothesised negative effects of impairments in motor regulation, cognitive flexibility, and visual perception were not supported while the effect involving visual STM was not expected. Of the observed effects, planning was related to both PRS parts but inconsistently: impairments in less difficult problems predicted poorer progress whereas deficits in more difficult problems predicted improvement. This might imply that patients with more severe impairment may be more responsive to a treatment programme designed to address such deficits (NICE, 2009). However, it might also reflect practice effects, as the CANTAB presents the SOC trials in order of increasing difficulty without counterbalancing (Cambridge Cognition, 2006), thereby leading to systematic error (McBurney & White, 2007).

Visual STM, on the other hand, was not impaired in psychopathy but nevertheless appeared to facilitate progress within the treatment programme (PRS Part A). This may reflect the same process as in ASPD, that is, memory enhancing acquisition of skills in treatment. However, it remained an isolated observation out of the three tasks assessing visual STM in psychopathy and, therefore, may have been the result of Type I error.

Overall, neuropsychological performance did not predict progress reliably in psychopathy, even though this group seemed to gain significantly less from therapy compared to other patients. This might suggest that neuropsychological difficulties are not particularly relevant in developing current treatments to meet the needs of this population better but might also be a reflection of a treatment programme designed primarily to cater for behavioural difficulties and ASPD rather than the interpersonal/affective aspects of psychopathy (McCarthy et al., 2012; NICE, 2009).

ASPD and psychopathy demonstrated some commonalities in terms of neuropsychological function compared to controls but there was virtually no overlap between the two operationalisations in terms of the relationship between neuropsychological function and progress in treatment. In fact, the presence of psychopathy within the ASPD group seemed to bias the findings at least in connection with motor regulation and response reversal, as removal of patients with psychopathy from analyses altered the way in which both functions predicted progress in treatment in ASPD. Dissimilar results between ASPD and psychopathy might reflect genuine differences and/or the ASPD orientation of treatment programme (McCarthy et al., 2012; NICE, 2009) but may also have occurred because of methodological reasons, as discussed below.

#### 7.2.2.1 Some cautionary notes

In light of the limited support for hypotheses and some of the unexpected results in both ASPD (verbal memory, visual learning, and risk-taking) and psychopathy (planning, visual STM), it is necessary to highlight some cautionary notes. Firstly, it is important to recognise lack of statistical power (particularly for psychopathy and the newer CANTAB tests AGN, VRM, and CGT), limitations of measurement (e.g. PRS), and the resulting inflation of standard errors (Maas & Hox, 2005), as possible explanations for the absence of the hypothesised effects especially in a demanding statistical method such as MLM (Maas & Hox, 2005; Tabachnick & Fidell, 2007). Secondly, the non-randomly selected and relatively smaller samples in the AGN, VRM, and CGT tasks as well as in analyses involving psychopathy may not have been representative of the heterogeneous antisocial populations. This implies that the relevant effects may not be replicable. Thirdly, as there were three tests of visual STM, the relevant effects in both ASPD and psychopathy may be the products of Type I error. Finally, the psychometric weaknesses of the CANTAB may have further inflated Type II error resulting in failure to detect small effects.

Although prior literature has documented a relationship between neurocognitive deficits and poorer progress in treatment in individuals with bipolar disorder (Torres et al., 2010), schizophrenia (deVille et al., 2011; Spiekermann et al., 2011; Üçok et al., 2006), substance abuse (Carroll et al., 2011), depression (Story et

al., 2008), and offenders (Fishbein et al., 2009), this is a new area of investigation in the antisocial personality. The present, initial findings will potentially provide a useful foundation to guide future research in the area but the above concerns imply that any results must be viewed with scepticism. Consequently, replication remains necessary.

## 7.3 Strengths and limitations

The present project contributed empirical evidence indicating neuropsychological deficits in the antisocial personality and undertook a novel examination of their relationship to progress in treatment. The neuropsychological evidence from this project extends the current understanding of cognitive impairment in the antisocial personality. However, the evidence regarding the relationship of neurocognitive deficits to treatment progress was less robust. Methodologically, the project demonstrated several strengths compared to prior research but there were also a number of weaknesses.

### 7.3.1 Methodological strengths

#### 7.3.1.1 Confounding variables

A range of confounders may present during neuropsychological assessment and can include age, comorbid mental illness, IQ, substance abuse, traumatic brain injury, education, and medication (Lezak et al., 2004; Strauss et al., 2006). As a result, the study incorporated a number of methods (via sampling or statistically) to control for these variables the only exception being advanced education in those comparisons involving healthy controls. Alongside the use of standardised measurements and clinical assessments, these placed the study in the "high" quality range (maximum score of 9.5/10) using the Quality Rating Scale developed within this project (Section 2.2.2). The only criterion of the scale that had not been met was handedness, due to lack of data, however, this should be of little consequence in the overall validity of the results as handedness was likely to be equally distributed in the samples.

The study by Dolan and Park (2002) was the only one to have received a higher quality rating (10/10) than the present project, having also controlled for handedness. The study also excluded participants with a history of SRD. However, as this diagnosis is highly prevalent in populations with antisocial personality (De Brito & Hodgins, 2009; Hare, 2003), the results of Dolan and Park might lack external validity compared to the present study which controlled for past SRD statistically rather than by exclusion.

#### 7.3.1.2 Parallel examination of ASPD and psychopathy

The systematic literature review indicated that prior research in the antisocial personality focused on a single operationalisation. Kosson et al. (2006), who compared ASPD with and without psychopathy, might be considered the only exception. As a result, it had not yet been possible to ascertain the extent to which differences between operationalisations may be attributed to the definitional diversity rather than methods, populations, and research groups. By considering both ASPD and psychopathy, the present project was able to reveal considerable differences between the two operationalisations in neuropsychological functions and their relationship to treatment. These were evident even though the antisocial samples originated from the same population of offenders and showed some overlap, which suggests that the observed differences could be particularly robust. However, this also highlights the potential obstacle that such lack of agreement in defining the antisocial personality might pose both in clinical and research work. Furthermore, it raises a cautionary note when evaluating research in the field as findings using one definition might not always generalise to another.

#### 7.3.1.3 The CANTAB

Use of a sensitive, detailed, standardised, computer-administered, and well-validated neuropsychological assessment battery such the CANTAB conferred several benefits compared to prior research. In spite of some limitations to its stability (Section 4.3.2), the battery's focus, standardisation, and good psychometric properties were potentially instrumental in discerning the sub-clinical deficits expected in the antisocial personality thus enabling the confirmation of previously ambiguous deficits in this project. This was not the case for many of the tests featuring in the reviewed literature where studies used less rigorously standardised assessments and custom tests with under-researched psychometric properties (e.g. the passive avoidance Go/NoGo variant, Stroop variants, emotion recognition/processing, etc., Section 2.3). Use of the CANTAB not only replicated some of the important findings of the two previous studies using the assessment (Dolan & Park, 2002; Mitchell et al., 2002) but also extended the support for deficits in additional neuropsychological operations. Furthermore, while the present project matched Dolan and Park in quality (using the

Quality Rating Scale, Section 2.2.2), it incorporated considerably better methodological controls than Mitchell et al.

#### 7.3.1.4 Multiple comparison groups, large samples, and cohort inclusivity

Recruiting the majority of a patient cohort offers an advantage against selection bias (McBurney & White, 2005) while the larger samples compared to most studies in the reviewed literature – at least for ASPD and older CANTAB tests – increased statistical power in detecting subclinical cognitive deficits. Furthermore, comparing offenders with antisocial personality to offenders with other personality disorders and healthy controls enabled the identification of neuropsychological deficits that were present in the antisocial personality (ASPD or psychopathy) but not other personality disorders. This was particularly important since the antisocial type is not the only personality disorder to exhibit cognitive impairments (Baer, Peters, Eisenlohr-Moul, Geiger, & Sauer, 2012; Ruocco, 2005; Schuermann, Kathmann, Stiglmayr, Renneberg, & Endrass, 2011) and prior research has not attempted to identify those unique to it. Therefore, using a healthy control group clarified the presence of impairment whereas incorporating an offender group with non-antisocial personality disorders matched on key variables facilitated the detection of deficits in the antisocial personality which were not observed in other personality disorders.

#### 7.3.1.5 Use of Multilevel Modelling and a structured measure of treatment progress

The present study involved retrospective data collection and there was no control over the clinical process of treatment reviews. This resulted in missing data and unequal intervals between treatment evaluation time-points, which would have been problematic to analyse with traditional ANOVA methods (Field, 2009; Tabachnick & Fidell, 2007). However, these are not obstacles for MLM (Field, 2009; Tabachnick & Fidell, 2007), which enabled the analysis of progress trajectories in this naturalistic dataset thereby enhancing statistical validity. At the same time, it was possible to factor in the differences in starting points and trajectories between each patient which better reflected both patient and group progress. Employing a structured, non-self-report measure to model treatment trajectories provided an

original methodological contribution in the field, since such a measure has not been available to date, and demonstrated promising validity though further corroboration remains necessary.

### 7.3.2 Limitations

In spite of several methodological strengths, the study also exhibited a number of weaknesses that potentially threaten the validity of the research. Four types of research validity are examined: internal, external, construct, and statistical (McBurney & White, 2007).

#### 7.3.2.1 Threats to internal validity

Internal validity refers to the extent to which a study provides evidence for the effects under scrutiny whilst minimising the plausibility of alternative explanations for the findings (McBurney & White, 2007). Although a number of steps were taken in the present study to rule out alternative explanations by controlling for potentially confounding variables, several threats to the internal validity of the research remained.

##### *7.3.2.1.1 Events outside the study*

The treatment regimen at the PDS has been broadly similar between patients but there is a range of factors that might have introduced bias, particularly as patient admissions took place over a decade. Different clinical teams, service targets, therapeutic approaches, life circumstances between the patients, and ongoing treatment developments may all have affected who was offered admission, how treatment progressed over the years, and how this progress was reflected in the reports. Although was not possible to control for all these biases reliably and realistically, the possibility that they were systematic at different times poses a threat to the internal validity of the research.

##### *7.3.2.1.2 Maturation*

Progress in treatment was quantified using PRS scores over time but this was uncontrolled (e.g. no baseline such as time in waiting list and no control group).

Therefore, change in PRS scores may reflect a treatment effect as well as natural maturation, a placebo effect, or other non-treatment-related processes. This limits the internal validity of the findings regarding the relationship between progress in treatment and neurocognitive performance.

#### *7.3.2.1.3 Sampling biases*

There were two sampling biases in the study: and admission rate of approximately 57% for referred patients and a participation rate of 76% for admitted patients. Although some reasons for exclusion from the service and the study supported the internal validity of the research (e.g. absence of major mental illness diagnosis) others may have introduced a degree of bias. For example, prospective or admitted patients who were excluded because of lack of motivation or disruptive behaviour are likely to reflect a common sub-type of individuals with antisocial personality and even imply pronounced neuropsychological impairment. Furthermore, whereas it was possible to establish that the 32 patients who were admitted but did not consent to the study or were discharged early were comparable to the remainder of the cohort on age, IQ, PCL-R scores, and number of personality disorders, differences may have existed in other variables including neuropsychological impairment. In addition, absence of such data on referred patients who were not offered admission does not permit such comparisons. Therefore, sampling bias remains a threat to the internal validity of the project in connection with both neuropsychological deficits and progress in treatment.

#### *7.3.2.1.4 Confounders and non-equivalent control groups*

Regarding the investigation into neuropsychological deficits, the groups were comparable on a range of demographic and clinical variables. However, use of different IQ and mental health measures between patients and healthy controls is likely to have introduced some error in establishing equivalence. Where differences between groups were detected on measured variables, statistical control examined their influence as covariates. This method provided some control for some potential confounders (e.g. age, IQ, years in education) but several threats remained, as discussed below.

*7.3.2.1.4.1 Substance abuse*

Although none of the participants in the study had any current substance misuse, one of the major confounders in the present project was comorbidity of the antisocial personality disorder with prior substance abuse. Substance abuse is overrepresented in populations with antisocial personality (De Brito & Hodgins, 2009; Hare, 2003) and can be deleterious to neuropsychological function (Lezak et al., 2004; Strauss et al., 2006) as well as being associated with substance abuse disorders (Baldacchino et al., 2012; Ersche & Sahakian, 2007; R. D. Rogers & Robbins, 2001). In the present project, a larger proportion of the ASPD and non-psychopathy groups had received a substance-related diagnosis than non-ASPD and psychopathy groups respectively. Though a history of SRD diagnosis was used to control for differences between patient groups where these occurred, this variable did not reflect different patterns of misuse between patients. Furthermore, details on substance abuse in the control group were not collected due to the ethical approvals of the project and substance-related diagnoses provide little information on the pattern and quantity of abuse (Spitzer & Endicott, 1978; First et al., 2002). As a result, it was not possible to control fully for prior substance abuse in the present project, which poses a threat to the internal validity of the findings.

*7.3.2.1.4.2 Socio-economic status and education*

Another potential limitation was the possibility that the healthy control and patient groups were not equivalent on socio-economic status and education. The majority of the healthy controls were recruited from ancillary and unqualified staff at the research setting. However, they spent more years in further education than the patients groups, which may have enhanced both cognitive ability and socio-economic status. While the groups seemed comparable in basic education, details on attendance and performance were not consistently reported in clinical records and most information was by self-report. As a result, it was not possible to control for educational differences fully.

*7.3.2.1.4.3 Medication*

Differences in prescribed medication may also pose a cause of concern. Participation for the healthy control group was conditional upon not receiving psychotropic medication but a proportion of the patient groups had been prescribed antidepressants, antipsychotics, or mood stabilisers. In addition, the ASPD and non-ASPD groups were different in the proportions of patient who were in receipt of antidepressant and antipsychotic medication. This did not seem to predict neuropsychological performance while none of these types of medication fall into the category of causing considerable cognitive side effects (Lezak et al., 2004). Nevertheless, some effects have been observed in relation to the CANTAB tests though they do not appear consistent for each medication type (e.g. second generation antipsychotics) and can vary considerably depending on the prescribed drug (Andersen et al., 2011; Chamberlain et al., 2006; Fagerlund, Mackeprang, Gade, Hemmingsen, & Glenthoj, 2004; McCartan et al., 2001; Tyson, Roberts, & Mortimer, 2004; Vollenweider, Barro, Csomor, & Feldon, 2006). The implication of this is that complex medication effects may have been present in the current findings but the analysis was not able to detect them. Though systematic bias might not be a cause of concern, medication effects may have inflated random error in the patient groups thus reducing statistical power.

*7.3.2.1.4.4 Intellectual functioning*

A mediating effect of IQ in the relationships between neuropsychological performance and progress in treatment was observed in motor regulation, risk-taking, and planning in ASPD. Control for IQ was possible using fixed effects models in the majority of the examined functions (models did not converge for the DMS and MTS tasks in ASPD). However, as the sample sizes were marginally sufficient for MLM in ASPD (except for the AGN, VRM, and CGT tests which were introduced more recently to the PDS) and below the recommended level in psychopathy, it is likely that the effect of IQ may have been underestimated in these cases due to loss of power. In addition, it was not possible to control fully for IQ using all necessary terms for MTS in psychopathy and several tests in ASPD (IED-response reversal, CGT-quality of decision-making, PAL-visual cued recall, VRM-verbal recall and recognition, SWM-

working memory). Furthermore, the models involving IQ did not converge at all for DMS, PAL errors, and MTS in ASPD therefore it was not possible to control for intellectual functioning in these cases. These considerations pose several threats to the internal validity of the study as they leave open the possibility that intellectual functioning may be able to explain a larger proportion of the observed relationships between neuropsychological performance and progress in treatment.

### 7.3.2 Threats to external validity

Threats to the external validity the project arose due to sample characteristics, the admission criteria of the PDS, and the study's inclusion criteria (which were necessary to maintain internal validity). The characteristics of the research setting posed further limits to the external validity of the research.

#### *7.3.2.1 Age and ethnicity*

As participants were adults and Caucasians, results may not generalise to younger individuals or individuals with other ethnic backgrounds. Furthermore, as individuals of older age were not represented adequately in the sample (mean age at first admission to the PDS was $M$=30.75, $SD$=8.64, with maximum age of 58.4 years), findings may not generalise to this population either.

#### *7.3.2.2 Intellectual functioning*

The PDS admission criterion of adequate intellectual abilities in combination with the study participation criterion of IQ≥70 suggests that the findings may not generalise to populations with learning difficulties. Furthermore, the sample may not be representative of individuals with higher intellectual functioning, as the average IQ scores of the participants tended towards the low average/average range (Wechsler, 1997).

#### *7.3.2.3 Major mental illness*

A diagnosis of major mental illness (psychosis & bipolar disorders) was reason for exclusion from the PDS and the study, as these conditions often involve substantial neuropsychological impairment (Fullam & Dolan, 2008; Lewandowski et al., 2011). It follows that findings may not generalise to individuals with ASPD and comorbid psychosis or bipolar disorder. However, this should not pose a significant threat to the external validity of the research as these disorders have not appeared representative of the psychiatric comorbidity encountered in either ASPD (De Brito & Hodgins, 2009) or psychopathy (Coid, Freestone, & Ullrich, 2012; Hare, 2003). In addition, while on the one hand including individuals with substance-related diagnoses threatened the

internal validity of the findings, on the other hand it supported the external validity of the research, as these are common in populations with antisocial personality (De Brito & Hodgins, 2009; Hare, 2003).

#### *7.3.2.4 Type of offending*

Both patient groups in the study were serving offenders and their socio-economic circumstances were poor. As the literature suggests that neuropsychological deficits between "successful" and "unsuccessful" individuals with psychopathy or offender and community samples can be different (e.g. Iria & Barbosa, 2009; Ishikawa et al., 2001), current findings both in neuropsychological performance and treatment progress may not generalise to individuals with antisocial personality who have not offended, have never been incarcerated, or have never had contact with the criminal justice service. Furthermore, as a history of sexual offences has been an exclusion criterion for the PDS, results may not generalise to this population well either.

#### *7.3.2.5 Voluntary referrals*

Referrals to the PDS are primarily voluntary, therefore patients are required to show a degree of motivation in order to be admitted and remain in treatment. Apart from this contributing the sampling biases discussed earlier in connection with internal validity, results may not generalise to individuals with antisocial personality who show little or no motivation to change from the outset.

#### *7.3.2.6 Single-site study*

The research was completed within a Medium Secure NHS Unit in England. Consequently, results may not generalise to low or high security and health services using different interventions and admission criteria (including the independent sector).

### 7.3.3 Threats to construct validity

These include limitations to the validity of both instruments and data (McBurney & White, 2007). A range of measures were employed in the present project for screening/sampling (e.g. diagnostic schedules, IQ, etc.) and for measuring variables in connection with the hypotheses (CANTAB & PRS). All measures except the PRS have been extensively researched and validated but limitations remain. In spite of some initial evidence supporting the validity of the PRS as a process measure, the need for further work was recognised. Differences between the measures for assessing patients and controls may also limit the construct validity of the findings.

### 7.3.4 Threats to statistical validity

A range of statistical analyses were conducted in this project and concerns with sample size, parametric assumptions, and power may pose considerable threats to the validity of some findings.

#### *7.3.4.1 Normality and homogeneity of variance*

Both assumptions appeared violated in several cases. Sample sizes were less than 30 in several analyses (particularly for the newer CANTAB tests AGN, VRM, and CGT and all analyses involving psychopathy) therefore violations to normality may have limited the validity of the results (Gravetter & Wallnau, 2009) whilst transformations were not helpful.

In addition, samples were not approximately equal between groups, therefore the ANOVA may have been less robust against heterogeneity of variance (Field, 2009). Although non-parametric tests confirmed findings where violations had been detected in between-subjects comparisons, it was not possible to examine this in mixed designs as there are no non-parametric alternatives to mixed ANOVA and MLM (Field, 2009). Consequently, for the SOC (number of moves to solution & thinking times), CGT, DMS, and SWM, violations of normality and homogeneity of variance may have reduced the validity of the results, particularly where samples were very small (CGT & psychopathy). MLM may have also been affected by violations to normality. Consequently, relevant findings should be viewed with caution and replication remains necessary.

#### *7.3.4.2 Statistical power and Type II error*

The relatively small samples in connection with in the newer CANTAB tests (AGN, VRM, and CGT) and psychopathy may indicate some loss of power. This can be particularly problematic for MLM where sample sizes under 50 may result in overestimated standard errors for fixed effects (Maas & Hox, 2005). Both reasons suggest inflated Type II error, particularly for small effects as was anticipated for most of the neuropsychological deficits. The WCST was such an example in the literature, where none of the identified studies reported a deficit in psychopathy but a meta-analysis revealed a significant effect.

The sensitivity of the CANTAB was considered a benefit in minimising measurement error and detecting relatively mild impairments, but some limitations were present. Although the battery has shown good reliability and internal consistency (Strauss et al., 2006), it has shown some lack of temporal stability. The majority of the tests have shown adequate or marginal test-retest reliability but measurements using the DMS, IED non-ED errors, the MTS, and SOC average number of moves have shown low test-retest coefficients (Cambridge Cognition, 2008; Strauss et al., 2006). These results could be due to practice effects (Cambridge Cognition, 2008) but nonetheless raise some concerns regarding the stability of the measurements. Therefore, tests of potentially lower stability may have resulted in inflated random error and therefore further loss of statistical power.

#### *7.3.4.3 Type I error*

The present project examined a range of neuropsychological tests and Bonferroni corrections were employed within the same clusters of analysis to limit familywise error. The large number of analyses, however, means that some effects may have resulted by chance. It follows that caution is required when interpreting the findings and replication with a narrower focus remains necessary.

#### *7.3.4.4 MLM convergence and random effects.*

Using MLM to examine progress in treatment enabled the use of data collected at different time points for each patient within the same model. Although this was a

superior technique compared to the alternative of repeated-measures ANOVA, some models did not converge when examining random effects. This occurred in varying degrees for most tests (except the RVP, AGN, and IED for psychopathy, which converged for all random effects). It follows that results remain tentative until further investigation enables the examination of the influence of random effects further.

### 7.3.5 Summary

The study demonstrated a range of methodological strengths including a range of controls for potential confounders, parallel examination of ASPD and psychopathy, sensitive assessment of cognitive function, multiple comparison groups, and relatively large samples formed from the majority of a patient cohort. Further advantages included the use of a structured measure to assess treatment progress (not previously available in the field) and MLM to enable detailed longitudinal analysis and enhance statistical validity.

In spite of a number of strengths, the present project also showed several weaknesses. Events outside the study, absence of a control group in the examination of progress in treatment, potential sampling biases, and confounding variables such as substance abuse may limit the internal validity of the research. In addition, sample characteristics and the single-site of the study may restrict external validity. Furthermore, measurement differences between participant groups and limitations to the validity of the instruments – particularly the PRS – may reduce construct validity. Finally, assumption violations, lack of statistical power, potentially inflated Type I error, and unexamined random effects in some multilevel models, may pose threats to the statistical validity of the research. Consequently, caution remains essential when considering the present findings.

## 7.4 Implications for future research

The examination of neuropsychological deficits in the antisocial personality provided further evidence of impairment in this population suggesting that examining cognitive function might be helpful in understanding the antisocial personality. However, the results regarding progress in treatment were limited with some aspects of neuropsychological performance able to predict only a subset of progress scores in ASPD but not psychopathy. Although the project demonstrated several strengths, its methodological weaknesses indicate the need for replication and further research.

Overall, replication with other, larger samples is necessary. This is particularly the case for psychopathy in connection both with some neuropsychological functions (motor regulation, verbal memory, and decision-making) and with the relationship between cognitive performance and progress in treatment where samples were small. Future investigations should also endeavour to limit sampling bias and particularly admission bias. In addition, it is important to investigate the mediating effect of potential confounders further, particularly prior history of substance abuse, which is likely to account for a substantial portion of neuropsychological impairment (Baldacchino et al., 2012; Ersche & Sahakian, 2007; Lezak et al., 2004; R. D. Rogers & Robbins, 2001).

### 7.4.1 Neurocognitive deficits

Although evidence suggested a range of neurocognitive impairments in the antisocial personality, findings merit further investigation. Offenders with antisocial personality performed sufficiently worse than controls on several occasions and the differences reached significance. However, on several occasions, offenders with other personality disorders seemed to perform somewhere between their antisocial counterparts and controls but these differences did not reach significance. Although this implied that significant effects of deficit in antisocial individuals compared to controls were more robust or consistent than deficits in individuals with other personality disorders, the absence of significant effects in connection with the latter limited conclusions. As a result, there is lack of clarity in the present findings regarding which deficits might underlie the antisocial personality and which might predict offending or personality disorder pathology generally. It is therefore important

that future research examine these effects further with the aim of establishing the extent of additional impairment in offenders with antisocial personality.

Further research in the similarities and differences between the operationalisations of antisocial personality is also required. The samples with ASPD and psychopathy were overlapping in the present project but only some control was possible via excluding individuals with psychopathy from the group with ASPD. It follows that it is important to replicate deficits in individuals with and without psychopathy who do not meet criteria for ASPD. In addition, the functions of motor regulation, response reversal, and visual perception merit further research as they appeared impaired in both ASPD and psychopathy. Because it seems these operations might be important in understanding the antisocial personality, future research should attempt to confirm findings and clarify the role of these functions in its development. Equally, findings indicated that ASPD may be more fronto-temporally impaired than psychopathy. The latter may reflect more affective deficits than ASPD but, as the scope of the CANTAB was limited in this respect, this remains an important question for future research.

Confirming findings in connection with unsupported hypotheses and further exploring related deficits will also be important. These concern potential deficits in WM and visual perception in ASPD and sustained attention in psychopathy, which had not been supported by prior literature and might reflect Type I error in this project. In addition, the absence of an attentional set-shifting deficit in psychopathy in the present project requires further investigation in better controlled conditions. This is because the potential impairment in psychopathy appears to be mild in the literature and the ability to detect an effect could be compromised by measurement error and loss of power.

ASPD appeared more impaired than psychopathy in the examined fronto-temporal functions. However, the literature has highlighted other potential deficits (with varying degrees of clarity), particularly in affective and language processes in psychopathy. As it was not possible to assess these functions in the present project due to a limitation of the CANTAB, it follows that further research should extend the investigation to these operations as they appear highly relevant in understanding the antisocial personality and contrasting its different conceptualisations.

Overall, divergence between ASPD and psychopathy appeared to complicate findings and impede conclusions. It follows, that future research may benefit by incorporating the triarchic conceptualisation of psychopathy as more compatible with neuropsychological findings (Patrick et al., 2009, 2012) which, therefore, may identify a less heterogeneous as well as more relevant population.

#### 7.4.1.1 Neurodevelopmental aetiology

Having demonstrated the presence of neuropsychological deficits and impairments in the antisocial personality not encountered in other personality disorders, future research could ask how these might have developed. On the one hand, there is support for a genetic predisposition in the antisocial personality (R. Blair, Peschardt, Budhani, Mitchell, & Pine, 2006; McGruffin & Thapar, 1998) which may direct neurological development and neurochemistry in this population (R. Blair, 2006; Viding, 2004; Viding, Blair, Moffitt, & Plomin, 2005; Viding, Jones, Frick, Moffitt, & Plomin, 2008). On the other hand, a range of environmental influences is also likely to play a key role in the process but research is lacking (Raine, 2008). Since affective, executive, and memory deficits seem to characterise the antisocial personality, focus should be on the limbic system and PFC (D. L. Clark et al., 2010), which is also consistent with the neurological observations in this population (R. Blair et al., 2005; Koenigs et al., 2011).

The limbic system and PFC are two highly interconnected regions (D. L. Clark et al., 2010). In addition, the development of the latter, which matures later in life, is affected by input from the former implicating inhibitory and social functions (Barber, Caffo, Pekar, & Mostofsky, 2013; Gogtay et al., 2004; Krüger, Brockmann, Salamon, Ittrich, & Hanganu-Opatz, 2012). Both regions appear susceptible to early life damage in environments involving stress, adversity, and trauma, with causes potentially including neurotoxic effects of cortisol secretion (Carrion & Wong, 2012; Dillon et al., 2009; Mychasiuk, Gibb, & Kolb, 2011) and even epigenetic influences (Kofink, Boks, Timmers, & Kas, 2013). This is important in light of strong evidence to suggest that early adversity is highly prevalent in personality disorders and particularly the antisocial type (Afifi et al., 2010; R. Blair et al., 2006; De Brito &

Hodgins, 2009; Grover et al., 2007) because it might explain limbic-PFC deficits in this population.

Developmentally, limbic-PFC impairments generally predict impulsiveness and socially unhelpful behaviour (Barber et al., 2013) which could also lead to substance abuse, which is highly prevalent in the antisocial personality (De Brito & Hodgins, 2009; Hare, 2003; Khalifa et al., 2012; Krueger, Markon, Patrick, Benning, & Kramer, 2007), and therefore further neurotoxicity (Lezak et al., 2004) thereby exacerbating the deficits. Neuropsychological findings might thus provide a plausible explanation for the development of the antisocial personality and provide a potentially fruitful avenue for further research with implications for prevention.

### 7.4.2 Progress in treatment

Results provided some initial but limited evidence to suggest that neuropsychological deficits may predict progress in treatment – at least in ASPD. Since findings did not appear robust, future research should focus on replication and addressing some of the methodological limitations that emerged in this project. These were particularly in connection with measuring progress and sampling for psychopathy.

As progress in treatment was measured with the newly developed PRS, further validation of the instrument appears necessary. This will also enable better interpretation of the results in connection with the different PRS parts. In addition, larger samples will be important to improve power, address threats to normality (Gravetter & Wallnau, 2009) and facilitate convergence of the models in order to maximise control for both IQ and random effects (Tabachnick & Fidell, 2007). The latter are essential in order to model individual progress accurately as patients exhibited both different starting points in treatment and variance in trajectories over time. Exploring additional mediating factors in MLM (e.g. personality) would also become possible with larger samples while using a suitable control group (e.g. patients on a waiting list) would provide some control for maturation, placebo, and cohort effects.

Further replication and investigation of some unexpected findings may also be of benefit. These include the positive relationship between risk-taking and progress in

treatment in ASPD and the conflicting findings regarding planning in psychopathy. It would be important to clarify whether risk-taking facilitates change (e.g. Lorian & Grisham, 2011) or whether it reflects benefits of a treatment programme designed to address such deficits. The effect of planning was more complex and future research should first attempt to replicate findings whilst addressing methodological limitations, for example a small sample and potential practice effects.

Finally, extending the investigation to include risk assessments alongside (or as alternative to) the PRS is also worthwhile. Risk is a key outcome in the treatment of personality disorders in secure settings (Ministry of Justice, 2011; NICE, 2009) and forensic healthcare in general (McMurran et al., 2009) while treatments generally operate on the basis of addressing criminogenic needs (Andrews, Bonta, & Wormith, 2011; Heilbrun, Yasuhara, & Shah, 2010). Risk assessments have already been used successfully as indicators of progress to investigate treatment effectiveness and results mirror those of the PRS (Wong & Olver, 2010). Consequently, examining change in risk assessments during treatment appears highly relevant in the endeavour to investigate the potential effect of neuropsychological deficits on progress.

## 7.5 Conclusions

The project attempted to further clarify the presence of neuropsychological deficits in the antisocial personality using the CANTAB and then investigate their relationship to progress in treatment. The antisocial personality has proven both difficult to define and challenging to treat with three divergent operationalisations in mainstream use, poor intervention outcomes, and high attrition rates. There is a substantial body of literature on the neuropsychological deficits associated with this personality type. The systematic review revealed some robust deficits but the majority of the findings lacked consistency thereby highlighting the need for further and more detailed investigation. Furthermore, in spite of the breadth of neuropsychological research in the field and evidence suggesting poorer treatment outcomes in various clinical populations, no study has yet examined the relationship between neurocognitive deficits and progress in treatment in the antisocial personality.

The results further supported the usefulness of neuropsychological research in fostering an understanding of the antisocial personality. An array of deficits characterised ASPD and psychopathy compared to healthy controls and other personality disorders. The broad network of deficits in ASPD included fronto-temporal, attentional, and visual processing operations, but psychopathy appeared less impaired than ASPD and did not exhibit deficits in temporal (memory) functions. Although many deficits may be associated with criminality/personality disorder more generally, impairments in motor regulation, risk-taking, WM, and particularly in response reversal, and visual perception, were present in the antisocial personality (ASPD and psychopathy) but not patients with other personality disorders. These results extend the current evidence base by clarifying the presence of impairments in a range of functions, examining ASPD and psychopathy in parallel, and comparing the antisocial personality to other personality disorders, but the effects of different operationalisations of the antisocial personality were difficult todelineate due to substantial overlap. Although results pave the way towards a better understanding of the aetiology and difficulties associated with this personality type, further research is required to confirm findings, address limitations, and extend the investigation to functions not assessed by the CANTAB such as affective and language operations.

On the other hand, the project was less successful in making an initial attempt to demonstrate a relationship between neuropsychological deficits in individuals with antisocial personality and progress in treatment. The instrument what was developed to measure progress showed promise but the evidence in relation to neuropsychological performance was limited showing negative associations between a subset of the impaired neuropsychological functions and progress through the forensic care pathway in ASPD only. Though cognitive impairment may indeed not predict many aspects of progress in treatment in the antisocial personality, unlike other clinical populations, methodological limitations indicate the need of replication and further research.

Perhaps one of the most critical observations throughout the project was the discrepancy between ASPD and psychopathy, notwithstanding the two groups being subsets of the same cohort. Though a degree of discrepancy was expected in light of the different conceptualisations between the two definitions, findings imply a more fundamental difference extending to the neuropsychological level. However, it is unclear how valid each definition is in defining the antisocial personality. As such lack of convergence might become an obstacle in both clinical and research work resulting in misleading and contradictory findings, it is vital to pursue better conceptual integration which remains informed by the scientific evidence.

In concluding this work, it might be helpful to consider some the wider issues surrounding it. The findings are in line with the literature suggesting a neurological basis for the antisocial personality, which, alongside evidence suggesting genetic, environmental, and societal influences in its development, raise some epistemological questions. Perhaps the most important one concerns the responsibility surrounding the antisocial personality and the extent to which it lies within the individual versus society. This raises questions regarding the extent to which treatments will ever be truly effective as long as they continue to focus on the individual and whether a more radical shift towards systems, society, and prevention, might confer greater benefits. In spite of the recent advances in the field, the evidence is still not sufficiently robust to answer such questions highlighting the need to continue investigating this debilitating condition with a critical and open mind.

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# 9 APPENDIX A: LITERATURE SEARCH TERMS

*Part 1: Antisocial Personality*

1. (antisocial personality disorder$ or dissocial personality disorder or Psychopathy).sh,id. [2]
2. (apd$1.tw. and (asocial$ or anti social$ or antisocial$ or character$ or dissocial$ or dis social$ or person$).mp.) or aspd$1.tw.
3. ((asocial$ or antisocial$ or anti social$ or dissocial$ or dis social$) adj3 (character$ or difficult$ or disorder$ or dysfunction$ or PD or person$)).tw. or ((asocial$ or antisocial$ or anti social$ or dissocial$ or dis social$) and personalit$).tw,hw.
4. (neuroPsychopath$ or Psychopath$3 or psycho path$3 or sociopath$ or socio path$).tw.
5. or/1-4

*Part 2: Neuropsychology*

General terms

PsycINFO. (cantab or neuropsych$ or neurocogniti$ or $frontal or prefrontal or orbitofrontal or parietal or temporal or occipital).mp. or exp Neuropsychology/ or exp Neuropsychological assessment/ or exp Neurocognition/

MEDLINE. (cantab or neuropsych$ or neurocogniti$ or $frontal or prefrontal or orbitofrontal or parietal or temporal or occipital).mp. or exp Neuropsychological Tests/ or exp Neuropsychology/

EMBASE. (cantab or neuropsych$ or neurocogniti$ or $frontal or prefrontal or orbitofrontal or parietal or temporal or occipital).mp. or exp Task performance/ or exp Neuropsychological test/ or exp Neuropsychology/

Frontal functions

PsycINFO. (executive or (rule and (acqui$ or revers$)) or ((Behavio$ or response) and (inhibition or disinhibition)) or set shift$ or self order$ or fluency or persever$).mp. or (decision making).mp. or exp Decision making/ or exp Executive function/ or exp Behavioral Disinhibition/

MedLine. (executive or (rule and (acqui$ or revers$)) or ((Behavio$ or response) and (inhibition or disinhibition)) or set shift$ or self order$ or fluency or persever$).mp. or (decision making).mp. or exp Decision making/ or exp Verbal behavior/

EMBASE. (executive or (rule and (acqui$ or revers$)) or ((Behavio$ or response) and (inhibition or disinhibition)) or set shift$ or self order$ or fluency or persever$).mp. or (decision making).mp. or exp Perseveration/ or exp Decision making/

Temporal functions

PsycINFO.

[2] MEDLINE & EMBASE: (antisocial personality disorder$ or dissocial personality disorder or Psychopathy).sh,af. The terms were entered in this original form in PsycINFO.

1. exp Memory/ or exp Explicit Memory/ or exp Spatial Memory/ or exp Memory for Designs Test/ or exp Episodic Memory/ or exp Verbal Memory/ or exp Autobiographical Memory/ or memory.mp. or exp Short Term Memory/ or exp Long Term Memory/ or exp Visuospatial Memory/ or exp Semantic Memory/ or exp Memory Decay/ or exp Iconic Memory/ or exp Visual Memory/ or exp Implicit Memory/ or exp Memory disorders/
2. exp Nonreversal Shift Learning/ or exp Learning Ability/ or exp Verbal Learning/ or learn$.mp. or exp Nonsense Syllable Learning/ or exp Paired Associate Learning/ or exp Perceptual Learning/ or exp Spatial Learning/ or exp Learning/ or exp Reversal Shift Learning/ or exp Perceptual Motor Learning/ or exp Sequential Learning/ or exp Nonverbal Learning/ or exp Serial Learning/ or exp Discrimination learning/
3. (recall or recognition or acquisition or ((auditory or information) and (process$ or perce$)) or visu$).mp. or exp Auditory perception/ or exp visual perception/ or exp visuospatial ability/

MEDLINE.

1. memory.mp. or exp Memory/ or exp Memory, Short-Term/ or exp Memory Disorders/
2. exp Paired-Associate Learning/ or exp Verbal Learning/ or exp Learning/ or exp Reversal Learning/ or learn$.mp. or exp Discrimination Learning/ or exp Avoidance Learning/ or exp Association Learning/ or exp Serial Learning/
3. (recall or recognition or acquisition or ((auditory or information) and (process$ or perce$)) or visu$).mp. or exp Mental recall/ or exp Pattern Recognition, Automated/ or exp Pattern Recognition, Visual/ or exp Pattern Recognition, Physiological/ or exp "Recognition (Psychology)"/ or exp Auditory perception/ or exp Visual perception/

EMBASE.

1. exp memory/ or exp short term memory/ or exp autobiographical memory/ or exp procedural memory/ or exp spatial memory/ or exp auditory memory/ or exp reference memory/ or exp associative memory/ or exp memory consolidation/ or exp tactile memory/ or exp working memory/ or exp visual memory/ or exp explicit memory/ or memory.mp. or exp implicit memory/ or exp sensory memory/ or exp memory disorder/ or exp declarative memory/ or exp verbal memory/ or exp long term memory/ or exp semantic memory/ or exp episodic memory/
2. learn$.mp. or exp learning test/ or exp discrimination learning/ or exp learning/ or exp paired associate learning/ or exp experiential learning/
3. (recall or recognition or acquisition or ((auditory or information) and (process$ or perce$)) or visu$).mp. or exp recall/ or exp word list recall/ or exp recognition/ or exp automated pattern recognition/ or exp word recognition/ or exp automatic speech recognition/ or exp pattern recognition/ or exp auditory discrimination/ or exp evoked auditory response/ or exp auditory orientation/ or exp auditory response/ or exp visual information/ or exp information processing/ or exp information retrieval/ or exp information storage/ or exp visual impairment/ or exp visual orientation/ or exp visual discrimination/ or exp evoked visual response/ or exp visual information/ or exp visual threshold/ or exp depth perception/

Parietal functions

PsycINFO.

1. (touch or tactile or tactual or apraxia or speech or verbal or reading or somatosensory).mp. or exp Sensory Neglect/ or exp Tactual Stimulation/ or exp Tactual Perception/ or exp Cutaneous Sense/ or exp Reading/ or exp Reading Comprehension/ or exp Speech perception/ or exp Verbal Fluency/ or exp Verbal Ability/ or exp Verbal Comprehension/ or exp Verbal Tests/
2. exp Spatial Organization/ or exp Spatial Perception/ or exp Spatial Distortion/ or exp Spatial Learning/ or exp Spatial Ability/ or exp Spatial Imagery/ or exp "Spatial Orientation (Perception)"/ or spatial.mp.

MEDLINE.

1. (touch or tactile or tactual or apraxia or speech or verbal or reading or somatosensory or neglect).mp. or exp Touch/ or exp Touch perception/ or exp Apraxias/ or exp Speech Discrimination Tests/ or exp Speech Perception/ or exp Speech/ or exp Speech Articulation Tests/ or exp Reading/ or exp Verbal behavior/
2. Spatial.mp. or exp Space Perception/ or exp Spatial Behavior/

EMBASE.

1. (touch or tactile or tactual or apraxia or speech or verbal or reading or somatosensory or neglect).mp. or exp touch/ or exp tactile discrimination/ or exp tactile stimulation/ or exp apraxia/ or exp "speech and language assessment"/ or exp speech/ or exp speech articulation/ or exp speech discrimination/ or exp speech intelligibility/ or exp "speech and language"/ or exp speech perception/ or exp reading/ or exp evoked somatosensory response/ or exp verbal behavior/
2. spatial.mp. or exp spatial discrimination/ or exp spatial orientation/

Occipital functions

PsycINFO. ((Colo$ or form or shape or movement or motion) and perception).mp. or exp "Form and Shape Perception"/ or exp Color Perception/ or exp Motion Perception/ or agnosia.mp. or exp Agnosia/

MEDLINE. ((Colo$ or form or shape or movement or motion) and perception).mp. or exp Color Perception/ or exp Color Perception Tests/ or exp Form perception/ or exp Motion perception/ or exp Agnosia/

EMBASE. ((Colo$ or form or shape or movement or motion) and perception).mp or exp color vision defect/ or exp color vision test/ or exp color discrimination/ or exp color vision/ or exp distance perception/ or exp movement perception/

Broader functions

PsycINFO.

1. (attention or vigilance).mp. or exp Sustained Attention/ or exp Divided Attention/ or exp Attention/ or exp Attention Span/ or exp Visual Attention/ or exp Selective Attention/ or emotion$.mp. or exp Emotions/ or language.mp. or exp Language/ or exp Language development/ or exp Language disorders/ or (perceptual orientation).mp. or exp Perceptual orientation/
2. Empathy.mp. or exp Empathy/ or ((complex and figure) and (test or task)).mp. or (affect and (recogni$ or process$)).mp. or (response modulat$).mp. or (Moral and (reason$ or judg$)).mp. or (Defining and Issues and (task or test)).mp.
3. (Theory of mind).mp. or exp "theory of mind"/
4. exp Prisoners Dilemma Game/ or prisoner$ dilemma.mp.
5. Attribution$.mp. or exp Attribution/

MEDLINE.

1. (attention or vigilance).mp. or exp Attention/ or emotion$.mp. or exp Emotions/ or language.mp. or exp Language Disorders/ or exp Language Development/ or exp Language Tests/ or exp Natural Language Processing/ or exp Language/ or (perceptual orientation).mp.
2. Empathy.mp. or exp Empathy/ or ((complex and figure) and (test or task)).mp. or (affect and (recogni$ or process$)).mp. or (response modulat$).mp. or (Moral and (reason$ or judg$)).mp. or (Defining and Issues and (task or test)).mp.
3. (Theory of mind).mp. or exp "theory of mind"/
4. prisoner$ dilemma.mp.
5. Attribution$.mp.

EMBASE.

1. (attention or vigilance).mp. or exp attention/ or exp selective attention/ or exp attention disturbance/ or emotion$.mp. or exp emotion/ or language.mp. or exp language processing/ or exp "speech and language"/ or exp language ability/ or exp language test/ or exp natural language processing/ or exp language/ or exp "speech and language assessment"/ or exp written language/ or exp language development/ or (perceptual orientation).mp.
2. Empathy.mp. or exp Empathy/ or ((complex and figure) and (test or task)).mp. or (affect and (recogni$ or process$)).mp. or (response modulat$).mp. or (Moral and (reason$ or judg$)).mp. or (Defining and Issues and (task or test)).mp.
3. (Theory of mind).mp.
4. prisoner$ dilemma.mp.
5. Attribution$.mp.

Intelligence

PhycINFO. exp Stanford Binet Intelligence Scale/ or intelligence.mp. or exp Intelligence/ or exp Wechsler Adult Intelligence Scale/ or exp Slosson Intelligence Test/ or exp Intelligence Measures/ or exp Culture Fair Intelligence Test/ or exp Wechsler Bellevue Intelligence Scale/ or exp Intelligence Quotient/ or IQ.mp. or WAIS.mp. or NART.mp.

MEDLINE. Exp Intelligence/ or exp Intelligence Tests/ or exp Wechsler Scales/ or WAIS.mp. or intelligence.mp.

EMBASE. Exp Wechsler Intelligence Scale/ or exp intelligence test/ or exp intelligence quotient/ or intelligence.mp. or exp intelligence/ or exp Stanford-Binet Intelligence Scale/ or intelligence.mp.

Motor functions

PsycINFO. exp Motor Performance/ or exp Perceptual Motor Coordination/ or exp Motor Processes/ or exp Motor Coordination/ or exp Perceptual Motor Development/ or $motor.mp. or exp Perceptual Motor Processes/ or exp Motor Skills/ or exp Gross Motor Skill Learning/ or exp Fine Motor Skill Learning/ or exp Perceptual Motor Learning/ or hand dynamometry.mp. or finger tapping.mp. or exp Finger Tapping/ or sequencing.mp.

MEDLINE. Exp Motor Activity/ or exp Motor Skills Disorders/ or exp Motor Skills/ or exp Psychomotor performance/ or $motor.mp. or hand dynamometry.mp. or finger tapping.mp. or sequencing.mp.

EMBASE. exp motor development/ or exp motor coordination/ or exp motor control/ or exp motor dysfunction/ or exp motor performance/ or exp motor activity/ or $motor.mp. or hand dynamometry.mp. or finger tapping.mp. or sequencing.mp.

Frontal tests

PsycINFO.

1. (ssp or "spatial span" or "visuospatial span" or corsi or "digit span" or knox).mp. or (ied or id ed or "id-ed" or extra dimension$ shift$ or extradimension$ shift$ or Wisconsin Card or WCS$).mp. or exp Wisconsin Card Sorting Test/ or (soc or "SOC" or tower$).mp. or swm.mp.
2. (Stroop or "category test" or "category task" or "Halstead-Reitan" or "Halstead Reitan" or healthy controlT or Porteus or "trail making" or "trail-making" or partington or "colo$ trail" or CTT or TMT).mp. or ("Gambl$ task" or "Gambl$ test" or IGT or CGT).mp. or ((Token or spelling or "phonetic discrimination") and (test or task)).mp. or exp Stroop Effect/ or exp Stroop Color Word Test/ or exp Halstead Reitan Neuropsychological Battery/
3. (agn or "go no go" or nogo or "no-go").mp.

MEDLINE.

1. (ssp or "spatial span" or "visuospatial span" or corsi or "digit span" or knox).mp. or (ied or id ed or "id-ed" or extra dimension$ shift$ or extradimension$ shift$ or Wisconsin Card or WCS$).mp. or (soc or "SOC" or tower$).mp. or swm.mp.
2. (Stroop or "category test" or "category task" or "Halstead-Reitan" or "Halstead Reitan" or healthy controlT or Porteus or "trail making" or "trail-making" or partington or "colo$ trail" or CTT or TMT).mp. or ("Gambl$ task" or "Gambl$ test" or IGT or CGT).mp. or ((Token or spelling or "phonetic discrimination") and (test or task)).mp. or exp Trail Making Test/
3. (agn or "go no go" or nogo or "no-go").mp.

EMBASE.

1. (ssp or "spatial span" or "visuospatial span" or corsi or "digit span" or knox).mp. or (ied or id ed or "id-ed" or extra dimension$ shift$ or extradimension$ shift$ or Wisconsin Card or WCS$).mp. or (soc or "SOC" or tower$).mp. or swm.mp. or exp Wisconsin Card Sorting Test/
2. (Stroop or "category test" or "category task" or "Halstead-Reitan" or "Halstead Reitan" or healthy controlT or Porteus or "trail making" or "trail-making" or partington or "colo$ trail" or CTT or TMT).mp. or ("Gambl$ task" or "Gambl$ test" or IGT or CGT).mp. or ((Token or spelling or "phonetic discrimination") and (test or task)).mp.
3. (agn or "go no go" or nogo or "no-go").mp.

Parietal and temporal tests

PsycINFO.

1. (((Discrimination or Seguin or Board or "tactile pattern$" or "line bisection" or Gollin or Mooney or dichotic or "logical stor$" or "right left differentiation" or "left right differentiation" or "right-left differentiation" or "left-right differentiation" or Kimura or McGill or Rey) and (test or task)) or Wechsler memory or WMS).mp. or exp Wechsler Memory Scale/
2. vrm.mp. or (pal or pair$ associate$ learn$).mp. or (dms or "delayed matching to sample" or matching).mp.

MEDLINE.

1. (((Discrimination or Seguin or Board or "tactile pattern$" or "line bisection" or Gollin or Mooney or dichotic or "logical stor$" or "right left differentiation" or "left right differentiation" or "right-left differentiation" or "left-right differentiation" or Kimura or McGill or Rey) and (test or task)) or Wechsler memory or WMS).mp. or exp Wechsler Scales/
2. vrm.mp. or (pal or pair$ associate$ learn$).mp. or (dms or "delayed matching to sample" or matching).mp.

EMBASE.

1. (((Discrimination or Seguin or Board or "tactile pattern$" or "line bisection" or Gollin or Mooney or dichotic or "logical stor$" or "right left differentiation" or "left right differentiation" or "right-left differentiation" or "left-right differentiation" or Kimura or McGill or Rey) and (test or task)) or Wechsler memory or WMS).mp. or exp Wechsler Memory Scale/
2. vrm.mp. or (pal or pair$ associate$ learn$).mp. or (dms or "delayed matching to sample" or matching).mp.

Attention and information processing tests

PsycINFO, MEDLINE, and EMBASE. (rvp or "continuous performance" or "concentration endurance").mp. or (mts or "speed accuracy trade$").mp.

Soft signs

PsycINFO, MEDLINE, and EMBASE. soft signs.mp.

Terms for completion: Cognition and planning

PsycINFO.

1. Cogniti$.mp. or exp Cognitive Ability/ or exp Cognitive Impairment/ or exp Cognitive Processes/ or exp Cognition/
2. Plan$.mp.

MEDLINE

1. Cogniti$.mp. or exp Cognition Disorders/ or exp Cognition/
2. Plan$.mp.

EMBASE.

1. Cogniti$.mp. or exp cognition/ or exp cognitive defect/ or exp mild cognitive impairment/
2. Plan$.mp. or exp strategic planning/

# 10 APPENDIX B: QUALITY RATING SCALE PLOTS

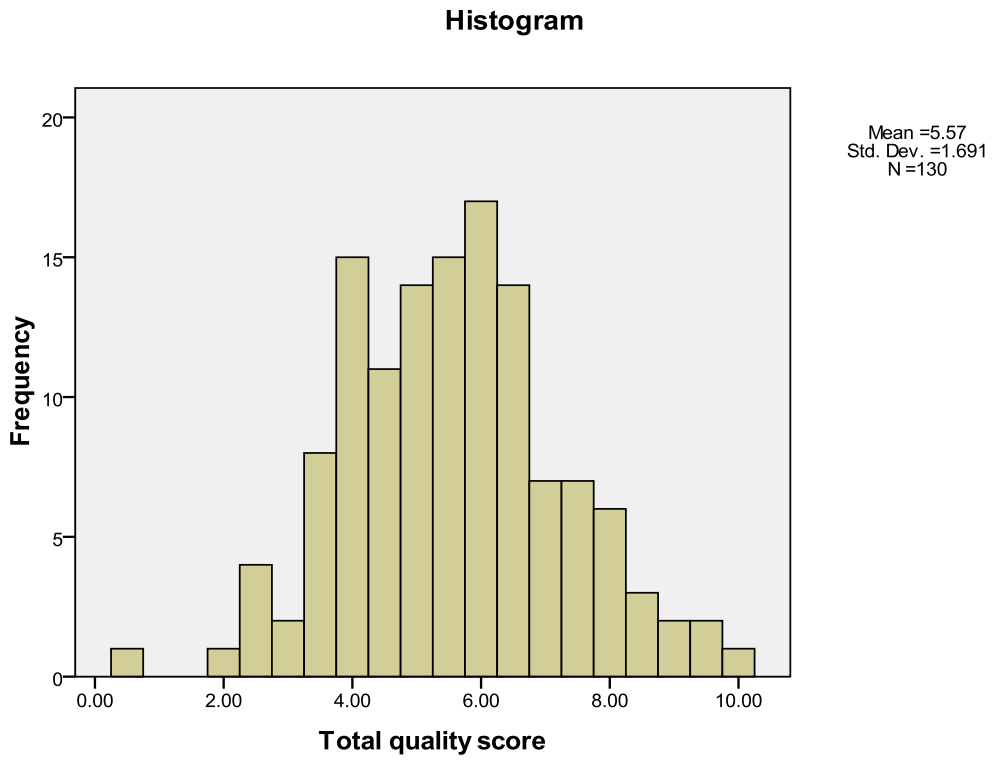


*Figure 10.1.* Distribution of quality scores of 130 publications and dissertations using the Quality Rating Scale, showing an approximately normal distribution.

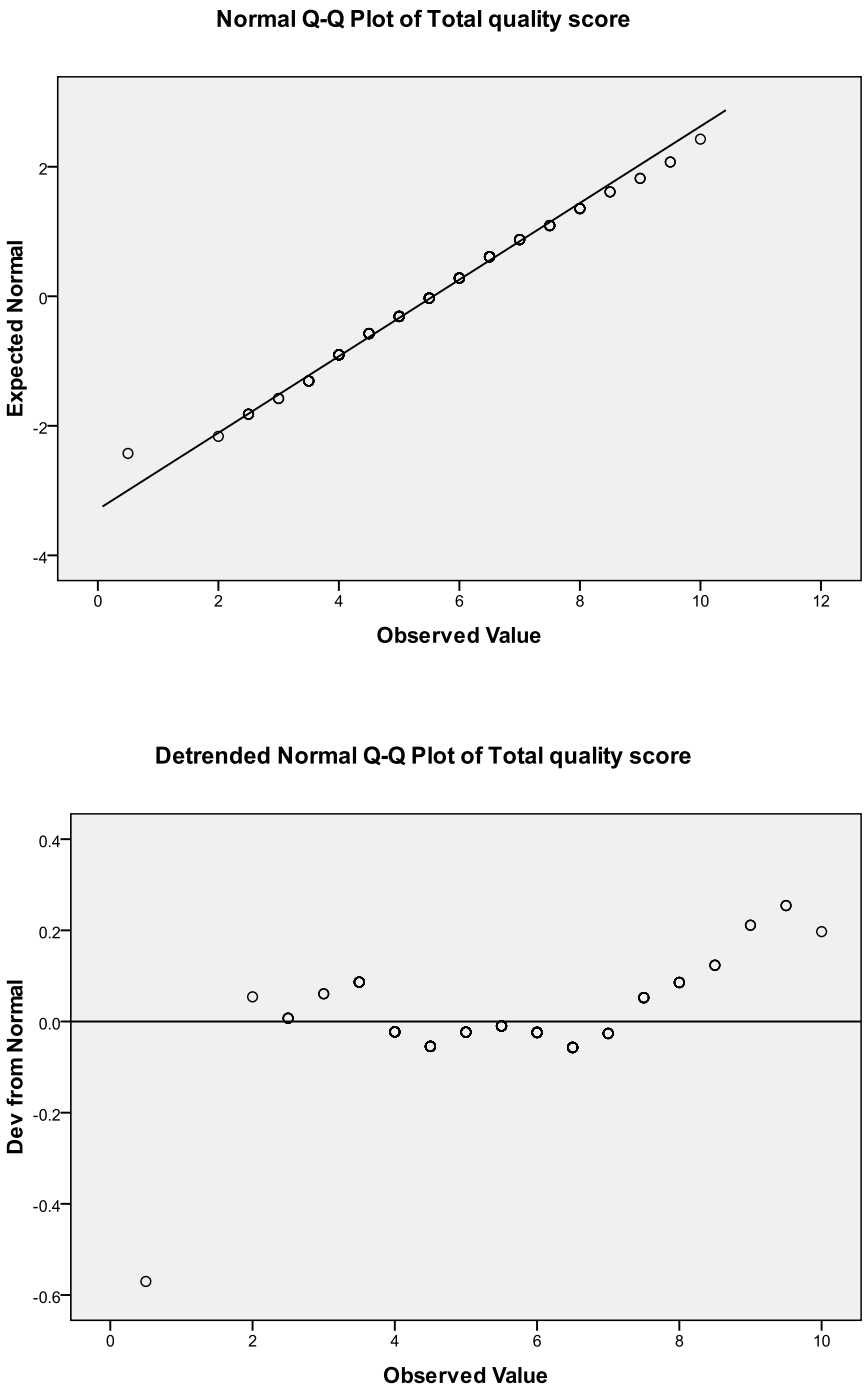


*Figure 10.2.* Normality plots of score distributions for 130 publications and dissertations using the Quality Rating Scale, supporting an approximately normal distribution.

# 11 APPENDIX C: STUDIES INCLUDED IN THE SYSTEMATIC REVIEW

Table 11.1. *Studies which examined cognitive functions in the antisocial personality.*

| **Publication/ study** | **Quality** | **Cognitive functions** | **Groups** | **Population** | **Diagnostic** | ***n*** | **Age (years)** | | **IQ** | | **Education (years)** | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | *M* | *SD* | *M* | *SD* | *M* | *SD* |
| ***ASPD*** | | | | | | | | | | | | |
| Barkataki 2005 | H | *Executive*: Planning; WM; Self-regulation (cognitive flexibility, motor regulation); Effective performance. | ASPD | Forensic inpatients | SCID-II | 12-14 | 33.5 | 10.5 | 94.9 | 11.8 | | |
| | | *Memory*: STM, LTM, WM<br>*Attention*: Sustained; Selective<br>*Intelligence*: FSIQ; VIQ, PIQ | HC | General public | SCID-NP | 12-15 | 32.1 | 7.47 | 104.3 | 14.5 | | |
| Barkataki 2008 | H | *Executive*: Self-regulation (motor regulation) | ASPD | Forensic inpatients | SCID-II | 14 | 33.5 | 10.5 | 94.9 | 11.8 | | |
| | | *Intelligence*: FSIQ | HC | General public | SCID-NP | 14 | 33.1 | 7.8 | 104.6 | 15.0 | | |
| Dolan 2002 | H | *Executive*: Planning; Self-regulation (cognitive flexibility, motor regulation) | ASPD | Forensic inpatients | SCID-II | 28-29 | 41.0 | 9.5 | 103.2 | 11.8 | 12.3 | 0.9 |
| | | *Memory*: STM<br>*Perception*: Visual<br>*Intelligence*: VIQ | HC | Hospital staff | SCID-II | 20 | 37.7 | 7.7 | 106.9 | 11.8 | 12.9 | 1.4 |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Gawda 2008a | H | *Language*: Academic skills (writing) | ASPD | Prison | DSM-IV-TR | 50 | 35.5 | 11 | 99 | 9 | 10.2 | 1.5 |
| | | *Intelligence*: FSIQ | Non-ASPD | | | 40 | 34.2 | 10 | 99 | 9 | 10.2 | 1.8 |
| | | | HC | General public | DSM-IV-TR | 50 | 33.5 | 9.8 | 100 | 9 | 10.4 | 1.5 |
| Gawda 2008b | M | *Affect*: Processing | ASPD | Prison | DSM-IV-TR | 60 | 35.5 | 11 | 102 | 10 | 10.3 | 1.8 |
| | | *Language*: Verbal expression (writing) | Non-ASPD | | | 40 | 34.2 | 10 | 99 | 9 | 10.4 | 1.5 |
| | | *Intelligence*: FSIQ; VIQ; PIQ | HC | General public | DSM-IV-TR | 100 | 33.5 | 9.8 | 100 | 9 | 10.4 | 1.5 |
| Howard 1997 | L | *Executive*: Self-regulation (motor regulation) | ASPD | Prison | QDIS-III-R | | | | | | | |
| | | | No ASPD | | QDIS-III-R | | | | | | | |
| Kumari 2006 | H | *Memory*: WM | ASPD | Forensic inpatients | SCID-II | 10 | 31.3 | 8.1 | 98 | 9.8 | | |
| | | *Intelligence*: FSIQ; VIQ | HC | General public | SCID-NP | 13 | 33.3 | 6.9 | 104.6 | 15.0 | | |
| Kumari 2005 | M | *Intelligence*: VIQ | ASPD | Forensic inpatients | SCID-II | 9 | 33.2 | 8.1 | 99.8 | 9.1 | | |
| | | | HC | General public | SCID-NP | 14 | 35.4 | 8.1 | 108.9 | 15.8 | | |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Kumari 2009 | M | *Intelligence*: VIQ | ASPD | Forensic inpatients | SCID-II | 13 | 32.9 | 10.6 | 96.9 | 9.9 | | |
| | | | HC | General public | SCID-NP | 14 | 33.1 | 6.6 | 107.4 | 16.5 | | |
| Lindberg 2004 | M | *NSS* | ASPD | Prison | SCID-II | 14 | 30.6 | 10.2 | | | 8.1 | 1.7 |
| | | | HC | Hospital staff | SCID-II | 10 | 29.5 | 8.1 | | | 14.1 | 2.6 |
| Lorenz Newman 2002c | L | *Affect*: Processing | ASPD | Prison | DSM-IV | 155 | | | | | | |
| | | *Language*: Semantic processing | Non-ASPD | | | 104 | | | | | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Raine 2000 | M | *Intelligence*: FSIQ | ASPD | General public | SCID-II | 21 | 31.9 | 6.8 | 98.4 | 12.8 | | |
| | | | HC | | SCID-II | 34 | 30.4 | 6.7 | 100.9 | 15.2 | | |
| Shamay-Tsoory 2010 | H | *Concept formation*: Abstraction | ASPD | Prison | DSM-IV-TR | 17 | 29.8 | 10.1 | | | | |
| | | *Social cognition*: Theory of Mind | HC | General public? | MINI | 20 | 27.7 | 8.4 | | | | |
| Stevens 2003 | H | *Executive*: Planning; Self-regulation (productivity, cognitive flexibility, motor regulation) | ASPD | General public | DIS-III-R | 34 | 23.4 | 1.8 | PIQ/VIQ 103/ 107.5 | 11.2/ 12.5 | 14.7 | 1.6 |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Concept formation*: Abstraction; Reasoning<br>*Memory*: STM; WM<br>*Attention*: Complex<br>*Language*: Verbal expression (fluency); Knowledge acquisition & retention<br>*Motor performance*<br>*Intelligence*: VIQ; PIQ | HC | | DIS-III-R | 32 | 22.5 | 1.3 | 105.4/ 113 | 14.5/ 13.0 | 15.4 | 1.3 |
| Swann 2009 | M | *Executive*: Self-regulation (motor regulation) | ASPD | General public | SCID-II | 34 | 38.7 | 10.3 | | | 12.7 | 2.1 |
| | | *Memory*: STM | HC | | SCID-II | 30 | 31.5 | 9.5 | | | 15 | 2.4 |
| | | *Attention*: Sustained | | | | | | | | | | |
| Völlm 2010 | H | *Executive*: Self-regulation (motor regulation) | ASPD | Prison & forensic inpatients | SCID-II | 25 | 42.1 | | 99.4 | | | |
| | | *Intelligence*: VIQ | HC | University staff & general population | SCID-II | 25 | 30.5 | | 103.9 | | | |
| ***ASPD & psychopathy*** | | | | | | | | | | | | |
| Dolan 2004 | H | *Affect*: Recognition | ASPD+ psychopathy | Prison | SCID-II, PCL:SV>18 | 28-30 | 31.0 | 5.4 | 105.4 | 13.8 | 12 | 0.2 |
| | | *Social cognition*: Theory of Mind | ASPD only | | SCID-II, PCL:SV<17 | 56-59 | 33.0 | 5.7 | 101.6 | 13.4 | 12.1 | 0.8 |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Intelligence*: VIQ | HC | Ancillary staff: prison & secure psychiatry | SCID-I & II | 20 | 31.7 | 7.7 | 106.9 | 11.8 | 12.2 | 0.6 |
| Dolan 2005 | H | *Affect*: Affect & memory | ASPD+ psychopathy | Prison & Forensic inpatients | SCID-II, PCL:SV top quartile | 20-21 | 31.2 | 5.9 | 107 | 13 | 12 | 0.2 |
| | | *Memory*: LTM | ASPD+ middle psychopathy [a] | | SCID-II, PCL:SV mid-quartile | 38 | 31.8 | 5.9 | 101.5 | 13.6 | 12.1 | 0.8 |
| | | *Intelligence*: VIQ | ASPD only | | SCID-II, PCL:SV low quartile | 26-27 | 34.2 | 5.5 | 102.3 | 13.6 | 12 | 0.7 |
| | | | HC | Ancillary staff: prison & secure psychiatry | SCID-II | 20 | 31.7 | 7.7 | 106.4 | 11.8 | 12.2 | 0.6 |
| Habel 2002 | H | *Affect*: Recognition | ASPD+ psychopathy | Prison & forensic patients | DSM-IV, PCL-R>20 | 17 | 33.4 | 5.8 | | | | |
| | | | HC | General public | No details | 17 | 33.5 | 7.6 | | | | |
| Kosson 2006 | L | *Affect*: Processing | ASPD+ psychopathy | Prison | DSM-IV, PCL-R≥30 | 25 | | | | | | |
| | | *Language*: Semantic processing | ASPD only | | DSM-IV, PCL-R≤20 | 26 | | | | | | |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | HC | | DSM-IV, PCL-R≤20 | 36 | | | | |
| Raine 2003 | M | *Interhemispheric integration* | ASPD+ psychopathy | General public | SCID-II, PCL-R≥23 | 15 | 31.6 | 6.6 | 97.7 | 13.7 |
| | | *Intelligence*: FSIQ; VIQ; PIQ | HC | | SCID-II, PCL-R≤14 | 25 | 28.8 | 6.5 | 101.6 | 15.2 |
| ***DPD & psychopathy*** | | | | | | | | | | |
| Dolan 2006 | H | *Affect*: Recognition | DPD+ psychopathy | Prison | ICD-10, PCL:SV≥17 | 22 | 35.2 | 10.3 | 106.6 | 10.9 |
| | | *Intelligence*: VIQ | DPD only | | ICD-10, PCL:SV<17 | 27 | | | | |
| | | | HC | | Axis I | 49 | 32.6 | 9.05 | 109.7 | 9.0 |
| Müller 2008/ Weber 2004 | M | *Executive*: Effective performance | DPD+ psychopathy | Forensic patients | PCL-R>28 | 10 | 33.1 | | | |
| | | *Affect*: Processing | HC | Not stated | PCL-R<10 | 12 | 33.2 | | | |
| | | *Attention*: Selective *Intelligence* | | | | | | | | |
| ***Psychopathy*** | | | | | | | | | | |
| Arnett 1993 | M | *Executive*: Self-regulation (motor regulation) | LA psychopathy | Prison | PCL-R≥30 | 13 | 25.3 | 3.9 | 99.6 | 8.8 |
| | | *Intelligence*: FSIQ | HA psychopathy | | | 16-18 | 24.8 | 4.2 | 95.6 | 13.2 |
| | | | LA non-psychopathy | | PCL-R≤22 | 16-18 | 26.9 | 4.9 | 93.7 | 15.1 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | HA non-psychopathy | | | 12-14 | 25.3 | 5.9 | 98.9 | 11.1 |
| Arnett 1997 - Exp. 1 | H | *Executive*: Self-regulation (motor regulation) | LA psychopathy | Prison | PCL-R≥30 | 13 | 27 | 3.9 | 93.9 | 11.0 |
| | | *Intelligence*: FSIQ | HA psychopathy | | | 16 | 27.4 | 6.4 | 98.4 | 11.9 |
| | | | LA non-psychopathy | | PCL-R≤20 | 19 | 28.8 | 5.1 | 99.9 | 9.4 |
| | | | HA non-psychopathy | | | 10 | 26.2 | 5.9 | 93.9 | 8.0 |
| - Exp. 2 | | *Executive*: Self-regulation (motor regulation) | LA psychopathy | Prison | PCL-R≥30 | 17 | 24.3 | 4.3 | 95.3 | 12.6 |
| | | *Intelligence*: FSIQ | HA psychopathy | | | 16 | 30.1 | 6.2 | 96.8 | 12.0 |
| | | | LA non-psychopathy | | PCL-R≤20 | 19 | 28.9 | 6.5 | 105.9 | 8.1 |
| | | | HA non-psychopathy | | | 12 | 28.6 | 5.6 | 98.9 | 12.5 |
| Assadi 2007 | L | *NSS* | Psychopathy | Prison | PCL:SV cut-off 18 | 64 | | | | |
| | | | Non-psychopathy | Prison & general public | PCL:SV cut-off 18 (prisoners only) | 286 | | | | |
| Bagley 2009 | H | *Affect*: Recognition | Psychopathy | Prison | PCL-R≥30 | 34 | | | | |
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL-R≤20 | 34 | | | | |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Bernstein 2000 | L | *Memory*: STM<br>*Intelligence*: FSIQ | Psychopathy<br>Non-psychopathy | Prison | PCL-R≥30<br>PCL-R<20 | 21<br>21 | | | | |
| Blair 1996 | M | *Social cognition*: Theory of Mind | Psychopathy | Prison & forensic inpatients | PCL-R≥30 | 25 | 31.6 | 6.7 | 94.1 | 17.3 |
| | | *Intelligence* | Non-psychopathy | | PCL-R≤20 | 25 | 33.1 | 9.5 | 97.8 | 15.8 |
| Blair, Morton 2006 | M | *Executive*: Self-regulation (cognitive flexibility) | Psychopathy | Prison | PCL-R≥30 | 21 | 36.7 | 7.5 | | |
| | | *Concept formation*: Abstraction | Non-psychopathy | | PCL-R≤20 | 19 | 32.2 | 9.1 | | |
| | | *Language*: Academic skills (reading) | | | | | | | | |
| Blair, Newman 2006 | L | *Executive*: Self-regulation (cognitive flexibility, motor regulation); Effective performance | Psychopathy | Prison | PCL-R≥30 | 17-19 | 35.5 | 7.7 | | |
| | | *Concept formation*: Abstraction | Non-psychopathy | | PCL-R≤19 | 18-19 | 36.9 | 9.6 | | |
| | | *Attention*: Selective<br>*Language*: Academic skills (reading) | | | | | | | | |
| Blair, Richell 2006 | M | *Concept formation*: Abstraction | Psychopathy | Prison | PCL-R≥30 | 18-24 | 35.2 | 9.8 | | |
| | | *Affect*: Processing; Recognition | Non-psychopathy | | PCL-R≤20 | 18-20 | 32.4 | 9.2 | | |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | *Language*: Academic skills (reading); Semantic processing | | | | | | | | |
| Blair 1995 | L | *Concept formation*: Abstraction | Psychopathy | Forensic inpatients | PCL | 7-10 | 33.3 | 7.7 | 91.6 | 17.2 |
| | | *Social cognition*: Moral reasoning | Non-psychopathy | | | 7-10 | 37.5 | 9.4 | 92.7 | 16 |
| | | *Intelligence*: FSIQ | | | | | | | | |
| Blair, Mitchell, Leonard 2004 | M | *Executive*: Self-regulation (motor regulation) | Psychopathy | Prison | PCL-R≥30 | 19 | 38 | 12.5 | | |
| | | *Concept formation*: Abstraction | Non-psychopathy | | PCL-R≤20 | 21 | 38.8 | 6.6 | | |
| Blair, Mitchell, Peschardt 2004 | M | *Concept formation*: Abstraction | Psychopathy | Prison | PCL-R≥30 | 19 | 33.6 | 9.2 | | |
| | | *Affect*: Recognition | Non-psychopathy | | PCL-R≤20 | 19 | 30.6 | 7.2 | | |
| Blair 2002 | M | *Concept formation*: Abstraction | Psychopathy | Prison | PCL-R≥30 | 19 | 34.5 | 9.1 | | |
| | | *Affect*: Recognition | Non-psychopathy | | PCL-R≤20 | 20 | 31.5 | 7.9 | | |
| Blair, Sellars 1995 | L | *Social cognition*: Theory of Mind | Psychopathy | Prison & forensic inpatients | PCL-R≥30 | 25 | 33.3 | 7.3 | 96.0 | 15.4 |
| | | *Intelligence* | Non-psychopathy | | PCL-R≤20 | 25 | 33.0 | 9.6 | 94.8 | 13.7 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Blair, Jones 1995 | L | *Social cognition*: Moral reasoning | Psychopathy | Forensic inpatients | PCL-R≥30 | 20 | 30.4 | 7.2 | 92.5 | 15.9 |
| | | *Intelligence* | Non-psychopathy | | PCL-R≤20 | 20 | 31.3 | 8.1 | 97.9 | 12.5 |
| Brazil 2009 | H | *Executive*: Effective performance | Psychopathy | Forensic inpatients | PCL-R≥26 | 16 | 39 | 9.5 | | |
| | | *Attention*: Selective | HC | Hospital staff | None | 18 | 37 | 6.4 | | |
| Brinkley, Bernstein 1999 | L | *Language*: Verbal expression (discourse) | Psychopathy | Prison | PCL-R≥30 | 37 | | | | |
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL-R≤22 | 41 | | | | |
| Brinkley, Newman 1999 | L | *Language*: Verbal expression (discourse) | Psychopathy | Prison | PCL-R≥30 | 18 | | | | |
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL-R≤22 | 21 | | | | |
| Brinkley 2005 - Exp. 1 | L | *Language*: Semantic processing | Psychopathy | Prison | PCL-R≥30 | 27 | | | | |
| | | *Memory*: Priming | Non-psychopathy | | PCL-R≤20 | 31 | | | | |
| | | *Intelligence*: FSIQ | | | | | | | | |
| - Exp. 2 | | *Executive*: Self-regulation (motor regulation); Effective performance | Psychopathy | Prison | PCL-R≥30 | 32 | | | 96.5 | |
| | | *Attention*: Selective | Non-psychopathy | | PCL-R≤20 | 37 | | | 102.4 | |

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|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Language*: Semantic processing | | | | | | | | | |
| | | *Intelligence*: FSIQ | | | | | | | | | |
| Budhani 2006 | M | *Executive*: Self-regulation (cognitive flexibility) | Psychopathy | Prison | PCL-R≥30 | 17-20 | 37.8 | 7.6 | | | |
| | | *Concept formation*: Abstraction | Non-psychopathy | | PCL-R≤20 | 16-17 | 34.5 | 10.6 | | | |
| Christianson 1996 | L | *Affect*: Affect & memory | Psychopathy | Prison | PCL-R≥30 | 27 | 28.9 | | | | 8 |
| | | *Memory*: STM | Non-psychopathy | | PCL-R≤20 | 37 | 28.8 | | | | 8.9 |
| Cima 2010 | H | *Social cognition*: Moral reasoning | Psychopathy | Forensic patients with personality disorders | PCL-R≥26 | 7-14 | | | 81.6 | 8.7 | |
| | | *Intelligence* | Non-psychopathy | | PCL-R<26 | 13-23 | | | 92.5 | 19.4 | |
| | | | HC | General public? | PCL-R<26 | 35 | | | | | |
| Craig 2009 | H | *Intelligence*: FSIQ | Psychopathy | Forensic inpatients | PCL-R≥25 | 9 | 34 | 12 | 94 | 7 | |
| | | | HC | General public | PCL:SV | 9 | 37 | 9 | 91 | 6 | |
| Day 1996 | M | *Affect*: Processing | Psychopathy | Prison | PCL-R≥30 | 20 | 29.8 | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Non-psychopathy | | PCL-R≤20 | 20 | 31.2 | | | | | |
| Dinn 2000 | H | *Executive*: Self-regulation (productivity, cognitive flexibility, motor regulation); Effective performance | Psychopathy | General public | PCL:SV≥16 | 12 | 27.8 | 4 | | | 13.9 | 1.7 |
| | | *Attention*: Selective | HC | | PCL:SV<16 | 10 | 28.9 | 6.9 | | | 13.9 | 1.7 |
| | | *Language*: Verbal expression (fluency) | | | | | | | | | | |
| Drugge 1998 | L | *Executive*: Self-regulation (motor regulation); Effective performance | Psychopathy | Prison | PCL-R>23.63 | 13 | 36.4 | 9.4 | 103 | 11.5 | | |
| | | *Concept formation*: Abstraction | Non-psychopathy | | PCL-R<23.63 | 12 | 45.1 | 12.4 | 101.8 | 10.6 | | |
| | | *Affect*: Processing | | | | | | | | | | |
| | | *Attention*: Selective | | | | | | | | | | |
| | | *Language*: Verbal expression (vocabulary); Academic skills (reading) | | | | | | | | | | |
| | | *Memory*: Priming | | | | | | | | | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Dvorak-Bertsch 2007 | L | *Executive*: Self-regulation (motor regulation); Effective performance | Psychopathy | Prison | PCL-R≥30 | 55 | | | | | | |
| | | *Attention*: Selective | Non-psychopathy | | PCL-R≤20 | 42 | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Forth 1989 | M | *Attention*: Reaction time | Psychopathy | Prison | PCL-R≥30 | 12 | 25.2 | 7.2 | | | 8.8 | 1.8 |
| | | | Non-psychopathy | | PCL-R<32 | 11 | 24.6 | 4.8 | | | 9.5 | 1.7 |
| Gacono 1990 | L | *Intelligence*: FSIQ | Psychopathy | Prison | PCL≥30 | 14 | | | | | | |
| | | | Moderate psychopathy | | PCL<30 | 19 | | | | | | |
| Gacono 1991 | M | *Intelligence*: FSIQ | Psychopathy | Prison | PCL≥30 | 21 | | | | | | |
| | | | Moderate psychopathy | | PCL<30 | 21 | | | | | | |
| Gacono 1992 | L | *Intelligence*: FSIQ | Psychopathy | Prison | PCL≥30 | 22 | 30.4 | 7.1 | | | | |
| | | | Moderate psychopathy | | PCL<30 | 21 | 26.6 | 6.0 | | | | |
| Gillstrom 1995 | H | *Concept formation*: Abstraction; Reasoning | Psychopathy | Prison | PCL-R≥30 | 17 | 32.2 | 9.2 | 102.6 | 12.7 | 11.6 | 2.1 |
| | | *Language*: Verbal expression (vocabulary) | Non-psychopathy | | PCL-R<30 | 28 | 31.3 | 8.4 | 105.4 | 10.8 | 11.3 | 1.6 |
| | | *Construction* | | | | | | | | | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Gillstrom 1988 | L | *Language*: Gestural | Psychopathy | Prison | PCL>33 | 10 | 25.1 | 8.4 | | | 8.5 | 2.2 |
| | | | Non-psychopathy | | PCL<23 | 10 | 21.7 | 3.8 | | | 8.9 | 1.4 |
| Glass 2006 | M | *Affect*: Recognition | Psychopathy | Prison | PCL-R≥30 | 50 | 32.6 | 7.1 | 100.8 | 9.6 | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL-R≤20 | 61 | 32.0 | 7.1 | 100.3 | 9.9 | | |
| Glass 2009 | M | *Affect*: Affect & memory | Psychopathy | Prison | PCL-R≥30 | 89 | 30.5 | 7.4 | 101.1 | 11.0 | | |
| | | *Memory*: Short-term | Non-psychopathy | | PCL-R≤20 | 150 | 32.2 | 7.5 | 100.9 | 11.1 | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Goldstein 1998 | H | *Executive*: Self-regulation (productivity, cognitive flexibility, motor regulation) | Psychopathy | Prison | PCL-R≥30 | 45-47 | 27.9 | 6.7 | 95.8 | 10.2 | 11.1 | 1.8 |
| | | *Concept formation*: Abstraction | Non-psychopathy | | PCL-R≤20 | 43-45 | 27.0 | 6.1 | 97.7 | 9.9 | 11.9 | 1.7 |
| | | *Language*: Verbal expression (fluency); Academic skills (reading) | | | | | | | | | | |
| | | *Perception*: Visual | | | | | | | | | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Hare 1988 | M | *Concept formation*: Semantic processing | Psychopathy | Prison | High PCL>32 | 13 | 28.9 | 6.1 | 102.7 | 12.6 | 11.1 | 3.0 |
| | | *Memory*: STM | Non-psychopathy | | Low PCL<23 | 13 | 30.2 | 7.2 | 102.1 | 12.8 | 10.4 | 3.3 |
| | | *Language*: Semantic processing | HC | General public | None reported | 13 | 30.8 | 8.3 | | | 10.5 | 1.3 |
| | | *Intelligence* | | | | | | | | | | |
| Harpur 1991 - Exp. 1 | M | *Executive*: Effective performance | Psychopathy | Prison | PC L≥ 31.5 | 19 | 28.8 | | | | 10.8 | |
| | | *Attention*: Selective | Non-psychopathy | | PC L<24 | 20 | 33.3 | | | | 10.9 | |

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| - Exp. 2 | | *Executive*: Effective performance | Psychopathy | Prison | PCL-R>30 | 18 | 28.8 | 10.8 |
| | | *Attention*: Selective | Non-psychopathy | | PCL-R<30 | 19 | 33.3 | 10.9 |
| - Exp. 3 | | *Executive*: Self-regulation (motor regulation); Effective performance | Psychopathy | Prison | PC L≥ 31.5 | 16-17 | 28.8 | 10.8 |
| | | *Attention*: Selective | Non-psychopathy | | PC L<24 | 15-19 | 33.3 | 10.9 |
| | | *Memory*: Priming | | | | | | |
| - Exp. 4 | | *Executive*: Effective performance | Psychopathy | Prison | PCL-R>30 | 19 | 28.8 | 10.8 |
| | | *Attention*: Selective | Non-psychopathy | | PCL-R<30 | 20 | 33.3 | 10.9 |
| | | *Memory*: Priming | | | | | | |
| - Exp. 5 | | *Executive*: Effective performance | Psychopathy | Prison | PCL-R>30 | 19 | 28.8 | 10.8 |
| | | *Attention*: Selective | Non-psychopathy | | PCL-R<30 | 20 | 33.3 | 10.9 |
| | | *Memory*: Priming | | | | | | |
| Hart 1990 - Sample 1 | H | *Executive*: Self-regulation (productivity) | Psychopathy | Prison | PCL-R≥30 | 22 | | |
| | | *Memory*: STM | Non-psychopathy | | PCL-R<20 | 27 | | |
| | | *Attention*: Complex | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Language*: Verbal expression (fluency)<br>*Perception*: Visual | | | | | | | | | | |
| - Sample 2 | | *Executive*: Self-regulation (productivity)<br>*Memory*: STM<br>*Attention*: Complex<br>*Language*: Verbal expression (fluency, vocabulary); Academic skills (reading)<br>*Construction*<br>*Intelligence*: FSIQ | Psychopathy<br>Non-psychopathy | Prison | PCL-R≥30<br>PCL-R<20 | 32<br>40 | | | 101.9<br>101.8 | 11.2<br>10.9 | | |
| Herpertz 2001 | H | *Intelligence*: FSIQ | Psychopathy<br>HC | Prison<br>General public | PCL:SV≥18 | 25<br>24 | 33.8<br>32.5 | 8.2<br>10.8 | 99.2<br>95.8 | 9.7<br>5.8 | 10.0<br>10.7 | 1.4<br>1.6 |
| Hervé 2003 | L | *Concept formation*: Reasoning<br>*Affect*: Recognition<br>*Language*: Academic skills (reading) | Psychopathy<br>Non-psychopathy | Prison | PCL-R cut-off 30<br>PCL-R cut-off 22 | 12<br>10 | | | | | 10.7<br>10.7 | |
| Hiatt 2005 - Exp. 2 | M | *Interhemispheric integration* | Psychopathy<br>Non-psychopathy | Prison | PCL-R≥30<br>PCL-R≤20 | 42<br>44 | | | | | | |
| - Exp. 3 | | *Interhemispheric integration* | Psychopathy | Prison | PCL-R≥30 | 23 | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL-R≤20 | 26 | | | | | | |
| Hiatt 2002 | M | *Affect*: Recognition | Psychopathy | Prison | PCL-R≥30 | 21 | 29.2 | 5.2 | 96.8 | 12.3 | 10.3 | 1.9 |
| | | *Attention*: Divided | Non-psychopathy | | PCL-R≤20 | 34 | 28.6 | 5.8 | 98.1 | 10.9 | 11.2 | 1.5 |
| | | *Perception*: Auditory<br>*Intelligence*: FSIQ | | | | | | | | | | |
| Hiatt 2007 | M | *Interhemispheric integration* | Psychopathy | Prison | PCL-R≥30 | 54 | 30.2 | 7.4 | 101.4 | 11.6 | | |
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL-R≤20 | 39 | 30.3 | 6.5 | 101.7 | 10.3 | | |
| Hiatt 2004 - Exp. 1 | M | *Executive*: Self-regulation (motor regulation); Effective performance | Psychopathy | Prison | PCL-R≥30 | 29 | 27.8 | 4.6 | 97.0 | 11.8 | | |
| | | *Attention*: Selective | Non-psychopathy | | PCL-R≤20 | 34 | 28.7 | 6.3 | 102.7 | 9.4 | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| - Exp. 2 | | *Executive*: Self-regulation (motor regulation); Effective performance | Psychopathy | Prison | | 27 | 29.2 | 6.0 | 96.7 | 10.8 | | |
| | | *Attention*: Selective | Non-psychopathy | | | 48 | 27.7 | 6.1 | 97.1 | 11.3 | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| - Exp. 3 | | *Executive*: Self-regulation (motor regulation); Effective performance | Psychopathy | Prison | | 26 | 27.5 | 5.7 | 97.3 | 11.5 | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Attention*: Selective | Non-psychopathy | | | 42 | 28.8 | 6.1 | 99.2 | 11.7 | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Howard 2007 | H | *Concept formation*: Semantic processing | Psychopathy | Prison | PCL:SV≥18 | 17 | 32.3 | 4.1 | 86.2 | 8.5 | | |
| | | *Affect*: Processing | Non-psychopathy | | PCL:SV≤12 | 17 | 34.3 | 4.8 | 85.6 | 10.2 | | |
| | | *Attention*: Sustained | | | | | | | | | | |
| | | *Intelligence*: PIQ | | | | | | | | | | |
| Howard 1997 | L | *Executive*: Self-regulation (motor regulation) | Psychopathy | Prison | PCL:SV≥14 | 19 | | | | | | |
| | | | Non-psychopathy | | PCL:SV≤10 | 16 | | | | | | |
| Howland 1993 | L | *Executive*: Effective performance | Psychopathy | Prison | PCL-R≥30 | 30 | | | | | | |
| | | *Attention*: Selective | Non-psychopathy | | PCL-R≤20 | 19 | | | | | | |
| | | | | | | | Offenders/Non-offenders | | | | | |
| Iria 2009 | H | *Executive*: Self-regulation (motor regulation) | Psychopathy (Offenders/ Non-offenders) | Offenders & general public | PCL:SV>18 | 22/ 16 | 30.1/ 28.1 | 11.3/ 14.6 | | | 8.0/ 8.9 | 4.7/ 2.1 |
| | | *Affect*: Processing | Non-psychopathy (Offenders/ Non-offenders) | | PCL:SV<12 | 11/ 13 | 27.4/ 28.3 | 7.6/ 12.7 | | | 8.0/ 8.9 | 2.6/ 2.6 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Ishikawa 2001 | L | *Executive*: Self-regulation (cognitive flexibility) | Unsuccessful psychopathy | General public | PCL-R highest tertile | 16 | 33.8 | 6.6 | 96.4 | 14.7 | | |
| | | *Memory*: STM; LTM | Successful psychopathy | | | 13 | 29.6 | 6.1 | 99.1 | 14.2 | | |
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL-R lowest tertile | 26 | 28.4 | 6.5 | 106.0 | 16.8 | | |
| | | | | | | | | | (stanine score) | | | |
| Johansson 2005 | L | *Concept formation*: Abstraction | Psychopathy | Prison | PCL-R≥30 | 93 | | | 5.6 | | | |
| | | *Language*: Verbal expression (vocabulary) | Non-psychopathy | | PCL-R≤19 | 277 | | | 6.0 | | | |
| | | *Construction* | | | | | | | | | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Jozef 1999 | L | *Concept formation*: Abstraction | Psychopathy | Prison | PCL-R cut off 25 | 11 | | | | | | |
| | | *Attention*: Complex | Non-psychopathy | | | 13 | | | | | | |
| | | *Construction* | | | | | | | | | | |
| Jutai 1987 | L | *Attention*: Sustained; Divided | Psychopathy | Prison | PCL≥34 | 11 | 28.4 | 6.5 | | | 9.3 | 2.7 |
| | | | Non-psychopathy | | PCL<24 | 13 | 29.7 | 6.7 | | | 9.1 | 2.5 |
| | | | | | | Sample 1/2 | | | | | | |
| Kiehl, Bates 2006 | H | *Attention*: Sustained | Psychopathy | Prison | PCL≥30 | 23/ 18 | 33.9/ 32.5 | | 103.2/ 105.5 | 11.85/ 10.8 | 11.0/ 10.4 | |

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|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Intelligence*: VIQ | Non-psychopathy | | PCL<30 | 21/ 18 | 35.8/ 34.1 | | 103.5/ 105.8 | 8.5/ 9.2 | 11.4/ 11.2 |
| Kiehl, Hare, Liddle 1999 | H | *Attention*: Sustained | Psychopathy | Prison | PCL-R≥29 | 11 | 27.0 | | | | 10.5 |
| | | | Non-psychopathy | | PCL-R≤27 | 10 | 33.0 | | | | 10.8 |
| Kiehl, Hare, McDonald 1999 | H | *Concept formation*: Semantic processing | Psychopathy | Prison | PCL-R≥30 | 8 | 29.0 | | | | 10.1 |
| | | *Affect*: Recognition | Non-psychopathy | | PCL-R≤20 | 9 | 33.0 | | | | 9.7 |
| | | *Language*: Semantic processing | | | | | | | | | |
| Kiehl, Laurens 2006 | H | *Language*: Semantic processing | Psychopathy | Prison | PCL≥30 | 25 | 32.5 | | 107.8 | 10.0 | 10.1 |
| | | *Intelligence*: VIQ | Non-psychopathy | | PCL<30 | 25 | 32.1 | | 106.8 | 11.4 | 10.9 |
| Kiehl 2000 | H | *Executive*: Self-regulation (motor regulation) | Psychopathy | Prison | PCL-R≥30 | 13 | 28.0 | | | | 10.3 |
| | | | Non-psychopathy | | PCL-R≤20 | 11 | 27.0 | | | | 10.2 |
| Kiehl 2001 | H | *Affect*: Affect & memory | Psychopathy | Prison | PCL-R>23.6 | 8 | 33.9 | 7.6 | 111.2 | 7.5 | |
| | | *Memory*: Short-term | Non-psychopathy | | PCL-R<23.6 | 8 | 37.1 | 7.1 | 115.5 | 5.9 | |
| | | *Intelligence*: VIQ | HC | General public | PCL:SV | 8 | 31.9 | 8.4 | 108.9 | 11.5 | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Kiehl 2004 | H | *Concept formation*: Semantic processing | Psychopathy | Prison | PCL-R>28 | 8 | 33.9 | 7.6 | 111.2 | 7.0 | 9.9 | 3.5 |
| | | *Language*: Semantic processing | HC | General public | None reported | 8 | 27.9 | 5.0 | 111.8 | 7.0 | 12.4 | 0.7 |
| | | *Intelligence*: VIQ | | | | | | | | | | |
| Klaver 2007 | L | *Language*: Verbal expression (discourse); Gestural | Psychopathy | Prison | PCL-R≥30 | 7 | | | | | | |
| | | | Non-psychopathy | | PCL-R<30 | 38 | | | | | | |
| Kosson 1990 | L | *Executive*: Self-regulation (motor regulation) | Psychopathy | Prison | PCL/-R≥30 | 30 | | | | | | |
| | | | Non-psychopathy | | PCL/-R≤20 | 29 | | | | | | |
| Kosson 1996 | H | *Attention*: Sustained; Divided | Psychopathy | Prison | PCL>29 | 30 | 26.0 | 4.8 | 96.7 | 7.5 | | |
| | | *Perception*: Visual; Auditory | Non-psychopathy | | PCL<21 | 30 | 27.9 | 6.4 | 97.8 | 12.8 | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Kosson 1998 | M | *Concept formation*: Abstraction | Psychopathy | Prison | PCL-R≥28.5 | 31 | 31.2 | 6.6 | | | | |
| | | *Attention*: Sustained; Divided | Non-psychopathy | | PCL-R≤18.5 | 37-38 | 29.7 | 6.3 | | | | |
| | | *Language*: Verbal expression (vocabulary) | | | | | | | | | | |
| | | *Perception*: Visual | | | | | | | | | | |
| Kosson 2007 | M | *Perception*: Visual | Psychopathy | Prison | PCL-R≥30 | 55 | 27.5 | 6.6 | 93.1 | 12.6 | 11.5 | 2.0 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL-R≤20 | 57 | 27.3 | 7.4 | 93.3 | 10.2 | 12.2 | 1.5 |
| Kosson 2002 | H | *Affect*: Recognition | Psychopathy | Prison | PCL-R≥30 | 34 | 27.0 | 6.6 | 93.8 | 11.5 | | |
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL-R≤20 | 33 | 27.0 | 6.5 | 96.1 | 9.7 | | |
| Lapierre 1995 | M | *Executive*: Planning; Self-regulation (cognitive flexibility, motor regulation) | Psychopathy | Prison | PCL-R≥30 | 30 | 33.5 | 8.5 | | | 9.6 | 2.0 |
| | | *Concept formation*: Abstraction | Non-psychopathy | | PCL-R≤20 | 30 | 32.5 | 8.6 | | | 9.8 | 2.2 |
| | | *Perception*: Olfactory<br>*Visuospatial skills* | | | | | | | | | | |
| Lee 2008 | L | *Language*: Verbal expression (discourse) | Psychopathy | Prison | PCL-R≥30 | 7 | | | | | | |
| | | | Non-psychopathy | | PCL-R<30 | 38 | | | | | | |
| Llanes 2006 | M | *Attention*: Sustained; Divided | Psychopathy | Prison | PCL-R≥30 | 26 | 26.0 | 6.2 | 99.4 | 11.5 | 10.7 | 1.8 |
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL-R≤22 | 46 | 26.5 | 6.6 | 97.3 | 11.2 | 11.8 | 1.9 |
| Lopez 2007 | H | *Perception*: Visual | Psychopathy | Prison | PCL-R≥30 | 25 | 26.9 | 7.2 | 88.3 | 10.9 | 11.0 | 1.3 |
| | | *Interhemispheric integration* | Non-psychopathy | | PCL-R≤22 | 29 | 25.8 | 6.8 | 91.7 | 11.9 | 10.9 | 1.5 |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Lorenz Newman | M | *Affect*: Processing | LA psychopathy | Prison | PCL-R≥30 | 11 | | | 100.8 | 11.5 | | |

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| 2002a | | | | | | | | |
| | | *Language*: Semantic processing | HA psychopathy | | | 17 | 98.0 | 11.0 |
| | | *Intelligence*: FSIQ | LA non-psychopathy | | PCL-R≤20 | 26 | 99.6 | 11.9 |
| | | | HA non-psychopathy | | | 20 | 96.5 | 12.7 |
| Lorenz Newman 2002b | M | *Affect*: Processing | Psychopathy | Prison | PCL-R≥30 | 23 | | |
| | | *Language*: Semantic processing | Non-psychopathy | | PCL-R≤20 | 39 | | |
| | | *Intelligence*: FSIQ | | | | | | |
| Lösel 2004 | L | *Executive*: Self-regulation (cognitive flexibility) | Psychopathy | Prison | PCL-R≥25 | 17 | | |
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL-R<25 | 32 | | |
| Louth 1998 | L | *Language*: Verbal expression (discourse) | Psychopathy | Prison | PCL-R≥27 | 10 | | |
| | | *Intelligence*: VIQ | Non-psychopathy | | PCL-R<27 | 10 | | |
| Marshall 1996 | L | *Affect*: Processing | Psychopathy | Prison | PCL-R cut-off 25 | 10 | | |
| | | *Language*: Semantic processing | Non-psychopathy | | PCL-R cut-off 25 | 10 | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Mayer 2000 | L | *Interhemispheric integration* (handedness)<br>*Intelligence*: FSIQ | Psychopathy<br>Non-psychopathy | Prison | PCL-R≥30<br>PCL-R≤20 | 137<br>111 | | | | | | |
| Mayer 2006 | M | *Executive*: Effective performance<br>*Attention*: Selective<br>*Intelligence*: FSIQ | Psychopathy<br>Non-psychopathy | Prison | PCL-R≥30<br>PCL-R≤20 | 20<br>35 | | | | | | |
| Mercer 2005 | M | *Executive*: Self-regulation (productivity, cognitive flexibility, motor regulation); Effective performance<br>*Concept formation*: Abstraction; Reasoning; Arithmetic reasoning<br>*Memory*: STM; WM<br>*Attention*: Selective; Complex<br>*Language*: Verbal expression (fluency, vocabulary); Knowledge acquisition & retention<br>*Construction*<br>*Intelligence*: FSIQ | Psychopathy<br>Non-psychopathy | Prison | PCL:SV cut-off 18<br>PCL:SV cut-off 18 | 143<br>187 | 33.9<br>32.0 | 7.4<br>8.6 | 88.7<br>94.5 | 10.7<br>10.7 | 11.0<br>11.6 | 1.9<br>1.9 |
| Mills 1995 - Exp. 1 | H | *Affect*: Recognition | Psychopathy | Prison | PCL-R≥30 | 12 | 33.7 | 10.3 | 101.8 | 12.0 | 10.6 | 2.0 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Attention*: Sustained | Non-psychopathy | | PCL-R≤24 | 12 | 28.8 | 3.8 | 98.8 | 9.1 | 10.6 | 1.8 |
| | | *Language*: Academic skills (reading) | | | | | | | | | | |
| | | *Perception*: Visual | | | | | | | | | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| - Exp.2 | | *Executive*: Effective performance | Psychopathy | Prison | PCL-R≥30 | 12 | 32.3 | 9.7 | 101.4 | 11.5 | 10.5 | 1.9 |
| | | *Affect*: Recognition | Non-psychopathy | | PCL-R≤24 | 12 | 30.8 | 6.4 | 99.9 | 8.3 | 9.8 | 1.8 |
| | | *Attention*: Sustained; Selective | | | | | | | | | | |
| | | *Perception*: Visual; Auditory | | | | | | | | | | |
| | | *Visuospatial skills* | | | | | | | | | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Mitchell 2002 | M | *Executive*: Self-regulation (cognitive flexibility) | Psychopathy | Prison | PCL-R≥30 | 21 | 33.6 | 8.0 | | | | |
| | | *Concept formation*: Abstraction | Non-psychopathy | | PCL-R<20 | 21 | 32.9 | 7.9 | | | | |
| Mitchell 2006 | M | *Concept formation*: Abstraction | Psychopathy | Prison | PCL-R≥30 | 16 | 33.4 | 9.1 | | | | |
| | | *Affect*: Processing | Non-psychopathy | | PCL-R≤20 | 19 | 31.2 | 10.0 | | | | |
| Mol 2009 | L | *Executive*: Self-regulation (cognitive flexibility) | Psychopathy | Forensic patients | PCL-R≥26 | 17 | | | | | | |
| | | | Non-psychopathy | | PCL-R<26 | 36 | | | | | | |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Moltó 2007 | L | *Executive*: Self-regulation (cognitive flexibility) | Psychopathy | Prison | PCL-R≥30 | 9 | | | |
| | | | Non-psychopathy | | PCL-R<20 | 11 | | | |
| Newman 1992 | M | *Executive*: Self-regulation (motor regulation) | LA psychopathy | Prison | PCL≥30 | 29 | 27.2 | 101.6 | 10.4 |
| | | *Intelligence*: FSIQ | HA psychopathy | | | 44 | 26.5 | 94.6 | 10.4 |
| | | | LA non-psychopathy | | PCL≤22 | 45 | 27.2 | 101.3 | 11.2 |
| | | | HA non-psychopathy | | | 40 | 26.2 | 95.6 | 10.6 |
| Newman 1990 - Study 1 | M | *Executive*: Self-regulation (motor regulation) | LA psychopathy | Prison | PCL-R≥32 | 15 | 26.4 | 110.0 | |
| | | *Intelligence*: FSIQ | HA psychopathy | | | 17 | 25.4 | 109.4 | |
| | | | LA non-psychopathy | | PCL-R≤22 | 14 | 26.2 | 107.6 | |
| | | | HA non-psychopathy | | | 13 | 28.8 | 108.9 | |
| - Study 2 | | *Executive*: Self-regulation (motor regulation) | LA psychopathy | Prison | PCL-R≥32 | 12 | 28.4 | 112.5 | |
| | | *Intelligence*: FSIQ | HA psychopathy | | | 10 | 25.2 | 108.1 | |
| | | | LA non-psychopathy | | PCL-R≤22 | 11 | 26.9 | 107.3 | |
| | | | HA non-psychopathy | | | 14 | 31.4 | 109.9 | |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| - Study 3 | | *Memory*: STM | LA psychopathy | Prison | PCL≥32 | 20 | 25.8 | | 109.2 | |
| | | *Intelligence*: FSIQ | HA psychopathy | | | 34 | 26.3 | | 104.3 | |
| | | | LA non-psychopathy | | PCL≤22 | 32 | 26.3 | | 108.7 | |
| | | | HA non-psychopathy | | | 36 | 27.0 | | 104.9 | |
| Newman 1987 | M | *Executive*: Self-regulation (cognitive flexibility) | Psychopathy | Prison | PCL≥31.5 | 36 | 25.5 | 4.9 | 109.2 | 9.2 |
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL≤20 | 36 | 26.7 | 6.0 | 109.2 | 8.0 |
| Newman 1998 | L | *Executive*: Self-regulation (motor regulation) | Psychopathy | Prison | PCL-R≥30 | 50 | | | | |
| | | | Non-psychopathy | | PCL-R≤20 | 58 | | | | |
| Newman 1997 - Caucasian sample | M | *Executive*: Effective performance | LA psychopathy | Prison | PCL-R≥30 | 12 | | | 100.5 | 9.0 |
| | | *Attention*: Selective | HA psychopathy | | | 19 | | | 95.8 | 9.8 |
| | | *Intelligence*: FSIQ | LA non-psychopathy | | PCL-R≤22 | 12 | | | 98.8 | 12.3 |
| | | | HA non-psychopathy | | | 11 | | | 96.6 | 15.5 |
| - African-American sample | | | LA psychopathy | Prison | | 12 | | | 89.3 | 13.8 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | HA psychopathy | | | 9 | | | 79.7 | 11.0 |
| | | | LA non-psychopathy | | | 7 | | | 82.5 | 13.6 |
| | | | HA non-psychopathy | | | 10 | | | 93.9 | 10.4 |
| Patterson 1990 - Exp. 1 | H | *Social cognition*: Social interpretation & knowledge | Psychopathy | Prison | PCL≥30 | 24 | | | 95.6 | 8.7 |
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL≤20 | 22 | | | 96.1 | 10.5 |
| - Exp. 3 | | *Social cognition*: Theory of Mind; Social interpretation & knowledge | Psychopathy | Prison | PCL≥30 | 31 | 27.5 | 5.4 | 98.3 | 11.1 |
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL≤20 | 30 | 26.3 | 4.4 | 97.2 | 9.4 |
| Pham 2000 | L | *Intelligence*: FSIQ | Psychopathy | Prison | PCL-R cut-off 23.9 | 14 | | | 92.9 | |
| | | | Non-psychopathy | | | 16 | | | 101.4 | |
| Pham 2003 | M | *Executive*: Planning; WM; Self-regulation (cognitive flexibility, motor regulation); Effective performance | Psychopathy | Prison | PCL-R≥25 | 18 | 29.1 | 10.5 | 95.1 | 6.6 |
| | | *Concept formation*: Abstraction; Reasoning; Arithmetic reasoning | Non-psychopathy | | PCL-R≤15 | 18 | 31.9 | 9.8 | 98.8 | 13.5 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | *Memory*: STM; WM<br>*Attention*: Sustained; Selective; Complex<br>*Language*: Verbal expression (vocabulary); Knowledge acquisition & retention<br>*Perception*: Visual<br>*Construction*<br>*Intelligence*: FSIQ | | | | | | | | |
| Raine 2004 | M | *Intelligence*: FSIQ | Unsuccessful psychopathy | General public | PCL-R≥23 | 16 | 33.8 | 6.6 | 96.4 | 14.7 |
| | | | Successful psychopathy | | | 12 | 29.5 | 6.4 | 97.3 | 13.2 |
| | | | Non-psychopathy | | PCL-R<23 | 23 | 28.4 | 6.6 | 105.1 | 16.9 |
| Raine 1988 | L | *Concept formation*: Abstraction; Reasoning; Arithmetic reasoning | Psychopathy | Prison | PCL median split | 12-14 | | | | |
| | | *Memory*: STM | Non-psychopathy | | | 14 | | | | |
| | | *Attention*: Sustained; Complex<br>*Language*: Verbal expression (vocabulary); Knowledge acquisition & retention<br>*Construction*<br>*Intelligence*: FSIQ | | | | | | | | |
| Reveillere 2003 | M | *Intelligence*: FSIQ | High-factor 1 psychopathy | | PCL-R cut-off 8 | 18 | 40.0 | 13.5 | 86.8 | 13.7 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Low-factor 1 psychopathy | | PCL-R cut-off 8 | 17 | 37.4 | 12.4 | 92.4 | 10.2 |
| Richell 2003 | L | *Concept formation*: Abstraction | Psychopathy | Prison | PCL-R≥30 | 19 | 32.2 | 6.8 | | |
| | | *Social cognition*: Theory of Mind | Non-psychopathy | | PCL-R≤20 | 18 | 33.3 | 8.1 | | |
| Richell 2005 | M | *Concept formation*: Abstraction | Psychopathy | Prison | PCL-R≥30 | 19 | 37.2 | 8.7 | | |
| | | *Social cognition*: Social interpretation & knowledge | Non-psychopathy | | PCL-R≤20 | 19 | 31.8 | 10.9 | | |
| Schmitt 1999 - African American sample only - Exp. 1 | L | *Executive*: Self-regulation (motor regulation); Effective performance | LA psychopathy | Prison | PCL-R≥30 | 13 | | | 97.6 | 11.7 |
| | | *Attention*: Selective | HA psychopathy | | | 12 | | | 96.0 | 10.3 |
| | | *Intelligence*: FSIQ | LA non-psychopathy | | PCL-R≤20 | 13 | | | 96.3 | 12.3 |
| | | | HA non-psychopathy | | | 13 | | | 97.8 | 10.3 |
| - Exp. 2 | | *Executive*: Self-regulation (motor regulation); Effective performance | LA psychopathy | Prison | PCL-R≥30 | 10 | | | 90.9 | 10.3 |
| | | *Attention*: Selective | HA psychopathy | | | 11 | | | 85.8 | 12.6 |
| | | *Intelligence*: FSIQ | LA non-psychopathy | | PCL-R≤20 | 10 | | | 86.4 | 13.0 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | HA non-psychopathy | | | 10 | | | 83.6 | 8.8 |
| - Exp. 3 | | *Executive*: Self-regulation (motor regulation); Effective performance | LA psychopathy | Prison | PCL-R≥30 | 11 | | | 88.8 | 12.8 |
| | | *Attention*: Selective | HA psychopathy | | | 11 | | | 84.5 | 13.3 |
| | | *Language*: Semantic processing | LA non-psychopathy | | PCL-R≤20 | 12 | | | 89.1 | 13.8 |
| | | *Intelligence*: FSIQ | HA non-psychopathy | | | 10 | | | 85.2 | 9.7 |
| - Exp. 4 | | *Executive*: Self-regulation (motor regulation); Effective performance | LA psychopathy | Prison | PCL-R≥30 | 10 | | | 90.6 | 11.2 |
| | | *Attention*: Selective | HA psychopathy | | | 9 | | | 94.8 | 12.9 |
| | | *Intelligence*: FSIQ | LA non-psychopathy | | PCL-R≤20 | 10 | | | 89.4 | 14.5 |
| | | | HA non-psychopathy | | | 8 | | | 85.7 | 13.2 |
| Schmitt Brinkley 1999 | L | *Executive*: Self-regulation (cognitive flexibility) | Psychopathy | Prison | PCL-R≥30 | 38 | | | | |
| | | | Non-psychopathy | | PCL-R≤20 | 51 | | | | |
| Smith 1999 | H | *Executive*: Self-regulation (motor regulation) | Psychopathy | Prison | PCL-R≥28 | 8 | 33.9 | 7.6 | 111.2 | 7.5 |
| | | *Intelligence*: FSIQ | Non-psychopathy | | PCL-R≤23 | 8 | 37.1 | 7.7 | 115.5 | 5.4 |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | HC | General public | PCL:SV | 8 | 32.5 | 7.7 | 118.7 | 3.7 | | |
| Smith 1992 | H | *Executive*: Self-regulation (productivity, motor regulation); Effective performance | LA psychopathy | Prison | PCL-R≥30 | 18 | 26.5 | 4.3 | 97.5 | 9.8 | 11.9 | 0.6 |
| | | *Concept formation*: Abstraction | HA psychopathy | | | 19 | 25.3 | 4.1 | 96.0 | 12.3 | 11.7 | 1.0 |
| | | *Memory*: STM; LTM; WM | LA non-psychopathy | | PCL-R≤20 | 18 | 26.9 | 4.2 | 98.6 | 9.3 | 11.5 | 0.9 |
| | | *Attention*: Selective/Complex | HA non-psychopathy | | | 14 | 24.9 | 4.6 | 95.9 | 8.1 | 11.4 | 1.0 |
| | | *Language*: Verbal expression (fluency) | | | | | | | | | | |
| | | *Construction* | | | | | | | | | | |
| | | *Motor performance* | | | | | | | | | | |
| | | | | | | Murderer sample/Non-murderer sample | | | | | | |
| Snowden 2004/ Gray 2003 | H | *Social cognition*: Social interpretation & knowledge | Psychopathy | Forensic inpatients | PCL-R≥30 | 23 (6/ 17) | 32.5/ 31.2 | 9.8/ 9.0 | 91.5/ 94.1 | 18.3/ 16.9 | | |
| | | *Intelligence*: VIQ | Non-psychopathy | | PCL-R≤19 | 51 (11/ 40) | 36.8/ 37.0 | 8.8/ 10.7 | 98.1/ 98.3 | 16.5/ 19.7 | | |
| Suchy 2005 - LHA | H | *Attention*: Divided | Psychopathy | Prison | PCL-R≥30 | 12 | 26.0 | 5.6 | 94.4 | 9.8 | | |
| | | *Perception*: Visual; Auditory | Non-psychopathy | | PCL-R≤21 | 19 | 27.4 | 6.6 | 98.8 | 9.3 | | |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| - RHA | | *Intelligence*: FSIQ | | | | 14 | 26.4 | 5.8 | 97.6 | 9.3 | | |
| | | | | | | 13 | 27.6 | 4.7 | 98.2 | 10.9 | | |
| Suchy 2006 | H | *Language*: Semantic processing | Psychopathy | Prison | PCL-R≥30 | 23 | 27.0 | 7.1 | 96.4 | 9.9 | 11.4 | 1.9 |
| | | *Perception*: Visual | Non-psychopathy | | PCL-R≤20 | 21 | 24.9 | 6.7 | 96.2 | 6.3 | 11.8 | 1.3 |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Swogger 2006 | L | *Executive*: Self-regulation (cognitive flexibility, motor regulation) | Psychopathy | Prison | PCL-R≥30 | 47 | 30.2 | 6.9 | 88.7 | 10.6 | | |
| | | *Language*: Academic skills (reading) | Non-psychopathy | | PCL-R≤20 | 38 | 26.7 | 7.1 | 91.4 | 10.0 | | |
| | | *Intelligence*: FSIQ | | | | | | | | | | |
| Williamson Harpur 1991 | M | *Affect*: Processing | Psychopathy | Prison | PCL>33 | 8 | 25.0 | | | | 10.7 | |
| | | *Language*: Semantic processing | Non-psychopathy | | PCL<25 | 8 | 23.0 | | | | 8.4 | |
| Williamson 1991 | L | *Language*: Verbal expression (discourse) | Psychopathy | Prison | PCL-R≥30 | 21 | | | | | | |
| | | | Non-psychopathy | | PCL-R≤20 | 15 | | | | | | |
| Yang 2005 | M | *Intelligence*: FSIQ | Unsuccessful psychopathy | General public | PCL-R≥23 | 16 | 33.8 | 6.6 | 96.4 | 14.7 | | |
| | | | Successful psychopathy | | | 13 | 29.6 | 6.1 | 99.1 | 14.2 | | |
| | | | Non-psychopathy | | PCL-R<23 | 23 | 28.4 | 6.6 | 105.1 | 16.9 | | |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Zeier 2009 | H | *Executive*: Effective performance | LA psychopathy | Prison | PCL-R≥30 | 14 | 31.8 | 9.2 | 99.8 | 13.8 |
| | | *Attention*: Selective | HA psychopathy | | | 22 | 31.6 | 6.8 | 96.7 | 10.3 |
| | | *Intelligence*: FSIQ | LA non-psychopathy | | PCL-R≤20 | 30 | 33.5 | 7.0 | 103.8 | 10.9 |
| | | | HA non-psychopathy | | | 25 | 33.1 | 7.6 | 103.8 | 9.4 |

*Note.* ASPD/DPD=Antisocial/Dissocial Personality Disorder; HC=Healthy control; LA/HA=Low/high-anxious; SCID-I/II/NP=Structured Clinical Interview for DMS Disorders-Axis I/II/Non-patient Version; DMS-IV-TR=Diagnostic and Statistical Manual, Version 4, Text Revision; Q/DIS-III-R=Quick/Diagnostic Interview Schedule for DSM-III-R; MINI=Mini International Neuropsychiatric Interview; PCL/-R/:SV=Psychopathy Checklist/-Revised/: Screening version; ICD-10=International Classification of Diseases and Related Health Problems, 10th Version; STM/LTM/WM=Short/long-term/working memory; FSIQ/VIQ/PIQ=Full-scale/verbal/performance IQ; NSS=Neurological soft signs; H=High; M=Medium; L=Low.

[a] Included as part of the ASPD diagnosis.

# 12 APPENDIX D: FUNNEL PLOTS

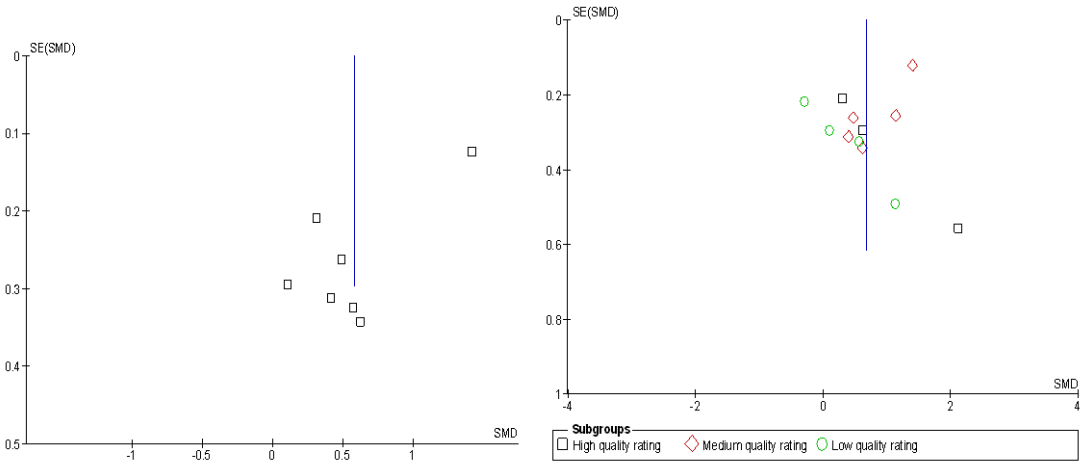


*Figure 12.1.* Respective funnel plots of attentional set shifting and overall cognitive flexibility data in psychopathy (strongest effects).

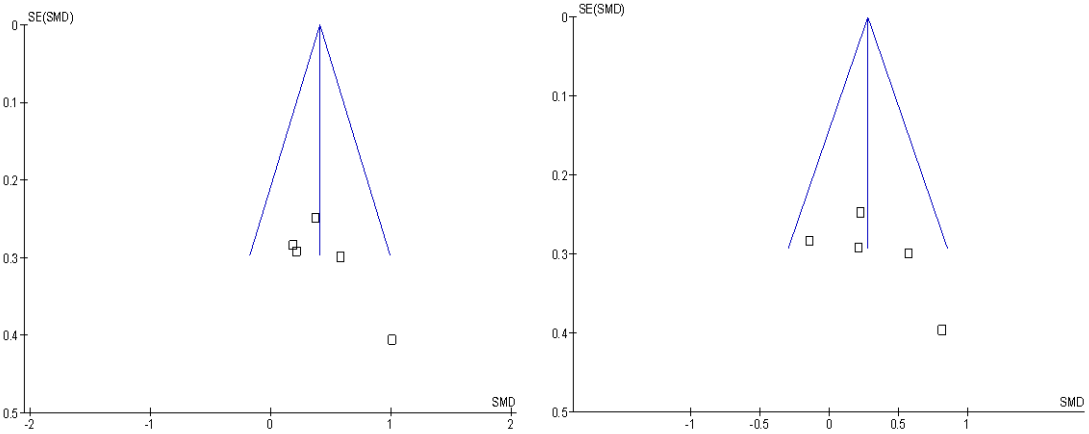


*Figure 12.2.* Respective funnel plots of motor regulation data in ASPD with strongest and weakest effects respectively.

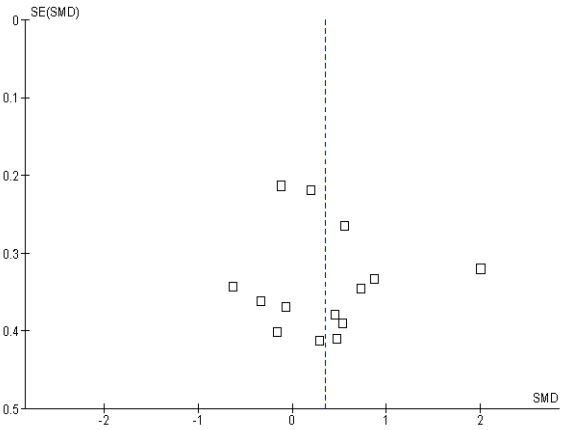


*Figure 12.3.* Funnel plot of response inhibition data in psychopathy (strongest effects).

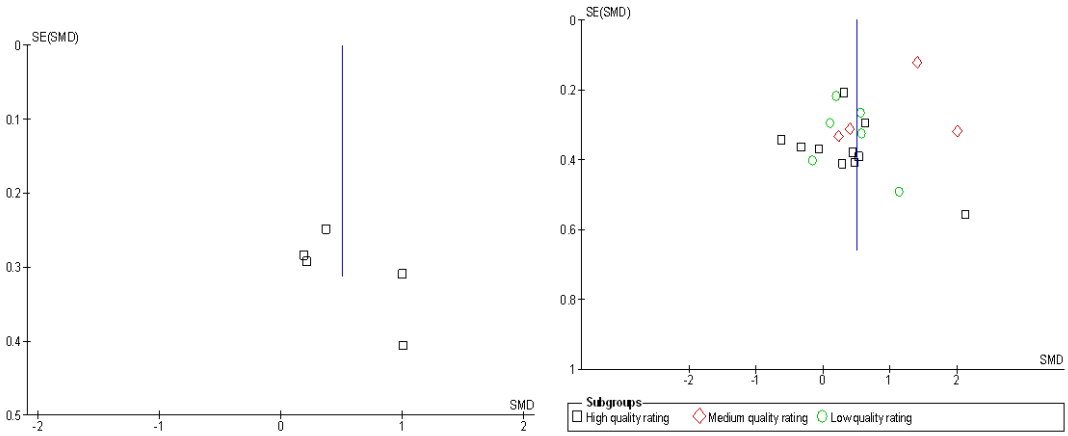


*Figure 12.4.* Respective funnel plots of self-regulation data in ASPD and psychopathy respectively (strongest effects).

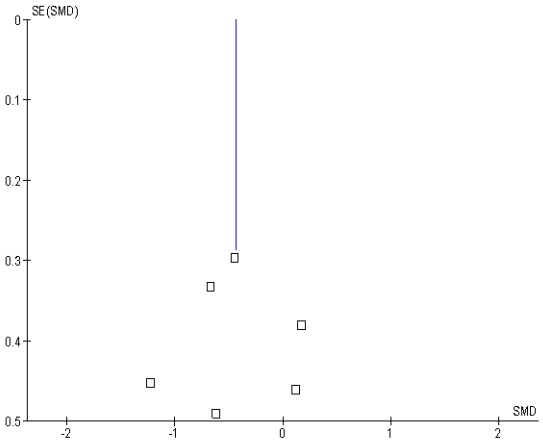


*Figure 12.5.* Funnel plot of interference data from effective performance studies in psychopathy which employed non-Stroop paradigms.

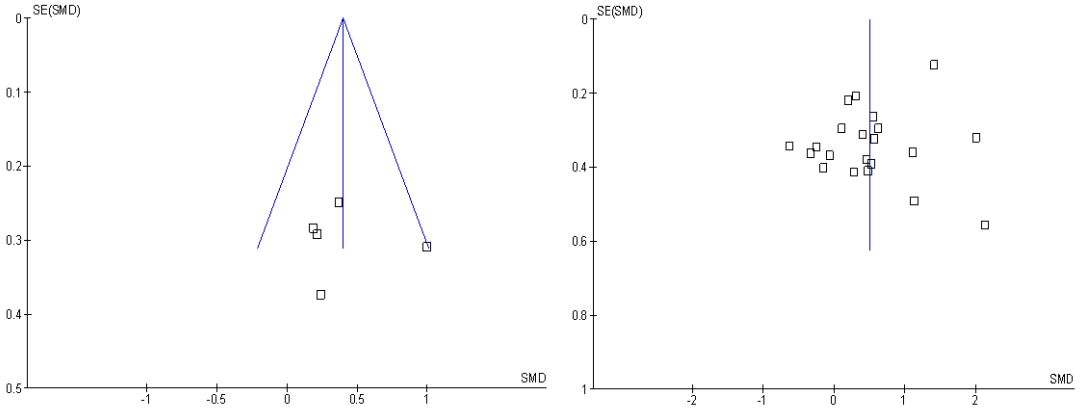


*Figure 12.6.* Respective funnel plots of executive function data (strongest effects) in ASPD and psychopathy with poor performance data.

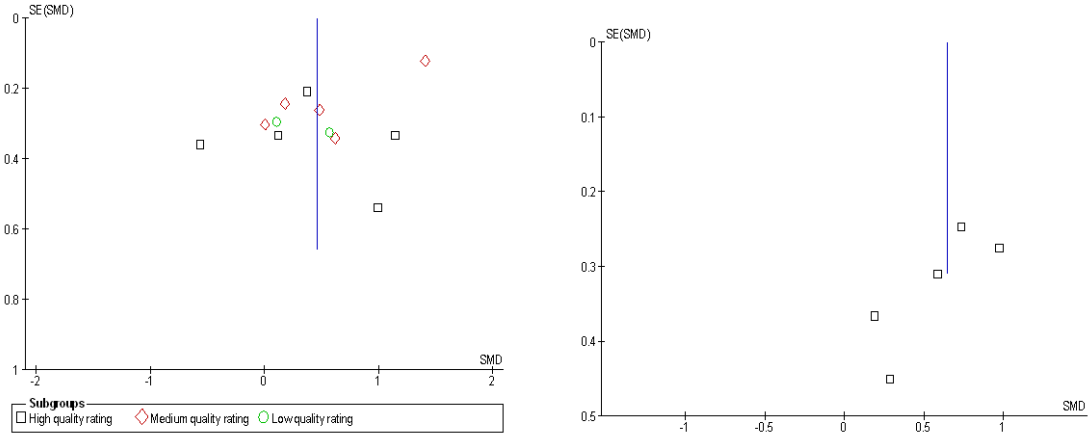


*Figure 12.7.* Respective funnel plots of overall abstraction and affective processing data in psychopathy (strongest effects).

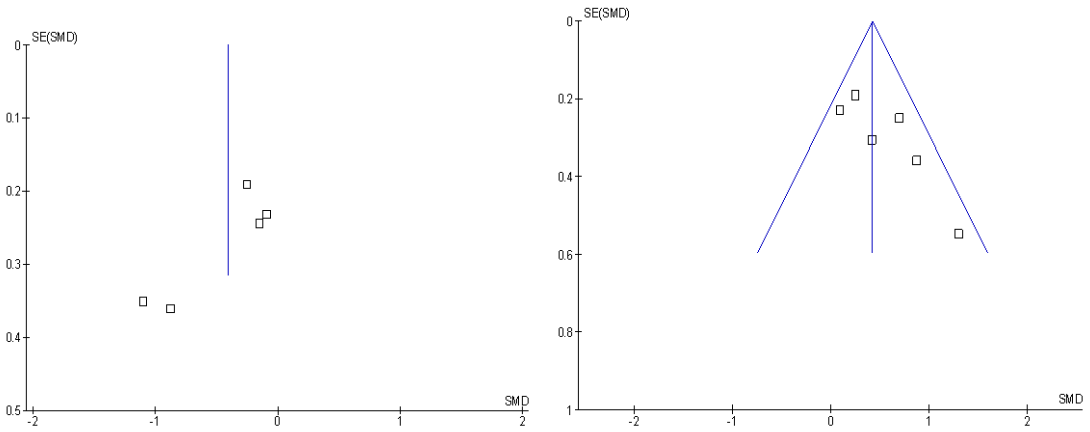


*Figure 12.8.* Respective funnel plots of visual and overall affect recognition data in psychopathy (strongest effects).

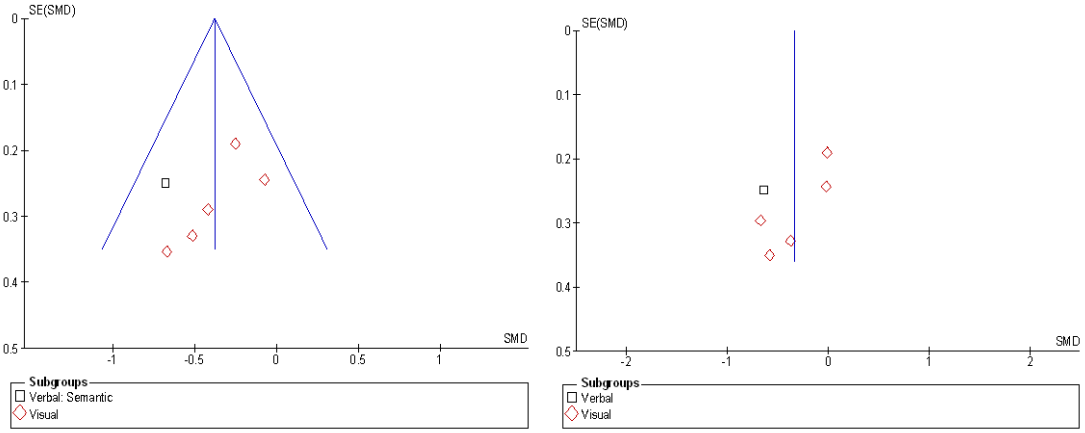


*Figure 12.9.* Respective funnel plots of happiness and sadness recognition data in psychopathy.

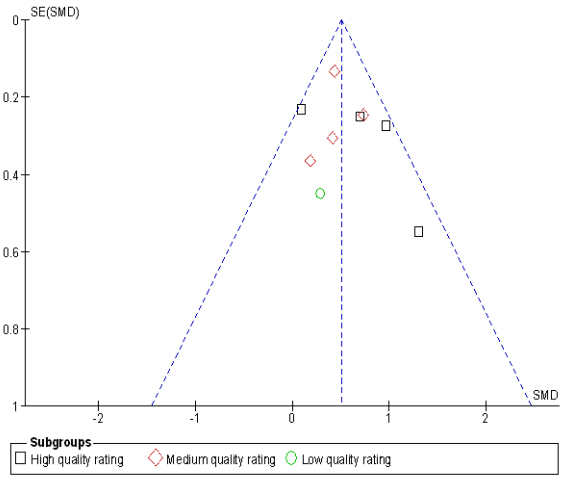


*Figure 12.10.* Funnel plot of data on affective operations in psychopathy (strongest effects).

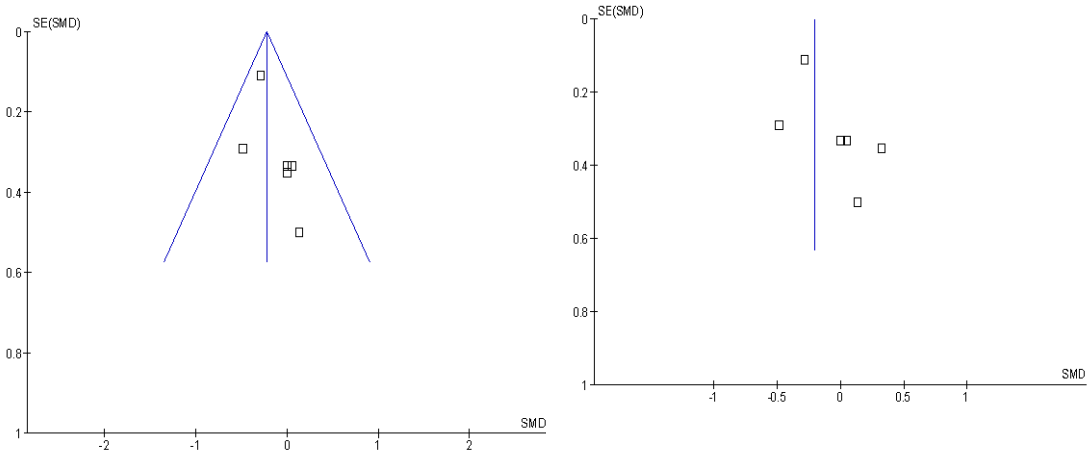


*Figure 12.11.* Funnel plots of verbal and STM data in psychopathy respectively (strongest effects).

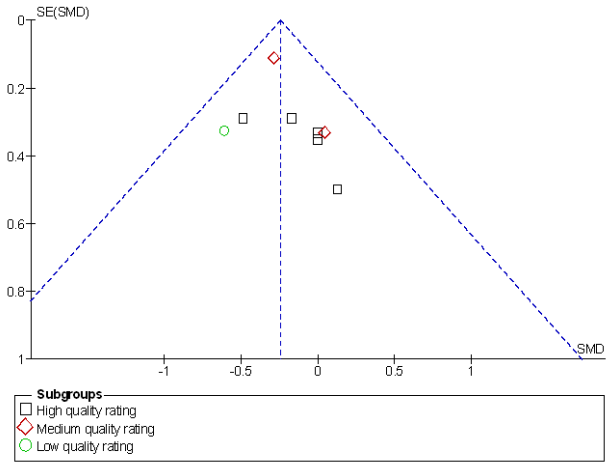


*Figure 12.12.* Funnel plot of memory data in psychopathy (strongest effects).

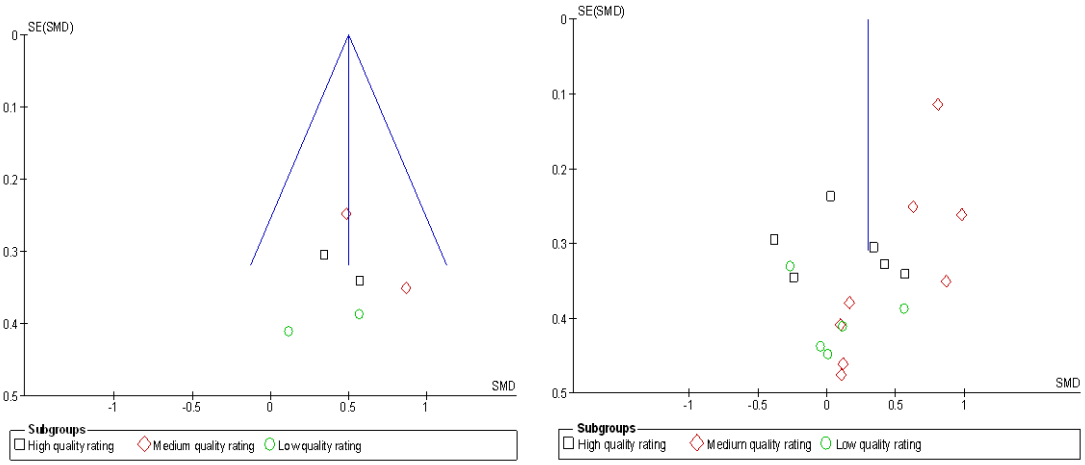


*Figure 12.13.* Funnel plot of sustained and overall attention data in psychopathy (strongest effects) respectively.

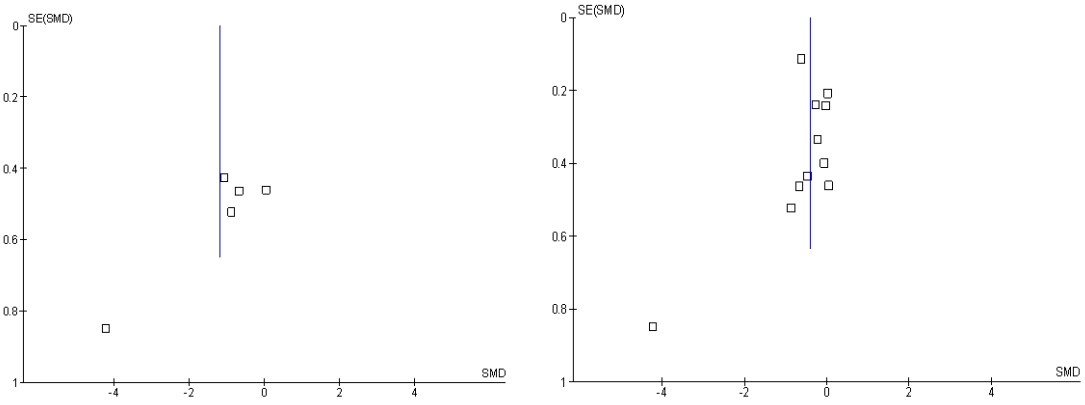


*Figure 12.14.* Funnel plot of syntax and overall verbal expression data in psychopathy (strongest effects) respectively.

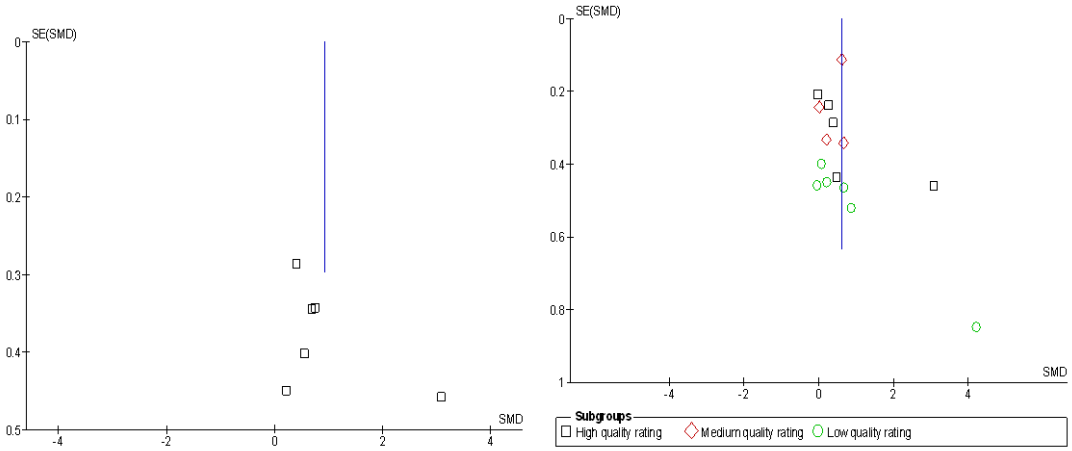


*Figure 12.15.* Funnel plots of semantic processing and language data in psychopathy (strongest effects) respectively.

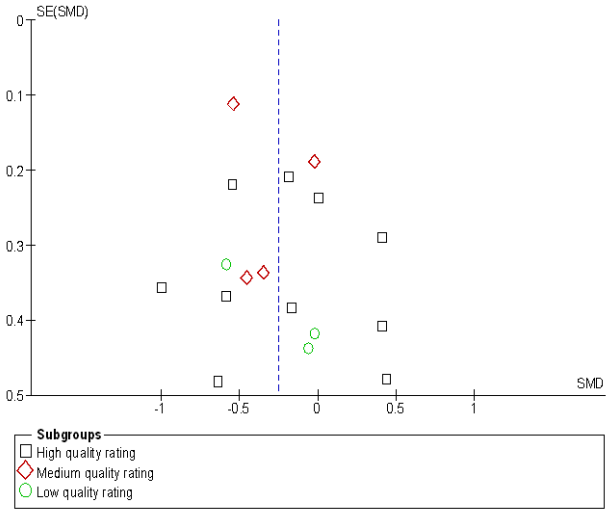


*Figure 12.16.* Funnel plot of intelligence (FSIQ) data in psychopathy (strongest effects).

# 13 APPENDIX E: HEALTHY CONTROL GROUP MATERIALS

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**Screening Questionnaire**
**(Interview Schedule)**

(Indicate Yes/No and comments)

1. Handedness: _____
2. Current medication use (psychotropic): _____
3. Traumatic brain injury (unconsciousness for over 2 hours): _____
4. Neurological condition (epilepsy, brain tumour, etc.): _____
5. Major mental illness: _____
6. Years in education: _____
7. Participants must not have **current** alcohol or drug abuse/dependence and no prior history of abuse/dependence: Still willing to participate? _____

Participant ID: _____ DOB:_____ Date:_____

**Main Interview Schedule**

*Brain Injury*

1. Ever had a physical brain injury or suffered from a neurological condition (e.g. epilepsy)? _____

2. Ever been knocked unconscious for over 24 hours/went to hospital (yes/no)? _____

    a. If no, ever been knocked unconscious and for how long (ascertain longer than 2 hours if possible)/went to hospital (yes/no)? _____

    b. Ever been hit on the head (note whether went to hospital and for what)? _____

# 14 APPENDIX F: THE PROGRESS RATING SCHEDULE

| *Item* | *Scale* | *Derived from* | *Notes* |
|---|---|---|---|
| **PART A** | | | |
| Engagement with therapeutic programme | Poor (0) – not engaging, all reports bad.<br>Limited (1) – some initial engagement, one or two (out of the five) positive reports.<br>Good (2) – at least three (out of five) positive reports.<br>Excellent (3) – all reports positive. | Overall CPA summary<br>Medical report<br>Psychology report<br>Nursing report<br>OT report<br>Education report | Scope: Attending & contributing to treatment groups (e.g. problem solving, substance misuse, etc.), attempts to use acquired skills (e.g. problem solving) outside the group, compliance with homework, any 1:1 psychology sessions, engagement in OT and education, compliance with medication, etc.<br><br>Scoring: Reports with some positive and some negative comments (e.g. engages well individually but not when in groups or at least 50% engagement) may count as half a positive report towards the total.<br>Scoring can be pro-rated where fewer than 5 individual reports are available. |
| Behaviour | Poor (0) – on probation, violent incident (actual physical violence in review period) or 3 or more relevant recorded incidents, more than one positive drug screen or room search, any other serious inappropriate behaviour (inc. that leading to discharge).<br>Reasonable (1) – up to two relevant entries in incident logs, not more than one positive drug screen or room search.<br>Good (2) – no incident log entries, positive drug screens or room searches but more subtle indications that further improvement is needed, e.g. rule breaking.<br>Excellent (3) – none of the above. | All CPA reports | Scope: Includes indicators of negative behaviour from 'on probation' status, relevant recorded incidents (e.g. incident logs for perpetrated aggression, security breaches including drugs, anger logs), comments about adherence to the rules, comments about boundary issues, any other inappropriate behaviour, results of any room searches or drug screens (refusal=negative result). |

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| Mental state | Poor (0) – some symptoms most of the time (> 50%), more than one episode of self-harm.<br>Variable (1) – Significant periods of symptomatology, one episode of self-harm, more than minimal interference with daily functioning<br>Good (2) – Minimal symptoms, transient, minimal interference with activities<br>Very good (3) – stable throughout whole review period, no more than normal day to day fluctuations, no interference of mental state with activities | Medical report<br>Psychology report<br>Nursing report | Scope: Generally Axis I including depression or low mood, anxiety (inc. PTSD), hypomania or mania, delusions, hallucinations; also violent or suicidal ideation and any self-harm.<br>Note: 1 episode of self-harm may include several incidents. |
| Interaction with peers and other non-staff individuals | Poor (0) – serious concerns about interaction with clear indications of inappropriateness with at least 1 peer/non-staff individual.<br>Reasonable (1) – Limited interactions with majority but no significant concerns or problematic interactions with majority but less severe than above.<br>Good (2) – Positive interactions with majority with minimal concerns, difficulties with some peers.<br>Very good (3) – Positive interactions with no concerns with (almost) all peers. | All CPA reports | Scope: All interactions with peers and, if off the service, with people outside (excluding family/friends). Would include participation in any formal social activities on/off the service. |

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| Interaction with staff | Poor (0) – serious concerns about interaction with clear indications of inappropriateness with at least 1 member of staff.<br><br>Reasonable (1) – Limited interactions with majority but no significant concerns or problematic interactions with majority but less severe than above.<br><br>Good (2) – Positive interactions with majority with minimal concerns, difficulties with some staff.<br><br>Very good (3) – Positive interactions with no concerns, refers to staff for problem solving, positive therapeutic relationship with (almost) all professionals. | All CPA reports | <u>Scope:</u> All interactions with staff. |
| Insight | Poor (0) – no insight, wants to go back to prison, constant ambivalence about staying or staying for the wrong reasons (e.g. parole), takes virtually no responsibility for actions/problems, does not recognise need for treatment (evidenced verbally or in overall presentation).<br><br>Reasonable (1) – some evidence of insight into own problems but assuming only a degree of responsibility for his actions/ problems (e.g. continues to blame others) evidenced verbally or in overall presentation.<br><br>Good (2) – good insight but with some further work to do.<br><br>Excellent (3) – Excellent insight, reflects on own problems, balanced, thoughtful view of own problems. | All CPA reports | <u>Scope:</u> Insight into personality function, risk, need for treatment, realistic future expectations. |

| **PART B** | | | |
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| Supportive relationship | No (0)<br>Maybe (½) – e.g. contact only recently re-established, quality of relationship uncertain.<br>Yes (1) | Social work report | Scope: Evidence of supportive relationship outside clinical team: family or otherwise, visits, regular phone/letter contact, attendance at care review meetings. |
| Risk / Violence | 1. Summary score HCR-20 C and R scales:<br>C: High (0) – Medium (½) – Low (1)<br>R: High (0) – Medium (½) – Low (1) | | Scoring: Low: <25$^{th}$ percentile; Medium: 25$^{th}$-75$^{th}$ percentile; High: >75$^{th}$ percentile.<br><br>Note: Dynamic parts of other actuarian risk assessments may be also suitable, e.g. the VRS (dynamic factors), provided suitable norms are available and the scoring sum for risk levels does not exceed current totals (0-1-2), for equivalence. |
| Employment | No (0)<br>Inside hospital (1)<br>Outside hospital (2) | All CPA reports | Scope: If applicable, any employment within or outside the service. Defined as a regular, ongoing work, including work placement (e.g. working in patient library, coffee shop, etc.), that is not as part of an OT session; further education/college. |
| Leave | No (0) – escorted (1) – unescorted (2) | All CPA reports | Scope: Any regular, escorted leaves, any unescorted leaves, exclude one off leave for hospital appointments, court appearances, etc. Only rate if applicable, ie if patient not suitable for leave for legal reasons (eg. prison transfer) omit item. |
| Final outcome (at discharge) | Negative (0): Transfer back to prison for non-engagement/serious concerns, transfer to high security.<br>Positive (1): Transfer to conditions of same or less security, move back to prison after treatment successfully completed. | Discharge summary<br>S.117 meeting minutes | |

| **PART C: Local items** | | | |
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| Examples:<br><br>Psychometrics, violent incidents, etc. | | | |