Using Mentalizing and Psychopathy to Explore a Dimensional Model of Antisocial and Borderline Personality Disorder

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Thesis declaration form

I confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

Signature:

Name: Josephine Carlisle

Date: 13th June 2014
Overview

Part one of the thesis reviews the literature on whether antisocial personality disorder (ASPD) and psychopathy represent distinct categories. This question was addressed by identifying studies with populations of individuals meeting criteria for ASPD and exploring the samples in terms of other constructs. Studies are divided into four areas; cluster analytic studies, studies of emotional processing, theory of mind and mentalizing, and executive functioning. The review suggests that those who meet criteria for ASPD represent a heterogeneous group, and that psychopathy is distinct from ASPD.

Part two consists of an empirical paper which measures the constructs of mentalizing and psychopathy in a sample of people with and without diagnoses of antisocial personality disorder (ASPD) or borderline personality disorder (BPD). This allowed for the testing of the mentalizing deficit theory of BPD, to explore mentalizing in an ASPD sample, and also to explore the construct of psychopathy, which has been used interchangeably with ASPD. BPD has also been suggested to be a phenotypic expression of psychopathy. Results supported a mentalizing deficit in BPD, and support the premise that ASPD is a heterogeneous group, and consists of at least two subtypes. The implications of findings in the context of a paradigm shift away from categorical towards a dimensional model of personality disorder are discussed, along with the limitations of the study and implications for future research.

In part three a critical appraisal of the research process is presented. Issues of research in the probation setting, risk and ethical issues of working with this population, and also the practicalities of working alongside a large scale research project are discussed, in order to guide future research in this area.
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Part one: Literature Review

Are Psychopathy and Antisocial Personality Disorder Distinct Disorders?
Abstract

Aims

This systematic literature review addresses the question of whether Psychopathy and Antisocial Personality Disorder (ASPD) exist as two distinct disorders, or whether they represent different points of one continuum.

Method

PsycINFO, EMBASE and MEDLINE databases were searched for studies spanning the past decade up until 31st March 2014 in order to select studies to be included in the review. PsycEXTRA was also searched, within the same time frame, in order to explore the grey literature. In total 12 studies were selected for review.

Results

The studies suggest that those scoring highly on measures of psychopathy seem to be unique from other ASPD offenders in terms of emotional processing, comorbid psychopathology, risk of violence, neuropsychological factors and structural differences in certain brain regions.

Conclusion

The diagnostic category of ASPD seems to encompass a heterogeneous group of people. These findings call for careful use of the diagnosis of ASPD and have implications for the use of the diagnostic category in terms of risk assessment, treatment planning, and access to services.
1. Introduction

People given a diagnosis of antisocial personality disorder (ASPD), broadly speaking, are those who repeatedly offend from a young age, are irresponsible, impulsive and lack remorse. It is estimated that around 4.1% of the general community in the United States meet criteria for a diagnosis of ASPD, and in the general British population, a prevalence of between 0.3 and 1.1% has been reported (Coid et al., 2006). British and American prison populations are estimated to have around a 10 times higher incidence of ASPD in comparison to community samples (Fazel & Danesh, 2002). Clearly a diagnosis of ASPD represents a significant cost to the individual, their friends, family and colleagues, and to the healthcare and criminal justice systems. It has been associated with increased risk of recidivism and therefore is a heavy influence on criminal justice pathways. Given the association of ASPD with poorer treatment outcomes, it is often used as an exclusion criterion in mental health services, restricting access to treatment for those given the diagnosis. Stigma continues to surround the diagnosis, despite the publication of the government document, “Personality Disorder: No Longer a Diagnosis of Exclusion” (Snowden & Kane, 2003). The implications both to society, and the lifelong implications to those labelled with ASPD highlight the importance of examining the diagnostic construct and how it is used.

As part of the revision of Diagnostic and Statistical Manual (4th edition, text revision, American Psychiatric Association, 2000) in preparation for DSM-5 (APA, 2013), a working party was formed which considered a revision of the way that personality disorders were classified (e.g. Widiger & Simonsen, 2005). Many proposed changes were discussed, such as the creation of the category “antisocial/psychopathic prototype” (Hesse, 2010). For personality disorder in general, a move was proposed away from the ten categories of personality disorder towards a dimensional model of personality pathology (Krueger et al., 2011). This is reflective of the work of leading theorists such as Joel Paris, who proposed that...
borderline personality disorder (BPD) and classic psychopathic presentations may represent different points of the same continuum (Paris, 1997), and of professor Jeremy Coid, who suggested that psychopathy represents the end point of a continuum of antisocial personality pathology (Coid & Ullrich, 2010).

A set of guidelines for the assessment and treatment of ASPD were produced by the National Institute for Health and Care Excellence (NICE) summarized by Kendall et al. (2009). They highlight those scoring highly on psychopathy screening measures as being of high risk to others and as having a “lifelong disability”. NICE were pessimistic about the treatability of the group of people encompassed in the ASPD guidelines. The guideline has been criticized for its reification of a potentially heterogeneous group (Pickersgill, 2009). Potentially, as the literature reviewed here explores, a wide range of people are being grouped together and labelled as “untreatable”, which highlights the importance of reviewing this area of the literature. Perhaps those at the higher end of a spectrum of constructs such as psychopathy are responsible for the assumption that ASPD is difficult to treat. The guidelines recommend that severity of ASPD is assessed using the Psychopathy Checklist-Revised (PCL-R, Hare, 2003), which is a measure of psychopathy. Therefore the guidelines seem to imply that ASPD and psychopathy are one and the same. Critiques of the guideline highlight that later in the document they are in fact treated as separate entities, citing the research finding that only a small proportion of those meeting criteria for ASPD also meet criteria for psychopathy (Pickersgill, 2009).

1.1 Psychopathy

The apparent confusion in the ASPD guidelines as to whether this is a disorder distinct from psychopathy, is symbolic of a debate spanning the last two centuries (Arrigo & Shipley, 2001). The “psychopath” was first described by Cleckley, who described a syndrome characterised by behavioural, interpersonal
and affective symptoms, such as antisocial behaviour, deceit and insincerity and lack of remorse. Cleckley’s book, “the mask of sanity” described how these symptoms were “masked” by features such as “good intelligence, superficial charm, lack of delusions, lack of irrational thinking, and an absence of neuroticism” (Cleckley, 1982). This definition was later refined and empirically validated by Robert Hare, who developed what continues to be the gold standard in measuring psychopathy, the PCL-R. Psychopaths were described by Hare as “human predators who coldly, callously, and ruthlessly use charm, deceit, manipulation, threats, intimidation, and violence to dominate and control others and to satisfy their own selfish needs and desires” (Hare & Hart, 1993).

There is evidence to suggest that those rating highly on measures of psychopathy are distinct from other “antisocial” individuals. More recent explorations of the construct of psychopathy have moved away from a focus on behavioural factors towards a neurodevelopmental and cognitive aetiology (Blair, 1995). The Integrated Emotions System (IES) model incorporates the fear recognition and amygdala dysfunction theories of psychopathy (Blair, Mitchell & Blair, 2005). The fear recognition theory is supported by research that shows inferior performance in facial emotion recognition paradigms in both adults and children rating highly on psychopathic traits (Dadds et al., 2006) and in patients with amygdala damage (Adolphs, 2002). A failure to recognize distress in others leads to a failure of the normal process of inhibition of behaviours which cause distress in others via classical conditioning (the violence inhibition mechanism, or VIM), leading to a failure to develop the moral emotions, such as empathy. The IES model describes three neural networks, interacting with the central (CeN) and basolateral nuclei of the amygdala (Blair et al., 2005). Rather than a single causal impairment, as in the VIM theory, separable pathways are proposed by the IES which are responsible for different types of conditioning. This explains why in patients with a lesion to the CeN, aversive conditioning may be absent but instrumental learning remains intact.
(Blair, 2005). The unique symptomatology of psychopathy could be understood, in terms of the IES, as a disruption in the pathology of these neural networks. This provides an explanation of the deficiency in moral socialisation, underdeveloped empathy and antisocial behaviour/aggression which is characteristic of psychopathy.

Evidence supports psychopathy as a construct unique from general antisocial pathology. Elevated levels of instrumental aggression have been observed in this group (Blair et al., 2004; Marsh & Blair, 2008). Psychopaths in general are at a greater risk of violent behaviour (Cooke, Michie, Hart & Clark, 2005). In forensic populations, research suggests that psychopathic individuals are more likely than non-psychopathic offenders to violently reoffend soon after release. One study found that psychopaths actually had a higher rate of recidivism after treatment in a Canadian therapeutic community, whereas non-psychopaths showed some improvement (Hobson, Shine & Roberts, 2000). Psychopaths may also start offending at an earlier age in comparison to other non-psychopathic offenders (De Brito, Viding, Kumari & Blackwood 2013).

Although there is a history of theorising about subtypes of psychopathy, only relatively recently have these been studied empirically. Cluster analytic studies of offenders have repeatedly identified subgroups within psychopaths that map on to primary and secondary psychopathy (Hare, 1991; Benning, Patrick, Hicks, Blonigen & Krueger, 2003; Marcus, Fulton & Edens, 2013). The “primary psychopath” describes Cleckley’s classic cold, callous, unemotional, low-anxious psychopath. In contrast, it is postulated that secondary psychopaths engage in antisocial behaviour as a result of negative internal states such as anxiety and guilt and have been described as having a low tolerance to stress, being prone to irritability and worry, thus expressing more reactive aggression (Karpman, 1948 cited in Blackburn, 1975; Lykken, 1995). Three (Cooke & Michie, 2001) and four (Hare, 2003) factor models of psychopathy have also been proposed.

The PCL-R remains the “gold standard” in the diagnosis of psychopathy,
which incorporates the primary and secondary factors described above. For the purposes of this review the conceptualisation of “psychopathy” that is ascribed to is that which is described by the PCL-R, which is commonly used in the studies described. In order to meet criteria for “psychopathy”, sufficient criteria (depending on the cut-off score used) must be met on both primary and secondary factors. There is of course the possibility that more than two factors exist, or that psychopathy may be better conceptualised as a dimensional construct. This will be informed by the following review of the literature.

1.2 Antisocial Personality Disorder

Cleckley’s description of a glib, low-anxious, insincere and superficial psychopath was criticised for being based largely on unobservable, unmeasurable traits. In the DSM-III (American Psychiatric Association, 1980) these features formed the bases of the description of Antisocial Personality Disorder (ASPD). Primary features were observable, behavioural characteristics like criminality, delinquency and irresponsibility. This was in turn criticised as these criteria arguably describes a large majority of the prison population and thus has lead to a vast number of people who engage in persistent criminal or antisocial behaviour to be labeled with this diagnosis. It could be argued that as a result one diagnostic category may include a very heterogeneous group. When the PCL –R was administered to 80 inmates, of which half met the criteria for ASPD, only 12.5% met the criteria for psychopathy (Hare, Hart & Harpur, 1991).

In order to distinguish what differentiates ASPD as a diagnosis as opposed to someone who engages in criminal behaviour it is necessary to consider the developmental aetiology of personality disorder. Table 1 lists the features that must be present in order to give a diagnosis of ASPD. Conduct disorder must have been present prior to the age of 18, however not every adolescent that engages in antisocial behaviour goes on to develop ASPD. For a diagnosis of a personality
disorder to be given these features must affect functioning in different domains and be prevalent across the lifespan. A toxic combination of biochemical, genetic, autonomic and environmental factors elevate the risk of conduct disorder, which, given a permissive environment, adverse life circumstances, and through processes such as social learning can go on to develop into ASPD, which is one of many developmental trajectories (Martens, 2000).

Table 1

_DSM-IV Criteria for ASPD_

A. There is a pervasive pattern of disregard for and violation of the rights of others occurring since age 15 years, as indicated by three (or more) of the following:
(1) failure to conform to social norms with respect to lawful behaviors as indicated by repeatedly performing acts that are grounds for arrest
(2) deceitfulness, as indicated by repeated lying, use of aliases, or conning others for personal profit or pleasure
(3) impulsivity or failure to plan ahead
(4) irritability and aggressiveness, as indicated by repeated physical fights or assaults
(5) reckless disregard for safety of self or others
(6) consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations
(7) lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another

B. The individual is at least age 18 years.

C. There is evidence of Conduct Disorder with onset before age 15 years.

D. The occurrence of antisocial behavior is not exclusively during the course of Schizophrenia or a Manic Episode.

1.3 ASPD and psychopathy, distinct disorders?

There is a divide amongst researchers as to whether psychopathy and ASPD should be considered as distinct disorders, or whether they represent
manifestations of the same disorder. Robert Hare’s development of the PCL-R was crucial in this debate. Studies showed that 70-80% of prisoners met the diagnosis for ASPD, but of those that met the criteria for ASPD only a third were classified as meeting the criteria for psychopathy (Widiger & Corbitt, 1995). It was argued that ASPD was capturing many people who were merely engaging in antisocial behaviour, and that most of these people did not possess the affective/interpersonal traits seen in psychopaths, which theorists regarded as an etiologically distinct subgroup (Lykken, 1995). This was supported by the finding, described above, that most people with a diagnosis of ASPD do not meet criteria for psychopathy. This evidence seemed to provide empirical support for ASPD and psychopathy as distinct categories. Henry and Moffitt (1998) warned that if we considered ASPD as a homogenous group, “we may be comparing apples and oranges”.

Table 2

<table>
<thead>
<tr>
<th>Factor 1</th>
<th>Interpersonal Affective</th>
<th>Factor 2 Behavioural</th>
<th>Other</th>
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<tbody>
<tr>
<td>Glibness / Superficial charm</td>
<td>Need for stimulation / proneness to boredom</td>
<td>Criminal Versatility</td>
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<td>Superficial sense of self worth</td>
<td>Parasitic lifestyle</td>
<td>Many short term marital relationships</td>
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<td>Pathological Lying</td>
<td>Poor behavioural controls</td>
<td>Sexual promiscuity</td>
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<td>Conning / Manipulative</td>
<td>Early behaviour problems</td>
<td></td>
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<tr>
<td>Lack of remorse or guilt</td>
<td>Lack of realistic goals</td>
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<td>Shallow affect</td>
<td>Impulsivity</td>
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<tr>
<td>Callous / lack of empathy</td>
<td>Irresponsibility</td>
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<td>Failure to accept responsibility</td>
<td>Juvenile delinquency</td>
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<td></td>
<td>Revocation of conditional release</td>
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</table>
Whilst this could be considered as evidence that the two are distinct, others argue that this merely reflects differences in the sensitivity and specificity of the tools used to measure ASPD and psychopathy (Widiger et al., 1996). The PCL-R has more items and a higher cut-off score than the ASPD DSM-IV criteria, which means a higher level of specificity, but it remains possible that these two measures are identifying a cohort of people with a similar underlying disorder (Kosson, Lorenz & Newman, 2006). See Tables 1 and 2 for a list of the diagnostic criteria for both ASPD and psychopathy.

In terms of differences between the two constructs, there is empirical evidence to suggest that people scoring highly on measures of psychopathy are unique from those who meet criteria for ASPD. They differ in terms of outcomes such as violent recidivism and treatment failure (Hemphill, Hare & Wong 1998). Of people leaving a secure psychiatric hospital, 40% violently reoffended, compared to a 77% violent recidivism rate in psychopathic offenders (Harris, Rice & Cormier, 1991). The two may differ in other areas such as emotional processing (Kosson, Lorenz & Newman, 2006), executive functioning (Dolan, 2012) and other neuropsychological indices such as startle response (Vaidyanathan, Hall, Patrick & Bernat, 2011). These, amongst others, are constructs which are used in the papers currently reviewed in order to investigate whether ASPD and psychopathy represent distinct groups.

2. Aims

Given the possibility that the predictive value of the diagnostic category of ASPD may be driven by those psychopathic individuals that are encompassed by the diagnosis, it was necessary to strictly select papers for the current review that separate participants into those that meet criteria for ASPD only, and those that meet criteria for both psychopathy and ASPD. By including studies that have
employed this methodology to investigate differences between the two groups, this review aims to further clarify whether ASPD and psychopathy are distinct disorders.

3. Method

Keyword searches of PsycINFO, EMBASE and MEDLINE databases were performed. The search term “psychopath* adj10 "antisocial personality disorder" “and the following limits were applied:

- Peer reviewed journal articles
- Adult subjects (over 18 years)
- Last ten years
- English language

The total number of papers from this search was 134 once duplicates were removed. Abstracts were read and screened and papers were selected based on the following inclusion criteria:

- Empirical research studies with adult participants

The study of antisocial behaviour in adolescents represents a vast volume of literature. The focus of this review is on ASPD and psychopathy, both of which require a minimum age of 18 for a diagnosis.

- Must have administered measures of ASPD and psychopathy in a clinical or forensic population

There is a growing body of evidence on psychopathic traits in community samples, but this review is concerned with how the diagnostic labels are applied in clinical and forensic settings, where they can have an impact on access to services.

- Quantitative, empirical papers, not meta analyses or reviews
The current review aims to evaluate very specific studies in order to answer a specific question about a clinical population, and therefore the search was limited to quantitative empirical papers in order to achieve greater generalizeability of findings.

- Male participants only

Research in this area historically has focused almost exclusively in males. There is evidence to suggest that psychopathy and ASPD are expressed differentially according to biological sex (Cale & Lilienfield, 2002). The expression of these conditions in females necessitates its own body of research and is therefore beyond the scope of the current review.

- Original paper available in English

Based on these criteria 11 papers were identified for review. The reference lists of all relevant papers were screened and one further paper was identified, giving a total of 12 to be included in this review.

3.1 Quality appraisal

A quality appraisal tool, the “standard quality assessment criteria” (Kmet, Lee & Cook, 2004) was used in order to assess the quality of the studies selected. This allowed for conclusions drawn from the current review to be evaluated in terms of the quality of the studies from which they were drawn. The quality appraisal tool (see Appendix A1) includes 14 items, which are rated on a three point scale, giving a maximum score of 22 for each paper. The criteria covers, for example, the clarity of the description of the objectives of the study, methodological issues such as sampling and robustness of measurement tools, quality of data analysis, and whether the conclusions presented are actually represented in the data. Two raters used the tool to independently rate the papers. The two sets of scores were then compared, and in cases where a different rating was given, a consensus was
agreed upon through discussion. See appendix A2 for the final ratings for each
paper.

3.2 Measures of ASPD and Psychopathy

The papers selected for review utilized a range of measures to assess for
ASPD and psychopathy. The psychometric properties of each are described here.
a) PCL-R

As mentioned in the introduction section, the gold standard for the
assessment of psychopathy is the PCL-R (Hare, 2003). The PCL-R is a 20 item
scale (see table 2). The items are rated on a three point scale, from zero (item does
not apply) to two (item definitely applies), for a maximum score of 40. Ratings
should be carried out by a trained rater, based on interview and file data. As
described in the introduction, most items load onto one of two factors (see table 2).
Studies have suggested that the instrument has a standard error of measurement of
about 3 points, and good test-retest and inter-rater reliability (Schroeder, Schroeder
& Hare, 1983). These estimates are based on studies of prison samples, and
suggest that in this population the PCL-R is a unidimensional and homogenous
scale. It has been replicated in research that the interpersonal/affective factor has a
higher threshold than the impulsive/antisocial factor. Different cut off scores can be
applied in order to establish clinical levels of psychopathy, which is further discussed
in the review.
b) PCL-SV

Given that the PCL-R is costly and time consuming to administer, a
screening version was created which is particularly useful for research purposes
(PCL-SV, Hart, Hare & Cox, 1995). The PCL-SV is a 12 item scale based on the
PCL-R, which gives briefer criteria and requires less corroborative information to
rate. A cut off score of 18 is recommended for establishing psychopathy criteria. An
item response theory approach was used to compare the PCL-SV to the PCL-R and
results suggested that it can be considered a reliable short form of the PCL-R (Cooke, Michie, Hart & Hare, 1999). It correlated highly with the PCL-R even when administered across samples by different raters (r < .80).

c) PPI

The Psychopathic Personality Inventory (Lilienfield & Andrews, 1996) is an 187 item self-report scale for assessing psychopathic personality traits. The scale was originally created for use in a general population, given that the PLC-R and PCL-SV are mostly used in offending samples. However the PPI has since been shown to have good internal consistency and test–retest reliability in undergraduate and prison samples (Lilienfield & Andrews, 1996) and has been found to correlate positively and significantly with the PCL-R and PCL:SV (Malterer, Lilienfield, Neumann & Newman, 2009).

d) PAI

The Personality Assessment Inventory (PAI, Morey, 1991) is a 344 item self-report instrument that assesses a wide range of personality constructs. The PAI has good construct and discriminant validity. It includes four scales which assess for the validity of responses. Internal consistency of clinical scales is high with alphas in the .80s for clinical, student and general populations (reported in Strauss, 2006).

e) SCID I and SCID II

The structured clinical interview for diagnosis of DSM-IV axis I disorders and axis II personality disorders are semi structured diagnostic interviews for axis-I disorders and axis-II personality disorders. A study of a mixed in and out patient sample with non-clinical controls suggested that the SCID I had moderate to excellent inter rater reliability (Lobbestael, Leurgans & Arntz, 2011). Reliability for most personality disorders, measured categorically and dimensionally, was excellent. Studies suggest that the SCID II has similar reliability and validity in comparison to other measures of DSM personality disorders but with the advantage
of being quicker to administer (Spitzer, Gibbon, Williams, Davies, Borus & Rounsaville, 1995).

4. Results

After reading the 12 papers it was discovered that each focused on one of four areas; studies employing cluster analytic methodology to investigate the validity of the diagnostic categories, emotional processing, neuropsychological factors and other neurological factors. For clarity, the 12 papers were divided into these four areas for review.

4.1 Clustering and subtypes of antisocial personality

As described in the introduction section, some argue for the existence of variants of psychopathy, such as primary and secondary psychopaths. A three factor model has also been suggested (Cooke & Michie, 2001). The studies reviewed here (table 3) used statistical clustering techniques to explore these subgroups within large forensic samples meeting the criteria for ASPD. These studies aimed to further refine the construct validity of these clusters by investigating how ASPD offenders varied in terms of many theoretically relevant and clinically useful variables, such as aggression, treatment outcome, and institutional infractions.

A methodological strength of all of the studies in this section is their large sample sizes. It can be difficult to recruit such large numbers from such a specific group as ASPD offenders. All participants were from either prison or court mandated residential drug treatment programmes, which impacts on the generalizability of findings to non-incarcerated populations. The first two studies, (Poythress et al., 2010 and Cox et al., 2013) used the same sample. The SCID-II was used to identify 691 men meeting the criteria for ASPD. The PCL-R was also administered to these men as a measure of psychopathy. They then selected further factors, based in
empirically supported theory, which would help to identify whether these 691 men, given the diagnosis of ASPD, actually differed on meaningful variables such as violence, aggression, anxiety, depression, impulsivity, dominance and passive avoidance learning. Therapist rating scales were used to assess treatment motivation and progress in therapy. A methodological strength of this study was the use of the well validated PAI scale to provide corroboration for therapist ratings.
<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>Sample size</th>
<th>ASPD / Psychopathy measures</th>
<th>Other factors measured</th>
<th>Key findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poythress et al. (2010)</td>
<td>Men serving prison sentences or court ordered drug treatment programs in the US</td>
<td>691 men with ASPD</td>
<td>SCID II PAI PCL-R</td>
<td>Anxiety, low mood, violence &amp; aggression, interpersonal dominance, impulsivity, passive avoidance learning, treatment outcome, institutional infractions, recidivism</td>
<td>ASPD was a heterogeneous group, four clusters emerged: primary psychopathy, secondary psychopathy, non-psychopathic ASPD, and fearful psychopathy</td>
</tr>
<tr>
<td>Cox et al. (2013)</td>
<td>Men serving prison sentences or court ordered drug treatment programs in the US</td>
<td>679 men with ASPD</td>
<td>SCID-II PAI PPI</td>
<td>As above</td>
<td>The PPI had discriminant validity in terms of the four clusters identified in the study above. Categories had predictive utility in terms of institutional misconduct</td>
</tr>
<tr>
<td>Coid &amp; Ullrich, 2010</td>
<td>Prisoners in England and Wales</td>
<td>496</td>
<td>SCID I and II PCL-R (cut off 25)</td>
<td>Comorbid psychopathology, demographic data, treatment received, criminal history</td>
<td>31.8% of ASPD met criteria for psychopathy. Psychopathic ASPD more severe than ASPD alone in terms of comorbid PD, violence and antisocial symptoms</td>
</tr>
</tbody>
</table>
Forensic records were used as a measure of institutional infractions, and follow up data was also gathered on offending at one year post release. These empirically based factors arguably have high clinical and predictive utility. If psychopathy measures could be used as a predictor of factors such as psychopathology, violence and criminal recidivism, and treatment outcomes, in a group that have all received the same diagnosis of ASPD, then this provides a challenge to the view that ASPD is a homogenous group.

A statistical clustering technique revealed four subtypes within the group of ASPD men. Two of these mapped onto Cleckley’s primary and secondary psychopathic subtypes (described in the introduction section, outlined in table 2). A third non-psychopathic ASPD group was revealed. A fourth, unexpected group was also revealed, which will be referred to as fearful psychopaths. Planned comparisons were carried out concerning the primary and secondary clusters.

This first subgroup of “primary psychopaths” seemed to map onto the classic primary psychopath first described by Cleckley. Primary psychopaths have been found to have a temperament very low in anxiety (Lykken, 1995). The current study provided support for this, finding significantly lower levels of internalising psychopathology relative to the other clusters, along with significantly lower levels of violence, aggression and impulsivity in comparison to the secondary group. Cleckley’s classic primary psychopath is described as failing to learn from prior experience. As predicted, this cluster made significantly more errors on the passive avoidance learning task compared to the secondary group.

A second cluster seemed to map onto the “secondary psychopath”. This group were more likely to be cited for infractions, displayed higher levels of internalising and externalising psychopathology. The secondary psychopaths were high in anxiety and depression, and engaged more in externalizing behaviours of violence and aggression, in comparison to the cold, callous and unemotional primary cluster. The data did not support predicted differences between primary
and secondary clusters in terms of interpersonal dominance. This prediction was based on literature that suggests primary and secondary psychopaths differ in terms of style, with the latter being more submissive and withdrawn, and the former being more dominant and extrovert (Blackburn, 1987). The failure to find the predicted difference in dominance could represent a type II error, however, given the sample size, and the established psychometric properties of the PAI, which is a well validated tool in correctional settings, it seems unlikely. The failure to find the predicted difference in dominance is also unlikely to be due to response bias, as the PAI includes subscales which identify biased responding. This study also failed to confirm the hypothesis, based on previous meta analyses, that secondary clusters would have higher rates of offending in the year post release. Of course recidivism can only be assessed based on those crimes which are recorded. Police records may well not be an accurate reflection of crimes being committed. The analysis accounted for the potential impact of treatment effectiveness on recidivism, however, “treatment effectiveness” was measured using counsellor ratings. A difficulty with this method of assessing effectiveness is subjectivity and inter-rater reliability. Completion of treatment programmes was also used as an index of effectiveness, which assumes completion is indicative of active engagement with an intervention. It is possible that offenders could appear compliant with treatment, and not engage in disruptive behaviours, which would cause them to be rated as having a good treatment outcome. This may not necessarily mean that therapy has been “effective” in terms of other indices such as symptom reduction, or reduced recidivism. The somewhat subjective and specific measures of effectiveness employed could have impacted on the failure to find predicted differences in treatment outcome between the two groups.

A third cluster was also generated by the analysis, consisting of those that met the criteria for ASPD but did not meet the PCL-R criteria for psychopathy. In answer to the literature review question, this provides support for ASPD as a
heterogeneous group, and ASPD and psychopathy as distinct disorders. Of course, as outlined in the introduction section, this could be representative of the differential sensitivity of the measures. A much lower level of antisocial behavior can attract an ASPD diagnosis, whereas, depending on the cutoff scores employed, higher levels of pathology are required before clinical levels of psychopathy are diagnosed.

A fourth cluster emerged that consisted of those with a highly anxious temperament which the authors hypothesised might map onto a “disadvantaged sociopath” (Mealey, 1995), which described individuals with antisocial features, lower intelligence and socioeconomic disadvantage. This provides further support for “ASPD” as a diagnostic term that describes multiple pathways to antisocial behaviour. Further validation of this subtype is required.

As mentioned, a methodological strength of this study is the unusually large sample of ASPD offenders. It would have been interesting to include in the analysis offenders who do not meet the criteria for ASPD, to see if they emerged as a fifth cluster. If they did not, this would have implications for the utility and specificity of the diagnosis of ASPD.

The study by Cox et al. (2013) aimed to replicate the findings of Poythress et al. (2010) but using a self-report psychopathy measure, the Psychopathic Personality Inventory (PPI). As outlined at the beginning of the results section above, there is evidence supporting the reliability of the use of self report measures of psychopathy in both community (Levenson, Kiehl & Fitzpatrick, 1995) and incarcerated populations (Lynam, Whiteside & Jones, 1999). According to the study by Cox et al. (2013) six of the eight subscales of the PPI had good discriminant validity, although overall the accuracy in terms of correctly identifying cluster membership was 43%. An interesting finding was that the PPI was most accurate in terms of identifying membership to the non-psychopathic ASPD group. If, as these two studies suggest, “ASPD” consists of different clusters which predict factors such as violence impulsivity and infractions in institutions, then the Cox et al. (2013) study
has promising implications in terms of using a quick and non-resource intensive tool in order to further inform risk assessment and formulation. The self-report data replicated the differences between primary and secondary psychopaths in terms of internalising and externalising psychopathology and impulsivity, although unexpectedly, the opposite was found in terms of externalising problems, with secondary psychopaths rating significantly lower than the primary group on these measures. Unlike in the original study, Cox et al. (2013) found that primary psychopaths were more likely to display higher levels of recidivism. The authors do not fully explain this anomaly, although do highlight the potential pitfalls of reification, and that a dimensional approach to these personality constructs is a valid alternative to somewhat “fuzzy” groups or clusters.

The third study reviewed (Coid & Ullrich, 2010), used an alternative methodology to investigate whether ASPD and psychopathy are distinct syndromes. The SCID-II and PCL-R was administered to 496 prisoners. The SCID-II ASPD criteria were broken down into their two constituent parts, childhood conduct disorder (CD), and adult antisocial syndrome (AAS). Psychopathy was divided into four facets; interpersonal, affective, impulsive and antisocial. A logistical regression was then carried out, including these six factors. Demographics, violent offending, prior treatment and comorbid psychopathology were also included in the analysis. The authors hypothesised that if psychopathy and antisocial personality disorder are distinct then there should be differences in the correlations between the different facets of psychopathy and the diagnostic criteria for ASPD. They also hypothesized that those participants meeting the criteria for both ASPD and psychopathy should differ in terms of the antisocial criteria they met from those who are ASPD but not psychopathic.

The authors concluded that rather than ASPD and psychopathy representing distinct groups, psychopathy may represent an extreme end of an antisocial trait dimension. They concluded that there were no significant differences between
ASPD with and without psychopathy in terms of their relationship to treatment. Close inspection of the results section shows that there were group differences which approached significance. There was a lack of power due to increasing the cut off point for psychopathy from 25 to 30, despite the empirically recommended cut-off point of 25 for UK samples (Cooke & Michie, 1999). After this change in cut off score, some previously significant group differences became insignificant, such as the finding that those with psychopathy and ASPD displayed higher levels of violent offending. A potential danger of increasing the cutoff point for the PCL-R is that this can cause people scoring higher on the psychopathy scale to fail to meet criteria and therefore be included into the ASPD only group.

Further evidence for the existence of subtypes within ASPD samples is provided by Kosson et al. (2006), reviewed in the following section. This study measured amount and type of offending, or “criminal versatility”. After the removal of those rated as psychopaths, ASPD was a significant predictor of number of violent and non-violent offenses. This finding demonstrates that ASPD as a diagnosis has predictive utility, which contradicts the argument that any predictive utility in ASPD groups is due to the high number of psychopaths which fall into the diagnostic category of ASPD.

Overall the papers reviewed in this section suggest that “ASPD” may represent a heterogeneous group. At least two of these groups map onto theoretical subtypes first identified in the 1940s, the primary and secondary psychopaths. The studies suggest that these categories have predictive utility in terms of violence and aggression, mood disorders, impulsivity and engagement in treatment. They also provide promise for the development of self report tools that can be used with some accuracy to inform researchers and clinicians about group membership. These studies also raise the question of the utility of a categorical view of antisocial behaviour, and highlight the issue of the potential pitfalls of reification. A potential
move towards viewing personality pathology in a dimensional manner was suggested by Coid & Ullrich (2010).

4.2 Emotional Processing

Deficits in emotional processing in psychopaths, children with psychopathic traits and antisocial individuals are fairly well established in the literature and are implicated in influential models of psychopathy (Blair & Coles, 2000; Marsh & Blair, 2008). Studies reviewed in this section compare emotional processing in individuals with ASPD to those classified as psychopathic (see table 4).

4.2.1 Affective facilitation

Research in non-clinical samples has repeatedly found that when people are presented with words and non-words, and are then asked to identify what constitutes a real word, affective words are better recognized than neutral words. This phenomenon has been termed “affective facilitation”. In the study by Kosson et al. (2006) it was found that ASPD only offenders did not differ from non ASPD offenders in terms of affective facilitation, whereas the ASPD plus psychopathy group displayed significantly lower levels of affective facilitation than the ASPD only group. The authors report initially a sample size of 472 inmates. After assessing for ASPD and psychopathy, and removing outliers, each of the three groups only contained between 25 and 36 offenders with complete emotional processing data. If previous estimates of the prevalence of ASPD in incarcerated samples are accepted (e.g. 35%, Black, Gunter, Loveless & Sieleni, 2010), then it would be expected that a much larger group of ASPD participants would be found in a sample of this size. This may be indicative of a lack of sensitivity in the ASPD measure used. The authors chose to create interview questions based on the DSM-IV criteria for ASPD rather than using a standardised measure such as the SCID-II. Whilst inter-rater reliability for the interviews was good (k=.92), the validity of the measure has not been empirically validated.
Table 4  
*Studies Comparing Emotional Processing in Psychopathic and Non-psychopathic ASPD*

<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>Sample size</th>
<th>ASPD / Psychopathy measures</th>
<th>Other factors measured</th>
<th>Key findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dolan &amp; Fullam (2004)</td>
<td>UK prison and high security hospital who met DSM IV criteria for ASPD - Staff used as control group</td>
<td>109</td>
<td>DSM-IV criteria for ASPD, PCL-SV</td>
<td>Empathy, First and second order ToM tasks, Complex “faux pas” task, Facial emotional expression task</td>
<td>ToM for ASPD and psychopathy relatively intact. ASPD slightly more of a mentalizing deficit than psychopathic ASPDs</td>
</tr>
<tr>
<td>Verona, Sprague, Verona, Sprague &amp; Sadeh (2012)</td>
<td>American offenders recruited via criminal justice system</td>
<td>45</td>
<td>DSM-IV criteria for ASPD, PCL-SV</td>
<td>Negative emotional processing and inhibitory control</td>
<td>Blunted negative emotion processing in psychopaths regardless of task demands. Enhanced processing of negative emotion in ASPD group despite competing demands of task, suggesting poor inhibitory control</td>
</tr>
</tbody>
</table>
Results demonstrated greater criminal activity and versatility in the psychopathic group, suggesting that psychopathy may be a predictor for greater number and variety of offences. T-tests revealed significant group differences, with psychopaths displaying less affective facilitation than ASPD offenders. ASPD offenders performed similarly to controls. It is fair for the authors to conclude that there may be differences between ASPD and psychopathy is terms of emotional processing.

Attempts to link performance in the affective facilitation paradigm to real world criminal behaviour were less convincing. There were a few significant interactions, for example, reduced affective facilitation was associated with more nonviolent offences in the psychopathic group, but not the ASPD group. The authors do not offer an explanation for this finding, so it remains unclear how performance in this paradigm would relate to non-violent offences. The VIM and IES models (Blair, 1995, Blair et al., 2005) provide accounts of emotion processing in violent behaviours. The role of emotional processing in non-violent offending is less clear.

4.2.2 Theory of mind and mentalizing

Broadly speaking, empathy is an emotional response to the emotional states of others, which some argue requires the ability to first form a mental representation of the emotional state in another person. This ability has been referred to as theory of mind, or the ability to “mentalize” the internal state of others. Blair (1995) developed the idea of the “violence inhibition mechanism”, by which we learn to inhibit responses which cause distress to others, and that this mechanism is key in the development of the moral emotions such as empathy. He proposed a disruption to the development of this neural network in psychopaths, thus a failure to develop empathy. The moral/conventional distinction is a paradigm which requires participants to make judgements about transgressions which are moral (e.g. someone injuring another person) or conventional (violation of social norms, such as
a male dressing as a female). Research has found a distinction between the judgments made on moral and conventional transgressions, with both adults and children usually judging the former to be more serious (Smetana & Braeges, 1990). Psychopaths (defined by Hare’s criteria) have failed to make this distinction (Blair, 1995), but when compared to non-psychopathic offenders do not display deficits on simple theory of mind tasks (Blair et al., 1996). Researchers have also failed to find a psychopathic deficit on a more complex theory of mind task (Richell et al., 2003).

The second study (Dolan & Fullam, 2004) reviewed in this section selected a sample of ASPD males from a secure hospital and a prison and divided them into with and without psychopathy groups, and compared them with a healthy control group of staff. They administered an empathy measure, the Interpersonal Reactivity Index (IRI; Davis, 1996), theory of mind tasks (Stone, Baron-Cohen & Knight, 1998) of three levels of complexity, and a facial emotional expression task using Baron-Cohen’s photographs of faces displaying seven basic emotions (Baron-Cohen, Wheelwright & Joliffe, 1997). The control, ASPD and ASPD with psychopathy groups did not differ on empathy, and there were no differences in terms of theory of mind ability on first and second order tasks. The interesting finding came from a complex “faux pas” task. There were no group differences in terms of the ability to identify that a faux pas had been committed and identifying who had committed it. However, ASPD and ASPD with psychopathy groups were impaired in comparison to the control group in terms of assessing the mental state of the listener and speaker. The ASPD only group was significantly worse than the staff control group and the ASPD with psychopathy group on recognizing basic emotions in faces. In terms of recognizing more complex emotional states from photos of facial expressions, the ASPD group was significantly poorer than the control group. Other group differences did not reach significance.

In interpreting these results it is necessary to note the difference between merely recognizing the emotion in the face of another person, and theory of mind, or
mentalizing, which refer to a felt sense of the emotional state of another person, and to recognise that others and our own emotional states are separate. The increasing complexity of the theory of mind paradigm used in the Dolan & Fullam (2004) study help to differentiate between recognition of emotion, and more complex attributions of the internal states of another. In this study this paradigm is taken to be a measure of “mentalization”. The construct of mentalization is a development of the concept of theory of mind, and the terms are used interchangeably in this study.

This study replicates findings that psychopaths do not have a specific mentalizing deficit, in fact on some aspects of the tasks they outperformed the staff control group. It provided further support for ASPD and psychopathy as distinct mechanisms, with the ASPD group displaying deficits in terms of the recognition of basic emotions in the faces of others. It could be the case that this group maps onto the secondary psychopathic group described in this review, who have adverse early life experiences. Studies show that violent adolescents from an adverse background respond to distress in others with aggression, which would explain the finding from the current study that the ASPD group had the most difficulty distinguishing between distress and sadness.

The use of correctional staff as a control group in the Dolan & Fullam (2004) study may present a methodological issue. The authors noted that interestingly, the staff group performed more poorly on an empathy task than would be expected in a general healthy population. There may be some impact of working in a forensic environment on mentalizing ability. It is also possible that correctional staff may more representative of certain populations (e.g. ex-military) rather than being representative of the wider population.

A potential limitation of this study is the possibility of a ceiling effect of a relatively simple task, given that all participants were of at least average intellect. Intellect could compensate for a difficult in mentalizing, which could mean that the failure to find mentalizing deficits in the psychopathy group represents a type II
error. Moreover, the theory of mentalizing in personality disorder posits that people may switch in and out of mentalizing modes depending on level of emotional arousal (Fonagy & Bateman, 2008). Given the low levels of emotional arousal in a laboratory setting, it could be the case that tasks can be completed adequately, which does not map onto real life experiences of mentalizing in interpersonal situations.

4.2.3 Negative emotional processing and inhibitory control

The literature has suggested that ASPD and psychopathy may differ in terms of behavioural and physiological response to neutral or emotive stimuli, with psychopathic groups showing a reduced response to emotive stimuli (Levenston, Patrick, Bradley & Lang, 2000). The impulsive aggressive behaviour exhibited by antisocial individuals may be associated with negative emotional reactivity and deficits in cognitive control, as demonstrated by performance in studies on inhibitory control mechanisms (Morgan & Lilienfield, 2000). The third study reviewed in this section (Verona, Sprague & Sadeh, 2012) was the first to investigate emotion processing and inhibitory controls in ASPD only versus ASPD with psychopathy. Their hypotheses were based on the theory that in psychopathy, the cognitive demands of a task would not affect the emotional processing, whereas those with cluster B personality disorders, such as ASPD, are more likely to have their inhibitory control affected by emotive situations, given the high level of emotional reactivity that is characteristic of this cluster. They hypothesized that this difficulty in emotional processing under conditions of inhibitory control would map onto everyday difficulties in inhibiting behaviour, as measured by incidents of aggressive behaviour. They used a go/no go task in which participants were required to press a button in response to words in normal font, and to inhibit this response when the word was presented in italicised font. Words were either neutral, generally negative, or offender related negative words (e.g. “jail” and “scum”). Results suggested that control offenders, those that did not qualify for a diagnosis of ASPD or psychopathy,
were able to suppress emotional processing in order to prioritise inhibitory control in no-go trials. The psychopathic group showed lower levels of emotional processing regardless of whether there were inhibitory demands or not. ASPD offenders had difficulty in processing negative emotion when under the demands of inhibiting a response. The authors interpreted these results as evidence of differences in emotional processing between ASPD only and ASPD with psychopathy groups. Psychopaths seemed to have lower levels of emotional processing, regardless of the cognitive demands placed or not placed on them, whereas ASPD offenders seemed to prioritise processing of negative emotional information even when it compromises task performance. The control group was able to suppress their emotional reactions in order to deal with the demands of the task, something which the ASPD group apparently struggled with. The tendency of the ASPD group to prioritize negative emotional processing and failure to inhibit this to deal with the demands of a situation may explain the increased impulsive aggression and self-harm seen in this and other cluster B personality disorders. It is important to note that whilst psychopaths outperformed ASPDs in terms of the current task, there was no group difference in terms of level of aggressive behaviour. It may be that the aetiology of these behaviours differs between the groups. Characteristics of psychopathy such as callous unemotional traits and low emotionality lead to poorer functional adaptation in many real world contexts. The link between these traits and increased risk of violent recidivism is documented in the literature (Walsh & Kosson, 2008). As noted by the authors, this was the first study of its kind and replication is required. The sample size was small relative to the other papers reviewed, and therefore type II errors are a possibility.

The studies in this section used a range of paradigms to investigate whether those assigned to ASPD or psychopathy groups varied in terms of emotional processing. Whilst strengths of these studies are the stringent methodologies and use of empirically validated paradigms, the validity of these paradigms represents a
problem for this area of research. The extent to which conclusions can be drawn about real world offending behaviour from, for example, the presentation of “emotional” word strings is limited.

4.3 Executive functioning

It has been hypothesized that those with ASPD are more emotionally reactive whereas those rating highly on both ASPD and psychopathy measures display emotional hypo-reactivity. The papers reviewed so far provide some support for this theory. Neuropsychology offers an explanation for this observation. It is theorized that deficits in executive functioning may explain the persistent and pervasive antisocial behaviour seen in ASPD individuals from teenage years throughout adult life, even despite repeated punishment (Blair, Mitchell & Blair, 2005; Raine et al., 2005). The studies reviewed in this section (summarized in table 5) investigated executive functioning in order to ascertain whether these deficits differed between those with ASPD only and those with ASPD and psychopathy.

Blair’s Integrated Emotions System (IES) model explains that ASPD is characterized by more affective, reactive aggression which is related to a broad range of executive functioning deficits, whereas psychopathy is associated with more instrumental, premeditated aggression, which may be related to more specific deficits in the amygdala and orbitofrontal cortex (Blair, 2006). Dolan (2012) administered a variety of executive functioning tasks to ASPD individuals and also measured psychopathy. The methodology differs slightly from others in the review, in that psychopathy was measured dimensionally, by dividing ASPD participants into low, medium and high psychopathy according to PCL-R scores, rather than nominal assignment to ASPD with or without psychopathy groups.
<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>Sample size</th>
<th>ASPD / Psychopathy measures</th>
<th>Other factors measured</th>
<th>Key findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dolan (2012)</td>
<td>Offenders from UK prison and medium and high secure hospitals, staff control group</td>
<td>96</td>
<td>SCID II PCL:SV</td>
<td>Spatial planning; Attentional set shifting; Response inhibition</td>
<td>ASPD had impairments in planning compared to healthy controls. Those with higher psychopathy scores performed similarly to control. Set shifting difficulties in ASPD as a whole, no psychopathy specific deficit.</td>
</tr>
<tr>
<td>De Brito, Viding, Kumari &amp; Blackwood (2013)</td>
<td>Violent ASPD offenders from UK community and community controls</td>
<td>66</td>
<td>SCID I and II PCL-R</td>
<td>-Digit span backwards; -Spatial alternation task; -Response reversal task; -Cambridge Gamble Task; -Passive avoidance learning</td>
<td>No significant differences between ASPD only and ASPD with psychopathy. Both showed deficit on verbal working memory and adaptive decision making in comparison to non offenders.</td>
</tr>
<tr>
<td>Zeier, Baskin-Sommers, Newman &amp; Racer (2012)</td>
<td>Caucasian males from maximum security US correctional institution</td>
<td>126</td>
<td>PCL-R</td>
<td>Cognitive control (response competition task) Welsh Anxiety Scale</td>
<td>ASPD performed less accurately on cognitive control tasks. Similar deficits also found in psychopathy, contrary to predictions.</td>
</tr>
</tbody>
</table>
Dolan found that those “ASPD” offenders which rated at the higher end of the psychopathy scale actually had significantly longer mean reaction times in an inhibition task than the healthy controls. The authors conclude that this provides support for Hare’s ‘classic psychopaths’ who engage in more instrumental, planned acts as opposed to reactive impulsive aggressive acts. However none of the planning tasks revealed any group differences, apart from overall a slight deficit in planning in the offending group as a whole compared to healthy controls. Set shifting ability also did not appear to co vary with psychopathy.

The finding that there were significant group differences when dividing psychopathy into low, medium and high suggests that this may be a useful methodology, rather than using a cutoff score to assign groups. This endorses the view that psychopathy can be more usefully conceptualized as a dimensional trait as opposed to the categorical approach adopted by many of the papers described here.

All in all the study seemed to employ a rigorous methodology, and controlled for extraneous variables such as axis I mental health diagnoses, intellect, trauma, and current substance use. A detailed analysis breaking down the tasks into their constituent phases and looking at all aspects of performance was carried out. It seems fair that the null hypothesis is accepted, i.e., there is no observed difference between those rating on psychopathy measures compared to controls in terms of planning and set shifting. This negative finding seems to fit theoretically, with the classic view of primary psychopathy, as being less reactive and more calculating. Whilst psychopathy may represent a continuum, or consist of various clusters, this study would suggest that executive functioning does not play a role in this variability.

The second study (De Brito et al., 2013) broke down executive functioning into cool and hot executive functioning; the former referring to primarily cognitive processes such as response inhibition, planning, working memory and attentional set shifting. Hot executive functions include those that are concerned with emotion,
reward and motivation such as the processes involved in affective decision making paradigms. Both groups of offenders in the study showed impairments in comparison to healthy controls in terms of decision making and verbal working memory. They failed to learn from punishment as indicated by performance in a passive avoidance learning task, which is an interesting finding as it goes some way to explaining the persistent offending by ASPD offenders despite the negative consequences. Repeated offending despite negative punishing consequences is a common feature of ASPD. This research finding suggests that such individuals may have a deficit in terms of their ability to learn from punishment, at a neurocognitive level. Whilst expected deficits on a range of cool and hot executive functioning tasks were found when comparing offenders as a whole to non-offenders, no differences were found between the two offending groups. There is the possibility of type II error given the small sample size (n=66).

Another aspect of executive functioning is cognitive control, or the ability to persevere in goal oriented action despite the presence of competing cognitive and behavioural demands. There is generally a relationship between cognitive control and ASPD, but studies have failed to find this effect in psychopaths, and in some cases they have outperformed healthy controls on tasks of cognitive control (Hiatt et al., 2004), specifically primary psychopaths. The Zeier et al. (2012) study used a response competition paradigm in order to explore cognitive control across different subtypes of antisocial offenders. An interesting finding was that dimensionally speaking, ASPD symptoms are negatively associated with cognitive control. Despite using the higher cut-off score of 30 to allocate to the psychopathic group, this group was relatively large (n=54) in comparison to other studies employing a similar design. Despite this larger sample size, they failed to replicate the finding that psychopaths have equal or superior cognitive control compared to other offenders. This could represent a true negative finding, or it could be attributable to a methodological flaw. For example, unlike other similar studies reported here, the
authors subdivided the psychopathic offenders by anxiety score. It may have been preferable to employ a simple four group design (ASPD with psychopathy, ASPD without psychopathy, non ASPD offenders, and healthy controls) and use anxiety as a covariate. This study neglected to include a control group, and therefore the previous finding that psychopaths outperform controls in terms of cognitive control could not be replicated. Another methodological flaw is that a standardized ASPD assessment (such as the SCID-II) was not used. Instead data from the same interview and file review used during PCL-R administration was used. Since ASPD was deduced from PCL-R data, the overlap in measures could contribute towards the overlap in cognitive control deficits between ASPD and psychopathic groups, whereas previous studies have found that the latter perform better in terms of cognitive control. It would be informative to see how each group performed in comparison to controls.

Executive functioning is a broad umbrella term encompassing a number of constructs, including planning, organization, selective attention, inhibitory controls and problem solving. As such, there is no pure, unambiguous test for executive dysfunction (Morgan & Lilienfield, 2000), which presents a challenge in this area of research. The extent to which the findings from the studies presented here can be applied to real world offending behaviour is limited. The applicability of findings from simple computerized tasks has limited utility in terms of explaining real world behaviour.

Whilst, perhaps due to methodological flaws, some group differences were not supported, these studies provide some interesting findings in relation to the question posed by this literature review. For example, the findings of the study by Zeier et al. (2012) support the view of ASPD as a continuous variable rather than a distinct category, with symptom severity correlating significantly with cognitive control deficit. The failure to replicate group differences could be attributable in part to the fact that a gold standard diagnostic measure such as the SCID-II was not
used. On the other hand this result could represent a true negative finding, supporting Coid & Ullrich’s (2010) view of ASPD and psychopathy as being on a continuum.

4.4 Neurological Factors

The literature searched produced three studies which compared ASPD and psychopathy in terms of neurological measures such as startle response, and structural brain differences (see table 6). These studies explored the neurological processes and structures which may add to the understanding of the emotional processing and executive functioning differences discussed in the previous sections.

The startle reflex is an automatic cortical event, generally accepted to be linked to the amygdala (Angrilli et al., 1996; Davis, 1992). It is triggered in response to a perceived threat, which, in laboratory paradigms, is usually generated by presenting a sudden loud noise. The function of the startle response is to interrupt whatever cognitive processing may be occurring in order to orient attention towards a potential threat. In healthy non-offending populations this startle response, as evidenced by blinking, is potentiated under conditions in which aversive or threatening stimuli is being viewed. In the Vaidyanathan et al. (2011) study, it was found that both offending groups did not exhibit this effect, i.e., their startle response was not amplified by viewing of aversive stimuli. Further analysis revealed that this effect was mainly driven by factor one psychopathy.

In the Drislane et al. (2013) study EEG was used to measure P3, a cortical response which initiates the startle reflex described above. The P3 has been found to be generated in the presence of an audio startle probe in laboratory paradigms. Again, this was measured in ASPD and psychopathic offenders when viewing neutral or affective stimuli.
<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>Sample size</th>
<th>ASPD / Psychopathy measures</th>
<th>Other factors measured</th>
<th>Key findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vaidyanathan Hall, Patrick &amp; Bernat, 2011</td>
<td>Incarcerated US adult males</td>
<td>108</td>
<td>PCL-R Structured interview questions based on DSM-IV ASPD criteria</td>
<td>Startle response as measured by blinking in response to a noise probe whilst viewing neutral and threatening stimuli</td>
<td>Deficits in startle reflex in aversive picture viewing associated more with psychopathic traits than ASPD</td>
</tr>
<tr>
<td>Drislane, Vaidyanathan &amp; Patrick, 2013</td>
<td>Incarcerated US adult males</td>
<td>143</td>
<td>PCL-R Interview questions based on SCID II</td>
<td>P3 (cortical event potentiated in response to sudden unexpected noise, measured by EEG)</td>
<td>Factor 1 psychopathy was related to reduced startle response. ASPD diagnosis did not affect startle response</td>
</tr>
<tr>
<td>Gregory, Ffytche, Simmons, Kumari, Howard, Hodgins &amp; Blackwood, 2012</td>
<td>UK probation service</td>
<td>66</td>
<td>PCL-R SCID I and II</td>
<td>Gray matter volumes as measured by MRI</td>
<td>ASPD+P had reduced gray matter volume in some brain regions compared to ASPD without psychopathy. No difference in key temporal areas such as amygdala</td>
</tr>
</tbody>
</table>
This study also found smaller P3 amplitudes in psychopaths in response to noise probes no matter the content of what they were viewing. Again further analyses suggested that this effect was accounted for mostly by factor one features of psychopathy. Both of these studies provide support for unique features of psychopathy, in that they provide empirical evidence for those rating highly on measures of psychopathy as exhibiting less of a response to perceived threat. This fits with the “low-anxious” psychopath described in this review. The EEG paradigms provide a direct measure of a neurological reaction, which adds further support to the validity of Vaidyanathan’s (2011) study.

In the Gregory et al. (2012) study MRI was used to measure grey matter volume in brain regions implicated in antisocial behaviour in ASPD men with and without psychopathy, recruited via the probation service. Whilst amygdala dysfunction has been widely implicated in models of psychopathy, only a few studies have found evidence of reduced amygdala volume in this group. This study failed to find a significant difference in amygdala volume between the two offending groups. However they did find reduced grey matter volume in the anterior rostral medial prefrontal cortex (arMPFC) and temporal poles in ASPD offenders with psychopathy compared to ASPD only offenders. The arMPFC is thought to be involved in the assessment of storage of social information and therefore may play a key role in the emotional understanding of other’s acts, which relates to the concept of mentalizing described by Fonagy and Bateman (2008). The authors explain that the failure to find predicted structural differences in the amygdala volumes of those with and without psychopathy could be related to the limitations of the imaging techniques, which rely on structural measures and do not give information on, for example, cortical thickness. This study is deemed to be a high quality study with a particularly stringent methodology. The authors controlled for the effects of comorbid axis-I disorders, and substance use, as these can impact the volume of brain structures. They also continually checked for substance use throughout the study, which is
important in a community sample where substances can be more freely accessed. Trained clinicians were used to make diagnoses, which were agreed upon by a secondary rater. File information provided reliable corroborative information, however a standardised tool such as the SCID-II was not used to assess for ASPD. A strength of the design was that groups were matched in terms of comorbid personality disorders and substance use disorders. They employed modern imaging techniques and statistical mapping software, to avoid the subjectivity and bias of manual tracing methods employed in previous similar studies. This is the first study to use these imagine techniques to investigate structural differences in ASPD offenders with and without psychopathy. The authors acknowledge that replication is necessary.

Studies reviewed in this section employed direct objective measures, such as EEG and structural MRI in order to test for group differences in brain function and structure in ASPD individuals with and without psychopathy. These were high quality studies with stringent methodologies, although they all neglected to use standardized, validated measures of ASPD such as the SCID-II. Overall the studies provide fairly convincing evidence that there may be features unique to psychopathy, such as a reduced startle response, which seems to map on to factor one type psychopathy, described in the first section of this review. The structural imaging study found modest differences (Gregory, 2012) although the stringent methodology suggests that this is a true negative finding. The authors also highlight that other measures, such as cortical thickness, may yield different results.

5. Discussion

These studies were carefully selected in order to address a specific question; whether ASPD and psychopathy are distinct disorders. In order to answer this question, studies from four areas were selected; studies employing statistical clustering techniques, studies of emotional processing, executive functioning, and
other neurological factors such as brain area volume and startle response. Due to the specific nature of the question, only a small number of papers were selected. After careful review, including a dual rated quality appraisal (Kmet, Lee & Cook, 2004), these papers are deemed to be high quality in terms of methodology, which gives credibility to the conclusions drawn.

A large number of people in the US and the UK offending populations meet the criteria for ASPD. Historically, the terms “psychopath” “sociopath” and “ASPD” have been used somewhat interchangeably. The current literature review explored four areas of research in ASPD samples in order to address the question of whether psychopathy and ASPD are distinct disorders.

Studies using sophisticated statistical clustering techniques found that psychopathy scores created meaningful clusters in samples of ASPD offenders. They differed in terms of factors such as impulsivity, aggression, anxiety, depression, adjustment to prison life, engagement in and outcome of treatment, and recidivism. These clusters seemed to map onto those previously described in the psychopathy literature, with at least four separate pathways to antisocial behaviour. One group (ASPD or secondary psychopaths) seem to point to aggression as a result of poor emotional regulation and the tendency to prioritize negative emotional content over adaptive response to situations. In another pathway (classic, primary "Hare" psychopaths) callous unemotional traits and a hypo-responsivity to emotion may be causal in antisocial behaviour and or aggressive behaviour.

Studies of emotional processing in groups of ASPD offenders again highlighted variation within the group. Those with features of psychopathy were distinct from other ASPD offenders in terms of affective facilitation and generally displayed blunted negative emotion processing. These studies also suggested that ASPD only groups may have poor inhibitory control. ASPD and psychopathy may also represent differences in terms of theory of mind and mentalizing. Some negative findings in this area may be due to a ceiling effect of relatively simple tasks,
and therefore merits further research and the development of paradigms which encompass the concept of “mentalisation”.

In terms of executive functioning, the ASPD group as a whole seemed to have deficits in planning, set shifting, verbal working memory, response inhibition and cognitive control, whereas psychopathic groups were found to perform similarly to a non-offending control group of prison staff. This negative finding could be interpreted as further evidence for multiple pathways to antisocial pathology, with one group being more “reactive” as a result of a failure to inhibit emotional responses and aggression, and another group, being more “cold and callous”. These groups theoretically map onto the “primary” and “secondary” psychopaths described in the introduction section.

The final section reviewed studies of neurological factors, such as startle response as measured by EEG, and volume of brain structure as measured by MRI and statistical mapping software. Again unique differences were identified in the psychopathic group, in terms of a reduced startle response, and some reduced gray matter volume. Expected structural differences in the amygdala were not observed, potentially due to the limitations of the imaging techniques. Further research using more advanced and specific measures of brain structures such as cortical thickness is required. Studies have suggested that cortical thickness may be a more reliable and valid research tool in comparison to gray matter volume (Winkler et al., 2010).

Overall the studies suggest that psychopathy and ASPD have distinctive features, and that the large number of people that meet criteria for ASPD represent a homogenous group. Different clusters emerged, suggesting numerous aetiologies for antisocial behaviour. This has potential implications in terms of clinical practice, research and policy.

In order to address the question of this review a rigorous search was conducted, yielding only a small number of suitable studies. In order to explore whether ASPD and psychopathy are distinct, it is necessary for researchers to
administer measures of both factors. Researchers have only begun to distinguish between the two relatively recently, and many papers could not be included in the review due to their failure to account for the potential influence of two separate constructs. It is necessary for future research to take into account the possibility that “ASPD” is a term that may encompass a heterogeneous group, and to make use of gold standard measures of both psychopathy and ASPD. This can be achieved with minimal resources with the use of a self-report psychopathy measure such as the PPI (Lilienfield & Andrews, 1996). Researchers need to simultaneously bear in mind the potential pitfalls of reification. It may be more useful in both research and clinical practice to conceptualize psychopathy as a continuum rather than as a diagnostic category (Coid & Ullrich, 2010). Using psychopathy score as a continuous variable would counteract the methodological issue presented by different cutoff points.

The papers reviewed suggest that the ASPD diagnosis encompasses a large group of people who vary on clinically relevant factors, such as internalising and externalizing psychopathology and risk of violent recidivism. Differential diagnosis could be used to inform risk assessments, for example, predicting the likelihood of aggressive and violent behaviour in prison. One study reviewed suggests that a simple, non-resource intensive self-report tool can be used to differentiate these subgroups. Psychopathy remains a controversial issue, and is commonly misunderstood to be associated with “untreatability” (Skeem, Monahan & Mulvey, 2002).

The findings of this review suggest that ASPD and psychopathy represent distinct constructs, or perhaps, different ends of a continuum. Either way, it is arguable that there is variability within the group of “antisocial” offenders as a whole. These findings provide support for a move away from considering a belonging to a diagnostic category as a risk factor, and a move towards a formulation based approach to risk assessment and treatment planning.

The conclusions drawn from this review are limited to North American and
UK male offending populations. The majority of the studies took their samples from incarcerated male populations, so care should be made when generalizing to community samples, due to potential effects of incarceration. Some, but not all studies controlled for ethnicity, or included only Caucasian males, in order to control for potential cross cultural variability in the construct of psychopathy. A meta-analysis has suggested that this may not be necessary, finding no significant difference between black and white males in terms of core psychopathic traits in prison, community and psychiatric samples (Skeem, Edens, Camp & Colwell, 2004). Studies have found that across the UK and North America, psychopathy is fairly consistent, although PCL-R measures vary, with UK samples obtaining a lower score for the same level of psychopathy (Cooke & Michie, 1999). This highlights the importance of researchers adjusting the cut off scores of measures used. In terms of gender, this review is solely focusing on males, given the differential effects of gender in terms of biological, social and psychological factors. The constructs discussed here are beginning to be researched in female subjects, (Anton et al. 2012; Sturek, Loper & Warren, 2008; Warren & South, 2006).
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*Personality Disorders 19*(2) 103-109.


Part two: Empirical Paper

Mentalizing and Psychopathy in Antisocial and Borderline Personality Disorder
Abstract

Aims
To investigate psychopathy and mentalization in a large sample of people with and without a diagnosis of Borderline (BPD) or Antisocial personality disorder (ASPD).

Method
60 participants from personality disorder (PD) services, 21 from probation and 81 non-clinical controls completed a battery of tests of mentalizing, psychopathy, and personality pathology, as part of an existing large ongoing research project.

Results
Both PD groups had lower mentalizing scores than controls. BPD pathology was predictive of mentalizing ability for two of three mentalizing scales. ASPD pathology was a modest predictor of one mentalizing scale. Both PD groups exhibited higher levels of psychopathy in comparison to controls but did not differ significantly from each other. The secondary factor of psychopathy was the strongest predictor of mentalizing across the sample.

Conclusion
The mentalizing deficit hypothesis of BPD was supported, a similar deficit may also be present in ASPD but replication with a larger ASPD sample is required. Overall the data provide some support for a move towards a dimensional model of personality disorders. The secondary factor of psychopathy was predictive of personality pathology in ASPD and BPD. Findings support ASPD as a heterogeneous group, and supports ASPD and psychopathy as distinct constructs.
1. Introduction

Borderline Personality Disorder (BPD) is characterized by affective, behavioural, cognitive and interpersonal difficulties (American Psychiatric Association, 2013). A pervasive pattern of affective instability and difficulty in regulating emotions is characteristic in BPD (Lieb et al., 2004), often leading to impulsive, suicidal and parasuicidal behaviours, such as self-mutilation. BPD is increasingly gaining recognition as a major public health problem with prevalence estimates at around 5.9% in the community (Grant, et al., 2008) and 24% in primary healthcare attenders in the UK (Moran, Jenkins, Tylee, Blizard & Mann, 2000). BPD is over-represented in incarcerated females in England and Wales at about 20% (Nee & Farman, 2005).

In the DSM-5 (American Psychiatric Association, 2013). Antisocial Personality Disorder (ASPD) criteria includes impairments in self functioning (e.g. ego centrism, failure to conform with lawful or culturally normative behavior), lack of empathy or intimacy, antagonism (manipulativeness, deceitfulness, callousness and hostility) and disinhibition (impulsivity, irresponsibility and risk taking). ASPD is relatively common in criminal populations with an estimated prevalence of 47% of males and 21% of females meeting criteria (Fazel & Danesh, 2002).

Psychopathy is a construct characterised by reduced guilt, empathy, and attachment to others, and a prevalence of antisocial behaviours (Blair, 2007). Psychopaths have been described as “human predators who coldly, callously, and ruthlessly use charm, deceit, manipulation, threats, intimidation, and violence to dominate and control others and to satisfy their own selfish needs and desires” (Hare, 2000, cited in Shipley & Arrigo, 2001, p.409). In terms of UK prevalence, it has been estimated that 7.7% of male prisoners, and 27% of homicide offenders are likely to meet criteria for psychopathy (Coid et al., 2009). Aside from those identified in forensic populations, it is likely that there is a prevalence of ‘successful psychopaths’ in society. These people may display psychopathic trait patterns but have not come into contact with the Criminal Justice System (Lynam, Whiteside &
Studies have identified many psychopaths working successfully in organisations, perhaps using skills of manipulation and influence to great success (Board & Fritzon, 2005). It has been estimated that approximately 0.6% of the UK general population may meet criteria for psychopathy (Coid et al., 2009).

Although prevalence is relatively low, it is arguable that psychopaths present a great challenge in terms of the criminal justice system, and indeed for the wider society. Psychopathy may represent a unique behavioural profile in comparison to other individuals who engage in antisocial behaviour. Psychopaths in general present greater risk of violent behaviour (Cooke, Michie, Hart & Clark, 2005). In terms of offending populations, high scores on measures of psychopathy are indicative of future risk of violent recidivism (Hare, 1991 cited in Blair, 2003). Psychopathy has also been found to be predictive of treatment failure, violent and non-violent offending, and substance misuse (Kosson et al., 2006). Significant resources in the UK have been allocated to attempts to find suitable treatment pathways for individuals scoring highly on psychopathy, for example, the development of the Dangerous and Severe Personality Disorder programme (DSPD, Department of Health, 1999).

Historically both treatment and research have viewed personality disorder and psychopathy in terms of a categorical diagnosis, indicated by cut off scores on gold standard measures such as the SCID-II (Structured Clinical Interview for Axis-II Personality Disorders; First, Gibbon, Spitzer, Williams & Benjamin, 1997) and the PCL-R (Psychopathy Checklist-Revised; Hare, 2003). There is increasing debate in the literature as to whether psychopathy and personality disorders may be more usefully viewed as dimensional constructs (Wright, 2009). The current categorical classification systems (DSM-5 and ICD-10) have been criticised for poor validity and reliability, high comorbidity, poor convergent and discriminant validity, and arbitrary cut offs (Verheaul, 2006; Sarker & Duggan, 2010). It is argued that the diagnostic criteria employed to date have been based on clinical consensus rather than
empirical data (Livesley, 2007). Prior to the most recent revision of the Diagnostic and Statistical Manual of Mental Disorders, (DSM-5, American Psychiatric Association, 2013), the possibility of a move towards a dimensional classification of personality disorders was explored (Widiger & Simonsen, 2005), and it seemed likely that a major shift in the way that Axis II disorders are conceptualised would be included. However the decision was made to retain the categorical structure from DSM-IV. Nonetheless a new hybrid categorical-dimensional section was added, in order to encourage further research in to dimensional approaches to the identification of personality disorder (APA, 2013).

Common processes have been identified in BPD and ASPD, including impulsivity, affective instability, and cognitive symptoms (Paris, 1997). Psychopathy may also represent a trait dimension which is related to both ASPD and BPD pathology. Elevated psychopathic traits have been found in people with a diagnosis of BPD and those with a diagnosis of ASPD (Blackburn & Coid, 1998). High psychopathy scores were found to predict borderline personality pathology in a large sample of community and incarcerated females (Sprague, Javdani, Sadeh, Newman & Verona, 2012). The authors posited that BPD may be a “female phenotypic expression of psychopathy”. Historically the term “psychopathy” has been used interchangeably with “ASPD”. There is ongoing debate as to whether or not psychopathy and ASPD represent distinct disorders (e.g. Ulrich & Coid, 2010).

Another construct of interest in the study of BPD and ASPD pathology is that of “mentalizing”. Mentalization, like psychopathy, may represent a trait dimension associated with personality disorder pathology. The ability to “mentalize” refers to the process of perceiving and understanding one’s own and others’ behaviour in terms of intentional mental states (Bateman & Fonagy, 2006). Early attachment relationships are key to the development of mentalization. In order to create internal representations of mental states the infant must experience “mirroring” from the primary caregiver. Bateman and Fonagy argue that many psychological disorders
can be understood in terms of difficulties with mentalizing. In a non-mentalizing mode, cognitions and emotions are experienced as real and concrete. A chronically depressed individual, for example, may experience negative self-appraisals as real, rather than “just thoughts” leading to low self-esteem. There is empirical evidence to support the premise that mentalisation is disrupted in those diagnosed with ASPD and BPD (McGauley, Yakeley, Williams & Bateman, 2011; Fonagy & Bateman, 2008) and in those categorised as “psychopaths” (Dolan & Fullam, 2004). Mentalization is a construct which has provided a promising focus for intervention for personality disorders which clinicians have historically viewed as difficult to treat (Fonagy & Bateman, 2006).

A recent doctoral research project investigated the hypothesis that the degree of ASPD symptomatology would predict the extent of mentalizing difficulties (Newbury-Helps 2011). In this study 82 male offenders on license in the community ASPD completed three computerised measures of mentalizing ability; the Movie Assessment for Social Cognition (MASC, Dziobeck, Fleck, Kalbe, Rogers, Hassenstab, Brand & Convit, 2006), The Perspective Taking Test (Dumontheil et al., 2010) and the Reading the Mind in the Eyes, Revised Version (Baron-Cohen, Wheelwright, Hill, Raste & Plumb, 2001). ASPD traits were measured using the Personality Assessment Inventory (PAI, Morey, 1991). Data from the London Probation Service’s offender management database was also used to provide a behavioural measure of the severity of ASPD pathology. The results revealed that those offenders with a diagnosis of ASPD seemed to have a mentalizing deficit in comparison to those without a diagnosis of ASPD. In terms of the hypothesised relationship between ASPD traits and mentalizing deficit, some modest correlations were revealed. Three of the mentalizing subscales had some predictive power in terms of ASPD severity.

The current study will extend the research of Newbury-Helps (2011) with a larger and more diverse sample, including people meeting criteria for BPD, ASPD
and non-clinical controls. Data on mentalizing could provide empirical support for the mentalization deficit theory of personality disorder (Fonagy & Luyten, 2009). This study will employ a mixture of computerized and self-report measures of mentalizing. This may be beneficial in that scores are less likely to be affected by intellectual ability in comparison to the complex computerized tasks employed in previous studies.

In accordance with the mentalizing deficit hypothesis of personality disorder, it was predicted that the ASPD and BPD groups would demonstrate a mentalizing deficit in comparison to the control group in terms of their scores on the three mentalizing measures.

In line with a dimensional model of personality disorder, it was hypothesised that personality pathology would vary with psychopathy when measured as dimensional constructs. Specifically, it was predicted that as personality disorder pathology increases, psychopathy scores would increase and mentalizing scores would decrease. Analysis of this data will also allow for testing of the premise that BPD is a “phenotypic expression of psychopathy” (Sprague et al., 2012), if elevated psychopathy traits were found in the BPD group. By comparing the three groups on psychopathy measures, this will also contribute to the ongoing debate as to whether ASPD and psychopathy are distinct disorders. If they are similar constructs then it would be expected that the ASPD group would have a significantly higher score on psychopathy measures compared to the BPD and control groups.

One study has previously explored the relationship between mentalizing and psychopathy (Dolan & Fullam, 2004), who found that psychopathy was associated with a deficit on subtle tests of mentalizing ability. A third, somewhat exploratory hypothesis was that psychopathy score would be related to mentalizing ability. Due to previous support in the literature for different subtypes of psychopathy (Poythress et al., 2010; Cox et al., 2013), it was predicted that primary and secondary subscales of a self-report psychopathy measure may
differ in terms of their relationship to mentalizing ability. A finding that these subscales differ in terms of mentalizing ability and personality disorder pathology would provide further support for the two factor theory of psychopathy.

1. Method

This study drew its sample from a large ongoing study which is investigating the neural and behavioural signatures of emerging and manifest BPD and APSD in adult and adolescent clinical and control populations (Montague and Fonagy, Wellcome grant). This involved completing a range of structured interviews, questionnaires, and behavioural measures, and engaging in computer tasks in an fMRI paradigm. The larger study recruited participants from outpatient personality disorder services across London, London Probation Services and MST (multi systemic therapy) trial sites. Ethical approval for the study was granted by the Research Ethics Committee (REC) for Wales (See Appendix B1).

2.1 Participants

A power analysis was conducted and assuming a power of 0.8 and an alpha of <.05, a minimum sample size of 59 participants was required. From the larger study database, cases which had complete datasets as of March 2014 were selected for analysis, for a total of 162 participants. Anyone with a diagnosis of ASPD according to the SCID-II was allocated to the ASPD group (N=21). The remaining cases were divided into those meeting criteria for BPD (N=60), and controls who did not meet criteria for either diagnosis (N=81).

2.2 Demographics

Socio demographic data was gathered on gender, ethnicity, education, parental income and education, physical health, and history of psychological therapy and details of medications prescribed. These are potential covariates which could be controlled for in analysis. For example, IQ may affect scores on measures of
mentalizing ability, as was found to be the case in previous research (Newbury-Helps, 2011).

Table 1 displays the demographic characteristics of the sample. The intention of the study was to match groups in terms of age, gender, ethnicity and intellectual ability. Gender was not evenly distributed across samples.

Table 1

Demographic Characteristics of Sample

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>BPD</th>
<th>ASPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>81</td>
<td>60</td>
<td>21</td>
</tr>
<tr>
<td>Age</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td></td>
<td>29.01 (10.52)</td>
<td>31.34 (10.03)</td>
<td>31.86 (13.59)</td>
</tr>
<tr>
<td>IQ</td>
<td>50.75 (5.73)</td>
<td>46.97 (8.44)</td>
<td>45.95 (6.72)</td>
</tr>
<tr>
<td>Intellectually impaired</td>
<td>3.70%</td>
<td>13.33%</td>
<td>15.00%</td>
</tr>
<tr>
<td>Definitely below average</td>
<td>29.60%</td>
<td>35.00%</td>
<td>30.00%</td>
</tr>
<tr>
<td>Average</td>
<td>54.30%</td>
<td>45.00%</td>
<td>50.00%</td>
</tr>
<tr>
<td>Definitely above average</td>
<td>4.90%</td>
<td>5.00%</td>
<td>5.00%</td>
</tr>
<tr>
<td>Intellectually superior</td>
<td>7.40%</td>
<td>0.00%</td>
<td>0.00%</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White British</td>
<td>51.90%</td>
<td>56.70%</td>
<td>61.90%</td>
</tr>
<tr>
<td>Other white</td>
<td>14.80%</td>
<td>15.00%</td>
<td>0.00%</td>
</tr>
<tr>
<td>Black British</td>
<td>8.60%</td>
<td>13.30%</td>
<td>9.52%</td>
</tr>
<tr>
<td>Mixed</td>
<td>13.60%</td>
<td>3.30%</td>
<td>9.52%</td>
</tr>
<tr>
<td>Asian British</td>
<td>8.70%</td>
<td>6.70%</td>
<td>9.52%</td>
</tr>
<tr>
<td>Other / not stated</td>
<td>2.40%</td>
<td>5.00%</td>
<td>9.52%</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>48.10%</td>
<td>21.67%</td>
<td>100.00%</td>
</tr>
<tr>
<td>Females</td>
<td>51.90%</td>
<td>76.67%</td>
<td>0.00%</td>
</tr>
<tr>
<td>Not specified</td>
<td>0.00%</td>
<td>1.66%</td>
<td>0.00%</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No GCSEs</td>
<td>6.20%</td>
<td>12.07%</td>
<td>23.81%</td>
</tr>
<tr>
<td>GCSEs less than 5 A* to C</td>
<td>8.60%</td>
<td>6.90%</td>
<td>23.81%</td>
</tr>
<tr>
<td>GCSEs 5 or more A*-C</td>
<td>23.50%</td>
<td>24.14%</td>
<td>28.57%</td>
</tr>
<tr>
<td>A level</td>
<td>35.80%</td>
<td>29.31%</td>
<td>14.29%</td>
</tr>
<tr>
<td>Higher education</td>
<td>16.00%</td>
<td>25.86%</td>
<td>4.76%</td>
</tr>
<tr>
<td>Postgraduate</td>
<td>9.90%</td>
<td>1.72%</td>
<td>4.76%</td>
</tr>
</tbody>
</table>
The gender distribution in the sample generally reflects the disproportionate percentage of females referred to personality disorder services, and males in the probation service. A series of t-tests revealed no significant difference between the groups in terms of age. A chi squared analysis suggested that there were no significantly unusual variations in ethnicity across the samples, $\chi^2(30, 162)=41.84$, $p=0.07$. There was evidence of significant variation in terms of the highest level of education reached across the samples, $\chi^2(12, 162)=20.86$, $p=0.05$. A Cramer’s V of .25 indicated a small effect size.

2.3 Procedure

Clinicians at clinical recruitment sites were briefed as to the nature of the study and inclusion criteria, and how to explain the study to potential participants. They were provided with information sheets for clinicians and for potential participants. Volunteers then completed a form granting their consent to be contacted by researchers from the larger study. The probation service used their computer database which identifies those likely to receive a diagnosis of personality disorder in order to focus recruitment to those likely to meet criteria for ASPD. Control participants were recruited via an online participant pool, and via posters in public places such as coffee shops, and were then screened by telephone for eligibility. Attempts were made to match the control participants to the clinical groups in terms of age and education. At the first testing session participants were given an information sheet (Appendix B2) and signed a consent form (Appendix B3). Upon completion of the tasks participants were given a debriefing form (Appendix B4). In order to mediate the risk of participant distress after leaving the research site, the debriefing form contained relaxation and distress tolerance techniques, and contact details of an experienced clinician and the overarching study supervisor. Participants were compensated £10 per hour, plus an additional payment calculated based on their performance on computerized tasks.
If participants declined or were not eligible for the fMRI aspect of the study, testing took place at clinical sites from which participants were recruited. Control participants and those eligible for fMRI tested at the Functional Imaging Laboratory, Queen Square. The overarching study required the completion of a large number of measures and an fMRI scan, and therefore two separate testing sessions were required. Each session was facilitated by one of a team of researchers from clinical and academic psychology disciplines. Where possible, the same researcher conducted both sessions.

2.4 Design

This study employed a cross sectional between groups design in order to explore group differences in mentalizing and psychopathy scores between those with and without a diagnosis of personality disorder. A correlational design was also employed in order to investigate psychopathy, mentalizing and personality pathology as covarying trait dimensions.

2.5 Measures

a) IQ: The Raven’s Progressive Matrices (Raven, Raven & Court, 2003) are an easily administered measure of intelligence as measured by abstract reasoning ability. The respondent is provided with a booklet of patterns, each with a missing section. The respondent is required to correctly identify the one missing item from six that are presented. There are five sets each containing 12 items for a total of 60 items, which become progressively more difficult. The test is widely used in research and has the benefit of being free from the influence of language and literacy skills, and easy to administer. Split-half and test-retest reliability are reported to be above .80 (Raven, 2000) which is deemed as high. The test correlates with other established measures of intelligence (r values between .5 and .7) including the Weschler scales (Burke, 1972). The results provide a raw score, which relate to
percentiles. The percentiles inform categories, ranging from “intellectually impaired” to “intellectually superior”. The raw score was used in the current study as a measure of fluid intelligence, and the distribution of categories is also presented.

b) Personality Disorder: Personality pathology was measured by the administration of the Structured Clinical Interview for Axis-II DSM-IV personality disorders (SCID-II: First, Gibbon, Spitzer, Williams & Benjamin, 1997). The scale remains valid for measuring DSM-V personality disorders, as the original DSM-IV categorical system was retained in the DSM-V. The SCID-II is a semi-structured interview in which respondents are asked about their behaviour, thoughts, emotions and relationships, for example, “do you have a lot of sudden mood changes?”. Responses are graded on a four point scale, from “?” which denotes insufficient information, to “three” indicating that the symptom is endorsed. Researchers were trained by a clinician in the administration and scoring of this semi structured interview. Previous studies have shown the SCID-II to have excellent inter-rater agreement, with kappa values of between 0.77 and 0.94, in terms of both categorical diagnosis of personality disorders and when using the tool to measure personality pathology dimensionally (Lobbestael, Leurgans & Arntz, 2011). The SCID-II was used to provide categorical diagnoses and also dimensional indicators of level of personality pathology, as defined by the number of items endorsed (indicated by the researcher assigning a score of three to an item). As per the protocol of the overarching study, the SCID-II was administered to all participants recruited from clinical and probation sites but not to community controls.

The Standard Assessment of Personality – Abbreviated Scale (SAPAS; Moran, Leese, Lee, Walters, Thornicroft & Mann, 2003) was administered to all participants. The SAPAS is a brief eight item screening measure. The authors recommend a score of four or above as indicative of clinical levels of personality pathology (Moran et al., 2003). Five control participants scored four or above and therefore it was
decided that they would also complete the SCID-II. None of these five participants reached criteria for any of the personality disorders.

As an additional measure of borderline personality pathology that would provide a continuous measure of borderline personality traits, the borderline subscale of the Personality Assessment Inventory (PAI-BOR; Morey, 1991) was included in the analysis. The PAI-BOR is a self report questionnaire containing 24 items relating to the core features of BPD; affective instability, identity and relationship problems, and self harm. Respondents are required to rate items such as “my moods get quite intense” and “my relationships have been stormy” on a four point scale, (0 to 3; false, slightly true, mainly true, very true). This scale has been validated in non clinical samples which makes it appropriate for the current study (Jackson & Trull, 2001). This scale has been suggest to have good internal consistency (α=.84; Trull, 1995), good convergent and discriminant validity, and high test-rest test reliability over a three to four week period (r=.86, Morey, 1991).

c) Psychopathy: The Levenson Self Report Psychopathy Scale (SRPS; Levenson, Kiehl & Fitzpatrick, 1995) is a 26-item self-report measure designed to evaluate both the behavioral and personality traits commonly associated with psychopathy. Items such as, “people who are stupid enough to get ripped off usually deserve it” are rated on a five point scale, from “strongly disagree” to “strongly agree”. The SRPS consists of two factors, (interpersonal and behavioural) which theoretically represent the two factors of the PCL-R. It is deemed to be a quick and reliable method of assessing the level of psychopathic traits in non-clinical populations, and also correlates well with the PCL-R in forensic populations (Brinkley, Schmitt, Smith & Newman, 2001). The SRPS has been found to have high test – retest reliability across a two month period, r=.83, p<.01. (Lynam, Whiteside & Jones, 1999). In the same study a factor analysis confirmed a two factor model, \( \chi^2(280, N = 1852) = 45, p <.001 \). In terms of internal consistency, Cronbach’s alpha
values for the total SRPS score, primary, and secondary factors have been reported as .85, .83 and .69 respectively (Brinkley, Schmitt, Smith & Newman, 2001).

d) Antisocial Behaviour: Given that the SCID-II would not be administered to control participants, the Life History of Aggression (LHA, Coccaro, Berman & Kavoussi, 1997) was administered as a supplementary measure of antisocial and aggressive behaviour. The LHA is an 11 item self report questionnaire, consisting of three subscales; aggression, antisocial behaviour/consequences, and self directed aggression. Respondents are required to indicate the frequency of the occurrence of events over the course of their lifetime, such as “got into physical fights with other people” and “had difficulties with the law or police which resulted in a warning, arrest or conviction for a misdemeanor or felony offense”. Items are rated on a scale from zero (never happened) to five (happened so many times I couldn’t give a number). Possible scores therefore range from 0 to 55, with a higher total score indicating a more aggressive history. It has been found to have excellent test-retest stability (0.91) and a Cronbach’s alpha of 0.88 was reported indicating good internal consistency (Coccaro et al., 1997).

e) Mentalizing ability: Given the lack of established and validated self report measures of mentalization, the Reflective Functioning Questionnaire (RFQ-54, Fonagy & Ghinai, in preparation) was used as a measure of mentalizing deficit. The subscales were analysed for internal consistency, and hypothesis testing using RFQ-54 scores as a continuous measure of mentalizing deficit may provide support for a non resource intensive, easy to administer self report measure of mentalizing. The Reflective Functioning Questionnaire (RFQ-54; Fonagy & Ghinai, in preparation) is a 54 item self-report measure of mentalization. This is an adaptation of the Reflective Functioning Scale (Fonagy, Target, Steele, & Steele, 1998). Items such as ‘I always know what I feel’, are rated on a 7-point Likert scale ranging from 1 = ‘disagree strongly’ to 7 = ‘agree strongly’. This RFQ-54 provides an overall mentalizing trait score and two subscale scores: mentalizing with respect to self
(Internal-Self) and mentalizing others (Internal-Other). The RFQ has been shown to have acceptable internal consistency with a cronbach’s alpha of .85 and .78 for the two subscales (Fonagy & Ghinai, unpublished manuscript).

Given that the RFQ-54 still requires empirical validation, a well validated existing measure of mentalizing was also used. The Movie Assessment for Social Cognition (MASC; Dziobeck et al., 2006) is a sensitive video-based test for the evaluation of subtle mindreading difficulties. Participant are shown a 15 minute film in which four characters get together for a dinner party. The video is paused 46 times for participants to answer multiple choice questions concerning the characters’ feelings, thoughts and intentions, such as “What is Sandra feeling?” and “why is Michael saying this?”. The MASC has good internal consistency, with a reported alpha of 0.84. Intraclass correlation coefficients suggested good test-retest reliability (ICC=0.97).

2.6 Statistical Analysis

All data from the overall study was continually entered onto an Excel spreadsheet. Questionnaire responses were checked and reversed appropriately for total scores to be correctly calculated before being exported to SPSS 22 (IBM Corp, 2013) for analysis. Data was explored for skewness, kurtosis and outliers in order to check whether assumptions for parametric testing were satisfied. Initial analysis was conducted in order to calculate the Cronbach’s alpha for the scores used, as in index of the internal consistency of the scales before proceeding to the hypothesis driven testing.

In order to test the hypothesis that ASPD and BPD are characterized by a mentalizing deficit, it was first necessary to explore group differences in terms of mentalizing measures. It was recommended (Fonagy, personal communication) that the RFQ54 be scored in terms of two subscales; Low Reflective Functioning – uncertain (LRFu) and the Low Reflective Functioning – certain (LRFc). The former
measuring uncertainty as to mental states of self and others, and the latter, a measure of certainty about the mental states of self and others. Three ANOVAs were conducted, in order to assess group differences on each of the three mentalizing measures (MASC, LRFu and LRFc). Post hoc tests allowed to allow group comparisons where a main effect was revealed in order to see which group differences were significant. To establish whether any significant group differences remained so whilst accounting for the variance that may be related to intellectual abilities. Using ANCOVA, Raven’s score was entered as a covariate into the models and again, post-hoc tests were used to assess which, if any, group differences would remain significant. In order to explore personality pathology as a continuous trait, Pearson’s correlations were conducted between each mentalizing measure and personality scale (LHA and PAI-BOR), as well as Raven’s score, to assess whether intellectual ability was related to scores on any of the mentalizing measures. For the subscales that were deemed likely to be affected by IQ, partial correlations were conducted with Raven’s score as a bivariate. The outcome of these correlations were then used to inform the decision to enter variables into three multiple linear regression models, in order to explore the predictive power of personality traits and IQ, with the three mentalizing scales as outcomes in three separate models. This would inform the hypothesis that PD traits are predictive of mentalizing pathology, accounting for the potential impact of IQ score. Due to the lack of prior research utilising the RFQ-54 subscales in this way the decision was made to enter all variables at the same time.

In order to inform the second hypothesis regarding the relationship between psychopathy and personality pathology, an ANOVA was conducted to assess for differences between the two PD groups and the control group, with total psychopathy score as an outcome variable. Significantly higher psychopathy scores in the BPD group would support the premise of BPD as a phenotypic expression of psychopathy. If ASPD and psychopathy are not distinct disorders then a significantly
higher psychopathy score in the ASPD group would be expected. Correlational analyses between severity of BPD and ASPD traits were then conducted in order to further explore the relationship between psychopathy and personality pathology. If the primary and secondary scales correlated differentially with severity of traits this would support psychopathy as a multidimensional construct. As in the testing of the first hypothesis, the outcomes of these correlational analyses were used to inform a regression model, in order to further assess the predictive power of any variables which seemed to be significantly correlated.

In order to test the third exploratory hypothesis around mentalizing ability and psychopathy, a series of correlations were conducted between the three mentalizing scales and the three self reported psychopathy scales. Finally, a multiple linear regression was conducted in order to assess the extent to which psychopathy was predictive of mentalizing deficit.

Throughout the analysis, the Games-Howell post-hoc test was used when the assumption of homogeneity of variance was violated as indicated by a significant Levene’s statistic. When this assumption was not violated, the Tukey’s post-hoc procedure was used (recommended in Morgan, Leech, Gloeckner & Barrett, 2012).

3. Results
3.1 Reliability of scales

Cronbach’s alpha was calculated for all scales and subscales used in the analysis. Values are presented in table 2 and indicate that the majority of the scales used had good internal consistency. The subscales with the lowest alpha values were the primary and secondary subscales of the SRPS, which seemed to have poor internal consistency.
Table 2
Internal Consistency of Scales

<table>
<thead>
<tr>
<th>Scale</th>
<th>Cronbach's alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td>RFQ54</td>
<td></td>
</tr>
<tr>
<td>LRFc</td>
<td>0.86</td>
</tr>
<tr>
<td>LRFu</td>
<td>0.85</td>
</tr>
<tr>
<td>PAI-BOR</td>
<td></td>
</tr>
<tr>
<td>Affective Instability</td>
<td>0.87</td>
</tr>
<tr>
<td>Identity Problems</td>
<td>0.77</td>
</tr>
<tr>
<td>Negative Relations</td>
<td>0.77</td>
</tr>
<tr>
<td>Self Harm</td>
<td>0.84</td>
</tr>
<tr>
<td>Total</td>
<td>0.94</td>
</tr>
<tr>
<td>SRPS</td>
<td></td>
</tr>
<tr>
<td>Primary</td>
<td>0.58</td>
</tr>
<tr>
<td>Secondary</td>
<td>0.58</td>
</tr>
<tr>
<td>Total</td>
<td>0.7</td>
</tr>
<tr>
<td>LHA</td>
<td></td>
</tr>
<tr>
<td>Aggression</td>
<td>0.89</td>
</tr>
<tr>
<td>Antisocial/consequences</td>
<td>0.73</td>
</tr>
<tr>
<td>Self directed aggression</td>
<td>0.88</td>
</tr>
<tr>
<td>LHA total</td>
<td>0.87</td>
</tr>
<tr>
<td>MASC</td>
<td>0.95</td>
</tr>
</tbody>
</table>

The subscales with the lowest alpha values were the primary and secondary subscales of the SRPS, which seemed to have poor internal consistency. Alpha values for the SRPS are slightly lower than those reported in previous studies alpha values of .85, .83 and .69 for total, primary and secondary factor scores respectively were reported by Brinkley et al. (2001).

3.2 Data exploration

Data were explored for normality and outliers. Starting with the control group, the LHA, PAI-BOR, MASC and LRFu scales were skewed, as indicated by dividing the skewness statistic by the standard error. Kolmogorov-Smirnov and Shapiro-Wilks statistics were also significant for these scales, providing further evidence of skewness. In order to address this, Z-scores were calculated and ten cases with a value >2 were identified. The decision was made to remove these cases in their
As a result, all scales, with the exception of the LHA, no longer demonstrated any evidence of skewness on indices. Four extreme values in terms of LHA score were identified and the cases removed, after which the LHA also met assumptions for normality on all indices described above. The remaining N for the control group was 67.

For the ASPD group, all scales did not violate assumptions of normality according to any of these indices, with the exception of the total SRPS score, which provided a value >2 when the skewness statistic was divided by the standard error. Calculation of Z scores revealed one extreme low value. All data for this entire case was removed, after which all assumptions were satisfied. The remaining N for the ASPD group was 20.

For the BPD group, all scales met all the above assumptions, with the exception of LRFu, which had a significant Kolmogorov-Smirnov statistic. Two cases were identified by SPSS as extreme values (one high, one low). These two cases also converted into Z scores >2. As such all data from these two cases were removed from the dataset, after which the data satisfied all tests described here and were deemed suitable for parametric testing.

After removal of these 17 cases the remaining N was 145 with 67 controls, 58 in the BPD group and 20 in the ASPD group.

### 3.3 Matching samples

Previous research suggests that measures of mentalizing such as the MASC can be affected by IQ. In order to assess whether samples were matched in terms of IQ, t-tests were conducted. Table 3 displays groups means for IQ, as indicated by the mean raw score on the Raven’s Standard Progressive Matrices (Raven, Raven & Court, 2003).
Table 3
*Mean Raw Raven’s Scores and Categories*

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>BPD</th>
<th>ASPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>67</td>
<td>58</td>
<td>20</td>
</tr>
<tr>
<td>Raven’s SPM raw score M (SD)</td>
<td>50.75 (5.73)</td>
<td>46.97 (8.44)</td>
<td>45.95 (6.72)</td>
</tr>
<tr>
<td>Intellectually impaired</td>
<td>3.70%</td>
<td>13.33%</td>
<td>15.00%</td>
</tr>
<tr>
<td>Definitely below average</td>
<td>29.60%</td>
<td>35.00%</td>
<td>30.00%</td>
</tr>
<tr>
<td>Average</td>
<td>54.30%</td>
<td>45.00%</td>
<td>50.00%</td>
</tr>
<tr>
<td>Definitely above average</td>
<td>4.90%</td>
<td>5.00%</td>
<td>5.00%</td>
</tr>
<tr>
<td>Intellectually superior</td>
<td>7.40%</td>
<td>0.00%</td>
<td>0.00%</td>
</tr>
</tbody>
</table>

The control group had a significantly higher IQ score as indicated by the Ravens raw score in comparison to the BPD group $t(126)=3.29, p=.001$, and the probation group, $t(88)=3.07, p=.002$. There was no significant difference between the two clinical groups in terms of IQ score.

### 3.4 Hypothesis testing

The first hypothesis was that there would be a mentalizing deficit in the personality disorder groups. A series of one way ANOVAs was conducted in order to examine group differences in mentalizing ability (see table 4).

Table 4
*Results of One Way Analysis of Variance for Group Differences in Mentalizing Measures*

<table>
<thead>
<tr>
<th></th>
<th>LRFu</th>
<th>LRFc</th>
<th>MASC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Control</td>
<td>8.55 (5.24)</td>
<td>25.83 (11.80)</td>
<td>35.45 (4.05)</td>
</tr>
<tr>
<td>BPD</td>
<td>25.57 (9.60)</td>
<td>13.19 (7.45)</td>
<td>32.47 (4.97)</td>
</tr>
<tr>
<td>ASPD</td>
<td>20.35 (8.28)</td>
<td>19.59 (13.09)</td>
<td>28.15 (4.21)</td>
</tr>
<tr>
<td>F value</td>
<td>76.53</td>
<td>21.60</td>
<td>22.09</td>
</tr>
<tr>
<td>Significance</td>
<td>p&lt;.001</td>
<td>p&lt;.001</td>
<td>p&lt;.001</td>
</tr>
<tr>
<td>Effect Size</td>
<td>.53</td>
<td>.24</td>
<td>.24</td>
</tr>
</tbody>
</table>

The LRFu is a deficit measure, therefore a higher score indicates more of a deficit in mentalizing. For the LRFu subscale, a significant group difference was revealed, $F(2,134)=76.53, p<0.001$. The assumption of homogeneity of variance was violated.
as indicated by a significant Levene’s statistic, $p<0.001$, therefore the Games-Howell post hoc procedure was performed. This indicated that control group had significantly less of a deficit in mentalizing compared to the BPD group ($p<0.001$) and the ASPD group ($p<0.001$). The difference in LRFu score between the two personality disorder groups was not significant. Overall an effect size of .53 indicates that 53% of the variance in LRFu score was accounted for by group membership. Using the benchmark for effect sizes recommended by Cohen (1988) this is deemed to be a large effect size.

For the LRFc subscale the model suggested that there was a significant difference between groups, $F(2,134)=21.60, p<0.001$. Again the assumption of homogeneity of variance was violated, indicated by a significant Levene’s result ($p=0.01$). Games-Howell post hoc multiple comparisons suggested that this finding was driven primarily by a significant difference between the control and BPD groups ($p<0.001$) with the control group scoring significantly higher than the BPD group in terms of their certainty around mental states of self and others. An effect size of .24 suggests a medium effect size, with 24% of the variance in LRFc score accounted for by group membership.

There was a significant group difference in terms of MASC score, $F(2,142)=22.09, p<0.001$. Tukey’s post hoc multiple comparisons suggested that the control group scored significantly higher on the MASC compared to the BPD group ($p=.001$) and the ASPD group ($p<0.001$). The ASPD group scored significantly lower than the BPD group ($p=.001$). These findings provide support for the hypothesis that PD is characterised by a mentalizing deficit. A partial ETA squared value of .24 indicates a medium effect size, with 24% of the variance in MASC score was accounted for by group membership.

Previous studies indicated that IQ may impact performance on measures of mentalizing, analysis of covariance was conducted to control for the impact of IQ scores on task performance.
Analysis of covariance (ANCOVA) was performed in order to test whether these significant differences remained after controlling for IQ. For LRFu scores the model remained significant, $F(1,134)=67.76, p<.001$. The effect size remained large, at .51. All group comparisons described previously remained unaffected. For the LRFc subscale scores, the model remained significant, $F(1,134)=19.07, p<.001$, with a similar medium effect size of .23. Again was driven, according to Sidak post hoc tests, by a significant difference between the control and BPD groups, $p<.001$.

For the MASC scores, the model remained significant after controlling for IQ, $F(1,142)=15.40, p<0.001$, however the effect size reduced to .18, indicating a medium effect size. Post hoc Sidak tests indicated that the difference between control and BPD groups remained significant, $p=.025$, as did the difference between the control and ASPD group, $p<.001$. The significant difference between the two personality disorder groups remained unchanged after controlling for IQ.

In order to test the hypothesis that mentalizing ability is related to personality pathology, correlations were conducted between the three MASC subscales and the two personality disorder trait measures. In order to account for the potential impact of IQ on performance on tests of mentalizing, IQ score was also correlated with each mentalizing scale (see table 5). Any positive correlations would inform the decision to carry out regressive analyses.

Table 5

<table>
<thead>
<tr>
<th></th>
<th>LRFu Mean (S.E)</th>
<th>LRFc Mean (S.E)</th>
<th>MASC Mean (S.E)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong></td>
<td>8.89 (.96)</td>
<td>25.55 (1.34)</td>
<td>34.94 (.54)</td>
</tr>
<tr>
<td><strong>BPD</strong></td>
<td>25.51 (1.05)</td>
<td>13.05 (1.47)</td>
<td>32.78 (.58)</td>
</tr>
<tr>
<td><strong>ASPD</strong></td>
<td>20.92 (1.89)</td>
<td>19.59 (2.64)</td>
<td>28.61 (.99)</td>
</tr>
<tr>
<td><strong>F value</strong></td>
<td>67.76</td>
<td>19.07</td>
<td>15.40</td>
</tr>
<tr>
<td><strong>Significance</strong></td>
<td>$p&lt;.001$</td>
<td>$p&lt;.001$</td>
<td>$p&lt;0.001$</td>
</tr>
<tr>
<td><strong>Effect Size (Eta squared)</strong></td>
<td>.51</td>
<td>.23</td>
<td>.18</td>
</tr>
</tbody>
</table>
Table 6

*Correlations Between Mentalizing and Personality Scales*

<table>
<thead>
<tr>
<th></th>
<th>PAI-BOR</th>
<th>LHA</th>
<th>IQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>LRFc</td>
<td>-.53**</td>
<td>-.33**</td>
<td>.14</td>
</tr>
<tr>
<td>LRFu</td>
<td>.66**</td>
<td>.46**</td>
<td>-.28**</td>
</tr>
<tr>
<td>MASC</td>
<td>-.30**</td>
<td>-.32**</td>
<td>.37**</td>
</tr>
</tbody>
</table>

* Significant at \( p<.001 \)

Small to moderate significant correlations were revealed between all mentalizing scales and the two personality scales. IQ score also had a small but significant correlation with the LRFu and MASC subscales, therefore partial correlations were conducted for these variables, to explore the relationship between severity of personality pathology and mentalizing deficit, whilst accounting for the potential effects of IQ. Since there was no significant relationship between IQ and LRFc, partial correlations were not carried out for this subscale.

Table 7

*Partial Correlations Controlling for IQ*

<table>
<thead>
<tr>
<th></th>
<th>PAI-BOR</th>
<th>LHA</th>
</tr>
</thead>
<tbody>
<tr>
<td>LRFu</td>
<td>.64**</td>
<td>.43**</td>
</tr>
<tr>
<td>MASC</td>
<td>-.24*</td>
<td>-.29*</td>
</tr>
</tbody>
</table>

**significant at \( p<.001 \)  *significant at \( p<.01 \)

After controlling for IQ Pearson's \( r \) values reduced slightly (see table 7) but all correlations remained significant. The strong positive correlation between the LRFu subscale and PAI-BOR, \( r=.64, p<.001 \), supports the hypothesis that severity of BPD pathology is related to an increase in mentalizing deficit. A moderate and significant correlation between the LHA and the LRFu suggests that an increase in severity of antisocial and aggressive traits is also associated with an increase in mentalizing deficit. Small but significant negative correlations between the MASC and PAI-BOR, \( r=.24, p<.01 \) and between the MASC and LHA, \( r=-.29, p<.001 \), suggest that a decrease in MASC score is associated with increasing severity of personality pathology, supporting the mentalizing deficit hypothesis. The LRFc measures the
certainty about mental states of others. A significant moderate negative correlation between LRFc score and PAI-BOR scores, $r=.53$, $p<.001$ suggests that increase in severity of BPD is related to a decrease in the certainty of mental states of self and others. The same was the case for antisocial traits, although the correlation was small rather than moderate, according to Cohen’s criteria (Cohen, 1988).

Given these findings, a linear regression was carried out in order to explore the extent to which personality pathology was predictive of mentalizing deficit (table 7). For the MASC and LRFu scales IQ was added to the model. LHA score was a significant predictor of MASC score, $b=-.24$, $t(134)=-2.26$, $p=.03$, whereas PAI-BOR was not a significant predictor of MASC score. IQ score was also a significant predictor of MASC score, $b=.32$, $t(134)=-4.14$, $p<.001$.

### Table 8

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Independent variables</th>
<th>$R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>MASC</td>
<td>PAI-BOR</td>
<td>.22</td>
</tr>
<tr>
<td></td>
<td>LHA*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>IQ score**</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PAI-BOR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LHA</td>
<td>.12</td>
</tr>
<tr>
<td>LRFu</td>
<td>PAI-BOR**</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LHA</td>
<td></td>
</tr>
<tr>
<td></td>
<td>IQ score</td>
<td>.45</td>
</tr>
<tr>
<td></td>
<td>PAI-BOR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LHA</td>
<td>.44</td>
</tr>
<tr>
<td>LRFc</td>
<td>PAI-BOR**</td>
<td>.28</td>
</tr>
<tr>
<td></td>
<td>LHA</td>
<td></td>
</tr>
</tbody>
</table>

*significant predictor $p<.05$ ** significant predictor $p<.001$

Overall the model (see table 8) accounted for 22% of the variance in MASC score, $R^2=.22$, $F(3,137)=12.62$, $p<.001$. However when IQ was removed from the model, only 12% of the variance in MASC score was accounted for $R^2=.12$, $F(2,140)=9.41$, $p<.001$.

In terms of the LRFu scale, the addition of IQ made only a small change to
the proportion of variance in LRFU score explained by the model. Together PAI-BOR and LHA scores accounted for 44% of the variance in LRFu score, \( R^2 = .44 \), \( F(2,132) = 50.46, p < .001 \). However, only PAI-BOR was a significant predictor of LRFu score, \( b = .32, t(134) = -4.14, p < .001 \).

PAI-BOR was a significant predictor of LRFc score, \( b = -.57, t(130) = -5.70, p < .001 \), but LHA was not. The model accounted for 28% of the variance in LRFc score, \( R^2 = .28, F(2,132) = 25.19, p < .001 \).

In order to test the second hypothesis, that level of psychopathy is related to severity of personality disorder traits, a one way analysis of variance was conducted. There was a significant effect of group on psychopathy score, \( F(2,142) = 15.62, p < .001 \). The ASPD group scored highest on the SRPS (M=72.63, SD=12.70) followed by the BPD group (M=67.21, SD=13.44). The control group scored the lowest on the SRPS (M=57.81, SD=10.54). Tukey's post-hoc comparisons suggested the ASPD group score was significantly higher than the control group, \( p < .001 \) as did the BPD group, \( p < .001 \). The differences between the two personality disorder groups were not significant. Correlations were performed in order to test the hypothesis that psychopathy would co-vary with severity of personality pathology (see table 9).

<table>
<thead>
<tr>
<th></th>
<th>LHA</th>
<th>PAI-BOR</th>
</tr>
</thead>
<tbody>
<tr>
<td>SRPS</td>
<td>.54**</td>
<td>.50**</td>
</tr>
<tr>
<td>SRPS primary</td>
<td>.18*</td>
<td>.34**</td>
</tr>
<tr>
<td>SRPS secondary</td>
<td>.64**</td>
<td>.75**</td>
</tr>
</tbody>
</table>

**p < .001  *p < .05

Total SRPS score had significant moderate positive correlations with both borderline, \( r = .50, p < .001 \), and ASPD traits, \( r = .54, p < .001 \). When this was broken down into the two psychopathy subscales the strongest correlation was between the secondary subscale of the SRPS and the PAI-BOR. To further explore the predictive
power of personality pathology in terms of psychopathy score a linear regression was performed (see table 10).

Table 10
Regression analyses of SRPS secondary subscale and severity of PD traits

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Independent variables</th>
<th>Variance in DV accounted for</th>
</tr>
</thead>
<tbody>
<tr>
<td>SRPS secondary subscale</td>
<td>LHA*</td>
<td>59%</td>
</tr>
<tr>
<td></td>
<td>PAI-BOR**</td>
<td>59%</td>
</tr>
<tr>
<td></td>
<td>PAI-BOR*</td>
<td>57%</td>
</tr>
</tbody>
</table>

**Significant predictor p<.001  *Significant predictor p<.05

PAI-BOR, b=.60, t(136)=8.09, p<.001 and LHA, b=.22, t(136)=2.93, p<.05 were both significant predictors of psychopathy score and together accounted for 59% of the variance in psychopathy score, $R^2=.59, F(2,138)=98.05, p<.001$. When LHA was removed from the model, PAI-BOR alone accounted for 57% of the variance in psychopathy score, $R^2=.57, F(2,138)=177.65, p<.001$.

In order to inform the hypothesis that psychopathy score would co-vary with mentalizing ability, correlations were carried out between the SRPS and its primary and secondary subscales, and the three mentalizing measures (table 11). In the case of the MASC and LRFu, partial correlations were conducted to control for the effect of IQ.

Table 11
Correlations Between Psychopathy and Mentalisation

<table>
<thead>
<tr>
<th></th>
<th>LRFc</th>
<th>LRFu</th>
<th>MASC</th>
</tr>
</thead>
<tbody>
<tr>
<td>SRPS total</td>
<td>-.35**</td>
<td>.36**</td>
<td>-.30**</td>
</tr>
<tr>
<td>SRPS primary factor</td>
<td>-.12</td>
<td>.07</td>
<td>-.22*</td>
</tr>
<tr>
<td>SRPS secondary factor</td>
<td>-.55**</td>
<td>.62**</td>
<td>-.29**</td>
</tr>
</tbody>
</table>

** p<.001  * p<.01

Total psychopathy score had small but significant correlations with all MASC measures. The negative correlation with LRFc, $r=-.35, p<.001$, suggests that as psychopathy increases, certainty about mental states of self and other decreases. The positive correlation between SRPS and the LRFu scale, $r=-.32, p<.001$ suggests...
that as psychopathy scores increase, mentalizing deficit also increases, and MASC scores decrease, $r = -.30, p < .001$. Interestingly, the primary factor of the SRPS did not correlate with any mentalizing measure, other than a modest negative correlation with the MASC, $r = -.22, p = .007$, whereas the secondary factor had small to moderate significant correlations with all measures of mentalizing. These findings support the two factor theory of psychopathy, and suggest that a mentalizing deficit may be more related to secondary than primary psychopathy. In order to further explore this hypothesis, a linear regression was conducted. Guided by the results of correlational analyses, LRFu was selected as the dependent outcome, and the secondary subscale of the SRPS was entered as a predictor (table 12).

### Table 12

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Independent variables</th>
<th>Variance in DV accounted for</th>
</tr>
</thead>
<tbody>
<tr>
<td>LRFu</td>
<td>SRPS secondary subscale**</td>
<td>43%</td>
</tr>
<tr>
<td></td>
<td>SRPS total*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SRPS secondary subscale**</td>
<td>39%</td>
</tr>
</tbody>
</table>

**significant predictor $p < .001$  *significant predictor $p < .01$

The secondary subscale of the SRPS was a significant predictor of LRFu score, $b = .62, t(133) = 9.12, p < .001$, and accounted for 39% of the variance in LRFu score, $R^2 = .39, F(1,134) = 83.25, p < .001$. The amount of variance accounted for when total SRPS was entered into the model increased only slightly, to 43%, $R^2 = .43, F(2,134) = 49.85, p < .001$. SRPS total was also a significant predictor of LRFu score, $b = -.34, t(133) = -3.24, p = .002$.

4. **Discussion**

The results suggest that those with a diagnosis of BPD do experience a mentalizing deficit compared to non clinical controls, providing support for the mentalizing model of BPD (Fonagy & Bateman, 2008). Borderline pathology was
predictive of mentalizing ability, particularly as measured by the LRFu subscale, suggesting that severity of borderline traits predicts level of uncertainty about mental states of self and others. The strongest predictor of MASC score was IQ score, suggesting that this may not be a useful measure of mentalizing. In terms of ASPD the data supports a modest relationship with mentalizing deficit. Whilst the ASPD group's scores were lower than controls and mentalizing subscales were significantly correlated with ASPD traits, as measured by the LHA, regression analyses did not find LHA score to be significant predictors of mentalizing deficit.

As expected, the ASPD group had the highest psychopathy scores. An interesting finding was that the BPD group's psychopathy scores were also significantly higher than the control group. The link between BPD and psychopathy was further supported when BPD traits were explored as a continuous trait measure. As BPD pathology increased, so did psychopathic traits. This contributes towards the debate in the literature as to whether BPD may represent a phenotypic expression of psychopathy (Sprague et al., 2012) however the overlap between the measures has been highlighted previously (e.g. Dolan & Coid, 1993). The two personality disorder groups did not differ significantly in terms of psychopathy score. The failure to find a difference between the two personality groups could be interpreted as providing support for the tenet that ASPD and BPD are representations of expressions of a collection of trait dimensions rather than distinct categorical entities (Paris, 1997). Some differences, however, were revealed when regression analyses were conducted to explore the predictive power of ASPD and BPD traits in terms of psychopathy score.

As described above, regression analyses supported BPD trait pathology as a significant predictor of mentalizing deficit, whereas LHA was not a significant predictor of any index of mentalizing, although it did correlate significantly with all three measures. This finding could be interpreted as evidence of a mentalizing deficit in ASPD. It is also possible that the modest statistical findings are affected by
the fact that “ASPD” may be comprised of many different trait dimensions. Previous research suggests that those meeting criteria for ASPD actually represent a heterogeneous and diverse group consisting of various subtypes. Studies have used factors such as internalizing and externalizing psychopathology, impulsivity, aggression and violent offending to further validate these subtypes (Poythress et al., 2010; Cox et al., 2013). At least two subtypes are well replicated in the literature and theoretically map onto primary and secondary psychopathy. Primary psychopathy, relates to the “classic” psychopath first described by Robert Hare as cold, callous, unemotional (Hare & Hart, 1993). This group are low in anxiety and are more likely to engage in instrumental aggression, their ability to understand the mental states of others does not differ from controls (Blair et al., 1996). Secondary psychopaths, the literature suggests, are characterised by higher internalizing and externalizing psychopathology, and are more impulsive and likely to engage in reactive aggression as a result of emotional dysregulation (Lykken, 1995). The data from the current study provide further support for the heterogeneity of antisocial personality pathology. Severity of psychopathy was related to increasing mentalizing deficit and this variance in mentalizing was best accounted for by the secondary factor of psychopathy. This finding supports the two factor theory of psychopathy, perhaps with secondary psychopathy representing a deficit in mentalizing as a result of emotional dysregulation. The current finding, that mentalizing deficit was most apparent in secondary psychopaths, could be interpreted as replication of the previous failure to find a theory of mind deficit in primary psychopathy (Dolan & Fullam, 2004). The findings from the current study provide some contribution towards the ongoing debate in the literature as to whether ASPD and psychopathy are distinct. The data supports, to some extent, the two as different expressions of a variety of trait dimensions rather than terms that can be used interchangeably, as they have in the past. Further replication is needed with more even sample sizes.

The categorical system of personality disorders adopted by the DSM has
been criticised for high comorbidity, arbitrary cutoffs, and poor validity and reliability (e.g. Verheul, 2006). The potential move towards a dimensional system has been debated in the literature for many years (Widiger & Samuel, 2005) particularly in the context of the recent revision of the DSM 5 (APA, 2013). One area of the literature that can inform this debate stems from Joel Paris’ research into whether ASPD and BPD are distinct, or rather different representations of a set of trait dimensions such as impulsivity and emotional dysregulation (Paris, 1997; 2004). Most recently it was concluded that ASPD and BPD are distinct disorders which share overlapping trait dimensions and risk factors. The current study utilised both group and dimensional measures of ASPD and BPD pathology and compared them on trait dimensions of mentalizing, and psychopathy. A failure to find group differences between BPD and ASPD on these trait dimensions provide support for the overlap between the two disorders highlighted by Paris. When measured as continuous traits, borderline pathology was predictive of mentalizing ability. The finding that the ASPD group did not significantly differ from the BPD group in terms of mentalizing deficit or psychopathy score provides further support for the view of BPD and ASPD as two phenotypic expressions of one set of traits, as posited by Paris (1997). It is acknowledged that the failure to find these group differences could be related to the small ASPD sample size and therefore replication is required.

The results support the reliability of non resource intensive, easy to administer measures of mentalization, borderline and antisocial personality traits, with good internal consistency indicated for most of the scales used. The primary and secondary subscales of the SRPS had poor internal consistency within this sample, therefore findings on psychopathy should be interpreted with caution. Severity of BPD and ASPD pathology as continuous measures yielded significant results in terms of predicting other constructs such as mentalizing deficit and psychopathy, which supports the utility of a dimensional approach to personality disorder research.
A challenge of the current study was one that has been cited as a criticism of the categorical approach adopted by the DSM (Sarkar & Duggan, 2010) which is the issue of comorbidity. Many participants recruited for the study met criteria for both BPD and ASPD and indeed for other personality disorders. A weakness in the methodology is that AXIS I disorders were not controlled for.

Another limitation of the current study is that group sizes were very uneven. Difficulties in recruiting from probation services led to a small sample of ASPD participants. The current study used psychopathy as a trait dimension on which participants with and without diagnoses of ASPD and BPD varied. A difficulty in the study of psychopathy is that even those rating highly on measures of psychopathy may not meet criteria for psychopathy on a gold standard measure such as the PCL-R and therefore it would be important to replicate these findings with a larger ASPD sample size. In a larger sample it would be more likely that people representing the full spectrum of psychopathy are captured.

This study attempts to measure the construct of mentalization in order to test the mentalizing deficit hypothesis of BPD, and explore this deficit in antisocial populations. The mentalization theory of BPD explains that individuals may switch into and out of a “mentalizing mode”, and that problems arise during emotional dysregulation where mentalizing capacity is reduced. By this account, mentalisation is not a stable personality trait which can be easily measured via self report. This also raises the issue of the effect of emotional states of participants on their task performance, particularly for those with a diagnosis of BPD. In those participants it is likely that the demands of the testing, combined with existing emotional dysregulation, and other likely factors such as disturbed sleep could have impacted on task performance. The data described here were drawn from a larger study, which required participants to complete a large battery of measures beyond those described here over the course of two lengthy sessions. The demands of the testing sessions may have influenced task performance due to boredom, frustration,
tiredness or difficulty concentrating.

The original intention was to measure ASPD pathology by adding the number of ASPD items on the SCID-II that were endorsed to create a total score. This would provide a continuous variable of ASPD pathology. Unfortunately there was insufficient SCID-II data available and therefore the LHA scale was used as a continuous measure of antisocial behaviour. It contains three subscales that map onto the SCID-II criteria. It does not just cover physical aggression but accounts for other factors which are included in the SCID-II criteria for ASPD, such as school and employment disciplinary problems, criminal behaviour not resulting in contact with police such as driving whilst intoxicated. It is acknowledged that the LHA scale is not widely used as a measure of severity of ASPD pathology. It would be interesting to replicate these findings with the use of the ASPD subscale of the PAI.

The current findings provide support for the continued development of non-resource intensive self-report tools measuring mentalizing ability. Therapies focusing on increasing the ability to mentalize are becoming increasingly used in NHS personality disorder services and further research into the validity of the construct of mentalizing and how it is measured is necessary in order to inform and evaluate these treatments. These findings are also relevant to the assessment of individuals in terms of their suitability for mentalizing based treatment. Rather than assume that all “antisocial” individuals would benefit from such a treatment it is important to assess mentalizing ability on an individual level.

There has been a historical tendency to consider all “antisocial” individuals as one distinct category, with the terms “ASPD” and “psychopathy” often being used interchangeably. The current findings provide some support for the position that these disorders may be better understood as a variety of trait continuums. At the very least these data support distinction between two factors of “psychopathy”. The data support the existence of a primary, more cold, callous, unemotional subtype with less of a difficulty in mentalizing and a secondary, more impulsive subtype with
more of a mentalizing deficit. The former may represent a group that have been previously found to have enhanced mentalizing ability, as displayed by those scoring highly on psychopathy who require these skills in order to deceive and exploit others (Bateman & Fonagy, 2006). This suggests that a more formulation based approach to assessment and treatment of those with antisocial personality disorder traits may be appropriate than the current categorical system. The addition of a new dimensional-categorical hybrid model of personality disorder to the DSM-V calls for more research into the utility and validity of a dimensional model. The current study contributes towards the body of data supporting the utility of a dimensional model of personality traits, with personality disorders representing extreme ends of various dimensions rather than as separate categorical entities. Significant differences between BPD and ASPD groups were not revealed in terms of trait dimensions such as mentalizing ability and primary and secondary psychopathy. Whilst unequal sample size is an issue, the failure to find these group differences could also be interpreted as evidence to support the tenet that they in fact do not represent two distinct groups.
References


Part Three: Critical Appraisal
1. Introduction

The following is a reflection on the process of conducting the research presented in this thesis. The advantages and disadvantages of conducting research as a part of a large scale existing research project are considered. Ethical and risk issues associated with conducting this research with a population of people with a diagnosis of personality disorder are presented. The methodological issue of recruiting and conducting research in organisations outside of the NHS such as the London Probation Service is discussed in relation to the challenges that arose whilst recruiting for this study. The conceptual issues of measuring personality traits, and refining the measurement of “mentalizing” are discussed. Finally the clinical implications of a dimensional model of personality disorder are discussed in relation to assessment, treatment and future research.

2. Working as part of a large scale existing research project

Having worked with and conducted postgraduate level research in the area of psychopathy before, this project appealed to my interests. The potential for a large clinical sample was particularly appealing, as my previous research had been limited to the study of personality traits in community samples. The study had many benefits, such as being well funded by a large grant from The Wellcome Trust, awarded to Professors Peter Fonagy and Read Montague. The project had links with clinical and probation services, and approximately ten other students collecting data at any one time. The large number of researchers, combined with the funding which allowed for financial incentives for participants, guaranteed a respectable sample size. Another benefit of working on an already established project was that ethical approval had already been granted, which meant that I could begin data collection at an early stage. This project also afforded the opportunity to gain experience in clinical and probation settings, and gain specialist knowledge in personality disorder. I would also have the opportunity to be trained in using
instruments such as the Structured Clinical Inventory for Axis-II Personality Disorders (SCID-II, First, Gibbon, Spitzer, Williams & Benjamin, 1997) and the Adult Attachment Interview (AAI, George, Kaplan & Main, 1985), and also gain experience in functional magnetic resonance imaging (fMRI).

Along with the benefits of joining a well funded and established research project, this was balanced with the challenges of having limited influence over the methodology and measures which would inform my project. The lead of the study, a computational neuroscientist, aimed to explore the neural networks of individuals with and without personality disorder using fMRI techniques, which have previously revealed differing patterns of brain activity in ASPD and BPD (Völlm et al., 2004). Therefore these interests were prioritised over the more psychological aspects of the study that were of relevance to myself. In exchange for our gathering data for the study we would receive access to the database of behavioural measures, but not fMRI data or data from the battery of computerised tasks. The larger study aimed to measure a very large number of factors, which continued to increase as the study progressed. Due to the large amount of questionnaire, interview, and computerised tasks to be completed, some of which took place in an fMRI scanner, the testing procedure was very long. This also sacrificed the quality of some of the data of interest to my project. For example, SCIDs were not administered to all participants, data was often incomplete due to time constraints, and the ASPD subscale of the PAI was not administered, which meant that this had to be replaced by the Life History of Aggression scale (LHA, Coccaro, Berman & Kavoussi, 1997) in my analysis. Whilst having a large number of researchers gathering data was reassuring in terms of having a sufficient number of participants, this also raises issues such as inter-rater reliability and increased likelihood of inconsistency in the administration of the tests.

The lengthy testing procedures required each participant to attend sessions on two separate days, each lasting three to four hours. Participants were required to
complete lengthy and repetitive computerised tasks, designed by researchers on the larger study. Participants were also required to complete a very large questionnaire pack, the Raven’s IQ test, which is again very lengthy and repetitive, amongst several other measures. It would be easy to imagine any participant becoming bored during the testing, and finding it difficult to concentrate. It is possible that this affected the quality of the data.

A major component of the overarching research study was a battery of computerised games, the “social exchange battery”. This required each participant to play against a computerised opponent in several different tasks involving investing, gambling, buying and selling. Points won or lost were converted into real financial reward which was added to the hourly rate earned by participants. These tasks were very repetitive, and required each participant to concentrate for several hours at a time. Task instructions were long and complex, taking participants around 20-30 minutes to read prior to the start of the study. Some participants struggled to understand the instructions and seemed somewhat frustrated. Others became very involved in the task and expressed frustration at the “opponent”, who essentially in some cases controlled the amount of actual money the participant would receive. I wondered whether the task length, complexity, and the competitive nature of the task could have an impact on participants’ mood state to the extent that their responses to other measures were impacted.

3. Recruitment from probation and personality disorder services

My original interest in the project was focused more on the antisocial personality disorder aspect of the study as opposed to the borderline personality disorder sample. My literature review was very much focused on this area as were my research questions for the empirical paper. The process of testing participants brought me into contact with staff and service users from personality disorder services, and I became increasingly interested in borderline personality disorder,
and eventually requested and was granted a year-long specialist placement in one of the services from which the study recruits. I then became more heavily involved in the recruitment process, and was able to recruit many participants both through my colleagues in the service and directly through my own clinical work. It was not difficult to recruit from the service, most participants were very attracted by the financial remuneration and some were interested in keeping images from the fMRI. Recruitment from probation services, however, was not so straightforward. Firstly, the political context cannot be ignored. At the time that recruitment from probation was taking place, probation services in the UK were undergoing and continue to undergo major organisational changes due to privatisation of offender management services (Ministry of Justice, 2013). This added pressure, on top of the existing demands of offender managers was likely to mean that recruiting for this study was perhaps a low priority for probation workers. This could be addressed in future similar projects by a more proactive recruitment strategy, which has been used successfully in previous research in these settings (Newbury-Helps, 2011). The probation service is still in a transitional period after The Bradley Report (Bradley, 2009) recommended a new interdepartmental strategy for managing personality disorder, and it is hoped that as this collaboration between the NHS and criminal justice system develops there will be more opportunity for research of this kind.

The study did not benefit from direct links with clinical staff on these sites, as with the NHS services and therefore an alternative recruitment strategy was employed by the overarching study. This strategy was intended to identify potential participants who were most likely to meet criteria for ASPD. A database is used by the probation service, the Offender Assessment System (OAsys). Within this database are 12 variables, such as childhood disturbance, impulsivity, and aggression which highlight an increased likelihood of the need for assessment for ASPD (Minoudis, Shaw, Bannerman & Craissati, 2012). This system was used to
put forward the names of those deemed at increased risk of meeting criteria for ASPD to project supervisors. Due to confidentiality issues which are pertinent when working across organisations, researchers were not provided with contact details for offenders. The strategy employed by the larger study was to approach the relevant offender managers, who were then tasked with passing on recruitment information to the relevant offenders or informing us when their next meeting would be so that someone from the project could attend, and wait to speak with them when they attended the probation office for their appointment. Offender managers have busy caseloads and may work across different locations, and as such it was difficult to contact them via telephone. Each offender manager’s caseload frequently changed, with offenders moving on from the area, or returning to prison. Often by the time the offender manager was contacted, they were no longer in contact with those offenders identified from the database. The next task was for a project worker who managed the testing sessions to contact them to arrange an appointment and arrange for their travel. This was important as appointments had to account for availability of researchers such as myself, and radiographers for those taking part in the fMRI procedure. On several occasions there was great difficulty getting into contact with the participant. They were often living somewhat chaotic lives, attending many appointments and did not have mobile telephones. The characteristics of ASPD include impulsivity and irresponsibility, and therefore these issues are to be expected when recruiting from this population. These individuals were also facing the challenge of adapting to life in the community after having served a prison sentence coupled with the demands of their probation order. When this is taken into consideration it is unsurprising that there was a difficulty in recruiting from probation services to central London for two long appointments.

Due to these difficulties with recruitment of those likely to meet criteria for ASPD the focus of the empirical paper shifted somewhat away from that described in the literature review towards incorporating BPD. The early stages of planning my
project took place in the lead up to the revision of the DSM-IV. It seemed likely that there would be significant changes in the way that personality disorder was conceptualised, with a move away from the categorical classification system towards a dimensional approach to the conceptualisation of personality disorders (Krueger, Skodol, Livesley, Shrout & Huang, 2007). This influenced my decision to design my project around exploring personality traits as continuous variables rather than categorical entities. Eventually these changes were rejected. However the appendix to DSM-5 was added, proposing a new synthesised categorical-dimensional approach, aimed to encourage research in this area. This provided support for the relevance of the approach of my empirical paper.

4. Ethical and risk issues

Deliberate self harm and parasuicidal behaviours are common in BPD. Recurrent suicidal threats and gestures have been considered a core feature of the disorder and place great demands on mental health services (Black, Blum, Pfohl & Hale, 2004). A history of suicide attempts has also been found to be related to ASPD diagnosis (Verona, Patrick & Joiner, 2001). Around three quarters of people with a diagnosis will attempt suicide, and 10 will complete (Paris & Zweig-Frank, 2001). Around 80% of women with a diagnosis of BPD have engaged in self mutilating behaviour (Shearer, Peters, Quaytman & Wadman, 1988). One of the key services recruited from was a specialist personality disorder therapy service. This service works exclusively with clients at high risk of self harming and suicidal behaviours. Clinical training and experience had prepared me for working with clients in distress and managing risk of harm. However working with risk issues in a research capacity presented different challenges. The larger study required the administration of the Adult Attachment Interview (AAI, George, Kaplan & Main, 1985). This interview requires participants to describe their earliest memories of attachment relationships and explores their childhood and adolescent experiences.
of these relationships, including interview questions about loss and trauma. Many participants became tearful during this interview, including control participants, many of which had experienced some form of emotional trauma at a young age. Despite the increased risk associated with emotional dysregulation in those diagnosed with BPD, this risk was well managed. A risk protocol was created by the NHS for the larger study. This included advice for researchers on how to support participants in tolerating distress and a handout for participants which included some relaxation strategies, and some contact numbers should they need further support in managing their distress. The added benefit was that all BPD participants were attached to a therapy service and therefore had an assigned clinician who could also provide support and be contact in the event of a risk situation. The control group, however, also exhibited some distress when discussing loss or trauma, understandably. They did not benefit from having an assigned therapist or being attached to a service, although they did receive the same debriefing procedure as clinical participants and were also encouraged to contact identified individuals for further support if required.

5. The construct of “mentalizing”

The empirical paper focuses on the construct of mentalizing, which is defined as a form of social cognition, the ability to understand and form representations of our own and others’ mental states (Fonagy & Luyten, 2009). The mentalizing deficit theory of BPD posits that a failure to form these representations is linked to emotional dysregulation in BPD. This theory forms the basis of mentalization based therapy (MBT) which is now a National Institute of Clinical Excellence (NICE, 2009) recommended treatment for borderline personality disorder, which has been shown to be effective in a randomised control trial in terms of reduced BPD symptomatology at 18 month follow-up (Bateman & Fonagy, 1999). Despite the successes of MBT, research is still attempting to clarify and measure the construct of mentalizing. Mentalizing is a broad and multifaceted construct (Choi-Kain &
Gunderson, 2008), perhaps overlapping with multiple constructs such as social cognition and theory of mind. Another difficulty of refining the measurement of mentalization is that it may not represent a stable trait, more a “mode” that we switch in and out of. It is theorized that in BPD a mentalizing deficit is only apparent once the attachment system has been activated (Fonagy & Bateman, 2006). This presents a challenge to researchers wishing to measure and improve the construct validity of mentalizing ability. Three measures were employed in the current study. The Movie Assessment for Social Cognition (MASC, Dziobeck et al., 2006) requires the participant to answer questions as to the thoughts and feelings of a set of fictional characters. The movie itself is clearly outdated, and the characters speak in German with an English voiceover. It is unlikely that participants emotionally connected to the material, and rather it is a test of mind reading ability and understanding of a storyline. Previous research, (Newbury-Helps, 2011), along with the current study, show that MASC scores are affected by IQ and therefore perhaps this is not a particularly useful measure of mentalizing ability. The Reflective Functioning Questionnaire (RFQ-54, Fonagy & Ghinai, unpublished) is an as yet unpublished scale. The subscales utilised in the empirical paper were advised in personal communication with the author, Peter Fonagy, and require further validation. It was advised that the RFQ-54 is a deficit measure, meaning that the higher the score, the higher the mentalizing deficit. The LRFu subscale measures a deficit in terms of being unsure about the mental states of self and others. The LRFc subscale relates to being certain of the mental states of others. It was unclear at which point being certain of the mental states of others represents a deficit, in other words, how certain is too certain? As one would expect, where one score correlated positively with LRFu, it correlated negatively with LRFc. This intuitively makes sense, as one becomes more uncertain of something, they become less certain of it. Clearly these subscales need further refinement and investigation.
6. Measuring personality

A challenge in the area of personality research as we move towards a dimensional model of personality disorder is measuring non-categorical traits. Researchers are faced with the challenge of developing measures that capture what they are intending to measure. Originally the plan for the current study was to take the number of SCID-II items endorsed for ASPD and BPD, ie those that were assigned a score of three, and totalling the number of items to create a continuous score which would indicate severity of BPD and ASPD pathology. The use of the SCID-II has been criticised for high levels of comorbidity (Verheul, 2006). The problem of comorbidity was noted anecdotally whilst working on the study, when it was noticed that many participants were endorsing criteria that would meet the cutoff point to indicate diagnosable personality disorders. As I learned over the course of this project in my own clinical work with personality disorder, “ticking the boxes” on the SCID-II is not sufficient to indicate a diagnosable personality disorder and a certain level of clinical judgment is necessary. The traits must be pervasive across time, and problematic for the individual across different domains of life, such as relationships and occupation (DSM-5, APA, 2013). It has been highlighted that the SCID-II has taken a system used for assessing more easily categorised axis-I disorders and attempted to apply it to the diagnosis of axis-II personality disorders (Westen, 1997) by asking direct questions to individuals about traits which they may lack the insight or knowledge to describe. When used as a research tool, as in the current study, the SCID-II is especially reliant on direct descriptions of participants in the absence of any corroborative information, clinical interview or previous therapeutic relationship. In the current study researchers were not all trained clinical and many had minimal diagnostic experience, further reducing its’ reliability. Given these issues it was decided that SCID data would not provide a reliable dimensional measure of PD pathology. Fortunately the overarching study included the PAI-BOR in its questionnaire battery. The PAI-BOR is a well validated scale with good
psychometric properties.

The measurement of antisocial traits is highly complex and represents a debate spanning decades and a large body of literature, as explored in detail in the literature review in part one of this thesis. In the past there has been a tendency to use the terms psychopathy and ASPD interchangeably. Increasingly, as concluded by the literature review, evidence suggests that to consider everyone meeting diagnostic criteria for ASPD as a categorical entity, would be, as suggested by Henry & Moffitt (1998) “to compare apples and oranges”. It is crucial that future research is careful to consider what is being measured when we are measuring ASPD pathology, and to avoid the pitfalls of reification of what previous and current research has found to be a diverse group of people.

In terms of measuring ASPD pathology, the intention was to use the ASPD subscale of the PAI. Unfortunately, due to the overarching study being out of my control, this scale was not administered for all subjects. Large amounts of missing data meant that this could not be used. The decision was made to replace it with another scale that was administered as part of the overarching study, the life history of aggression scale (LHA, Coccaro, Berman & Kavoussi, 1997). This consists of three subscales measuring aggression, antisocial behaviour/consequences, and self directed aggression.

7. Clinical implications

The current study adds to the growing body of literature which proposed a move towards a dimensional model of personality disorder (Krueger et al., 2007) in the context of the recent addition of a hybrid dimensional-categorical model of personality disorder in the DSM-5. If this approach were to be adopted this would have implications for the assessment of PD in clinical practise. It has been suggested that a dimensional approach would present clinical challenges due to the lack of cutoff points which are currently used by clinicians to make decisions, for
example about treatment and access to services (Widiger & Trull, 2007). Conversely, it is argued that a dimensional model could provide multiple cut off points which can be used to make different clinical decisions (Trull, 2005). An assessment process has been proposed (Widiger & Trull, 2007) which would involve initial assessment of trait dimensions using self report measures, which would then inform further assessment as to the extent of social and occupational impairment related to these traits. Decisions on the clinical significance of these traits would then be decided using a tool such as the Global Assessment of Functioning (GAF, American Psychiatric Association, 2000). The GAF has been shown to have good reliability even after brief training (Startup, Jackson & Bendix, 2002), whereas existing categorical cutoff points which are used as indices of clinical levels of impairment are somewhat arbitrary. Diagnosis based on examining a variety of trait dimensions could have more clinical utility in terms of providing specific treatment implications.

The study of mentalization is particularly relevant in ASPD given the lack of a robust evidence for effective treatments in this area (McMurran, 2002). Due to a lack of evidence and perhaps a “sense of therapeutic pessimism” (McGauley, Yakeley, Williams & Bateman, 2011) frequent in clinicians towards this client group, those assessed and assigned a categorical diagnosis of ASPD may not be accepted for treatment in personality disorder services. However given that there is growing evidence, supported by the current study that BPD and ASPD may represent shared trait dimensions, such as impulsivity and emotional dysregulation, there is hope that treatments designed for BPD may also be shown to be beneficial for ASPD. Based on the premise that ASPD is a developmental disorder characterised by disrupted attachment, and that mentalizing is also related to attachment, it was hypothesised that MBT may be an effective treatment for ASPD (McGauley et al., 2011). A pilot study of MBT for male outpatients with a diagnosis of ASPD revealed a decrease in severity of aggression towards self and others and symptom related distress. The
current study supports that tenet that those meeting the current criteria for a
diagnosis of ASPD represent a heterogeneous group, and therefore a more
dimensional approach to assessment would more specifically inform treatment
pathways. The current research further supports psychopathy as a variable trait
dimension. Historically treatment outcomes are poorer with those at the low end of
the anxious and emotional trait dimensions, who engage in less reactive aggression,
may benefit less from treatment than others. This further highlights the importance
of a formulation based, dimensional approach to clinical assessment rather than
relying on categorical diagnoses.

8. Future research

Research in the field of mentalization presents a challenge in that it is a
broad and multifaceted concept, potentially consisting of several overlapping
constructs such as theory of mind and social cognition. Measures of mentalizing are
still in their infancy and many more studies are required in order to refine and
empirically validate measures which are providing promise, such as the RFQ-54.
Researchers should proceed with caution around certain measures which are
heavily influenced by intellectual ability, such as the MASC. The mentalizing theory
of BPD posits that the failure to mentalize is present only when the attachment
system is activated (Fonagy & Bateman, 2006). In order to test this hypothesis
research paradigms are required, in which mentalizing measures are administered
prior to and after activation of the attachment system. This requires careful
consideration of ethical and risk factors. In terms of ASPD, the results described in
the empirical study require replication with a larger sample size in order to further
clarify how mentalization deficits may or may not manifest in those with ASPD. MBT
may represent a promising treatment modality for ASPD, however much more
robust empirical research is required in order to further establish the possible role of
mentalizing in ASPD. The current study did not account for attachment status, as
coding of the AAI data collected is yet to be completed. Given the hypothesis presented by McGauley et al. (2011), that mentalizing deficits in ASPD may be a function of early attachment relationships, it will be important for future research into the link between ASPD pathology and mentalizing to take attachment into consideration.

9. Summary

Overall this project adds to a body of literature which represents a shift in the approach to assessment and treatment of personality disorder, an area which historically has been surrounded by some stigma amongst mental health professionals (Lewis & Appleby, 1988). It is hoped that research in this area continues to contribute towards promising treatments such as MBT. Increased understanding of the aetiology of ASPD and BPD may foster a more hopeful attitude in clinicians towards the treatment of personality disorder.
References


Appendices

**Part one: Literature Review**
- Appendix A1: Quality Appraisal Tool
- Appendix A2: Results of Quality Appraisal

**Part two: Empirical Paper**
- Appendix B1: Ethical Approval Letter
- Appendix B2: Participant Information Sheet
- Appendix B3: Participant Consent Form
- Appendix B4: Participant Debrief Form
### Appendix A1: Quality appraisal Tool

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<thead>
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<td>2  Study design evident and appropriate?</td>
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<tr>
<td>3  Method of subject/comparison group selection or source of information/input variables described and appropriate?</td>
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<td>8  Outcome and (if applicable) exposure measure(s) well defined and robust to measurement / misclassification bias? Means of assessment reported?</td>
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<td>9  Sample size appropriate?</td>
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<td>11 Some estimate of variance is reported for the main results?</td>
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<td>13 Results reported in sufficient detail?</td>
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<td>14 Conclusions supported by the results?</td>
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### Appendix A2: Results of Quality Appraisal

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<tr>
<td>Cox <em>et al.</em> (2013)</td>
<td>22</td>
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<tr>
<td>Magyar <em>et al.</em> (2013)</td>
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<tr>
<td>Coid &amp; Ullrich (2010)</td>
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<td>Kosson <em>et al.</em> (2006)</td>
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<td>Verona <em>et al.</em> (2012)</td>
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<tr>
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<tr>
<td>Zeier <em>et al.</em> (2012)</td>
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Appendix B1: Ethical Approval Letter

Dear [Name]

Study title: Probing Social Exchanges – A Computational Neuroscience Approach to the Understanding of Borderline and Anti-Social Personality Disorder

REC reference: 12/CA/0593

Thank you for your letter of 26 September 2012, responding to the Committee’s request for further information on the above research and submitting revised documentation.

The further information was considered by a sub-committee of the REC at a meeting held on 36 October 2012. A list of the sub-committee members is attached.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised, subject to the conditions specified below.

Ethical review of research sites

NHS sites:
The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHSHSC R&D office prior to the start of the study (see “Conditions of the favourable opinion” below).

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission (“R&D approval”) should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements.

Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at http://www.risforum.nhs.uk.

Where a NHS organisation’s role in the study is limited to identifying and referring potential participants to research sites (“patient identification centre”), guidance should be sought from the...
K&O office on the information it requires to give permission for this activity.

For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.

Sponsors are not required to notify the Committee of approvals from host organisations:

- The Clinical/Probation Service Information Sheet, page two paragraph one, has the phrase "which is a psychiatric interview" twice; one of these instances should be removed;
- The word "however" should be removed from the start of the first paragraph of page three under "What are the possible disadvantages and risks of taking part?";
- The second paragraph of the same section is the same sentence repeated twice, and one of these instances should be removed;
- The Healthy Volunteers Information page three, the word "however" should be removed from the start of the first paragraph of page three under "What are the possible disadvantages and risks of taking part?"

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study on the initiation at a particular site (as applicable).

You should notify the REC in writing once all conditions have been met (except for site approval from host organisations) and provide copies of any revised documentation with updated version numbers. Confirmation should also be provided to host organisations together with relevant documentation.

**Approved documents**

The list of documents reviewed and approved by the Committee is as follows:

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<thead>
<tr>
<th>Document</th>
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<th>Date</th>
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<td>Evidence of insurance or indemnity</td>
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<td>GPC/Consultant Information Sheets</td>
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<tr>
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<td>Other: Consent to contact form</td>
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<td>Participant Information Sheet: Genetics</td>
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</table>
Participant Information Sheet: Clinical / Probation service

REC approval: 21 August 2012

Response to Request for Further Information: 25 September 2012

Statement of normalisation
The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and operates fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Reporting requirements
The attached document "After ethical review – guidance for researchers" gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Notification of serious breaches of the protocol
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

Feedback
You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

Further information is available at National Research Ethics Service website > After Review

121W4823 Please quote this number on all correspondence

With the Committee's best wishes for the success of this project

Yours sincerely

Chairman

Email: corinna.soadd@wales.nhs.uk

Enclosures: List of names and professions of members who were present at the meeting and those who submitted written comments

*After ethical review – guidance for researchers*

Copy to:
REC for Wales

Attendance at Sub-Committee of the REC meeting on 09 October 2013

Committee Members:

<table>
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<tr>
<th>Name</th>
<th>Profession</th>
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<tr>
<td>Alternate Vice Chairman</td>
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<tr>
<td>/ Hospital Consultant</td>
<td>(Cardiologist)</td>
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<tr>
<td>Arthritis Research UK</td>
<td>Yes</td>
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<tr>
<td>BBC Research Coordinator</td>
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<tr>
<td>Chairman / Statistician</td>
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<td>Vice Chairman / Clinical</td>
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<tr>
<td>Physiologist</td>
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Also in attendance:

<table>
<thead>
<tr>
<th>Name</th>
<th>Position (or reason for attending)</th>
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<tr>
<td></td>
<td>Co-ordinator</td>
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Appendix B2: Participant Information Sheet

Understanding the Social Brain in Healthy Volunteers and People with Psychological Difficulties.

This study has been approved by the Research Ethics Committee for Wales (Project ID Number): 12/WA/0283.

We would like to invite you to participate in this research project.

You are being invited to take part in a research study. You should only participate if you want to. Before you decide whether to take part, this sheet will give you some more information about why the study is being carried out, what you would be asked to do if you decide to take part, and how the study will be conducted. Please take some time to read this sheet, and to discuss it with other people if you wish. You are also very welcome to ask any further questions about the study, or if you find anything on this sheet unclear.

Why is this study being done?

With the proposed project we plan to investigate the brain activation patterns of people suffering from personality disorders (both in adults and adolescents) and compare them with healthy control participants. Only little is known about the neurobiology of Borderline and Antisocial Personality Disorders. Our study design will address some of these. This will hopefully allow us to gain a better understanding of the disorders and to develop more informed and effective treatments from which clients will benefit.

Why have you been invited to take part?

You have been invited to take part in the study because you have recently been assessed at one of the clinical or probation services currently collaboration with the research team.

Do I have to take part?

No. Taking part in the study is entirely voluntary. It is your choice whether or not you would like to participate. Deciding not to take part in the study will not affect the care you receive from services either now or in the future. If you do decide to participate, you will be given this information sheet to keep, and you will later be asked to sign a consent form stating that you wish to take part. If you do give consent to take part in the study, you are still free to leave the study at any point, without giving a reason. This will not affect the care you are currently receiving, or will receive in the future. If you leave, any information that we have already collected from you will be destroyed.
Appendix B3: Participant Consent Form

Please complete this form after you have read the Information Sheet and/or listened to an explanation about the research.

Project Title:

Understanding the Social Brain in Healthy Volunteers and People with Psychological Difficulties.

This study has been approved by the Research Ethics Committee for Wales (Project ID): 12/WA/0283.

Thank you for your interest in taking part in this research. Before you agree to take part, the person organising the research must explain the project to you.

If you have any questions arising from the Information Sheet or explanation already given to you, please ask the researcher before you to decide whether to join in. You will be given a copy of this Consent Form to keep and refer to at any time.

Participant’s Statement

I

- have read the notes written above and the Information Sheet, and understand what the study involves. I am also aware that I can consent to certain aspects of the study in order to participate in them whereas I can withhold my consent for others parts.
- understand that if I decide at any time that I no longer wish to take part in this project, I can notify the researchers involved and withdraw immediately.
- consent to the processing of my personal information for the purposes of this research study.
- understand that such information will be treated as strictly confidential and handled in accordance with the provisions of the Data Protection Act 1998.
- understand that some of the MRI data will be transferred for analysis to the Principal Investigator’s second laboratory at Virginia Tech University in the USA and will therefore no longer be subject to EEA data protection laws but that this data will be anonymised and no identifiable personal information will be shared or transferred.
- agree that the research project named above has been explained to me to my satisfaction and I agree to take part in this study.
- I agree that my non-personal research data may be used by others for future research. I am assured that the confidentiality of my personal data will be upheld through the removal of identifiers.
- I understand that part of my participation will be audio-recorded (the interviews) and I consent to the anonymous use of this material as part of the project.
- I agree to be contacted in the future by UCL researchers who would like to invite me to participate in follow-up studies.
I understand that the information I have submitted will be published as a report and that I can request a copy. Confidentiality and anonymity will be maintained and it will not be possible to identify me from any publications.

I agree that the research team might re-contact me in case that additional data has to be obtained or for follow-up studies.

Please initial the statements below if you agree with them:
Initial here

I agree to take part in the general part of the PD-CPA study as outlined in the information Sheet and to all points listed above. (a separate consent for the MRI, tattoo component, and genetics component follows below).

I agree to the audio recording of interviews and I consent to the anonymous use of this material as part of the project.

I agree that some of the study data will be shared with the collaborating laboratory at Virginia Tech University in the USA.

I understand that relevant sections of medical and or probation notes and data collected during my clinical assessment and during the study from me, may be looked at by individuals from the PD-CPA research team, my clinician or from the NHS Trust, where it is relevant to our taking part in this research. I give permission for these individuals to have access to my records.

I agree that the PD-CPA research team can contact me about coming in for up to two follow-up sessions over the next three years.

I agree that I can be contacted after the end of this study about possible future research and follow-up with PD-CPA and related groups.

I agree that my GP can be told that I am participating in this study.

GP’s name: _____________________ Surgery: _________________________
Address: ______________________________________________________

MRI and Cognition:

I agree to have an MRI scan and I understand what will happen in the scan.

I have had an MRI safety check and I am confident that there is no reason why I can’t have a scan, such as a recent operation.

I agree that my test results can be held by the Wellcome Trust and shared with other research groups, and I understand that this data will be anonymous.
and not contain any personal information.

**Genetics:**

You do not have to agree to provide blood or saliva samples to take part in the research. You do not have to agree that any samples you do give can be stored for future testing. By giving a sample, you consent to be contacted by BioResource about the possibility of joining their panel, but you are under no obligation to join BioResource.

I agree to give a sample of **blood and saliva** (delete as appropriate) for medical research and for details about me and any samples I provide to be kept on a secure database. I agree that BioResource, the study collaborator on genetics, can store my samples and can contact me to invite me to join their panel.

I agree that the samples and information I provide can be stored for use in future medical research, subject to ethical approval.

I understand that I will not benefit financially if my samples are used in research leading to a new treatment or medical test being developed.

In the unlikely event that an abnormality is picked up from tests carried out on my sample, I agree to be informed, and with my consent my GP can be told.

**Thank you for your help.**

By completing and returning this form, you are giving us your consent that the personal information you provide will be treated as strictly confidential and handled in accordance with the provisions of the Data Protection Act 1998.
Appendix B4: Participant Debrief Form

Thank you for taking part in our study, we appreciate that you gave up your time to take part and hope that you found it interesting.

Summary of the Research Project

The aim of our study is to understand how mind and brain work in order to better understand patients with psychological difficulties. We hope that this will have an impact on the development of specific treatment interventions.

Most of our tasks are designed to look at how we think about ourselves and others (called "mentalisation"), how we regulate our emotions, value co-operation or experience close relationships and how problems can sometimes develop in these relationships.

Getting a better sense of the different strategies that people apply in these areas can help us understand more about when people experience mental health problems that can lead them to find certain social interactions and situations challenging. We hope to use these findings so that treatments can be tailored to help improve the domains where a patient’s difficulties may lie.

We are also interested in how someone’s experiences in childhood and his or her parenting at that time impact on the performances in the tasks and the functioning of the brain areas that underpin them. For instance, the long interview can tell us more about the quality of your bonding with parents.

Some of the topics discussed in the course of the study may have brought about thoughts or feelings which you had not previously considered or may have made you recall memories which could be perceived as distressing or lead you to feel tense or ruminate on thoughts. Therefore, we have provided some exercises at the back of this sheet which may help you to cope with any such feelings which you may experience.

What to do if you continue to feel concerned

If you continue to feel concerned after taking part in the study it may be useful to talk to a family member, a friend or your GP. Your Lead Clinician (care co-ordinator) or Probation Worker will also be able to support you, if you have one.

In addition to this support there is also free and confidential advice provided by the Mental Health charity Mind which can be found on their website: http://www.mind.org.uk/ or by calling their advice line 0300 123 3393.

If you feel at immediate risk do not hesitate to contact Dr (details overleaf).
Contact Details

If you still have concerns or wish to contact the research team to discuss any of the information further or any concerns you have about the study, then please do so by getting in touch with the members of the research team listed below:

Thank you very much for taking the time to read this information sheet.
Relaxation Exercises

Progressive Muscle Relaxation Technique
{Pause between instructions}

Begin by finding a comfortable position either sitting or lying down in a location where you will not be interrupted. Allow your attention to focus only on your body. If you begin to notice your mind wandering, bring it back to the muscle you are working on.

Take a deep breath through your abdomen, hold for a few seconds, and exhale slowly. Again, as you breathe notice your stomach rising and your lungs filling with air.

As you exhale, imagine the tension in your body being released and flowing out of your body. And again inhale…..and exhale. Feel your body already relaxing.

As you go through each step, remember to keep breathing.

Now let’s begin. Tighten the muscles in your forehead by raising your eyebrows as high as you can. Hold for about five seconds. And abruptly release feeling that tension fall away.

Now smile widely, feeling your mouth and cheeks tense. Hold for about 5 seconds, and release, appreciating the softness in your face.

Next, tighten your eye muscles by squinting your eyelids tightly shut. Hold for about 5 seconds, and release.

Gently pull your head back as if to look at the ceiling. Hold for about 5 seconds, and release, feeling the tension melting away.

Now feel the weight of your relaxed head and neck sink.

Breath in…and out.

In…and out.

Let go of all the stress

In…and out.
Now, tightly, but without straining, clench your fists and hold this position until I say stop. Hold for about 5 seconds, and release.

Now, flex your biceps. Feel that buildup of tension. You may even visualize that muscle tightening.

Hold for about 5 seconds, and release, enjoying that feeling of limpness.

Breath in...and out.

Now tighten your triceps by extending your arms out and locking your elbows. Hold for about 5 seconds, and release.

Now lift your shoulders up as if they could touch your ears. Hold for about 5 seconds, and quickly release, feeling their heaviness.

Tense your upper back by pulling your shoulders back trying to make your shoulder blades touch.

Hold for about 5 seconds, and release.

Tighten your chest by taking a deep breath in, hold for about 5 seconds, and exhale, blowing out all the tension.

Now tighten the muscles in your stomach by sucking in. Hold for about 5 seconds, and release.

Gently arch your lower back. Hold for about 5 seconds, relax.

Feel the limpness in your upper body letting go of the tension and stress, hold for about 5 seconds, and relax.

Tighten your buttocks. Hold for about 5 seconds…, release, imagine your hips falling loose.

Tighten your thighs by pressing your knees together, as if you were holding a penny between them.

Hold for about 5 seconds…and release.

Now flex your feet, pulling your toes towards you and feeling the tension in your calves. Hold for about 5 seconds, and relax, feel the weight of your legs sinking down.
Curl your toes under tensing your feet. Hold for about 5 seconds, release.

Now imagine a wave of relaxation slowly spreading through your body beginning at your head and going all the way down to your feet.

Feel the weight of your relaxed body.

Breathe in…and out…in…out….in…out.

**Mindfulness Exercise**

*Read the following instructions*

Sit comfortably, with your eyes closed and your spine reasonably straight.

Bring your attention to your breathing.

Imagine that you have a balloon in your tummy. Every time you breathe in, the balloon inflates. Each time you breathe out, the balloon deflates. Notice the sensations in your abdomen as the balloon inflates and deflates. Your abdomen rising with the in-breath, and falling with the out-breath.

Thoughts will come into your mind, and that’s okay, because that’s just what the human mind does. Simply notice those thoughts, then bring your attention back to your breathing.

Likewise, you can notice sounds, physical feelings, and emotions, and again, just bring your attention back to your breathing.

You don’t have to follow those thoughts or feelings, don’t judge yourself for having them, or analyse them in any way. It’s okay for the thoughts to be there. Just notice those thoughts, and let them drift on by, bringing your attention back to your breathing.

Whenever you notice that your attention has drifted off and is becoming caught up in thoughts or feelings, simply note that the attention has drifted, and then gently bring the attention back to your breathing.

It's okay and natural for thoughts to enter into your awareness, and for your attention to follow them. No matter how many times this happens, just keep bringing your attention back to your breathing.