Executive Function and Theory of Mind in Borderline Personality Disorder.

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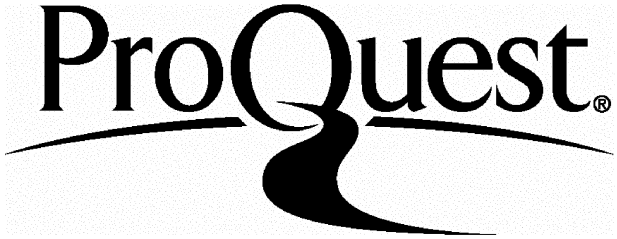

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Abstract

Recent years have seen an increasing interest in the question of whether there may be organic as well as psychosocial factors that are important in accounting for the aetiology and symptoms of Borderline Personality Disorder (BPD). Various brain abnormalities have been reported or proposed in the disorder, particularly involving the frontal lobes, the hippocampus and the amygdala. However, the overall body of previous research has shown only mixed evidence of neuropsychological impairments associated with these brain regions in individuals with BPD. The present study investigates two cognitive functions that have been linked to the frontal lobes: executive functioning and theory of mind. Theoretical and empirical evidence from a number of sources suggests that deficits in these functions may help to account for some of the symptoms of BPD, including interpersonal difficulties, poor affect regulation, dissociation, and impulsivity.

In the present study, a group of participants diagnosed with BPD (n=17) was compared to healthy controls (n=13) on a variety of executive function and theory of mind measures. These included two executive function tasks which were designed to be open-ended and to mimic real-life situations (Predicaments test, Channon and Crawford, 1999; Modified Six Elements test, Wilson et al., 1996). Such tasks may have better ecological validity than the more abstract measures of executive function which have previously been used in studies with the BPD population. The theory of mind tasks were chosen to be developmentally advanced, to enable subtle deficits in theory of mind ability to be detected (Story Comprehension test, Channon and Crawford, 2000; Happe Strange Stories test, Happe, 1994).

The BPD group performed significantly worse than controls on one of the theory of mind measures (Story Comprehension test). There was no difference between the groups on the other theory of mind task (Happe Strange Stories test), but it is argued that this result may be due to the task measuring theory of mind skills at a lower level. The results of the study indicate that there may be a subtle theory of mind deficit in BPD, but

this probably applies only to a proportion of individuals with the diagnosis. However further analysis underlined the possibility that this poorer theory of mind performance may be attributable to co-morbid depression rather than BPD diagnosis, and further research with a depressed control group is needed to investigate this issue properly. The theory of mind findings of the study are discussed in relation to the reflective function hypothesis of BPD. In particular, the reflective function hypothesis suggests that a deficit in theory of mind should be largely restricted to contexts which are relevant to that person's attachment history, but this study raises the possibility that the deficit may also apply to more neutral contexts.

The results of the executive function measures yielded only tentative evidence for executive dysfunction BPD, a finding that fits in with the overall picture of inconsistency from previous studies. A number of possible reasons are discussed for the lack of clear findings in this area, including the small sample sizes of studies, the possibility that only sub-groups of individuals with BPD have executive difficulties, the possibility that executive dysfunction is related to emotional arousal, and the possibility that BPD is not characterised by executive dysfunction at all. Suggestions for future research into both executive functioning and theory of mind in BPD are proposed.

Chapter 1: Introduction

1.1 The nature of Borderline Personality Disorder

1.1.1 Description of Borderline Personality Disorder.

Stern (1938) used the term "borderline" to describe a particular group of patients who, amongst other characteristics, were prone to narcissism, psychological rigidity, hypersensitivity, negative therapeutic reactions, deep insecurity, and difficulties with reality testing in interpersonal contexts. Many of these characteristics seem to map on to the modern concept of Borderline Personality Disorder, but it was not until the introduction of the multiaxial classification system of DSM-III (American Psychiatric Association, 1980) that the term gained formal nosological recognition along with the other personality disorders and learning disabilities in Axis-II.

Although DSM-III was ostensibly atheoretical, some (e.g. Silk, 2000) have argued that the split between Axis I and II disorders involved a tacit assumption that, while Axis I disorders were coming increasingly under the realm of biological psychiatry, Axis II could remain the territory of the psychoanalytic community, with psychosocial causation being seen as primary. This assumption has started to be questioned in recent years, with research increasingly addressing the question of whether there may be organic as well as psychosocial factors in the aetiology of personality disorders. The present study can be seen as part of this trend, with its hypotheses drawn from both psychosocial and organic models of Borderline Personality Disorder.

The most recent edition of DSM (DSM-IV, American Psychiatric Association, 1994) describes personality disorder as an "enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual's culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over

time, and leads to distress or impairment."

With respect to Borderline Personality Disorder, DSM-IV provides the following diagnostic criteria:

"A pervasive pattern of instability of interpersonal relationships, self image, and affects, and marked impulsivity beginning by early adulthood and present in a variety of contexts, as indicated by at least five of the following:

(i) Frantic efforts to avoid real or imagined abandonment

(ii) A pattern of unstable and intense interpersonal relationships characterised by alternating extremes of idealisation and devaluation.

(iii) Identity disturbance: markedly and persistently unstable self-image or sense of self.

(iv) Impulsivity in at least two areas that are potentially self-damaging.

(v) Recurrent suicidal behaviour, gestures, threats or self-mutilating behaviour.

(vi) Affective instability due to a marked reactivity of mood (e.g. intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days).

(vii) Chronic feelings of emptiness.

(viii) Inappropriate, intense anger, or difficulty controlling anger (e.g. frequent displays of temper, constant anger, recurrent physical fights).

(ix) Transient, stress related paranoid ideation or severe dissociative symptoms.

1.1.2 Epidemiology and natural course (figures from DSM-IV, APA, 1994)

The prevalence of BPD is estimated at 2% of the general population, 10% among outpatient mental health patients, and 20% in psychiatric inpatients. It is one of the most common personality disorders, with prevalence in specialist personality disorder clinics of 30 to 60%. It is thought that around 75% of those diagnosed with BPD are female. Physical and sexual abuse, neglect, conflict, and early parental loss or separation are common in the childhood histories of those with BPD. Although there is considerable variability in the course of BPD, the most typical pattern is of chronic instability in early adulthood, with the level of impairment and disruption gradually reducing as individuals reach their 30s and 40s, and beyond.

1.1.3 Treatment issues.

Individuals with BPD can be amongst the most challenging groups for mental health professionals to work with, with evidence that patients with BPD are more likely to be "dreaded" by clinicians than patients with other diagnoses (Bongar et al., 1991). Indeed, some have argued that the diagnosis of BPD has acquired a stigma within the mental health system (Nehls, 1998), and that clinicians are less likely to respond empathically to individuals if they have a diagnosis of BPD (Fraser and Gallop, 1993).

Therapeutic progress with patients with BPD can be difficult to achieve, and Layden et al., (1993) describe a number of common features of therapy with BPD which can contribute to this difficulty. These include poor insight into how difficulties have arisen, deficient awareness of how the patient's own behaviour contributes to their interpersonal difficulties, poor motivation to change personality problems, an extreme all-or-nothing thinking style, and the missing of numerous sessions. Fonagy (1999) adds that the associations of BPD patients can be hard to follow in therapy as they often fail to take into account the mental states of others, and that this failure of "mentalisation" can also

manifest itself in cruelty to others or violence.

Several approaches for the treatment of BPD have been developed specifically for the disorder, two of which have been subjected to randomised control trials: Dialectical Behaviour Therapy (Linehan, 1993; Shearin and Linehan, 1994), and Psychoanalytically Oriented Partial Hospitalisation (Bateman and Fonagy, 1999). Dialectical Behaviour Therapy is a manualised treatment in which patients receive individual psychotherapy and group-based skills training. The overall aim of the treatment is to enable patients to learn skills that help them to manage the emotional dysregulation which is characteristic of BPD. Psychoanalytically Oriented Partial Hospitalisation, described by Bateman and Fonagy (1999), is an approach which conceives of BPD as a disorder of attachment, separation tolerance and reflective functioning (the capacity to understand one's own and others' behaviour in mental state terms: Fonagy, 1999). Treatment consists of individual and group psychoanalytic psychotherapy, and group expressive therapy. Both treatment approaches were found to produce good outcomes on a number of measures, including frequency of suicidal behaviour, number of days in hospital and social adjustment.

1.1.4 Validity of the Borderline Personality Disorder diagnosis.

Some have questioned the diagnostic validity of BPD, arguing, for example, that borderline states should be viewed as variants of Axis I disorders (Akiskal, 1994). Tyrer (1994) notes that there are a number of features of BPD which argue against its status as a personality disorder: for example its relatively good long-term prognosis marks it out as being at odds with paradigmatic characterisation of a personality disorder being stable in the long-term. Also Tyrer et al., (1988) reported that when the Personality Assessment Schedule (Tyrer et al., 1988) was administered to a large group of patients, no borderline cluster emerged from the data. Tyrer (1994) notes that comorbidity of BPD with affective disorders is much more common than comorbidity of BPD with other personality disorders (e.g. Fyer et al., 1998), and suggests that this supports the notion

that individuals with BPD should be considered to be suffering from some form of primary affective disorder.

Against these arguments, Gunderson (1994) asserts that there are significant phenomenological differences between the affective experiences of individuals with BPD and those with a primary affective disorder (for example emptiness and anger are more typical of BPD than unipolar depression, whereas guilt and a preoccupation with failure are more typical of unipolar depression than BPD). Gunderson (1994) notes that studies of the responsiveness of BPD to pharmacological treatment do not show any consistent pattern of response to anti-depressants and mood stabilisers, and argues that this counts against suggestions of a strong link with affective disorders. Also Maffei and Fossati (1999) conducted a study in which structured interviews were administered to a large sample of patients with a variety of diagnoses, from which they concluded that DSM-IV criteria for BPD generally had good sensitivity and specificity, and correlated better with each other than with diagnostic criteria of other personality disorders.

In summary, the validity of the BPD diagnosis remains a contested issue. Arguments against its current status as a personality disorder include its relatively good long-term prognosis and the high levels of comorbidity with mood disorders. However, there are also arguments in favour of BPD's diagnostic validity, such as phenomenological differences in affective experience between BPD and primary mood disorders, and its response to pharmacological treatment.

1.1.5 Models of the aetiology and symptoms of BPD.

A large variety of models of the aetiology and symptoms of BPD have been proposed, many of which overlap to some extent. A selection of these will now be briefly presented.

(i) Psychodynamic models.

A number of models of BPD have been proposed within the psychodynamic field. These include suggestions that a derailment in the separation-individuation process is important (e.g. Maher and Kaplan, 1966), or that the borderline condition is a defence against powerful and unacceptable aggressive drives (Kernberg, 1976). One of these psychodynamic models, the reflective functioning model of BPD, will be described here in more detail as it is particularly relevant to the theory of mind hypothesis of this study. Fonagy et al. (2000) propose that poor reflective functioning is a core deficit in BPD, which can help to explain many of the symptoms of the disorder, including unstable sense of self, impulsivity, emotional instability, suicidality, and chronic feelings of emptiness. It is proposed that poor reflective functioning in BPD is a result of an ingrained defensive inhibition of mentalising, which develops as a result of childhood maltreatment or neglect occurring the absence of a secure attachment relationship. This defense originally occurs as a way of protecting the child from conceiving of the content of the abusive or neglectful caregiver's mind, and is a pattern which persists into adult life. However Fonagy et al. (2000) proposes a key qualification of this model: that poor reflective capacity in BPD is restricted to certain problematic internal working models and attachment contexts, and that reflective capacity is retained in some form at other times. This suggestion helps to account for anecdotal evidence that individuals with BPD at times appear to have an acute awareness of the mental states of others and can sometimes display a high level of apparent competence.

(ii) Cognitive-behavioural models.

Layden et al., (1993) proposes that early experiences of abuse or neglect lead to the formation of maladaptive schemas, which are defined as "...fundamental core beliefs: the basic rules that an individual uses to organise his or her perceptions of the world, self, and future, and to adapt to life's challenges." (Layden et al., 1993, p7). These maladaptive schemas result in the severe disturbances of interpersonal relationships,

affective regulation, and cognitive functioning seen in BPD patients. Layden et al. (1993) comment that individuals with BPD can be especially difficult to treat as they tend to hold numerous, often contradictory, schemas. For example, a patient might simultaneously hold schemas of dependence and mistrust, resulting in a continual dilemma about whether to seek closeness with others (thus putting themselves in a position where they could be vulnerable to maltreatment), or whether to withdraw from interpersonal contact (and risk feeling unable to cope alone).

(iii) Family based aetiologies.

Several researchers have proposed that a pathogenic family background may be important in the development of BPD. For example, Golomb et al. (1994) suggests that a families of BPD patients are typically chaotic, and that mothers of individuals with BPD tend to treat their offspring as a means of satisfying their own needs. Berzirganian et al. (1993) reports that a combination of maternal inconsistency and over-involvement predicted the development of BPD in a community sample of 776 adolescents. Also Linehan (1993) (in the context of the Biosocial model: see below) proposes that individuals with BPD often come from "invalidating" family environments, where the developing child's emotional reactions are labelled as wrong or invalid in some way.

(iv) Genetic predisposition.

A handful of studies have investigated the possibility of a genetic component to BPD. Torgersen (1984) performed a small twin study which suggested that genes have little influence on the development of BPD, but Torgersen (2000) reports preliminary results of a larger twin study which indicates there may be a much greater heritable component than previously thought. In addition, Torgersen (2000) points out that there is evidence that many of the personality variables associated with BPD diagnosis (such as neuroticism and affective lability) have been shown to have genetic components.

(v) Brain abnormality models.

There is a growing body of evidence from physiological and neuropsychological studies indicating that there may be brain abnormalities in BPD which might be linked to the clinical presentation of the disorder. For example Lyoo et al. (1998) report evidence that individuals with BPD have smaller frontal lobes compared to matched controls, and Judd and Ruff (1993) report that verbal and non-verbal memory are impaired in BPD. There have also been suggestions that neuro-chemical abnormalities may underlie some symptoms in BPD: for example Gurvits et al., (2000) hypothesise that impairment of seratonergic function may be linked to impulsivity in the disorder, and that acetylcholine abnormalities may relate to affective instability.

(vi) Biosocial model

Linehan's (1993) Biosocial model is an attempt to bring together biological and psycho-social factors into a coherent integrative account of BPD. The model proposes that BPD is a disorder of emotional dysregulation, the aetiology of which is influenced by the combination of a biological vulnerability to emotional reactivity and an early environment which does not allow the child to learn skills to regulate affect. As mentioned above, Linehan proposes that the family environments of individuals who develop BPD are typically invalidating. Many of the symptoms of BPD (such as suicidal and self-harming behaviour) are seen as maladaptive ways of coping with emotional dysregulation.

Summary

There have been numerous models proposed to account for the aetiology and symptoms of BPD, and this diversity reflects the historical shift from the disorder being viewed as exclusively psychosocial in aetiology, to recent views which suggest that organic factors may be also important. The brain abnormality hypothesis of BPD is the most relevant from the point of view of this study, as it forms the background from which

the major research questions of the study are derived. The issue of brain abnormality in BPD will now be considered in more detail.

1.2 Evidence for brain abnormality in BPD

As outlined above, some researchers have proposed that there may be brain abnormalities in individuals with BPD which go some way towards accounting for the clinical presentation of the disorder. Many of these proposed brain abnormalities are within the limbic system (which includes the hippocampus and the amygdala) and the frontal cortex; areas that have been strongly implicated in the regulation of emotion and personality (Teicher et al., 1994). The possibility of abnormalities in these structures therefore links in well with the affective and interpersonal abnormalities seen in the clinical syndrome of BPD.

1.2.1 General neurological abnormalities.

It is thought that "neurological soft signs" (e.g. involuntary movements, apraxias, difficulty with rapid movements) can be associated with general neurological damage when there is no specific neurological disease (e.g. Shaffer et al., 1985). Stein et al., (1993) reported that a sample of 28 patients with BPD had increased left-sided neurological soft signs compared to control subjects, suggesting non-specific neurological abnormalities in the disorder. Other studies have similarly found an increase in neurological soft-signs in BPD (e.g. Gardener et al., 1987).

EEG studies have also indicated neurological abnormality in BPD: for example, De La Fuente et al. (1998) reported that 40% of a group of 20 patients with BPD had diffuse abnormalities in their EEGs (no focal abnormalities were identified), again suggesting non-specific neurological dysfunction. Other studies have also reported a raised prevalence of abnormal EEG in BPD (e.g. Cowdry et al., 1985; Snyder and Pitts, 1984).

In summary, a number of neurological and EEG studies have suggested that there may be underlying organic abnormalities in BPD, and this has prompted research into whether there may be more specific brain regions and functions which are impaired in the disorder.

1.2.2 Frontal lobe abnormalities.

The frontal lobes are large and complex structures, which have been associated with a great variety of cognitive, motor and affective processes. The most posterior parts of this region are the sensory, motor and pre-motor cortices, which are involved in sensation, execution of movement, and the integration of motor skills into complex sequences. The more anterior section is the pre-frontal cortex. Lesions in this area may affect attention, working memory, the ability to inhibit pre-potent responses, the regulation of emotions and social behaviour, and the ability to appropriately initiate and terminate behaviours (Lezak, 1995). Other functions, such as the retrieval of autobiographical memories (Conway et al., 1999), and theory of mind (Stone et al., 1998) have also been associated with pre-frontal areas.

The pre-frontal region is of particular interest in the search for an organic basis for BPD, as lesions in this region have been known to produce personality and emotional changes. For example, Blair and Cipolotti (2000) report a single case of acquired anti-social personality disorder following right frontal lobe damage. Changes in subjective emotional experience and emotional expression following frontal lobe damage have also been well documented (Stuss et al., 1992).

The best direct evidence of structural frontal lobe abnormalities in BPD come from Lyoo et al. (1998) who compared the magnetic resonance imaging (MRI) scans of 25 BPD subjects to 25 age and gender matched healthy controls. It was found that BPD subjects had significantly smaller frontal lobes (a mean difference in volume of 6.2%). Previous structural brain scanning studies had found little or no evidence of abnormalities (Snyder

et al., 1983; Lucas et al., 1989). However, these used computerised tomography (CT) scans which are less likely to detect abnormalities than MRI scans (Na et al., 1991). In addition, the study by Lyoo et al. (1998) employed stricter criteria for diagnosis of the BPD subjects and exclusion of co-morbid diagnoses, so it remains the most reliable structural brain scanning study to date.

There have also been studies employing positron emission tomography (PET) scans, which have also produced evidence of frontal lobe abnormalities in BPD. PET scans measure the rate of metabolism (and thus brain activity) in different regions of the brain. Goyer et al. (1994) reported that a sample of 17 BPD subjects showed a significant decrease in metabolism in their frontal lobes when compared to controls. Also De La Fuente et al. (1994) found that BPD subjects (n=10) displayed a decrease in metabolism in the prefrontal and pre-motor areas.

These findings of structural and metabolic abnormalities in the frontal lobes in BPD patients would suggest that one may find corresponding impairments in cognitive functions that are associated with the frontal lobes. A number of studies have investigated executive functioning[1] in BPD, with mixed results. Table 1 summarises the results of these studies.

All of the studies used healthy volunteer control groups, except van Reekum et al. (1993) who compared the results to normative data. Tests used include the Wisconsin Card Sorting Test (Grant and Berg, 1948), the Stroop colour-word test (Stroop, 1935) the Trail-making test (Reitan, 1958) and the Porteus mazes (Porteus, 1955).

[1]Executive functioning is a term which refers to a collection of cognitive processess which enable an individual to adapt to novelty: see section 1.5.1 for further discussion.

Table 1. Summary of studies investigating executive function in BPD

Study	Group size	Tests	Result
Swirsky-Sacchetti et al. (1993)	10	WCST[a]	No impairment
		Stroop	Impaired
Carpenter et al. (1993)	17	Stroop	Impaired
		Trails B	Impaired
Judd and Ruff (1993)	25	Stroop	No Impairment
Sprock et al., (2000)	18	Stroop	No Impairment
		Trails B	No Impairment
		Porteus mazes	No Impairment
Van Reekum et al. (1993)[b]	9	WCST	Impaired
		Trails B	Impaired

[a]Wisconsin Card Sorting Test
[b] Comparison made with normative data.

As can be seen from Table 1, studies investigating executive functioning in BPD have not produced consistent evidence of impairments, despite brain-scanning evidence of frontal lobe abnormalities in the disorder. There are a number of reasons why this may be. Firstly, sample sizes in most of the studies are small, and this obviously reduces the ability of studies to detect between group differences. The studies also vary in their sample characteristics, due to differing exclusion criteria, gender balance, type of referring institution (e.g. inpatient versus outpatient), which may account for some of the heterogeneity of the results.

Finally, there is also the possibility that the inconsistent results are due to poor ecological validity of the executive functioning tests used so far with BPD patients. Some have argued that abstract neuropsychological tests of executive function provide more structure and information than open-ended real-life situations, and thus may not always pick up executive functioning difficulties which might be manifested in day to day life (e.g. Shallice and Burgess, 1991). Thus far, no studies of executive functioning in BPD have used tasks which have been explicitly designed to be open-ended and mimic real-life scenarios or demands (e.g. Shopping Errand Task, Shallice and Burgess, 1991) and this may be a reason for the mixed results so far in this area. This point is discussed in more detail in section 1.5.3 (iii).

It is worth also noting that some researchers have reported that poor performance on executive function tests has been associated with depression (e.g. Channon, 1996). This raises the possibility that executive function impairments, if found in BPD, are due to co-morbid affective disorders such as depression, rather than personality disorder. Sprock (2000) directly addressed this issue with the inclusion of a depressive disorder control group, but in the end did not find any significant difference between the groups on executive function measures.

Theory of mind (the ability to attribute mental states to others: e.g. Premack and Woodruff, 1978) has also been linked to the frontal lobes. For example Stone et al., 1998 reported that patients with orbito-frontal lesions were impaired compared to controls on a theory of mind task. Also Flecher et al., (1995) found that the comprehension of stories which required mental state attribution activated an area of the medial frontal cortex which was not active during comprehension of control stories which did not involve mental states. There have as yet been no published studies investigating theory of mind in BPD, but there have been studies which have looked at closely related constructs, the results of which indicate that this may be a fruitful area of cognition to study in the disorder. Firstly, Levine et al., (1997) reported that BPD patients were impaired on an emotional processing test which required the attribution of emotions to characters in stories: a task which closely resembles some theory of mind tests such as the Happe Strange Stories test (Happe, 1994). Secondly, Fonagy et al. (1996) reported that patients with BPD had significantly lower scores on a measure of reflective functioning compared to patients of other diagnoses in a therapeutic community. Reflective functioning is defined as the capacity to understand ones own and others' behaviour in mental state terms (e.g. Fonagy, 1999) and thus appears to be a broader, but related construct to theory of mind, which focuses just upon the understanding of others' mental worlds.

It should be noted, however, that a key difference between the measurement of theory

of mind and reflective functioning is that the latter is generally assessed in the context of an individual's close attachment relationships. Formal assessments of reflective capacity are generally conducted by rating responses given in the Adult Attachment Interview (George et al., 1985), a structured interview which elicits narratives about childhood attachment relationships. By contrast, theory of mind measures employ hypothetical situations which are not designed to be relevant to a particular individual's own relationships.

Finally, autobiographical memory has also been investigated in BPD. This function has been associated with the frontal lobes, through PET scanning studies (e.g. Conway et al., 1999). Conway et al. suggest that the role of the frontal lobes may be to regulate the construction of autobiographical memories from more posterior regions. Jones et al., (1999) found that 28 patients with BPD were impaired in comparison to controls on a test of autobiographical memory (Autobiographical Memory Test; Williams and Broadbent, 1986). The borderline group found it harder than the control group to access specific memories in response to cue words. Jones et al., (1999) argue that this deficit may link with dissociation in the disorder (see section 1.3.4 for a further discussion of this point). It is worth noting that autobiographical memory impairments have also been reported in patients suffering from depression, (e.g. Williams and Scott, 1988) which raises the possibility that the deficits reported by Jones et al., (1999) were due to co-morbid depression rather than personality disorder.

In summary, brain-scanning studies have indicated that there may be structural and metabolic abnormalities in the frontal lobes of individuals with BPD. At present there is only mixed evidence for executive dysfunction in BPD, a finding which may be related to small sample sizes, varying exclusion criteria and the fact that there have been no studies using executive tests which have been designed to be open-ended and mimic real-life scenarios or demands (e.g. Shopping Errand task, Shallice and Burgess, 1991). There have as yet been no published studies investigating theory of mind in BPD, although studies of related constructs have indicated that this may be a fruitful area of

research. Finally, there have been studies suggesting that individuals with BPD have autobiographical memory deficits.

1.2.3 Abnormalities of the hippocampus and amygdala

The hippocampus and the amygdala are adjacent sub-cortical structures, located just beneath the temporal lobes, which have been shown to be important in memory and emotional processing. Hippocampal lesions tend to produce anterograde memory deficits, and it appears that the major role of the hippocampus may be in the initial encoding of memory traces, which are later stored in other cortical areas (Lezak, 1995). The amygdala has been implicated in providing emotional "tags" to memories (e.g. Rolls, 1990), and may be involved in the processing of emotional elements of stimuli, including facial expressions (Young et al., 1996).

There is some indirect evidence from structural brain scanning that individuals with BPD may have hippocampal abnormalities. Stein et al. (1997) found that the average hippocampal volume (as measured by MRI) of 21 women reporting childhood sexual abuse was significantly smaller than that of matched controls. Other types of traumatic experience have also been linked to smaller hippocampal volume (e.g. combat trauma: Bremner et al., 1995). The significance of these findings for BPD is that childhood sexual abuse and other early traumatic experiences are a common feature of the histories of individuals with BPD (e.g. Zanarini et al., 1997, found that 91% of a sample of patients with BPD reported some sort of childhood abuse or neglect). Given this, one might therefore hypothesise that similar results in terms of hippocampal volume in BPD may be found in future studies.

This indirect evidence of the possibility of hippocampal abnormalities in BPD predicts that individuals with the disorder may be impaired upon tests of memory function, especially those involving the encoding of new information. A variety of studies have tested this prediction, and the results of these are summarised in Table 2.

For verbal memory, all of the studies used the original or revised version of the Wechsler Memory Scale (WMS) logical memory sub-test (Wechsler, 1945, 1987), and one study also used the Selective Reminding Test (Buschke and Fuld, 1974). For non-verbal memory, all of the studies used the Rey-Osterrieth figure recall test (Osterrieth, 1944) These tasks have been shown to be sensitive to the effects of hippocampal damage (e.g. Miller et al., 1998, Sass et al., 1990).

Table 2. Summary of studies investigating memory function in BPD

Study	Group size	Tests	Results
O'Leary et al. (1991)	16	WMS logical memory Rey-Osterrieth	Impaired (delayed recall) Impaired (delayed recall)
Swirsky-Sacchetti et al. (1993)	10	WMS-R logical memory Rey-Osterrieth	No impairment Impaired (delayed recall)
Carpenter et al. (1993)	17	WMS-R logical memory Rey-Osterrieth	No impairment Impaired (delayed recall)
Judd and Ruff (1993)	25	WMS logical memory SRT[a] Rey-Osterrieth	Impaired (delayed recall) Trend to impairment Impaired (delayed recall)
Sprock et al. (2000)	18	WMS-R logical memory Rey-Osterrieth	No impairment No impairment

[a]Selective Reminding Test

As can be seen from Table 2, attempts at finding memory impairments in BPD have produced mixed results. With regard to verbal memory, the two studies using the original WMS logical memory task reported impairments in the BPD group, but three studies using the revised version of the same test reported no impairment. Judd and Ruff (1993) reported a trend towards their BPD group being impaired on the Selective Reminding Test. For non-verbal memory, four studies reported that BPD patients were impaired on the recall portion of the Rey-Osterrieth test, and one study reported no impairment. As with tests of executive function (see previous section), this variability may be due to small sample sizes and differing exclusion criteria across the studies.

Two studies have investigated cognitive functions associated with the amygdala in BPD. The first is Levine et al., (1997), who reported that a group of 30 individuals diagnosed with BPD performed poorly compared to controls on a test involving identifying emotional expressions of faces. This is an impairment which has been associated with patients who have had amygdalectomies (e.g. Young et al., 1996). However, Wagner and Linehan (1999) reported that a group of 21 women with BPD were unimpaired on a test of facial expression identification, and displayed heightened sensitivity to the recognition of fear. Wagner and Linehan suggest that one possible reason for the discrepancy between the two studies is that while Levine et al. (1997) used a multiple choice design, Wagner and Linehan used a free response format with answers coded by raters, which they argue is a more accurate assessment of facial emotion expression identification. In all, evidence of cognitive impairments associated with the amygdala in BPD is at best mixed.

In summary, there is indirect evidence from brain-scanning studies of trauma victims that individuals with BPD may have structural abnormalities of the hippocampus. Studies investigating memory function in BPD have produced mixed results, and this variability may be due to small sample sizes and varying exclusion criteria. There have also been mixed results in studies investigating facial emotion perception in BPD, a function associated with the amygdala .

1.2.4 Neuro-chemical abnormalities

A number of recent studies have investigated the hypothesis that neurotransmitter abnormalities may underlie aspects of the clinical presentation of BPD, particularly impulsive aggression and affective instability. With regard to impulsivity, low levels of serotonin have been associated with impulsive (as opposed to pre-meditated) aggression (Linnoila et al., 1983). Studies of BPD patients have shown weak response to serotonin enhancing agents, suggesting reduced serotonin activity (e.g. de Vegvar et

al., 1994). There have been suggestions that deficient serotonergic modulation of the limbic system (which has a central role in the regulation of action) may underlie impulsive aggression in BPD (Gurvits et al., 2000). Gurvits et al. also hypothesise that levels of the neurotransmitter norepinepherine (which regulates levels of arousal and engagement with the environment) may determine whether impulsive aggression is directed outwards (i.e. at others) or inwards (i.e. self harm, suicidal behaviour).

With regard to affective instability, abnormalities in the function of the neurotransmitter acetylcholine have been related to mood disorders (e.g. Risch et al., 1983). Studies have indicated that BPD patients have a more intense depressive response compared to healthy controls following administrations of substances which mimic acetylcholine (e.g. Steinberg et al., 1997). In this last study, it was found that the magnitude of depressive response correlated with the number of affective instability traits in the patients.

In summary, it appears that several neurotransmitter abnormalities may be important in influencing impulsive aggression and affective lability in BPD, and that a combination of these abnormalities may be important (alongside psychosocial stressors) as predisposing factors for the development of the disorder (Gurvits et al., 2000).

1.3 Possible relation between BPD symptoms and brain abnormalities.

The following sections will consider how the physiological, cognitive and affective elements of the brain abnormality model of BPD may be helpful in accounting for some of the symptoms of the disorder.

1.3.1 Interpersonal difficulties.

Judd and Ruff (1993, p281) suggest that the memory deficits that have been reported in BPD subjects may relate to the interpersonal difficulties that are characteristic of the disorder. Difficulties in encoding new information (e.g. Judd and Ruff, 1993) and

retrieving autobiographical memories (e.g. Jones et al., 1999), possibly due to hippocampal and frontal lobe abnormalities, could result in a reduced ability to learn from and integrate information relating to interpersonal experiences, thus hampering the possibility of future social behaviour being guided in an adaptive way.

Hypothesised executive dysfunction in BPD could also relate to interpersonal difficulties. It could be argued that many interpersonal situations impose executive demands, as they may, for example, require the individual to inhibit their immediate response to a situation and weigh up the consequences of different courses of action before acting (e.g. Channon and Crawford, 1999).Therefore dysexecutive difficulties may make it hard to negotiate interpersonal relationships with sufficient social judgement, thus making relationships vulnerable to instability.

As mentioned above, there have so far been no studies directly investigating theory of mind in BPD, but if a subtle deficit in this function were to be found in this population, one could speculate that this could also contribute to interpersonal difficulties. For example, it could help to explain the dramatic shifts between idealisation and denigration characteristic of the disorder. When a significant other is unable to immediately gratify the patient's needs, a theory of mind deficit may make it harder to understand the other's behaviour as an expression of legitimate needs and concerns which differ from the patient's.

1.3.2 Poor affect regulation and inappropriate anger.

There is evidence that areas of the pre-frontal cortex are important for the regulation of affect. For example, Dias et al. (1996) reported that monkeys with orbito-frontal lesions were impaired in shifting their behaviour in response to changes in the emotional significance of stimuli. Meares et al. (1999) also suggests that descending inhibitory tracts from the orbito-frontal cortex are important in the modulation of affect. It is well established that injury to the pre-frontal cortex can lead to affective dysregulation, as can

be seen in some patients with frontal lobe injuries (e.g. Stuss et al., 1992). Therefore in BPD, one might speculate that frontal lobe abnormalities which have been reported (e.g. Lyoo et al., 1998) may contribute to the poor affect regulation and inappropriate anger seen in the disorder. In addition, abnormalities in neurotransmitter systems, particularly acetylcholine (e.g. Steinberg et al., 1997), may also have a role in affective instability and anger in BPD.

A hypothesised deficit in theory of mind may also contribute to these affective symptoms. For example, poor understanding of the motivation behind another's behaviour may lead to a mis-construal of a situation involving a perceived violation of personal rights, to which the individual with BPD may react with understandable anger. A deficit in theory of mind may render interpersonal relationships baffling for much of the time, and this reduced ability to predict and understand the likely behaviour of others would leave the individual particularly vulnerable to emotional instability triggered by interpersonal events (e.g. a temporary separation).

1.3.3 Impulsivity.

Symptoms of impulsivity (e.g. acting without thinking, inability to inhibit responses) are commonly seen in brain injured patients with dysexecutive problems (e.g. Wilson et al., 1996), and these problems are particularly associated with frontal lobe injuries (e.g. Lezak, 1995). As mentioned above, some (but not all) studies have reported deficits in executive functioning in BPD, and this leads one to speculate that frontal lobe abnormalities in the disorder may be linked to impulsivity symptoms through executive dysfunction. Neurotransmitter abnormalities (e.g. in the serotonergic system) could also be linked to impulsive aggression in the disorder (e.g. Gurvits et al., 2000).

1.3.4 Dissociation.

Dissociative experiences can involve drastic failures of autobiographical memory (e.g. Schacter et al., 1982), a function has been linked to the frontal lobes (e.g. Conway et al., 1999). Jones et al. (1999) report that individuals with BPD are impaired in their ability to retrieve specific autobiographical memories. In this study there was also a significant negative correlation between autobiographical memory performance and the Dissociative Experiences Scale (Carlson and Putnam, 1993), indicating that poor autobiographical memory (when not dissociating) may relate to the likelihood of having dissociative experiences. Thus, one might hypothesise that frontal lobe abnormalities in BPD may be linked (through abnormal autobiographical memory function) to a tendency to dissociate in the disorder (Jones et al., 1999). The proposal that abnormal autobiographical memory may be linked to dissociation ties in with suggestions that the function of dissociation may be to avoid the emotional consequences of recalling past trauma (e.g. van den Hout et al., 1996). There have also been suggestions that hippocampal abnormalities may relate to dissociative amnesia, as the hippocampus may have a role in bringing together memory traces from disparate areas of the cortex (Bremner et al., 1996).

Some researchers have proposed that dissociative experiences in BPD may also be related to attentional abnormalities (e.g. Meares et al, 1999). Guranlnik et al. (2000) propose that depersonalisation may partly result from a failure of effortful control of the focus of attention, and in support of this they report that a group of subjects with depersonalisation disorder were impaired on a test of selective attention (Continuous Performance Test: Vigil, 1996). Studies have linked attentional processes to the pre-frontal cortex (e.g. Dias et al., 1996), and thus one might speculate that frontal lobe abnormalities in BPD may be linked to dissociation through attentional deficits.

1.3.5 Identity disturbance and emptiness.

There have been suggestions that pre-frontal cortex activity may be important in meta-cognition and providing a sense of self (e.g. Stuss, 1991). The pre-frontal cortex has been proposed as the site of the executive functions, which have a role in co-ordinating cognitive activity (e.g. Shallice, 1988). Meares et al., (1999) suggests that this co-ordination of cognition enables a coherent stream of consciousness and a sense of self to emerge. It has also been suggested that disruptions in one's sense of self may be related to autobiographical memory disturbance (Klein, 2001), a function which has been related to the frontal lobes (e.g. Conway et al., 1999). Therefore, a disruption of executive function and autobiographical memory, both due to frontal lobe abnormalities, may help to produce the unstable sense of identity which is characteristic of BPD.

There is also some evidence that identity disturbance and emptiness may be related to neurotransmitter dysfunction in BPD: Steinberg et al. (1997) reported that affective instability traits (including identity disturbance and chronic feelings of emptiness) correlated significantly with a measure of acetylcholine dysfunction those with the disorder (i.e. depressive response to physostigmine).

1.3.6 Summary.

Brain abnormalities which have been reported or proposed in individuals with BPD have been theoretically linked to many of the symptoms of the disorder, including poor affect regulation, impulsivity, dissociation, identity disturbance, feelings of emptiness, and interpersonal difficulties. Thus the brain abnormality hypothesis of BPD appears to have the potential to be a useful tool in accounting for some aspects of the clinical presentation of the disorder. However, only a few studies have so far tried to empirically link particular brain abnormalities with particular symptoms in the BPD population (one example is Steinberg et al., 1997, referred to above, in which affective instability traits in BPD were correlated with acetylcholine dysfunction).

1.4 Possible explanations for the presence of brain abnormalities in BPD.

The previous section reviewed how brain abnormalities in BPD may relate to the symptoms of the disorder. The question of how such abnormalities may have been acquired in BPD has recently been the subject of increasing theoretical and empirical work (e.g.. Meares et al., 1999). At least three types of explanation for the existence of brain abnormalities in BPD have been proposed, and these will now be considered.

1.4.1 Childhood abuse and neglect.

Childhood abuse and neglect is a common feature of the histories of individuals with BPD (e.g. Zanarini et al., 1997), and several researchers have suggested that brain abnormalities in BPD may develop as a result of such traumatic experiences (e.g. Meares et al., 1999).

The pre-frontal cortex, the hippocampus and the amygdala are amongst the most plastic of brain regions, and pre-frontal areas are the latest developing part of the cortex, with significant changes continuing into adolescence (Teicher et al., 1994). The plasticity and late development of these areas (some of which have been proposed as sites for brain abnormalities in BPD) makes them plausible candidates for being adversely affected by childhood trauma.

There are a number of mechanisms by which childhood trauma might produce brain changes. Changes in neuronal excitability may come about as a result of high levels of stimulation (a process known as kindling), and van der Kolk and Greenberg (1987) propose that repeated traumatic experiences may lead to kindling in the limbic system (which includes the hippocampus and amygdala), resulting in behavioural disruption. There is also evidence from animal models that stress can result in cell death and atrophy in the hippocampus, which may be due to the high levels of glucocorticoids

associated with stress (Uno et al., 1989).

Some studies have produced support for the suggestion that childhood trauma in particular may be linked to brain changes. For example, Green et al., (1981) reported that in a sample of abused children, there was a raised prevalence of neurological soft signs and EEG abnormalities. Also, Stein et al., (1997) found that adult female survivors of sexual abuse had reduced hippocampal volume compared to controls (measured by MRI). Finally, Teicher et al. (1994) reported that in a large sample of out-patients, a history of childhood abuse was associated with higher scores on the Limbic System Checklist (Teicher et al., 1993), a self report questionnaire of symptoms indicative of limbic system dysfunction (e.g. dissociative experiences).

Although these studies were not conducted on BPD samples, they provide some support for the hypothesis that there may be link between childhood trauma and brain abnormalities in BPD. However Teicher et al. (1994) caution that the direction of causation is as yet unestablished. For example some have suggested that neurological and neuropsychiatric abnormalities (such as seizures, impulsivity and depersonalisation) may put a child at increased risk of abuse as they are less able to resist the abuser (Davies 1978-1979), or that smaller hippocampal volume may be a risk factor for the development of the psychological sequelae of trauma (Stein et al., 1997).

1.4.2 Genetic inheritance.

In accounting for the existence of brain abnormalities in BPD, one possibility may be that these abnormalities occur as a result of genetic inheritance. A number of studies have tried to assess whether there may be a genetic component to BPD. Torgersen (2000) reviews several family studies investigating the prevalence of BPD in first degree relatives of individuals diagnosed with the disorder, and he argues that these are inconclusive. So far there is one published twin study which compared the diagnostic concordance of mono-zygotic versus di-zygotic twins in BPD (Torgersen, 1984), but this

did not find significant evidence of heritability. However Torgersen (2000) reports preliminary results of a larger twin study, which hints at a greater genetic role in the development of BPD.

Some have suggested that it may be more fruitful to consider whether the underlying dimensions of BPD are heritable (such as impulsivity and emotional lability), rather than trying to establish that BPD is heritable in itself (Gurvits et al., 2000). For example, Gurvits et al. argue that there is evidence that serotonergic abnormalities may by partially inherited, and in view of the fact that serotonergic abnormalities have been reported in BPD (De Vegvar et al., 1994), they propose that this may provide a genetic basis for impulsive aggression in the disorder. Torgersen (2000) argues that there are many personality traits associated with BPD which display a significant degree of heritability (possibly through variations in neurotransmitter function) including neuroticism, negative affectivity, affective lability, and identity problems.

In summary, although at present the evidence of the heritability of BPD itself is sparse, there are indications that underlying traits associated with BPD, such as impulsive aggression, may have a genetic basis. A genetic component may help to explain the presence of brain abnormalities which have been reported in BPD, particularly those involving neurotransmitter systems.

1.4.3 Head injury.

Many studies have found that there is an unusually high prevalence of head injuries in patients with BPD. For example, Streeter et al., (1995), reported that a significantly higher proportion of a sample of 43 BPD patients had a history of acquired brain injury, as compared to control psychiatric patients (42% versus 4%). Van Reekum et al., (1996) also reports a raised prevalence of head injury in BPD. It is well documented that head injury can produce changes in personality and emotional regulation (e.g. Stuss et al., 1992) and there have been reports of head-injured patients presenting with symptoms

which amount to acquired personality disorder (e.g. Blair and Cipolotti 2000, Franulic et al., 2000). This raises the possibility that (at least for some individuals with BPD) a head injury may be a significant aetiological factor in the development of the disorder, and that some of the brain abnormalities which have been reported in BPD may be related to prior head injury.

Against this suggestion, it is plausible to suggest that the diagnosis of BPD may itself be a risk factor for head injury (e.g. through impulsivity). However Streeter et al. (1995) report that in all but one of the cases in the BPD group, the head injury was sustained before the development of the full clinical symptoms of the disorder. Streeter et al., argue that this finding supports the hypothesis that head injury can be a contributory factor to the development of BPD. Regardless of whether head trauma precedes the development of BPD, one could argue that the presence of head injury in such a large proportion of those diagnosed with the disorder means that it may be an important contributory factor to the cognitive, behavioural and emotional symptoms of many individuals with BPD.

1.4.4 Summary

Three major types of explanation for the existence of brain abnormalities in BPD have been put forward. One proposal is that childhood abuse and neglect may produce neurological changes in individuals with BPD, through processes such as kindling and atrophic changes due to glucocorticoids. Although there hasn't so far been a specific empirical link between such neurological changes and childhood abuse in BPD, there is evidence from other populations that childhood trauma can be linked to smaller hippocampal volume, limbic system dysfunction, and neurological soft signs. Others have suggested that brain abnormalities in BPD may be explained by genetic inheritance, with the proposal that underlying dimensions of BPD, such as emotional lability and impulsivity, may be heritable through neurotransmitter dysfunction. Finally, the unusually high prevalence of head injury in BPD has led some to suggest that prior

head injury may be a cause of some of the brain abnormalities reported in BPD, and that it may be an significant aetiological factor for a proportion of individuals with the disorder.

1.5 Review of relevant literature relating to executive functioning.

Before formally presenting the hypotheses of this study, a brief review of relevant literature and current debates relating to executive functioning (this section) and theory of mind (section 1.6) will be presented, along with implications for the study of these functions in BPD.

1.5.1 Definitions of executive functioning

Shallice (1988) notes that from the early 19th century onwards, many considered the frontal lobes to be the seat of higher order control of thought and action, observing that patients who had suffered frontal lesions showed deficits in planning, responding appropriately and adapting to new situations. Such cognitive abilities have become known as the executive functions, and there is a current consensus that these can be broadly defined as a set of processes which facilitate adaptation to novel situations (Burgess, 1997).

Numerous attempts have been made to provide a detailed functional definition of executive processes. Just one of these is provided by Norman and Shallice (1986), who argue that there are five types of situation where executive functions are required for optimum performance:

(1) Those that involve planning or decision making

(2) Those involving error correction or trouble shooting

(3) Situations where responses are not well-learned or contain novel sequences of

actions.

(4) Dangerous or technically difficult situations.

(5) Situations which require the overcoming of a strong habitual response or resisting temptation.

Rabbitt (1997) collected together a selection of recent attempts to distinguish between executive and non-executive functioning, and produced an extensive list which includes: dealing with novelty where planning and goal identification are required, initiating new sequences of behaviours, preventing inappropriate responses, carrying out novel dual-task combinations, monitoring performance to correct errors, enabling sustained goal-directed attention, and searching for specific autobiographical memories.

The extensiveness of Rabbitt's list illustrates the difficulty of providing a definitive functional definition of executive processes, and Burgess (1997) proposes that this difficulty stems primarily from the fact that executive processes are involved in the co-ordination of lower-order functions. This means that executive functioning can potentially be required in any novel situation requiring the regulation of these lower-order processes, with the result there is no clear association between executive functioning and any particular type of behaviour. Indeed, Rabbitt (1997) argues that the degree of involvement of executive processes in any one task is a function of its novelty for that particular individual, rather than being due to any intrinsic property of the task. This is in contrast to other cognitive functions, where a particular underlying cognitive processes (e.g. face recognition) will contribute to performance on a well defined set of tasks. Therefore, while it is clear that in order to investigate whether a patient suffers from dyscalculia one should ask them to complete some sums, it is less clear what a definitive operationalisation of executive dysfunction should be.

Burgess (1997) argues that this difficulty has led to a continuing reliance on an anatomical definition of executive function (e.g. referring to dysexecutive difficulties as "frontal lobe syndrome"). This contrasts with the practice in other areas of cognitive

function, such as memory, where deficits are defined in functional rather than anatomical terms.

1.5.2 Models of executive functioning.

There have been many models of executive functioning, and one of the most influential was developed by Norman and Shallice (1986). In common with most other models, it relies on the distinction between automatic and controlled processing, proposing that habitual and novel actions are handled in different ways.

Under the model, routine processing is carried out by thought and action schemas, which contain the cognitive and motor programming for carrying out particular tasks, and are triggered by internal or external stimuli. Each schema will have acquired associations with particular triggers over time (e.g. for example, the sight of a glass of water will be associated with a schema containing the cognitive and motor programming required for picking up the a glass and drinking from it). Schemas that require the same information processing resource compete for dominance by inhibiting one another when activated; a process known as contention scheduling. However, for novel situations, the supervisory attention system becomes active, which selects or overrides schemas in order to enable the individual respond in a flexible manner.

Under the Norman and Shallice model, patients with frontal lobe injuries and dysexecutive problems can be seen as having an impairment of their supervisory systems, leaving contention scheduling to operate in an unmodulated fashion in all situations. This proposal helps to account for many of the behavioural phenomena seen in patients with frontal lobe injuries. For example when a particular schema is very strongly activated, it becomes hard for the patient to switch to a different response, resulting in perseveration. On the other hand, if the patient is in an environment where no one schema is strongly activated by external triggers, then irrelevant stimuli capture control of cognition resulting in high levels of distractibility or inappropriate behaviour. It

could be argued that utilisation behaviour (where patients automatically begin using an object that is put in front of them: Lhermitte, 1983) is an example of such a process. More recent extensions of this model have included attempts to fractionate the supervisory system into constituent processes (e.g. Burgess, 1997). In this paper, Burgess reports data which suggests that response initiation and response suppression, both considered to be aspects of executive functioning, may be damaged independently by brain injury.

Another influential model of executive processes is Baddeley's (1986) central executive component of working memory. In this, the central executive co-ordinates the activities of two slave working memory processes: the phonological loop and the visuo-spatial sketchpad. Under this model, tasks which require the simultaneous storage and manipulation of material would be sensitive to deficits in central executive ability (which, it is argued, explains poor dual task performance in Alzheimer's disease). Baddeley conceives of the central executive as being similar to Norman and Shallice's Supervisory Attention System. Other models include the one proposed by Stuss (1991), which again is broadly similar to the Norman and Shallice model, except that an additional level of self-reflectiveness/meta-cognition is proposed. This level is placed above the supervisory system, and its function is to enable the individual to reflect on thinking and action. It is proposed that the self-reflectiveness/meta-cognition level may also be damaged by injuries to the frontal lobe.

1.5.3 Special difficulties in the study of Executive Functioning

Aside from the difficulty (referred to above) in providing a satisfactory functional definition executive processes, the study of executive function poses a number of other challenges, which are outlined below.

(i) Issues of task purity.

Any task designed to measure a particular cognitive function is likely to suffer from additional variance due to the involvement of processes other than the one of interest (e.g. a deficit in visual processing ability may affect performance in a recognition memory task). However, Burgess (1997) argues that measures of executive function are especially prone to this source of error, as they necessarily involve the regulation and control of lower-order functions. This means that it is generally more difficult to rule out the effects of other functions in executive tasks than for measures of lower-order abilities. Certain executive function tasks, such as the Wisconsin Card Sorting Test (Grant and Berg, 1948) have been particularly singled out for criticism of task impurity, with some arguing that the involvement of lower-order processes (such as memory) is so great that the task can sometimes present greater difficulties for patients with lesions outside the frontal lobes than patients with frontal abnormalities (e.g. Corcoran and Upton, 1993). However, Rabbitt (1997) points out that too zealous an effort to control performance variables may result in a reduction in the "executive" nature of a task, as executive functioning involves the simultaneous management of different functional processes.

(ii) Test-retest reliability.

Rabbitt (1997) notes that tests of executive function generally have poor test-retest reliability, and that this problematic psychometric feature increases measurement error. Rabbitt argues that poor test-retest reliability is due to the fact that executive functions rely on novelty for their validity, as the more times a task is repeated, the more routine (and non-executive) its execution becomes.

(iii) Ecological validity

One of the paradoxes of the assessment of executive function is that, while a formal

testing situation must necessarily impose a degree of structure upon the behaviour of the patient, executive difficulties tend to manifest themselves in situations where there are fewest environmental constraints on behaviour (e.g. Lezak 1995). Many tests of executive function have been criticised for being too structured and too explicitly defined when compared to the open ended tasks of daily living which present such difficulties for many patients with dysexecutive syndrome (e.g. Shallice and Burgess, 1991; Wilson et al., 1996).

To illustrate this point, Shallice and Burgess (1991) reported three case studies of patients with frontal lobe damage, whose IQs were in the superior range but who had severe impairments in activities of daily living due to executive difficulties. Two of the patients were unimpaired on 13 tests thought to be associated with frontal lobe damage, and the third was impaired on just three of these. However, all three patients were impaired on tests designed to simulate the type of open-ended activity the patients had difficulty with in everyday life (a shopping errand task and a version of the Modified Six Elements Test used in this study). Other studies have also found that traditional tests of executive functioning are poor at picking out the sorts of difficulties experienced by patients with frontal lobe damage: for example Anderson et al., (1996) reported that the Wisconsin Card Sorting Test (Grant and Berg, 1948) and the Halstead Category Test (Halstead, 1947) were unable to distinguish patients with frontal versus posterior lesions.

1.5.4 Implications for the assessment of executive functioning in BPD.

As with any study of executive functioning, the extensive involvement of non-executive processes in many executive function tasks means that some care should be taken in the interpretation of the results of testing. In particular, the possibility that there may be memory impairment in BPD (e.g. Judd and Ruff, 1993), means that the potential contribution of memory failures to poor performance on executive function tasks should be borne in mind.

It may also be useful to use a range of different executive measures, given evidence that executive processes may be frationable into relatively independent sub-components (e.g. Burgess, 1997).

It was mentioned above that some have claimed that many commonly used tests of executive function have poor ecological validity, and that open-ended tasks may be better at detecting dysexecutive problems (e.g. Shallice and Burgess, 1991, Wilson et al., 1996). It is notable that to date, no studies of executive functioning in BPD have used open-ended tasks such as those described by Shallice and Burgess (1991), and it could be argued that this may be one reason for the inconsistency of findings with regard to executive functioning impairment in BPD (see section 1.2.2). With this in mind, two of the executive function tasks chosen in this study have been explicitly designed to be open-ended: the first is the Modified Six Elements Test (Wilson et al., 1996) in which the subject must organise attempting six tasks in ten minutes whilst obeying a rule, a variant of a task used by Shallice and Burgess (1991). The second is the Predicaments test (Channon and Crawford, 1999) in which the subject must suggest solutions to awkward social situations. This would appear to be particularly suitable for use with the BPD population given the interpersonal nature of many of the difficulties seen in the disorder. These tasks are described in more detail in chapter 2.

1.6 Review of relevant literature relating to theory of mind

The term "theory of mind" refers to the ability to attribute mental states to others (Premack and Woodruff, 1978). Deficits in theory of mind have been proposed as important in explaining the social impairments seen in autism (e.g. Baron-Cohen, 1985) and, more recently, schizophrenia (Frith, 1992). As yet, there have been no studies empirically testing theory of mind ability in BPD.

Many supporters of the concept of theory of mind have argued that it is a distinct cognitive module (e.g. Stone et al., 1998). Stone et al. propose that theory of mind is a

modular function in the same sense that language ability is: it may be selectively impaired or spared (however see section 1.6.2), it is culturally universal, and it emerges according to a particular developmental sequence. Others have argued for alternative viewpoints, such as the suggestion that theory of mind skills can simply be seen as a form of executive functioning (e.g. Russell, 1997). The following sections will briefly review this debate, and will then consider the implications for the present study.

1.6.1 Evidence for Theory of Mind as a discrete cognitive module.

(i) Graded developmental emergence of theory of mind skills.

It appears that theory of mind skills develop amongst normal children according to a consistent developmental timetable. At around four years, children begin to understand first order false belief (Wimmer and Perner 1983). The classic version of the first order belief task is the Sally-Ann task (Baron-Cohen et al., 1985). In the test, a child sees Sally (a doll) hiding a marble in one basket, before leaving the room. Whilst out of the room, another doll moves the marble to a different basket. Upon Sally's return, the child is asked where Sally will look for the marble; a question which requires the child to be able to ascribe a mental state to another (false belief) which is different to the child's own.

This paradigm was extended by Baron-Cohen (1989) with the second order false belief task, which is passed by normally developing children at around seven years of age. Passing a second order false belief task requires the subject to be able to ascribe to someone a belief about another's belief. At between nine and eleven years old children begin to pass faux-pas detection tasks, in which the subject must decide whether or not a character in a story has said something they shouldn't have (Stone et al., 1998).

As far as is known, this developmental sequence appears to be standard across cultures (Avis and Harris, 1991). This pattern of the emergence of theory of mind skills fits in with the notion that theory of mind is a dedicated cognitive mechanism with a biological basis

(e.g. Bach et al., 2000). However it could be argued that this developmental pattern is also consistent with alternative viewpoints, such as the proposal that theory of mind is an aspect of executive functioning (e.g. Russell, 1997).

(ii) Selective impairment/sparing of theory of mind skills in developmental disorders.

Many have argued that the core cognitive deficit in autism is a selective theory of mind impairment, and empirical studies of cognitive functioning in autism have provided some of the key pieces of evidence supporting the notion of a theory of mind module. The hypothesis of a selective theory of mind deficit is supported by the fact that autistic children perform poorly on theory of mind tasks compared to controls matched for IQ (e.g. Baron-Cohen et al.,1985) and autistic individuals show preserved abilities in non-social cognitive areas, such as non-mental analogues of theory of mind tasks which require the representation of physical rather than mental states (e.g. the false photograph task, Leekam and Perner, 1991). Preserved abilities have also been reported on certain visuo-spatial tasks such as the block design sub-test of the Wechsler Intelligence scales (Shah and Frith, 1993), or the Children's Embedded Figures Test (Shah and Frith, 1983).

The cognitive pattern seen in autism has been contrasted to that found in Williams' syndrome. Individuals with this condition show poor verbal IQ, but are often affectionate and have good social skills, performing well on theory of mind tasks (Karmiloff-Smith et al., 1995). This suggests that theory of mind skills can be preserved against a background of other cognitive impairments. (However, Tager-Flusberg et al., 2000, challenge the notion that all aspects of theory of mind are spared in Williams syndrome).

In summary, research into the cognitive profiles of individuals with autism and with Williams' syndrome indicates that it may be possible for theory of mind skills to be selectively impaired or spared. This fits in with the notion that theory of mind forms a distinct cognitive module.

(iii) Neuroimaging studies and brain damaged patients

A number of recent neuroimaging studies have suggested that theory of mind may be localised to regions within the frontal lobes. For example, Fletcher et al. (1995) reported that an area within the left frontal lobe showed greater activity when subjects were engaged in a task which involved mental state attribution, compared to non-mental control task.

This link between theory of mind and the frontal lobes is also suggested by studies of patients with frontal lobe damage. Happe, Malhi and Checkley (2000), and Channon and Crawford (2000) report results of testing with brain damaged patients which suggest that damage to locations within the frontal lobe may produce an acquired theory of mind deficit.

As well as localising evidence, studies of brain damaged patients have to some extent supported the notion that theory of mind skills may be independent of executive functioning: Happe, Brownell and Winner (1999) reported that right hemisphere stroke patients were less impaired on control non-mental tasks than the theory of mind tasks, suggesting that general reasoning skills were left relatively intact (in this study, more precise localisation of damage was unavailable). Also, Bach et al., (2000) describes the case of a man with orbito-frontal brain damage who was impaired upon tests of executive functioning, but performed normally on advanced theory of mind tasks. Together these results tentatively suggest a double dissociation between theory of mind and executive functioning.

In summary, studies of patients with brain damage and neuroimaging studies indicate a specific link between theory of mind skills and regions within the frontal lobes, with some evidence for the independence of theory of mind and executive functioning. This supports the notion that theory of mind may be a distinct and modular cognitive function.

1.6.2 Doubts over the independence of theory of mind from executive functioning.

It has been suggested that the false belief task, upon which much of the empirical research into theory of mind rests, imposes significant executive function demands. Russell et al. (1999) argue that in order to pass a false belief task, the child must suppress a pre-potent response (their own belief) and select a less salient response (the other's false belief) which has been held in working memory, and they argue that this gives the task a definite executive structure. This leaves open the possibility that autistic children fail the false belief task because of a deficit in executive functioning.

The standard argument used by theory of mind advocates against such a line of attack is to point out that autistic individuals are not impaired upon non-mental comparison tasks with the same executive structure, such as the false photograph task (Leekam and Perner, 1991). In the false photograph task, (Zaitchick, 1990) the child sees a Polaroid photograph being taken with a particular object in front of the camera. While the photograph is developing, the object in front of the camera is changed, and the child must predict what will appear in the photograph.

Russell et al. (1999) argue that the executive demands of this task may be much lower than the false belief task, because difference in salience between the developing photo and the scene in front of the child may not necessarily be very great. With the assumption that the presence of an object is definitely more salient than its absence, Russell et al., (1999) performed a modified version of the false photograph task, in which a photo is taken of a blank screen, and then an object is placed in front of the screen while the photo is developing. It was reported that on this task children with autism were impaired relative to controls (showing a tendency to predict that the photo will be of the object rather than the blank screen), supporting the idea that executive dysfunction may be a cause of autistic children's failure in false belief tasks. However, Russell et al. (1999) concede that other types of theory of mind task failed by individuals with autism

are less obviously executive in nature, such as faux pas detection (Stone et al., 1998).

Whether or not theory of mind tasks impose executive demands, studies of the relation between theory of mind and executive dysfunction in autism raise doubts about the selectivity and primacy of the theory of mind deficit in the disorder, which is one of the key pieces of evidence that has supported the notion of a theory of mind module. For example, Ozonoff et al., (1991) reported that executive dysfunction is more prevalent in individuals with autism than impaired theory of mind, which fits with the hypothesis that impaired executive function is a more primary deficit. Their study also found that poor theory of mind was significantly correlated with executive dysfunction, which calls into question the selectivity of poor theory of mind in the disorder.

In summary, it appears that many theory of mind tasks, especially false belief tasks, could also be viewed as tests of executive function, which calls into question whether theory of mind and executive function are separate constructs. This raises the possibility that autistic individuals fail theory of mind tasks because of executive problems, a proposal which is supported by studies which suggest that individuals with autism *are* impaired on non-mental analogues of theory of mind tasks if the executive demands of the task are of an equivalent level. In addition, the primacy and selectivity of theory of mind impairments in autism is called into question, as executive dysfunction may be more prevalent in the disorder than impaired theory of mind, and as there is evidence that impairments in theory of mind and executive function are strongly correlated in the disorder.

1.6.3 Implications for the study of theory of mind in BPD.

The disagreements over the status of theory of mind as a construct have not yet been resolved, and the present study is not specifically designed to produce evidence pointing to a resolution of this debate. However, there are implications for this study in terms of the choice of theory of mind tests and the interpretation of results.

The first of these implications is that, regardless of whether one views theory of mind as separable from executive functioning, it seems likely the possession of theory of mind skills is not something which is "all-or-nothing", and that there are developmental gradations of mentalising ability (e.g. Baron-Cohen et al., 1997). This means that a developmentally advanced theory of mind test will be most likely to pick up deficits in any given population. This study used the Happe Strange Stories test (Happe, 1994) and the Story Comprehension test (Channon and Crawford, 2000), both of which were designed to be developmentally advanced. The tasks are described fully in chapter 2.

Another implication for this study of the debate over the status of theory of mind is that the interpretation of the results should bear in mind the possibility of executive dysfunction being a cause of poor performance in the TOM tests used. It could be argued (e.g. Channon and Crawford, 2000) that many of the stories used in tests such as the Happe Strange Stories test or Story Comprehension test involve the subject having to suppress an explicit or superficial reading of the situation in order to come up with the correct explanation of the behaviour in the story (e.g. in stories that involve understanding sarcasm, figurative speech, pretence, white lies, or misunderstandings). If this is so, then it would appear that such tasks impose an executive demand on the subject.

As both theory of mind and executive functioning will be investigated in this study, the pattern of performance on executive functioning tests may help to clarify the reasons for any deficits on theory of mind tests found in the BPD group. For example, if the BPD group were to perform poorly on both theory of mind and executive function measures, then this would leave open the possibility that the poor performance on the theory of mind tasks were due to executive difficulties. However, if the BPD group was to perform poorly on theory of mind measures, but adequately on executive tasks, then this would count against suggestions that the poor theory of mind performance was due to dysexecutive problems.

1.7 Hypotheses of the study.

(i) Individuals with BPD have a deficit in Executive Function.

It is hypothesised that individuals with BPD have a deficit in executive function, detectable through cognitive testing.

The hypothesis is based upon evidence of frontal lobe abnormalities in BPD (e.g. Lyoo et al., 1998), and tentative evidence of executive dysfunction in BPD from neuropsychological testing (e.g. van Reekum et al., 1993). The hypothesis is also prompted by the possibility that an executive function deficit may help to account for some the symptoms of BPD, such as impulsivity and unstable interpersonal relationships (see section 1.3). In order to test the hypothesis, it was decided that a greater variety of executive function measures would be used than in previous studies, in view of is evidence that different aspects of executive functioning may be separately impaired (e.g. response initiation and response suppression: Burgess, 1997). Two tests were included which have been designed to be open ended and realistic (Predicaments Test, Channon and Crawford, 1999, and the Modified Six Elements Test, Wilson et al., 1996), as there have been suggestions (e.g. Shallice and Burgess, 1991) that such tasks may be more ecologically valid than the more abstract type of executive measures previously used in BPD. It is predicted that executive function deficits will be most likely to be detected on these two tasks. The interpersonal nature of the Predicaments test is an additional reason why deficits are particularly predicted on this task for individuals with BPD.

(ii) Individuals with BPD have a subtle deficit in theory of mind.

It is hypothesised that individuals with BPD have a subtle deficit in theory of mind,

detectable through cognitive testing.

This hypothesis is based upon reports of frontal lobe abnormalities in BPD (Lyoo et al., 1998), together with suggestions that theory of mind may be localisable to the frontal lobes (e.g. Stone et al., 1998). The hypothesis is also informed by the reflective function hypothesis of BPD (e.g. Fonagy, 2000) and by evidence that individuals with BPD have deficits in cognitive abilities which appear to be closely related to theory of mind, such as reflective functioning (Fonagy et al., 1996), and the ability to correctly attribute emotions to others (Levine et al., 1997). Finally, the hypothesis draws support from the possibility that a deficit in theory of mind may help to account for some of the symptoms of the disorder, such as unstable interpersonal relationships (see section 1.3). In order to test the hypothesis, developmentally advanced theory of mind tasks were chosen, (Happe Strange Stories Test, Happe, 1994; Story Comprehension Test, Channon and Crawford, 2000) as these would be most likely to detect a subtle impairment in theory of mind ability in the disorder.

Chapter 2: Methods

2.1 Design of the study.

In order to test the hypotheses of the study, two participant groups were recruited: a group of individuals diagnosed with Borderline Personality Disorder, and a comparison group of healthy volunteers. Both groups completed a battery of psychometric tests designed to test theory of mind and executive functioning (described below).

The study recruited only female participants for both groups, in common with some other studies of neuropsychological performance in BPD (e.g. Sprock et al., 2000, Carpenter et al., 1993, Swirsky-Sacchetti et al, 1993). This was because of suggestions that that males and females with BPD may differ significantly with respect to certain characteristics (such as history of brain insults and familial history of depression), with females being more likely to fall into a non-organic sub-type (Andrulonis and Vogel, 1984). There are also indications that healthy male and female subjects may perform differently on some theory of mind tasks (Baron-Cohen et al., 1997). The exclusion of one gender therefore reduces the number of variables to take into account in the interpretation of the results, and it was decided that male participants should be excluded as around 75% of individuals diagnosed with BPD are female (DSM-IV, American Psychiatric Association, 1994).

As performance on many of the tests would be likely to be influenced by general intelligence, it was thought that estimated IQ should be controlled for as a factor wherever possible in the statistical analyses. Therefore analysis of co-variance was considered to be the most appropriate statistical test for between group analyses (provided the variables concerned were normally distributed).

In order to give a tentative indication as to the possible influence of co-morbid depression upon cognitive performance within the BPD group, it was planned that correlations within the BPD group would be performed between any cognitive measure upon which the group showed an impairment and a self report measure of depression (BDI-II, Beck et al., 1996).

2.2 Recruitment procedures.

The recruitment procedures for each of the participant groups is given below. Full information about the numbers included and group characteristics is given in chapter 3.

(i) Borderline Personality Disorder group.

Participants for the BPD group were recruited from community mental health teams, day hospitals, and a specialist personality disorder service. All participants in the BPD group were approached for recruitment to the study by their key worker or another professional involved in their care. If the potential participant showed an interest in taking part, the investigator contacted them directly to discuss the study and arrange a time for participation.

Participants were either diagnosed with BPD by the Structured Clinical Interview for DSM-IV Axis-II Personality Disorders (SCID-II: First et al., 1996), which was administered as part of their involvement in a specialist personality disorder treatment service, or were diagnosed with BPD during the course of standard clinical care before recruitment to the study. When invited for testing, all of BPD participants scored above threshold on the personality disorder screening scale of the Inventory of Interpersonal Problems (Pilkonis et al., 1996).

Inclusion criteria for the BPD group were: age 18-65, diagnosis of BPD, female, English as a first language (or native-speaker proficiency).

Exclusion criteria were: co-morbid diagnosis of psychosis (established through communication with key-worker), and learning disability (established through performance on the National Adult Reading Test - Revised; Nelson and Willison, 1991).

(ii) Healthy control group.

Participants for the healthy control group were recruited through advertisements placed on community notice-boards and in newspapers, inviting interested parties to contact the investigator to discuss the study further before arranging a time to take part. Before inviting participants for testing, the Structured Clinical Interview for DSM-IV screening module (First et al., 1995) was administered over the telephone as well as a general interview about mental health history, to exclude potential participants likely to be suffering from current mental illness. In order to recruit sufficient numbers of control participants to the study, it was necessary to include some participants who had experienced a past episode of depression. Potential participants with a history of neurological disease or head injury were also excluded at this point. Information about educational attainments was obtained in an attempt to select control participants who would match the demographic and general intellectual characteristics of the BPD group.

Inclusion criteria for the healthy control group: age 18-65, female, English as a first language (or native-speaker level).

Exclusion criteria were : current mental illness, past mental illness other than an episode of depression, history of neurological disease or head injury, and learning disability (established through performance on the National Adult Reading Test - Revised). In addition it was planned that participants who scored above the cut-off on a personality disorder screening scale (the IIP-PD: see section 2.3, number 6 below) would be excluded. However see section 3.1 for a description of difficulties encountered with this procedure.

2.3 Measures.

Measures used in the study are described in the order they were administered in the battery.

1. National Adult Reading Test - Revised (Nelson and Willison, 1991).

   This test consists of fifty irregular words which the subject reads out aloud to the examiner. The number of pronunciation errors on this test has been shown to give a good estimate of WAIS-R full-scale IQ (Nelson and Willison, 1991). Performance on the test is thought to be relatively resistant to brain damage, (e.g. dementia), and thus gives an estimate of pre-morbid IQ. The test was used in this study in order to provide a quick estimate of participants' general intelligence.

2. Story Comprehension test (Channon and Crawford, 2000).

   The Story Comprehension test is a theory of mind task, involving explaining motivations behind the words or actions of story characters in 12 brief vignettes. In this task the reasons behind the words or actions of the characters in the story are not explicit from the text, and subjects typically have to make a non-literal interpretation in order to arrive at the correct answer. Neutral prompts (e.g. "can you explain that a bit further") are given in response to ambiguous answers. An example of one of the stories is given below:

   Samantha is having a party to celebrate her birthday at the weekend. Her next-door neighbour, who Samantha does not like, has complained in the past when Samantha has played loud music, so Samantha decides to warn her about the party. When she sees her neighbour Samantha says "I'm glad I saw you, I just wanted to let you know that I'm having a party on Saturday night, it should finish quite early." Her neighbour replies "Oh thank you so much, I would love to come".

Why did the neighbour say that?

Channon and Crawford (2000) reported that patients with left anterior lesions were impaired on this task relative to those with right anterior lesions, posterior lesions, and controls. The Story Comprehension test appears to be developmentally advanced, as control subjects score 89% correct on this task. This compares to 99% correct for the control group in the original report of the Happe strange stories test (Happe, 1994), which is considered to be developmentally advanced because able autistic individuals who passed 2nd order theory of mind tasks were impaired on this task. In the present study the participants' responses were rated by the same scoring criteria used by Channon and Crawford (2000), yielding a total score out of 12 for each participant.

3. Strange Stories test (Happe, 1994; Janes et al., 2000)

The Strange Stories test is a theory of mind task consisting of a number of short vignettes in which characters make non-literal utterances. The subject is required to explain why each character said something which is not literally true (such as telling a white lie in order to please someone). No prompting to clarify ambiguous answers is given. Happe (1994) argues that the task is an advanced theory of mind test, as she found that able autistics who were able to pass 2nd order theory of mind tasks were impaired on the test relative to control groups of normal adults, normally developing children, and learning disabled individuals. The measure has been used in a number of other studies as an indicator of advanced theory of mind abilities (e.g. Blair et al., 1996, Bach et al., 2000). Happe (1994) speculates that the stories are more difficult for autistic subjects than traditional first or second order theory of mind tasks because they are more contextually embedded and realistic, and thus less easy for someone with poor theory of mind to work out a strategy for solving the test using general intellectual abilities. An example of one of the stories is given below:

Jill wanted to buy a kitten, so she went to see Mrs. Smith who had lots of kittens she didn't want. Now Mrs. Smith loved the kittens and she wouldn't do anything to harm them, though she couldn't keep them all herself. When Jill visited she wasn't sure she wanted one of Mrs. Smith's kittens, since they were all males and she had wanted a female.

But Mrs. Smith said, "If no one buys the kittens I'll just have to drown them!"

Was it true, what Mrs. Smith said?

Why did Mrs. Smith say this to Jill?

The present study used modified scoring criteria by Janes et al., (2000), which were developed for a longitudinal study of child psychotherapy outcome. These extend the original scoring system to distinguish answers which explain the story in mental state terms (which are given 2 points) from those which give a correct but purely physical state explanation (which score 1 point). The Janes et al. (2000) scoring criteria also reduce the number of stories presented from 24 to 9, in order to reduce administration time and eliminate items which were found to have poor discriminative validity.

4. Predicaments test (Channon and Crawford, 1999).

The Predicaments test is a test of problem solving in real-life-type situations. Subjects read eight vignettes about difficult social situations, and are asked a number of questions about how the situation might be resolved. The Predicaments test is designed to be more open-ended and realistic than many abstract neuropsychological tests of executive function, and thus might be more sensitive to the types of difficulties seen in patients with dysexecutive problems (e.g. Shallice and

Burgess, 1991). Channon and Crawford (1999) found that patients with anterior lesions were significantly poorer than patients with posterior lesions in selecting appropriate problem solutions when asked how they themselves would respond to the situation (personal solution quality - see below). There were also group differences in the number of ideas generated as potential solutions to the problems (solution fluency - see below). Channon and Crawford (1999) argue that this result is most plausibly attributed to executive dysfunction in the anterior lesion group. An example of a Predicaments test scenario is given below:

Anne is in her office when Tony comes in. She asks how he is, and he says he is alright, but tired. She agrees that he looks tired, and asks what is the matter. He has new neighbours who moved into the flat above his a couple of weeks ago. They are nice people, but they own dogs and keep them in their kitchen at night, which is directly above Tony's bedroom. All night, and every night since they moved in, the dogs jump around and bark. He finds it impossible to get to sleep. He says he has had a word with the neighbours, and although they were very reasonable, they said they had nowhere else to put the dogs as it is a block of flats.

To ensure participants are aware of all the key details of the story, they are asked to recall as much as they can about what happened in the scene. If the participant has missed any key ideas from the story, they are asked whether they recall any further details (prompt 1). If after this prompt there are still missing details, the subject is given the story to read once more and asked for further details they have missed (prompt 2). If the subject has still not given the all the key ideas from the story after this prompt, they are supplied by the examiner (prompt 3). Following this, participants are asked what the main character could potentially do in the situation, giving as many ideas as possible in two minutes (referred to as the fluency section of the test). Participants are then asked what the main character *ought* to do in the situation (optimum solution), and what the subject would do herself in the situation (personal solution). Responses are scored according to problem appreciation, social

appropriateness, and effectiveness. These sub-measures combine to form the overall solution quality score for each idea generated. The test yields several measures: the main ones are optimum solution quality (average solution quality for optimum solutions) and personal solution quality (average solution quality for personal solutions). Additional variables are solution fluency (sum of the number of ideas generated in the fluency sections of the test), and average solution quality (average solution quality of ideas generated in fluency sections). The number of prompts given is also recorded to give an indication of the participants' memory for the scenarios.

5. Beck Depression Inventory - 2nd edition (Beck et al., 1996).

   The Beck Depression Inventory - 2nd edition (BDI-II) is a self-report questionnaire consisting of 21 items, which aims to measure levels of depressive symptomatology. Each item has four statements, which relate to different levels of symptom severity. The subject chooses the item which best describes how they have been feeling in the last two weeks including the day of testing. Dozois et al., (1998) recommend that that a total score of 0 to 12 should be regarded as non-depressed, 13 to 19 as dysphoric, and 20 to 63 as depressed.

6. Inventory of Interpersonal Problems (Shortened self-report version: Pilkonis et al., 1996).

   This questionnaire consists of 47 self-report items designed to reflect the nature of the interpersonal problems characteristic of most patients with personality disorders. Each item presents an interpersonal problem (e.g. "I am too sensitive to rejection") which is rated by the subject on a 0 to 4 scale ("not at all" to "extremely"). There are five sub-scales: interpersonal sensitivity, interpersonal ambivalence, aggression,

need for social approval, and lack of sociability. The first three of these combine to form a screening scale for presence versus lack of personality disorder (referred to as the IIP-PD). Pilkonis et al. (1996) reported that amongst a sample of 145 in-patients and out-patients with non-psychotic diagnoses, an item average of 1.1 on these three scales provided the best cut-off point for distinguishing between presence versus absence of personality disorder. This cut-off point yielded a sensitivity of 0.71 and a specificity of 0.67. In addition to its use as a screening tool in this study, it was intended that the IIP-PD would be used to investigate the relation between any poor performance of the BPD group on executive function or theory of mind tasks and personality pathology.

7. Barratt Impulsiveness Scale (11th version) (Patton et al., 1995)

The Barratt Impulsiveness Scale (BIS-11) is a self-report questionnaire which aims to measure degree of impulsivity as a dimensional personality trait. There are 34 items, which consist of statements relating to impulsivity (e.g. "I act on the spur of the moment"). Each item is rated on a 4 point scale by the subject, according to how strongly they identify with the statement. Patton et al. (1995) reported that total scores on the BIS-11 for psychiatric in-patients and prison inmates were significantly higher than scores obtained by college undergraduates. The BIS-11 was included in the study primarily to investigate the relationship between any poor performance of the BPD group on executive function tasks and impulsivity.

8. Trail-making test. (Reitan, 1958).

The test consists of two parts, A and B. In part A the subject must join a series of numbered circles in order as quickly as they can, and the time to complete each part is recorded. In part B, the subject joins the same number of numbered and lettered circles, alternating between the two sets of sequences. Many consider part B to be a

test of executive ability that taps functions such as set shifting, and it has been reported that part B significantly correlated with other tests of executive functioning (Arbuthnott and Frank (2000). Some advocate subtracting part A from part B or dividing part B by A, in an attempt to control for processing speed and visuo-spatial ability. However, Spreen and Strauss (1998) note that trails A is not as good a control task for trails B as is sometimes assumed, as the total length of the trail is different, and as the line to be drawn for trails B passes close to more distracting items. In the present study, part B alone was used as the variable for analysis, for reasons explained in section 3.4.1.

9. Rule Shift test (Wilson et al., 1996).

The Rule Shift test is an executive function task which aims to measure subjects' ability to shift from applying one rule to applying another. The test is divided into two parts. In each part, a booklet of playing cards is turned over one at a time, and the subject must say yes or no according to a rule which is left in front of them to refer to. In the first part the rule is "Say yes to red, no to black". In the second part, the rule changes to "Say yes if the colour of the card is the same as the last one, otherwise say no." The Rule Shift test was found to correlate significantly with informants' ratings of dysexecutive problems in patients with a variety of neurological disorders (Wilson et al., 1996).

10. Modified Six Elements Test. (Wilson et al., 1996)

The Modified Six Elements is an executive function test requiring the subject to attempt three tasks (dictation, picture naming and arithmetic) in ten minutes, each of which is divided into two parts, A and B. The subject is told that they are to attempt a least something from all six parts of the test within the time limit, whilst obeying a rule which states that they must not perform two parts of the same task consecutively (for

example, they cannot do arithmetic part A and then immediately do arithmetic part B). The aim of the test is to measure how well the subject organises themselves to do the six elements of the test, rather than measuring how they perform on any individual part. Burgess (1997) argues that the Modified Six Elements Test taps a subject's ability to plan, organise, monitor behaviour, and utilise prospective memory (i.e. ability to remember to carry out an intention at a future time). Wilson et al., (1996) found that the test correlated significantly with informants' ratings of dysexecutive problems in patients with a variety of neurological disorders.

11. Hayling test (Burgess and Shallice, 1997)

The Hayling sentence completion test is an executive function task which aims to measure response initiation and response suppression. It is divided into two parts, each of which consists of fifteen sentences with the last word missing. In the first part the examiner reads each sentence aloud and the subject must complete the sentence in a sensible manner as quickly as possible. In the second part, however, the subject is required to supply a word which is unconnected to the sentence that has just been read to them, meaning that they must suppress the tendency to complete the sentence with a connected word. The test yields three measures related to executive function: a measure of response initiation (total latency on part 1 of the test) and two measures of response suppression (total latency on part 2, and an error score). Patients with anterior lesions were found to be significantly poorer than healthy controls on all of these measures and were significantly poorer than patients with posterior lesions on both of the response suppression measures (Burgess and Shallice, 1997).

12. Letter Fluency (Spreen and Strauss, 1998).

In this test, subjects are given a letter and are asked to say as many words as they can beginning with that letter in one minute, excluding proper nouns, numbers, and

the same word with different suffixes. The three letters used in this study are "FAS", the most commonly used combination (Lezak, 1995). Depressed scores on this test have been associated with frontal lesions, especially on the left side (Miceli et al., 1981). The association between strategy use and high scores on this task has been used as an argument for the task being viewed as executive in nature (e.g. Phillips, 1997).

13. Similarities (Wechsler, 1981)

This sub-test of the Wechsler Adult Intelligence Scale-Revised (WAIS-R) is designed to test conceptual reasoning. In the test, the subject is given a series of pairs of words graded in difficulty, and must explain what each pair has in common. The Similarities has been treated by several authors as an executive function task (e.g. Lafleche, et al., 1995), and functional imagining suggests that performance of this task is related to increases in glucose metabolism in frontal and temporal areas (Chase et al., 1984). Poor performance on this test is associated with frontal and temporal lesions, although it can also be sensitive to injury in other areas of the brain (Lezak, 1995).

2.4 Testing procedure.

(i) Testing rooms

Most testing took place at an adult mental health psychology department, with some other testing taking place at hospitals, day hospitals or health centres where BPD participants were receiving their care. The rooms were arranged so that the investigator and participant faced one another across a desk or table.

(ii) Consent and preliminary information.

Upon arrival participants were given an information sheet to read (see appendix), and were given the opportunity to discuss the study and ask questions. Once they were satisfied they had received enough information, they were given a consent form to complete (see appendix). The following information was then collected: age, occupation, and education attainments.

(iii) Psychometric testing

Psychometric testing then proceded, with the tests administered in the order in which they are described above. Testing normally took between 2 and 3 hours, with breaks given as required.

(iv) Payment

Following completion of the testing battery, participants were paid £10.

(v) Feedback

Participants were given the opportunity to receive feedback on their results if desired.

2.5 Ethical permission.

The study received ethical permission from the Camden and Islington NHS trust local research ethics committee (see appendix for copy of ethical permission).

Chapter 3: Results.

3.1 Participants.

(i) Borderline Personality Disorder group.

19 women were recruited to the study for the BPD group. One of these participants was excluded as her concentration during testing was so poor that she was unable to assimilate much of the information presented in the test items. A second participant (who was originally given a diagnosis of BPD by her community mental health team) was excluded because she was subsequently assessed by a specialist personality disorder service and was not given a diagnosis of BPD. This left 17 participants with BPD whose results were entered into the analysis. 13 of these had had their diagnoses of BPD established through the administration of the SCID-II (First et al., 1996) as part of their involvement with a specialist personality disorder service. The rest of the BPD participants had had their diagnosis of BPD established in the course of standard clinical care (e.g. in community mental health teams), and all of these scored above the cut-off value on the personality disorder screening scale of the Inventory of Interpersonal Problems (or IIP-PD).

Three of the participants had had a head injury which had resulted in unconsciousness or brief hospitalisation. A further three had received injuries to the head as a result of childhood abuse, but which did not result in hospitalisation (although hospitalisation was unlikely as the abuse was perpetrated by caregivers) and which did not result in unconsciousness. The rest of the participants did not report any head injury. One of the participants reported that she had had a few epileptic seizures when seven years old, but these had not recurred since. None of the other participants reported a history of neurological disease.

(ii) Healthy control group.

20 participants were seen for testing after preliminary screening over the telephone. All scored below cut-off for depression on the BDI-II. However, 7 scored above the cut-off point of the IIP-PD. It was originally planned that participants who scored above this cut-off would be excluded from the study, which would leave a much reduced sample of 13 in the healthy control group.

The original sample used to validate the IIP-PD scale as a screening tool for personality disorders was drawn from mental health in-patients and out-patients with non-psychotic disorders (Pilkonis et al., 1996). A cut-off of 1.1 for the item average of the IIP-PD scale was found to be the most satisfactory for screening purposes. As yet, there has not been a published study investigating what a suitable cut-off point for the non-clinical population might be for this measure. Scarpa et al., 1999, reported that high scores on the IIP-PD were associated with high scores on the SCID-II in a college sample, but the small sample size meant that the issue of cut-off points could not be investigated. Recent personal communication with Joseph Prioetti (a researcher working in the group which developed the IIP-PD) revealed that there is a study in progress designed to address the issue of cut-off points in the non-clinical population. Preliminary figures from this research suggest that 28% of their non-clinical sample score above the cut-off of 1.1 suggested by Pilkonis et al. (1996), a similar figure to the 7 out of 20 (i.e. 35%) of the control group in the present study.

In order to make best use of the data, it was decided that in addition to the primary statistical analyses using the 13 healthy volunteers who scored below the IIP-PD cut-off (referred to as the Control-I group), secondary analyses would be performed using all 20 participants tested for this group (referred to as the Control-II group). While the secondary analyses would have to be interpreted with caution (as there would be uncertainty as to the personality disorder status of some of the participants included in the group), they may produce some tentative additional information that the primary

analyses would be unable to provide due to small sample size. It could be argued that if the inclusion of individuals of questionable personality disorder status into the healthy control group were to introduce a bias, then this bias would reduce rather than inflate the possibility of finding group differences as the groups would be rendered more similar. It should also be noted that none of the seven participants whose IIP-PD scores were above the cut-off point had had any prior contact with mental health services.

3.2 General variables.

Table 3 summarises the means and standard deviations for age, years of education, estimated intelligence, BDI-II, IIP-PD, and BIS-11 for the three groups used in the analysis.

Table 3: Group means for general variables (standard deviations in brackets).

Variable	BPD (n=17)	Control-I (n=13)	Control-II (n=20)
Age	34.6 (7.6)	34.8 (9.5)	36.2 (10.7)
Education (years)	14.3 (2.3)	14.3 (1.8)	14.8 (2.3)
Estimated FSIQ[a]	101.13 (14.92)	104.23 (9.61)	108.06 (10.01)
BDI-II[b]	37.41 (11.01)	4.92 (5.28)	6.30 (4.96)
IIP-PD[c]	2.203 (0.716)	0.706 (0.262)	0.991 (0.460)
BIS-11[d]	73.48 (12.68)	65.01 (10.32)	63.45 (9.39)

[a] Full scale IQ estimated by the NART-R
[b] Beck Depression Inventory-II
[c] Personality disorder screening scale – Inventory of Interpersonal Problems
[d] Barratt Impulsiveness Scale

Tests of skewness and kurtosis indicated that the underlying distributions were normal, so t-tests were used to investigate whether there were significant differences between the BPD group and either of the control groups on these variables. The BPD group did not differ significantly in age from either the Control-I group ($p=0.950$) or the Control-II group ($p=0.609$). The BPD group also did not differ in years of education from the Control-I group ($p=0.986$) or the Control-II group ($p=0.505$). There was no significant difference between the BPD group and the Control-I group in estimated full scale IQ

NART-R (p=0.497). This was also the case for the Control-II group (p=0.115), but the level of significance is close to that which is conventionally regarded as a trend (i.e. 0.1). However, estimated general intelligence was used as a co-variate for where possible for all between-group comparisons of executive function and theory of mind measures. As would be expected, the BPD group scored significantly higher than both control groups on the BDI-II and IIP-PD ($p<0.001$ for all of these comparisons). The BPD also scored significantly higher on the BIS-11 compared to the Control-II group (p=0.009) and there was a trend for higher scores when compared to the Control-I group (p=0.060).

3.3 Theory of Mind variables.

Two theory of mind variables were included in the test battery, the Happe Strange Stories test, and the Story Comprehension test. All participants in the study completed both tests, and there were no outlying scores (i.e. scores which were more than three standard deviations from the group mean). Tests of skewness and kurtosis indicated that underlying distributions were normal. Table 4 summaries the means and standard deviations of these tests for all three groups. As the Story Comprehension and Happe Strange Stories tasks aim to measure the same underlying construct, a MANCOVA was used to analyse the two variables together, with the number of NART-R errors used as a co-variate. The results of this analysis are presented in Table 5.

Table 4: Mean scores and standard deviations (brackets) on theory of mind tasks

Measure	BPD (n=17)	Control-I (n=13)	Control-II (n=20)
Happe Strange Stories	14.70 (2.14)	14.76 (2.64)	15.38 (2.41)
Story Comprehension	9 .00 (2.06)	10.54 (1.23)	10.70 (0.97)

Table 5: Statistical tests for group differences on theory of mind variables.

Comparison	Variables	Statistical test	F-value	df[c]	p-value
BPD vs. Control-I	HSST[a] and SCT[b]	MANCOVA	3.91	26	0.033*
	HSST	ANCOVA	0.01	27	0.923
	SCT	ANCOVA	6.00	27	0.021*
BPD vs. Control-II	HSST and SCT	MANCOVA	3.89	33	0.030*
	HSST	ANCOVA	0.123	34	0.728
	SC	ANCOVA	7.19	34	0.011*

[a] Happe Strange Stories test
[b] Story Comprehension test
[c] Error degrees of freedom. Group degrees of freedom is 1 in all cases.
*Significant at the 0.05 level.

As can be seen from table 5, MANCOVA analyses of the two theory of mind variables (with NART-R used as a co-variate) revealed a significant effect of group for both BPD versus Control-I (p=0.033) and BPD versus Control-II (p=0.030). Given this significant overall result for the theory of mind variables, follow-up analyses were performed (univariate ANCOVA using NART-R as a co-variate) and it was found that there were significant differences between groups on the Story Comprehension task for both BPD versus Control-I (p=0.021) and BPD versus Control-II (p=0.011). However, the BPD group did not differ significantly from either control group on the Happe Strange Stories test.

In order to investigate whether performance on the Story Comprehension task in the BPD group was related to the level of depressive symptoms, a correlation between BDI-II scores and Story Comprehension scores was performed within the BPD group, with NART-R errors partialled out as a factor. This revealed no significant relationship between the variables (R= -0.3476, p=0.187). However, when BDI-II score was entered (as well as NART-R errors) as a co-variate for ANCOVA comparisons between the

groups, this rendered the between-group comparisons non-significant both for BPD versus Control-I (F=0.014, df=26, p=0.906) and BPD versus Control-II (F=0.002, df=33, p=0.965). Section 4.4.2 discusses the implications of this result with regard to BPD, depression and theory of mind.

Partial correlations were also performed within the BPD group between SCT score and IIP-PD (R=-0.0962, p=0.733), and between SCT score and BIS-11 (R=0.057, p=0.835) were also not significant, suggesting that there was no relationship between SCT performance and either personality pathology or impulsivity as measured by these instruments within the BPD group.

3.4 Executive function variables (excluding Predicaments test).

Excluding the Predicaments test, which is analysed in a separate section later on, there were six executive function tests included in the battery (Rule Shift, Modified Six Elements test, Hayling test, Trail-making test, Similarities and Letter Fluency). It was decided that it would not be appropriate to use a MANCOVA to analyse the results of the executive function measures together, as there are increasing suggestions that executive function may be fractionated into different sub-components, some of which may be relatively independent from one another (such as response initiation and response suppression: Burgess, 1997). Therefore a series of univariate ANCOVAs were performed, with NART-R errors used as a co-variate. However any significant results from this procedure must be interpreted cautiously in light of the increased risk of type-I error. For some of the variables, tests of skewness and kurtosis indicated that the underlying distributions were not normal and transformations were performed. Details are provided in the next section.

3.4.1 Measures entered into the analyses

For clinical purposes the number of errors on the Rule Shift test is normally converted

into a profile score with only five values (a range of 0 to 4), however for the purposes of statistical analysis it was decided that the number of errors would be entered as the dependent variable, because the relative crudeness of the profile score would probably reduce the power of the measure to detect group differences. As tests of skewness and kurtosis indicated that the underlying distributions were not normal, a log transformation was performed. The transformed variable was normally distributed for all groups.

The Hayling test yields three measures, latency on part A (which involves straight-forward sentence completion), latency on part B (where participants must supply an word unconnected to the sentence), and an error score for part B (which reflects the number and seriousness of errors). All three measures are normally converted into profile scores for clinical purposes, but again it was decided that raw scores would be entered into the analysis as this may increase the power of the measures to detect group differences (this practice was adopted by Channon and Crawford, 1999, for their analyses of these measures). As tests of skewness and kurtosis indicated that the underlying distributions for all three measures were not normal, log transformations were performed. The transformed variables were normally distributed for all groups.

The Modified Six Elements Test also has a profile score (out of 4) which is converted from raw scores (out of 6). The pattern of results from this measure presented difficulties for formal statistical analysis, which are discussed later.

For the Trail-making test, the time to complete part B of the test (where participants must alternate between number and letter) was entered into the analysis. As mentioned in section 2.3, some advocate subtracting part A from part B, or dividing part B by A, in an attempt to control for processing speed and visuo-spatial ability. However it was decided that the trails B time would be used for analysis, in order to make the results of this study more comparable to previous studies using the trail-making test in BPD (e.g. Sprock et al., 2000, van Reekum et al., 1993). Group differences were greater on trails B than either B-A or B/A. Tests of skewness and kurtosis indicated that time to complete trails B

was not normally distributed, so a log transformation was performed. The transformed variable was normally distributed for all groups.

Finally, the total number of words produced for all three letters in the Letter Fluency, and the raw score of the Similarities sub-test of the WAIS-R were used as the variables for these measures. Both of these variables were found to be normally distributed for all groups.

3.4.2 Statistical comparisons between groups.

A series of univariate ANCOVAs were performed on each of the executive function measures using NART-R errors as co-variate. Results from the Modified Six Elements test were not entered into formal statistical analyses, for reasons explained later in this section.

Some variables were transformed, as detailed above and in the footnotes of Tables 6 and 7. The numbers of participants who completed each test varied slightly (e.g. due to time running out for testing). Some values were excluded from the analyses as they were more than 3 standard deviations from the group mean, and details of these outliers are given in footnotes to the results tables. Comparisons between the BPD group and the Control-I group are shown in Table 6.

Table 6: ANCOVAs comparing the BPD group with the Control-I group on executive function measures and WAIS-R similarities, with NART-R errors used as a co-variate.

Variable	BPD	n	Control-I	n	F-value	df[c]	p-value
Rule-Shift errors[a]	2.71 (2.95)	17	1.23 (1.59)	13	1.357	34	0.254
Hayling A time[a]	12.5 (7.96)	16	9.31 (8.19)	13	2.645	33	0.116
Hayling B time[a]	31.44 (17.45)	16	32.69 (22.35)	13	0.068	32	0.796
Hayling errors[a]	7.75 (9.10)	16	5.38 (9.45)	13	0.924	32	0.345
Letter Fluency	44.50 (13.39)	16	42.58 (9.05)	12[b]	0.222	31	0.642
Trails B[a]	85.31 (43.14)	16	62.85 (24.22)	13	3.308	33	0.080
Similarities	16.12 (6.20)	17	19.46 (3.45)	13	2.943	34	0.098

[a]Log transformations were performed on these variables for the analysis. The group means and standard deviations provided in the table are not transformed.
[b]One participant excluded from this group for this analysis as the score obtained was more than 3 standard deviations above group mean.
[c] Error degrees of freedom. Group degrees of freedom is 1 in all cases.

As can be seen from Table 6, the BPD group was not significantly different from the Control-I group on any of the executive function measures when analysed by ANCOVA with NART-R errors used as a co-variate. There was a trend for the BPD group to score lower on Similarities (p=0.098) and Trails B (p=0.080), and the comparison between the two groups for Hayling A time (p=0.116) approached the level of significance conventionally regarded as a trend. The analyses were repeated using the Control-II group, and are presented in Table 7.

Table 7: ANCOVAs comparing the BPD group with the Control-II group on executive function measures and WAIS-R similarities, with NART-R errors used as a co-variate.

Variable	BPD	n	Control-II	n	F-value	df[c]	p-value
Rule-Shift errors[a]	2.71 (2.95)	17	1 .10 (1.33)	20	1.461	34	0.235
Hayling A time[a]	12.5 (7.96)	16	9.30 (8.46)	20	3.198	33	0.083
Hayling B time[a]	31.44 (17.45)	16	31.84 (19.97)	19[b]	0.051	32	0.823
Hayling errors[a]	7.75 (9.10)	16	4.85 (8.03)	19	0.982	32	0.329
Letter Fluency	44.5 (13.39)	16	43.83 (10.00)	18[b]	0.346	31	0.561
Trails B[a]	85.31 (43.14)	16	62.95 (22.89)	20	3.118	33	0.087
Similarities	16.12 (6.20)	17	20.55 (3.28)	20	4.45	34	0.042*

[a]Log transformations were performed on these variables for the analysis. The group means and standard deviations provided in the table are not transformed.
[b]One participant excluded from this group for this analysis as the score obtained was more than 3 standard deviations above group mean.
[c] Error degrees of freedom. Group degrees of freedom is 1 in all cases.
*Result significant at the 0.05 level.

Table 7 illustrates that the BPD group were significantly poorer than the Control-II group on the Similarities (p=0.042), and that there was a trend towards poorer performance in the BPD group for Trails B (p=0.087) and Hayling A time (p=0.083). None of the other ANCOVA comparisons between the BPD group and the Control-II group was significant.

In order to investigate whether poorer performance on the Similarities in the BPD group was related to the level of depressive symptoms, a correlation between scores on this task and on the BDI-II was performed within the BPD group, with NART-R errors partialled out as a factor. This revealed a trend towards a negative relationship between

BDI-II and Similarities (i.e. higher levels of depressive symptoms were possibly associated with lower scores on Similarities: R= -0.430, p=0.085). Similar partial correlations between the Similarities and the IIP-PD (R=0.437, p=0.103) and the Similarities and the BIS-11 (R=0.396, p=0.128) were not significant. This suggests that there was no relationship between SCT performance and either personality pathology or impulsivity as measured by these instruments, although the partial correlation between Similarities and IIP-PD approached the level of significance conventionally regarded as a trend.

As mentioned earlier, the results for the Modified Six Elements test were not entered into formal analysis. This was because on both the raw and profile scores for the test, there were only two scores obtained across all participants (5 or 6 out of 6 for raw scores, and 3 or 4 out of 4 for profile scores), reflecting those who had scored perfectly on the test (6 on raw scores, 4 on profile scores) and those who had made one error (5 on raw scores, 3 on profile scores). In addition, there were very few errors made in any of the groups (1 in the BPD group, 1 in the Control-I group, and 3 in the Control-II group). It was thought that a formal statistical analysis would not be appropriate as the results would be dependent on such a small proportion of the sample. However, the substantive point which can be taken from participants' performance on the Modified Six Elements is that the BPD group did not experience significant difficulties with this task, and performed comparably to the control groups.

3.5 Predicaments test

The Predicaments test yields two main measures: the personal solution quality and optimal solution quality. Each of these is made up of scores for problem appreciation, social appropriateness and effectiveness. Other variables from the Predicaments test are the number of prompts given (an indication of participant's memory for the scenarios), the solution fluency score (number of solutions generated in the fluency sections of the test) and the average solution quality (average solution quality of ideas

generated in fluency section). The Predicaments test was completed by all of the BPD group, and all but one of the control subjects (this subject had to cut testing short due to another commitment).

T-tests revealed that there were no significant differences in the number of prompts given between the BPD group and the Control-I group (p=0.898), or the Control-II group (p=0.697).This suggests that the memory ability of the groups was comparable (underlying distributions were normal for this variable).

Tests of skewness and kurtosis indicated that a number of the other measures from the Predicaments test had underlying distributions which were not normal. Unfortunately transformations were unsuccessful in remedying this (probably because some of the measures had a strong ceiling effect). It was therefore decided that the rest of the major measures from the Predicaments would be analysed with non-parametric statistics, which meant that it was not possible to use estimated general intelligence as a co-variate.

Comparisons between the BPD group and both the Control-I and Control-II groups revealed no significant difference on the main measures of personal and optimal solution quality (see tables 8 and 9), although the difference between the BPD group and the Control-II group on personal solution quality approached the level of significance conventionally regarded as a trend (p=0.107). In order to investigate whether there were group differences on the sub-measures which make up optimal and personal solution quality, but which were masked by the summing process, a series Mann-Whitney tests was performed on these sub-measures (bearing in mind that any significant results should be interpreted with caution in view of the increased risk of type-I error produced by this procedure). None of these follow-up comparisons was significant. The results of these analyses are also provided in Table 8 (BPD versus Control-I) and Table 9 (BPD versus Control-II).

Table 8: Mann-Whitney tests comparing BPD and Control-I groups for solution quality, problem appreciation, social appropriateness and effectiveness (from both personal and optimal perspectives.

Perspective	Measure	BPD (n=17)	Control-I (n=12)	U[a]	p
Personal	Solution Quality	18.82 (3.17)	20.42 (2.78)	70.0	0.166
	Problem Appreciation	7.41 (0.80)	7.42 (9.00)	100.0	0.948
	Social Appropriateness	5.59 (1.58)	6.42 (1.51)	71.5	0.180
	Effectiveness	5.82 (1.55)	6.58 (1.00)	74.0	0.227
Optimal	Solution Quality	20.12 (3.53)	20.58 (3.12)	97.0	0.845
	Problem Appreciation	7.35 (0.93)	7.50 (0.90)	92.5	0.679
	Social Appropriateness	6.35 (1.32)	6.33 (1.72)	97.0	0.845
	Effectiveness	6.41 (1.66)	6.75 (1.06)	99.0	0.913

[a]Mann-Whitney U test statistic.

Table 9: Mann-Whitney tests comparing BPD and Control-II groups for solution quality, problem appreciation, social appropriateness and effectiveness (from both personal and optimal perspectives.

Perspective	Measure	BPD (n=17)	Control-II (n=19)	U[a]	p
Personal	Solution Quality	18.82 (3.17)	20.37 (2.31)	110.0	0.107
	Problem Appreciation	7.41 (0.80)	7.36 (0.76)	153.0	0.802
	Social Appropriateness	5.59 (1.58)	6.37 (1.30)	114.0	0.138
	Effectiveness	5.82 (1.55)	6.63 (0.83)	112.0	0.121
Optimal	Solution Quality	20.12 (3.53)	20.90 (2.83)	145.0	0.616
	Problem Appreciation	7.35 (0.93)	7.47 (0.84)	151.5	0.754
	Social Appropriateness	6.35 (1.32)	6.47 (1.50)	148.5	0.684
	Effectiveness	6.41 (1.66)	6.94 (0.97)	143.0	0.573

[a]Mann-Whitney U test statistic.

Given the absence of significant group differences for the main measures of the Predicaments test, the solution fluency and average solution quality measures are of less interest than they might otherwise have been. However, for the sake of completeness these were analysed using Mann-Whitney tests. There were no significant differences between the BPD group and the Control-I group on solution fluency

(p=0.152) or average solution quality (p=0.711). There was equally no significant difference between the BPD group and the Control-II group on average solution quality (p=0.661). However the BPD group scored significantly less on solution fluency compared to the Control-II group (p=0.049). It must be borne in mind that it was not possible to partial out general intelligence for these analyses, so this significant result must be treated with caution, especially as the difference between the BPD group and Control-II group on estimated general intelligence approached the level of significance conventionally regarded as a trend (see section 3.2).

Chapter 4: Discussion

4.1 Re-statement of hypotheses, previous evidence and main statistical results.

The aim of this study was to test two hypotheses: (i) that individuals with BPD have measurable deficits on tests of executive functioning, and (ii) that individuals with BPD have a deficit in theory of mind which is detectable with an appropriately advanced task. Evidence from a variety of sources was used to arrive at these proposals. With regard to the first hypothesis, brain-scanning evidence of frontal lobe abnormalities in the disorder (e.g. Lyoo et al., 1998, De la Fuente et al., 1994) suggests that there may be deficits in cognitive functions which are associated with the frontal lobes, including executive functioning. Such deficits may help to explain some of the symptoms of the disorder, including interpersonal difficulties, impulsivity, and dissociation. Previous studies have produced only tentative evidence of executive dysfunction in BPD, (e.g. van Reekum et al., 1993), but it was hypothesised that the tasks in this study which were designed to be open-ended would be more likely to detect impairments (as there is some evidence that such tasks have better validity: e.g. Shallice and Burgess, 1991).

Brain-scanning evidence of frontal lobe abnormalities also informed the hypothesis of a theory of mind deficit in BPD, as there have been suggestions that theory of mind ability may be linked to frontal lobe function (e.g. Stone et al., 1998, Flecher et al., 1995). A deficit in theory of mind may help to account for some of the symptoms of BPD, including interpersonal difficulties, affective instability and inappropriate anger. Although there have been no previous studies of theory of mind in BPD, there is evidence that individuals with the disorder have deficits in abilities which appear to be closely related to theory of mind. One of these is reflective functioning (Fonagy et al., 1996), which is proposed by some as a core deficit in BPD (e.g. Fonagy et al., 2000). The other is what

Levine et al., (1997) termed "emotional processing", which was assessed in their study using a task that closely resembles some theory of mind measures.

The major significant results of the study were that the BPD group performed more poorly compared to both control groups on the Story Comprehension test, and had poorer scores compared to the Control-II group on the Similarities. There were no significant group differences on any of the other main measures used in the study.

4.2 Theory of mind measures

4.2.1 Interpretation of the theory of mind results.

The results of the study indicated that the BPD group performed significantly worse on the theory of mind measures (when analysed together in a MANCOVA) than either the Control-I group or the Control-II group. Follow-up analyses suggested that this overall result was due to performance on the Story Comprehension test (SCT), and that there were no group differences in performance on the Happe Strange Stories test (HSST). Section 4.2.2 discusses additional between-group analyses in which depressive symptoms were co-varied, and the implications of the results for the relationship between BPD, depression and theory of mind.

The findings of the study appear to provide some support for the hypothesis of a theory of mind deficit in BPD. However, as both the HSST and the SCT aim to measure the same construct, the disjunction between the groups' performance on these two tasks requires some examination. One possible explanation for this result is that the HSST was unable to detect a subtle mentalising deficit in the BPD group because it may measure theory of mind at a lower level than the SCT. Plausibility is lent to this suggestion by the fact that the HSST was developed primarily for use with children and individuals with developmental disorders, and by the fact that in the original studies reporting the two measures, normal adults scored 99% correct on the HSST (Happe,

1994) compared with 89% correct on the SCT (Channon and Crawford, 2000). However, suggesting that the HSST measures theory of mind at a lower level must be squared with the fact that none of the groups performed at the test's ceiling (BPD group mean =14.70; Control-I group mean=14.76; Control-II group mean=15.28; all scores out of 18). On the face of it, this non-ceiling performance suggests that the HSST was assessing theory of mind at a high enough level for the control groups to not score perfectly.

The probable reason for the difference in control group performance between this study and the study by Happe (1994), in which normal adults scored 99% correct, is that a different set of scoring criteria was used in this study (Janes et al., 2000). The major relevant difference between the two scoring systems is that the Janes et al. criteria gives two points to correct answers that use mental state explanations, and only one point to correct answers that give physical state explanations, whereas Happe's original system does not make such a distinction. One could propose that the use of these more sophisticated scoring criteria made the HSST a more advanced test of theory of mind skills, (which explains the non-ceiling performance of the control groups in this study) and means that the measure should have been able to detect a deficit in theory of mind ability in BPD, if there were one.

Against this suggestion, it could be argued that expanding the scoring criteria may have produced a non-ceiling performance because it resulted in the scoring system making some distinctions between answers that did not genuinely reflect different levels of understanding of the story. This point may be illustrated with the following story from HSST:

Today, Katy wants to go on the swings in the playground.
But to get to the playground she knows she has to pass old Mr Jones' house.
Mr Jones has a nasty fierce dog and every time Katy walks past the house the dog jumps up at the gate and barks. It scares Katy awfully, and she hates walking past the house because of the nasty dog. But Katy does so want to play on the swings.

Katy's mother asks her, "Do you want to go out to the playground?" Katy says, "No".

Is it true what Katy says?

Why does she say she doesn't want to go to the playground, when she so wants to go on the swings that are there?

Under the Janes et al. criteria, "because she is scared" counts as a two-point (mental state) answer to the second question, whereas "because of the scary dog" is awarded only one point (as it counts as a physical state answer). It does not seem clear that an adult giving the second answer has a less rich understanding of the motivations behind Katy's reply than an adult giving the first answer. So one explanation why none of the groups in the study scored at ceiling for the HSST may have been that the Janes et al. criteria resulted in some answers being marked down for reasons which merely reflected the particular way in which an answer was phrased, rather than a substantive difference in theory of mind ability. This would mean that the failure of the control groups to perform at ceiling on the HSST in this study should not necessarily be taken as an indication of the test having a high enough level of sophistication to detect group differences in theory of mind.

It is notable that the SCT does not distinguish between different levels of sophistication of correct answers (i.e. all answers are scored either 1 or 0), and that the instructions for its administration allow the use of neutral prompts in instances where an answer is ambiguous, or where it is unclear whether the participant has a correct understanding of the motivations behind the character's behaviour (unlike the HSST, which does not allow such prompts). One might argue that these two differences mean that a participant on the SCT who has a correct understanding of the story is less likely to be marked down because of the way in which they happen to phrase their answer. Thus, the non-ceiling performance of the Control-I and Control-II groups on the SCT (10.54

and 10.74 out of 12 respectively) seems more likely to reflect the measurement of theory of mind at an appropriately advanced level.

In summary, the results of the study appeared to provide support to the hypothesis of a subtle theory of mind deficit in BPD, as the BPD group performed worse overall than the Control-I or Control-II group on the theory of mind tests when both measures were analysed together (however see section 4.2.2 for a discussion of the possible influence of depression on theory of mind performance). Further examination revealed that this result appeared to be due to just to group differences on the Story Comprehension test, as there were no significant group differences on the Happe Strange Stories test. However the lack of a significant difference on the HSST is consistent with the hypothesis if one proposes that the theory of mind deficit was detectable on the SCT, but did not manifest itself on the HSST because this test measures theory of mind ability at a lower level.

4.2.2 Possible influence of depression on theory of mind result.

There was no significant correlation between SCT scores and the BDI-II within the BPD group, when estimated general intelligence (number of NART-R errors) was partialled out as a factor. However, when BDI-II scores were used as an additional co-variate for ANCOVA analyses of SCT scores, this rendered the effect of group non-significant for comparisons with both the Control-I and Control-II groups. This result underlines the possibility that TOM deficits in BPD may be related to depression rather than a diagnosis of BPD specifically. If this were the case, then it may have important implications for cognition in major depression and other disorders in which depressed mood is prominent, as thus far there have been no published studies investigating theory of mind ability in depression.

However, it could be argued that an ANCOVA comparison between a group of BPD participants and healthy controls using BDI-II scores as a co-variate is not the most ideal

method for teasing apart whether any theory of mind deficits are attributable to BPD or depression, and that a control group of depressed participants would be better placed to do this (as was originally envisaged for this study). In this study's sample, there was no overlap in BDI-II scores between the BPD group and either the Control-I or Control-II groups, and thus the association between group membership and depressive symptoms in the study was such that one could correctly assign group membership for every participant in the study by reference to their BDI-II score. This state of affairs means that all information about theory of mind at high levels of depression is provided by members of the BPD group, and that all information about theory of mind at low levels of depression is provided by members of the healthy control group. From this, one might argue that the study's ability to use co-variation as a tool to determine whether the BPD group's poor performance was due to co-morbid depression or BPD is hampered by the fact that only BPD participants had significant levels of depressive symptoms. A control group of depressed participants would provide the opportunity of seeing whether theory of mind is influenced by high levels of depressive symptoms in the absence of any axis-II pathology, something which was not possible with the two groups recruited for this study. However, as mentioned above, the result of the ANCOVA using BDI-II as a co-variate does underline the possibility that future studies may find that a theory of mind deficit in BPD may be better attributed to co-morbid depression than personality disorder.

4.2.3 Relationship between theory of mind and BPD symptoms.

It was argued in section 1.3 that a deficit in theory of mind may contribute to the production of BPD symptoms and characteristics, such as interpersonal difficulties and inappropriate anger. However it is difficult to assess what explanatory power the apparent theory of mind deficit found in this study may have in terms of the symptoms of the disorder. At present it is not clear whether the statistical difference between the BPD group and the control groups on the SCT would translate into a clinically significant difference in the ability to understand others' mental states in real life (and thus

contribute to BPD symptoms). Correlational analysis of the relationship between SCT performance and the IIP-PD within the BPD group was not significant (with estimated intelligence partialled out), which suggests that there was no clear link between the degree personality pathology (as measured by this instrument) and theory of mind. At first sight, this lack of association between degree of theory of mind deficit and personality pathology within the BPD group would seem to go against the suggestion that theory of mind has a role in producing BPD symptoms (as one might expect that a greater theory of mind deficit would translate into more severe personality pathology). However, the IIP-PD was developed as a screening tool for personality disorder in general, and which leaves open the possibility that future studies may find a relation between theory of mind and a more specific measure of BPD symptomatology (e.g. the Diagnostic Interview for Borderlines: Zanarini et al., 1989). In this context it is notable that Fonagy et al. (1996) found that patients with BPD had poorer reflective functioning than patients of other diagnoses, many of whom also had personality disorders, which suggests that a general measure of personality pathology may be less able to predict mentalising capacity in BPD than a measure which is more specific to the disorder.

There was also no significant relation between SCT performance and the BIS-11 (with estimated intelligence partialled out), which suggests that there was not a clear link between degree of impulsivity and theory of mind ability. However such a link was more specifically predicted for the executive function measures (see section 1.3.3 and 4.3.3)

4.2.4 Wider discussion of theory of mind in BPD.

As reported in the review of literature provided in section 1.2.2 there have as yet been no other published studies investigating theory of mind in BPD. However the results of the study broadly fit in with those of Fonagy et al. (1996), who assessed reflective functioning in BPD, and Levine et al., (1997), who measured emotional processing in a task which is similar to many theory of mind tests.

If there is a deficit in theory of mind in individuals with BPD, it is worth considering what the characteristics of this deficit might be. From the evidence of this study, one important consideration is that the deficit seems likely to be a subtle one: the BPD group performed at an equivalent level to the control groups on the Happe Strange Stories test (HSST), which has been used in other studies as a benchmark of advanced theory of mind ability (e.g. Blair et al., 1996, Bach et al., 2000). It was only on the SCT (which section 4.2.1 argues is a more advanced task) that group differences were found in this study.

Some (e.g. Channon and Crawford, 2000) have suggested that tests such as the SCT and HSST can be seen as having an executive component, as the subject typically must suppress an explicit or superficial reading of the story in order to arrive at a correct explanation of the behaviour of the main character. However the results of this study suggest that a deficit in theory of mind in BPD is not likely to be secondary to executive dysfunction, as there was only tentative evidence of executive function deficits (see section 4.3.1), whereas there was stronger evidence that the BPD participants had difficulty with the most advanced theory of mind measure used. This is particularly striking when one considers the results of the comparisons between the BPD and the Control-I group: there were no significant differences between the groups on a wide variety of executive function measures, yet the BPD group performed significantly more poorly on the SCT.

Another important consideration is whether a theory of mind deficit in BPD should be conceived of as a stable trait which applies equally across different situations, or whether the deficit is only manifest under certain conditions. In autism, the archetypal instance of a disorder associated with theory of mind difficulties, the proposed deficit would appear to be of the former, global, type (e.g. Baron-Cohen et al., 1985). One might argue that the difficulty with proposing a global theory of mind deficit in BPD is that there is anecdotal evidence that at times individuals with BPD appear to have an acute awareness of the mental states of others (e.g. during periods where the individual

functions with a high degree of apparent competence). The reflective function hypothesis of BPD (e.g. Fonagy et al., 2000) deals with this issue by proposing that poor mentalising ability in BPD is restricted to certain problematic internal working models and attachment contexts (e.g. ones relating to previous maltreatment or abuse). In such contexts, it is proposed that individuals with BPD inhibit reflective functioning, a defensive style which has its roots in early maltreatment, when it was protective for the child not to reflect upon the content of the care-giver's mind. Outside of these contexts, however, it is proposed that reflective capacity is retained in some form.

The results of this study would seem to go against this suggestion of a circumscribed deficit in mentalising proposed by Fonagy et al., (2000), as theory of mind in this study was measured in the context of hypothetical stories rather than personal attachment history. However, it could be argued that although the scenarios in the Story Comprehension test (SCT) were not designed to be personally relevant to any individual participant, some of the stories may have been reminiscent in some way of difficult relationships for the BPD participants, and may have activated problematic internal working models. A qualitative examination of some of the replies for the BPD group suggests that this may have been the case for some of the participants. The following story from the SCT provides an example:

> *Marie dreaded her trips to meet her husband's relatives because they were so boring. Most of the time they all sat in awkward silence, and this occasion was no different. On the way home, Marie's husband asked her how she found the visit. Marie said "Oh, marvellous. I could hardly get a word in edgeways."*
>
> *Why did Marie say that?*

Some of the BPD participants answered with the suggestion that maybe Marie was frightened of her husband and she had to lie to protect herself. In these cases one could plausibly speculate that the story may have activated internal working models relating to

previous experiences of maltreatment, which distorted the participants' explanations of the motivation behind Marie's utterance. Having said this, such clear cut examples of the potential influence of problematic internal working models upon SCT answers were not very common (although no formal analysis of responses for signs of such influence was carried out). In general the errors of participants in the BPD group appeared to be more straightforward in their misinterpretations (e.g. suggesting that Marie had changed her mind about her husband's relatives and had decided that she liked them after all). Overall it is probably difficult to determine the degree to which poor performance of BPD group may have been influenced by the activation of problematic internal working models, but the apparent neutrality of most of the stories in the SCT suggests that this is unlikely to be the principal reason for poorer performance.

Another possible way of reconciling the poor performance of the BPD group on the SCT with anecdotal evidence of sometimes acute awareness of others' mental states, is to suggest that where this apparent awareness occurs, it is partly non-conscious. Thus, although on occasion the individual with BPD may be able to act as though he or she were able to skilfully fathom the mental worlds of others, they may not be able fully to reflect upon this process. This is an idea proposed by Fonagy et al., (2000), who draws on suggestions that there can sometimes be a dissociation between verbal responses and implicit understanding in theory of mind tasks. Clements and Perner (1994) found that most children between the ages of 2 years and 11 months and 4 years and 6 months correctly indicated by their gaze that they were expecting the protagonist in a false belief task to look in the wrong location for an object they had previously hidden. However, most children between these ages did not give the correct *verbal* response. If such a dissociation between being able to verbalise and being able to act upon mental state information were to sometimes apply for individuals with BPD, it might mean that the BPD participants were poorer at explaining the motivations of the characters in the SCT because an explicit verbal explanation of motivation was required in the task.

However it is also worth noting that not all participants in the BPD group scored poorly on the SCT: 5 out of 17 scored above the group means for the control groups. This seems to suggest that, if there is a theory of mind deficit in the BPD, then it probably only applies to a proportion of individuals who have received the diagnosis. This in itself may help to explain the anecdotal phenomenon of acute awareness of others' mental states in BPD: it may be that it is only those with more advanced theory of mind abilities who are able to display the type of behaviour which would suggest such an awareness.

At present there is not enough evidence to choose between these possibilities, and the question of how global a theory of mind deficit in BPD is will have to be addressed in future research. The answer to this question may have implications for what is the most plausible account of the cause of a theory of mind in BPD. One might argue that a deficit which appears to be confined to particular situations (e.g. ones which are relevant to the attachment history of the individual) would be most plausibly explained as being the result of a psychological defence. However a more global deficit in theory of mind ability (such as is proposed in autism: Baron-Cohen et al., 1985) might fit in more easily with the notion of a biologically based impairment of a theory of mind "module", perhaps in the frontal lobes (e.g. Stone et al., 1998).

4.2.5 Summary.

The findings of this study broadly concur with those of two other studies in which constructs closely related to theory of mind were investigated in BPD, although (as mentioned in section 4.2.2), the role of depression in any theory of mind deficits in BPD will have to be considered carefully in future studies.

The performance of the BPD group suggests that if there is a deficit in theory of mind in BPD then it is a subtle one, which probably only applies to a sub-set of individuals diagnosed with the disorder. There is only limited evidence that this deficit is secondary to executive dysfunction. At present there is not enough evidence to say whether a

deficit in theory of mind is likely to apply equally across all situations, or whether it may be more apparent in certain contexts relevant to the individual's attachment history.

4.3 Executive function measures

4.3.1 Interpretation of executive function results.

Analysis of the results revealed that there were no significant differences between the BPD group and the Control-I group on any of the more abstract[1] executive function measures, with a trend for the BPD group to score more poorly on the Similarities and the Trails B. However, comparisons between the BPD group and Control-II group on these measures indicated that the BPD group was significantly poorer at the Similarities (with a trend for poorer performance in the Trails B and the Hayling A). With regard to the Predicaments test, there were no significant differences between the BPD group and either of the control groups on any of the main measures, although the comparison between the BPD group and the Control-II group on personal solution quality approached the level of significance conventionally regarded as a trend. There was a significant difference between the BPD group and the Control-II group for solution fluency.

This pattern of results suggests that the hypothesis of executive dysfunction in BPD is given only tentative support by the study. This is despite the use of a wide range of executive measures, which aimed to measure a variety of different aspects of executive functioning, including response inhibition (Hayling B and Hayling errors), response initiation (Hayling A), set-shifting (Rule shift, Trails B), planning (Six Elements), conceptual reasoning (Similarities), and problem solving (Predicaments). The significant difference between the BPD group and the Control-II group on the Similarities is

[1] i.e. excluding Predicaments test.

interesting, suggesting that concrete thinking and a lack of conceptual flexibility may be more common in individuals with BPD. However, given that many statistical comparisons were carried out for the executive function measures, this single significant result should be interpreted with caution.

Although there were no significant differences on the main measures of the Predicaments, the finding of reduced solution fluency in the BPD group compared to the Control-II group is interesting, as it suggests that individuals with BPD may consider fewer options before deciding on a course of action in interpersonal situations. However, this did not translate into any significant difference in the quality, problem appreciation, social appropriateness, or effectiveness of solutions to the problems. Also, it was not possible to co-vary for intelligence for the analysis of solution fluency, so some of the group difference on this measure may be attributable to the estimated general intelligence of the Control-II group (the group difference between Control-II and the BPD participants for estimated IQ approached the level of significance regarded as a trend: see section 3.2).

If the hypothesis of executive dysfunction in BPD were to be correct, then one could propose a number of reasons why the results of this study provide only tentative support for it. The first of these is that sample sizes were small (especially in the Control-I group), which obviously reduced the power of statistical analyses to find group differences which might have been apparent in larger samples. The second possibility one might propose is that the inclusion of several individuals in the Control-II group whose personality disorder status was not certain may have rendered the group more similar in its cognitive profile to the BPD group, and thus biased against finding group differences between the Control-II and BPD groups. However, an inspection of the group means for the Control-I and Control-II groups does not seem to bear this suggestion out, as there is little difference between the two groups on any of the variables (see tables 6, 7, 8 and 9).

With regard to the Predicaments test, one could argue that another reason for the failure to find group differences is that the administration of this task provides the participant with a ready-made problem-solving structure. This is because participants are asked to generate as many solutions as possible to the dilemma (the solution fluency section of the test) before selecting their best and personal solutions to the problem. This procedure is essentially the same as the brain-storming technique which is sometimes recommended in cognitive-behavioural approaches to problem-solving (e.g. Hawton and Kirk, 1989). One hypothesis might be that the BPD group were able to benefit from this problem-solving structure as it guided them to consider several alternatives before choosing a course of action, and that if they had been asked to provide a solution without this procedure, a clearer deficit may have been apparent. This hypothesis would assume that the anterior lesion group in the original study reporting the Predicaments (Channon and Crawford, 1999), who *were* impaired on the task compared to controls, were so severely dysexecutive that they were not able to benefit significantly from the problem-solving structure provided by the test.

4.3.2 Possible influence of depression upon executive function result.

In order to investigate the relationship between poor performance of the BPD group on the Similarities (when compared to the Control-II group) and depressive symptoms, correlations within the BPD group were performed with the BDI-II, with NART-R errors partialled out as a factor. This revealed a trend for a negative correlation with Similarities, which suggests that the level current depressive symptoms may have been a factor in the BPD group's performance on this task. This raises the possibility that the poor performance of the BPD group on the Similarities (compared with the Control-II group) may have been influenced by co-morbid depression. However, section 4.4.1 discusses the possible advantages of using a depressed control group in investigating the relationship between depression and executive functioning in BPD in future studies.

4.3.3 Relationship between executive functioning and BPD symptoms.

As with the theory of mind results, it is difficult to assess what the explanatory power of the tentative indications of executive dysfunction found in this study may be. It was argued in section 1.3 that executive dysfunction may help to account for a number of symptoms of BPD, including impulsivity and interpersonal difficulties. Although there were significant differences in performance between the BPD group and the Control-II group on the Similarities, correlational analyses within the BPD group between this measure and the IIP-PD were not significant. This suggests that poor performance within the BPD group did not relate to degree of personality pathology as measured by this instrument (although the level of significance approached that conventionally regarded as a trend). However, (as argued in section 4.2.2 with regard to the theory of mind measures) it may be more appropriate to investigate the relationship between the Similarities and a more specific measure of BPD severity. There was equally no significant correlation between the Similarities and the total score from the Barratt Impulsiveness Scale, which suggests that poor Similarities performance was not related to impulsivity (which is a possible symptom of BPD). Thus the link between the poor performance on the Similarities (in comparison to the Control-II group) and BPD symptoms is as yet unclear.

4.3.4 Wider discussion of executive functioning in BPD

In the review of previous studies investigating executive functioning in BPD (section 1.2.2), it was argued that the findings of this body of research were inconsistent and that there was so far only tentative evidence of executive dysfunction in BPD, despite brain-scanning evidence of frontal lobe abnormalities in the disorder. This study appears to add to rather than disambiguate this inconclusive picture, as again only tentative evidence of executive dysfunction was obtained. It is notable that the study used a much wider variety of executive function measures than previous ones (see section 1.2.2), and

it included measures which were designed to be open-ended and realistic, which some have suggested may have greater ecological validity.

There are a number of ways one might interpret this pattern of results with regard to the hypothesis of executive dysfunction in BPD. One of these is to suggest that the hypothesis has yet to be tested properly because sample sizes of all the studies so far conducted are all relatively small, which obviously reduces the likelihood of finding group differences. This is an important possibility to consider, and it may be that these small sample sizes reflect particular difficulties in the recruitment of individuals with BPD for such studies (e.g. disruption of testing plans due to frequent crises, reluctance to trust an unfamiliar researcher, or unwillingness to elect to enter a testing situation because of feelings of paranoia). However, many studies of executive dysfunction in other populations have used similar sample sizes (e.g. Channon and Crawford, 1999; Ozonoff et al., 1991).

Another interpretation of the failure of studies to find clear evidence of executive dysfunction would be to suggest that deficits only apply to a sub-group of individuals with BPD. For example, Streeter et al. (1995) propose that there are three distinct aetiological sub-categories in BPD: those with acquired brain damage (e.g. history of head injury), those with long-standing developmental disorders (e.g. learning disability or ADHD) and those without any history of acquired or developmental brain dysfunction. If such distinct sub-groups were to exist, then it could be that executive dysfunction applies only to the organic groups, which would have important implications for studies of executive function in BPD. Firstly, this would make the problem of small sample sizes more acute, as cognitive heterogeneity in the BPD group of any given study would make it harder to detect group differences. A second implication would be that the varying exclusion criteria of different studies would have a strong impact on the result obtained. Some studies treat head injury and neurological disease as complicating factors to be excluded (e.g. Judd and Ruff, 1993; Sprock et al., 2000), while others treat these factors as central to possibility of cognitive impairments in BPD, and therefore include

participants with "organic" histories (van Reekum et al., 1993). Such differences may obviously impact upon the likelihood of any one study finding group differences on executive function measures, and thus probably contribute to the overall picture of inconclusiveness in this area. The present study also did not exclude BPD participants with a history of head injury or neurological disease, although the numbers of such participants were low. Only three of the sample had had an injury that resulted in unconsciousness or a brief hospitalisation, and one participant had had a brief period of epileptic seizures during childhood.

A further explanation for the inconclusive findings may be that the hypothesised executive dysfunction in BPD is most evident at times of high emotional arousal (perhaps showing itself in impulsiveness, poor judgement, or difficulties in selecting socially appropriate courses of action) and does not manifest itself to the same extent in more neutral situations (such as neuropsychological testing). Sprock et al. (2000) attempted to address this question by including an emotional Stroop task in their study. In this, the time taken to name the ink colours of a list of anger and sadness words is compared to the time taken to name ink colours of neutral words, with the assumption that individuals who are more susceptible to emotional interference on the task will be show a greater difference in total time between the two conditions. In the end Sprock et al. found that the BPD group showed no difference between the conditions, but they propose that this experimental manipulation may have been too weak to produce emotional interference.

A sufficiently powerful experimental manipulation of emotional arousal on an abstract test of executive function may be difficult to achieve, but a modified Predicaments test using vignettes which focus upon different themes (e.g. threat or loss, rather than awkwardness) may be a way of testing this hypothesis. If such an approach did find that individuals with BPD had impairments in interpersonal problem solving with the emotionally arousing stories, the challenge would be to try and tease out whether this was because the executive abilities of individuals with BPD were *differentially* affected

by emotional arousal compared to controls, or whether it was simply because they experienced more affect in response to the story. If the latter were the case, this would suggest that the poorer performance was not related to an executive deficit per se.

Finally, another interpretation of the pattern of results in this area is to suggest that there is indeed no clear executive dysfunction in BPD. It may be that the frontal lobe abnormalities which have been reported in the disorder are not robust, or that they simply do not have any particular impact on executive abilities. For example Baddeley and Della Sala (1998) argue that frontal lesions do not necessarily produce executive function deficits, and that an anatomical link between the executive functions and the frontal lobes should not be assumed. It might be that frontal abnormalities in BPD are more related to other functions which may be important in accounting for the symptoms of BPD, such as emotional regulation (e.g. Meares, 1999) or theory of mind (e.g. Stone et al., 1998).

4.3.5 Summary

Several possible explanations for the inconclusive evidence of executive dysfunction in BPD have been discussed: the small sample sizes of studies conducted so far, the possibility that only sub-groups of individuals with BPD have executive difficulties, the possibility that executive dysfunction is related to emotional arousal, and the possibility that BPD is not characterised by executive dysfunction at all. At present there does not appear to be sufficient evidence to chose between these different explanations.

4.4 Concluding comments

4.4.1 Limitations of the study.

This study has several limitations which restrict its findings to some degree. The first of these is that sample sizes were small, which restricted the power of statistical analyses.

This reduces the confidence with which the results can be seen as tests of the study's hypotheses in the case of measures where no group differences were found.

The second limitation was the composition of the healthy control group. Due to recruitment difficulties, there were only 13 healthy control participants who scored below the cut-off for the IIP-PD (the Control-I group), and therefore comparisons between the BPD group and the Control-II group (which was comprised all of those recruited as healthy controls, regardless of IIP-PD score) are limited by the uncertain personality disorder status of some of this sample. However, as argued in section 3.1 part (ii), this is likely to bias against rather than for finding group differences, and in the end there was little difference between the Control-I and Control-II group means. The study would have been strengthened if it had been possible for all participants to have been given a complete structured diagnostic interview for axis-I and axis-II disorders, although this would have lengthened the battery considerably and may have reduced the proportion of potential participants who were willing to take part.

Although a majority of individuals diagnosed with BPD are female (DSM-IV, APA, 1994), the fact that only women were recruited to the study restricts the generalisability of its findings. There have been suggestions that the males and females with BPD may differ with respect to certain characteristics (e.g. Andrulonis and Vogel, 1984, propose that females are more likely to fall into a non-organic sub-type of the disorder) which may mean that different results would be obtained if this study were replicated in a male sample. Also Baron-Cohen et al. (1997) report findings which suggest that males and females may perform differently on some theory of mind tasks, and again this may restrict the generalisability of the results with regard to males with BPD.

One might argue that the lack of a depressed control group in this study is another limitation. The study performed correlations between BDI-II scores and those measures where deficits were found, in order to try to assess whether these deficits were related to the severity of depression in the BPD group. However this is not ideal as it assumes that

if co-morbid depression were a factor in poor performance, then the cognitive impairment would be related to the degree of depressive symptoms, rather than simply the presence of depression. Additional between-group analyses were also performed in which depressive symptoms were co-varied, but again this was not ideal, study's only source of information about theory of mind at high levels of depression came from the BPD group. A depressed control group (such as was used by Sprock et al., 2000), would have better addressed this issue, as this would have provided the opportunity of seeing whether theory of mind is influenced by high levels of depressive symptoms in the absence of any axis-II pathology.

4.4.2 Clinical implications of study

The major clinical implication of this study's findings is that theory of mind skills may be a worthwhile focus for intervention which in BPD, especially if subsequent studies indicate that poor theory of mind is related to particular symptoms of the disorder (e.g. interpersonal difficulties) or overall severity. The Bateman and Fonagy (1999) model of psychotherapeutic intervention in BPD already has the improvement of mentalisation skills as an explicit focus for treatment, and this treatment package has been shown to be successful in producing good clinical outcomes in a randomised controlled trial. It may be that other treatment approaches that do not have such an explicit aim of improving mentalisation (e.g. Dialectical Behaviour Therapy: Linehan, 1993), would benefit from such a focus. The results of this study also raise the possibility that a mentalisation deficit in BPD may apply even in neutral contexts which are not specifically related to attachment history, contrary to the proposal of Fonagy et al., (2000). This may mean that it could be useful to target the improvement of mentalisation skills in a wide variety of types of situation, including some which appear to be more neutral for the patient. The lack of consistent evidence for executive dysfunction in this study and others means there is not presently any support for the introduction of interventions which specifically target executive difficulties in BPD. However, if subsequent research is able to reliably identify sub-groups of individuals (e.g. Streeter et al., 1995) for whom

executive dysfunction is an issue, then it may be worth considering adapting some interventions which have been used for head trauma patients with dysexecutive difficulties (e.g. Mateer, 1999).

4.4.3 Suggestions for future research

There are a number of possibilities for future investigation of executive functioning and theory of mind in BPD which are suggested by the results of this study.

As with any new finding, the theory of mind result obtained in this study with the SCT will require replication in order to establish whether it is a robust phenomenon, ideally with the use of a depressed control group in order to tease apart the relative influences of BPD diagnosis and co-morbid depression upon any theory of mind deficit. The use of other developmentally advanced theory of mind tests in the BPD population would also be useful with regard to replication, and may help to confirm (as argued in section 4.2.1) that the SCT assesses theory of mind at a higher level than the HSST. Suitable tests might include the Eyes task (Baron-Cohen et al., 1997) and the Faux-pas detection test described by Stone et al. (1998).

This study failed to find a relationship between SCT performance and personality pathology within the BPD group, as measured by the IIP-PD. As suggested in section 4.2.3 this may have been because the IIP-PD is a general measure of personality pathology which is not designed to be specific to BPD. Future research in this area may be able to demonstrate a relationship between theory of mind performance and a more specific measure of BPD symptomatology (e.g. DIB: Zanarini et al., 1989), which would give a stronger indication of whether theory of mind ability is related to BPD symptoms.

Another way of investigating the relationship between theory of mind and BPD symptoms would be to measure theory of mind skills before and after psychotherapeutic intervention, which would enable one to determine whether positive therapeutic outcome

was associated with changes in mentalising ability. This design may also shed light on whether higher levels of theory of mind may skills be a predictor of good psychotherapy outcome.

If the theory of mind result of this study were to be replicated, future studies may also wish to further investigate whether a mentalisation deficit in BPD is likely to apply equally across all types of situation, or whether it may be more apparent in certain contexts which are relevant to the individual's attachment history. One possible way of doing this would be to add extra stories to the SCT which would be more likely to be relevant to the attachment history of BPD participants, in order to see if theory of mind performance on these stories was any different to the neutral stories. (This would probably require a pilot study in the normal population to establish that the "attachment stories" were of the same level of difficulty as the neutral ones.)

With regard to future investigations of executive dysfunction in BPD, a study with larger sample sizes may be able to address the issue of whether some sub-groups of patients with BPD (e.g. those with a history of brain insults: Streeter et al., 1995) may have clearer executive function deficits than have been hitherto found in the disorder. If this were to be the case, and if these deficits were found to relate to BPD symptoms, then this may support the introduction of interventions targeting executive dysfunction in a proportion of BPD patients.

Section 4.3.1 suggested that a modified Predicaments test which included scenarios with emotionally charged themes (e.g. threat or loss), would provide the ideal context in which to investigate the potential effect of emotional arousal upon executive functioning in BPD. It may also be useful to omit the of the solution fluency section of the test, in order to ascertain whether individuals with BPD in this study were being helped by being asked to think of many solutions before giving their best answer (as this is a similar procedure to that seen in some CBT approaches to problem-solving: e.g. Hawton and

Kirk, 1989). This change in administration may make the task a more sensitive assessment of executive ability in an interpersonal context.

4.4.4 Conclusion.

This study investigated two hypotheses: (i) that individuals with BPD have a deficit in executive functioning and (ii) that individuals with BPD have a deficit in theory of mind skills. These hypotheses were prompted by evidence from a number of sources, including reports of frontal lobe abnormalities in BPD from brain-scanning studies (Lyoo et al., 1998, De la Fuente et al., 1997), tentative evidence from previous studies of executive dysfunction in BPD (e.g. van Reekum et al., 1993) and evidence that individuals with BPD are impaired on constructs which appear to be closely related to theory of mind (Fonagy et al., 1996; Levine et al., 1997).

The performance of the BPD group in this study indicates that there may be a subtle theory of mind deficit in the disorder, but which probably applies only to some individuals who have received the diagnosis. The results of the study support the notion that interventions focusing upon the improvement of theory of mind skills in BPD may be helpful. However, future research is needed to establish that the finding is a robust one, and that the deficit in theory of mind can be linked to BPD symptoms. At present there is not enough evidence to determine whether this deficit is likely to apply equally across all situations, or whether it may be more apparent in contexts relevant to the individual's attachment history. In addition, further analysis in this study underlined the possibility that this poorer theory of mind performance may be attributable to co-morbid depression rather than BPD diagnosis, and further research with a depressed control group is needed to investigate this issue properly.

The results of the executive function measures yielded only tentative support for dysexecutive difficulties in BPD, a finding which broadly fits in with the body of previous research conducted in this area. A number of reasons are discussed for this, including

the small sample sizes of studies, the possibility that only sub-groups of individuals with BPD have executive difficulties, the possibility that executive dysfunction is related to emotional arousal, and the possibility that BPD is not characterised by executive dysfunction at all. Future research will be needed to determine which of these explanations is closest to being correct.

References

Akiskal, Hagop S. (1994). The temperamental borders of affective disorders. Acta Psychiatrica Scandinavica. Vol 89 (379, Suppl).

American Psychiatric Association (1980). Diagnostic and statistical manual of mental disorders (3rd edition). Washington DC, American Psychiatric Association.

American Psychiatric Association (1994). Diagnostic and statistical manual of mental disorders (4th edition). Washington DC, American Psychiatric Association.

Anderson, C.V., Bigler, E.D., Blatter, D.D. (1995). Frontal lobe lesions, diffuse damage, and neuropsychological functioning in traumatic brain-injured patients. Journal of Clinical & Experimental Neuropsychology. Vol 17(6), 900-908.

Andrulonis, P.A., Vogel, N.G., (1984). Comparison of borderline personality subcategories to schizophrenic and affective disorders. British Journal of Psychiatry. Vol 144, 358-363.

Arbuthnott, K., Frank, J., (2000). Trail Making Test, Part B as a measure of executive control: Validation using a set-switching paradigm. Journal of Clinical & Experimental Neuropsychology. Vol 22(4), 518-528.

Avis, J., Harris, P.L. (1991). Belief-desire reasoning among Baka children: evidence for a universal conception of theory of mind. Child Development. Vol 62, 460-467.

Axelrod, B.N., Henry, R.R. (1992). Age-related performance on the Wisconsin Card Sorting, Similarities, and Controlled Oral Word Association Tests. Clinical Neuropsychologist. Vol 6(1), 16-26.

Bach, L.J., Happe, F., Fleminger, S., Powell, J. (2000) Theory of mind: Independence of executive function and the role of the frontal cortex in acquired brain injury. Cognitive Neuropsychiatry. Vol 5(3), 175-192.

Baddeley, A. (1986). Working memory. Oxford: Clarendon Press.

Baddeley, A., Della Sala, S (1998). Working memory and executive control. In: Roberts, A.C., Robbins, T.W. (Eds) The prefrontal cortex: Executive and cognitive functions. (pp. 9-21). New York, NY, USA: Oxford University Press.

Baron-Cohen, S., Jollife, T., Mortimore, C., Robertson, M., (1997). Another advanced test of theory of mind: evidence from very high functioning adults with autism or asperger syndrome.
Journal of Child Psychology and Psychiatry. Vol 38, 813-822

Baron-Cohen, S., Leslie, A.M., Frith, U (1995). Does the autistic child have a "theory of mind?" Cognition. Vol 21, 37-46.

Baron-Cohen, S. (1989) The autistic child's theory of mind: A case of specific developmental delay. Journal of Child Psychology and Psychiatry. Vol 30, 285-297.

Bateman, A., Fonagy, P. (1999), "Effectiveness of Partial Hospitalization in the Treatment of Borderline Personality Disorder: a Randomized Controlled Trial", American Journal of Psychiatry, v. 156, p. 1563-1569.

Beck, A.T., Steer, R.A., Brown, G.K. (1996). Beck Depression Inventory manual, (2nd edition). San Antonio: Psychological Corporation.

Bezirganian, S., Cohen, P., Brook, J.S., (1993) The impact of mother^child interaction on the development of borderline personality disorder. American Journal of Psychiatry. Vol 150(12), 1836-1842.

Blair, R. J. R., Cipolotti, L. (2000). Impaired social response reversal. A case of 'acquired sociopathy'. Brain. Vol 123(6), Jun 2000, 1122-1141.

Blair, J., Sellars, C., Strickland, I., et al., (1996). Theory of mind in the psychopath. Journal of Forensic Psychiatry. Vol 7(1), 15-25.

Bongar, B., Markey, L.A., Peterson, L.G. (1991).Views on the difficult and dreaded patient: A preliminary investigation. Medical Psychotherapy: an International Journal. Vol 4, 1991, 9-16.

Bremner, J.D., Krystal, J.H., Charney, D.S., Southwick, S.M. (1996). Neural mechanisms in dissociative amnesia for childhood abuse: relevance to the current controversy surround the "false memory syndrome". American Journal of Psychiatry. Vol 153(7) Festschrift supplement.

Bremner, J.D., Ng, C.K., Staib, L., et al. (1995). MRI-Based measurement of hippocampal volume in combat-related post-traumatic stress disorder. American Journal of Psychiatry. Vol 152, 973-981.

Burgess, P. (1997). Theory and methodology in executive function research. In: Rabbitt, P (Ed) Methodology of frontal and executive function Hove: Psychology Press.

Burgess, P., Shallice, T. (1997). The Hayling and Brixton Tests (manual). Bury St. Edmonds: Thames Valley Test Company.

Buschke H., Fuld P.A. (1974) Evaluating storage, retention, and retrieval in disordered memory and learning. Neurology. 24(11):1019-1025,

Carpenter, C.J., Gold, J.M., Fenton, W. (1993). Neuropsychological testing results in borderline inpatient. Poster presentation at the 146th Annual Meeting of the American Psychiatric Association. Reported in: O'Leary K.M. (2000). Neuropsychological testing results. In: The Psychiatric Clinics of North America. Vol 23(1).

Carlson, E.B., Putnam, F.W. (1993) An update on the Dissociative Experiences Scale. Dissociation: Progress in the Dissociative Disorders. Vol 6(1), Mar 1993, 16-27.

Channon, S., Crawford, S. (2000). The effects of anterior lesions on performance on a story comprehension test: left anterior impairment on a theory of mind-type task. Neuropsychologia. Vol 38, 1006-1017.

Channon, S., Crawford, S., (1999). Problem-solving in real-life-type situations: the effects of anterior and posterior lesions on performance. Neuropsychologia, 37, 757-770.

Channon, S. (1996). Executive dysfunction in depression: the Wisconsin Card Sorting Test. Journal of Affective Disorders. Vol 39, 107-114.

Chase, T.N., Fedio, P., Foster, N.L. et al., (1984). Wechsler adult intelligence scale performance: cortical localization by fluorodeoxyglucose F 18 positron emmision tomography.
Archives of Neurology. Vol 41, 1244-1247.

Clements, W.A., Perner, J., (1994) Implicit understanding of belief. Cognitive Development. Vol 9(4), 377-395.

Conway, M.A., Turk, D.J., Miller, S.L., Logan, J., Nebes, R.D. Meltzer, C., Becker, J.T. (1999). A positron emission tomography (PET) study of autobiographical memory retrieval. Memory. Vol 7(5-6), 679-702.

Corcoran, R., Upton, D., (1993). A role for the hippocampus in card sorting? Cortex. Vol 29(2), 293-304

Cowdry, R.W., Pickar, D., Davies, S. (1985). Symptoms and EEG findings in the borderline syndrome. International Journal of Psychiatry in Medicine, Vol 15, 205-211.

Davies R.K. (1978-1979). Incest: some neuropsychiatric findings. International Journal of Psychiatry in Medicine. 9(2):117-21, 1978-79.

De la Fuente, J.M, Lotstra, F., Goldman, S., Biver, F., et al. (1994) Temporal glucose metabolism in borderline personality disorder. Psychiatry Research: Neuroimaging. Vol 55(4), 237-245.

De la Fuente, J.M., Tugendhaft, P., Mavroudakis, N., (1998) Electroencephalographic abnormalities in borderline personality disorder. Psychiatry Research. Vol 77(2), Feb 1998, 131-138.

De Vegvar, M., Siever, L.J., Trestman, R.L., Impulsivity and serotonin in borderline personality disorder. In: Silk, Kenneth R. (Ed); et al. (1994). Biological and neurobehavioral studies of borderline personality disorder. Progress in psychiatry, No. 45. (pp. 23-40). Washington, DC, USA: American Psychiatric Press, Inc.

Dias, R, Robbins, T.W, Roberts, A.C. (1996). Dissociation in prefrontal cortex of affective and attentional shifts. Nature. Vol 380, Mar 1996, 69-72.

Dozois, D.J., Dobson, K.S., Ahnberg, J.L., (1998). A psychometric evaluation of the Beck Depression Inventory-II. Psychological Assessment. Vol 10(2), 83-89.

First, M.B., Spitzer, R.L., Gibbon, M., et al., (1996). Structured clinical interview for DSM-IV Axis-II personality disorders (SCID-II version 2.0). Biometrics Research Department, New York State Psychiatric Institute.

First M.B, Spitzer R.L., Gibbon M., Williams J.B.W. (1995) Structured Clinical Interview for DSM-IV Axis I Disorders (SCID). Washington, DC: American Psychiatric Press

Flecher, P.C., Happe, F., Frith, U., et al. (1995). Other minds in the brain: a functional imaging study of "theory of mind" in story comprehension. Cognition, 57, 109-128.

Fonagy, P.,Target, M., Gergeley, G. (2000). Attachment and borderline personality disorder: A theory and some evidence. Psychiatric Clinics of North America Vol 23(1) 103-122.

Fonagy, P. (1999). Attachment, the development of the self, and its pathology in personality disorders. In Derkesen et al. (Eds) Treatment of Personality Disorders, New York: Plenum.

Fonagy, P., Leigh, T., Steele M., et al., (1996). The relation of attachment status, psychiatric classification, and response to psychotherapy. Journal of Consulting and Clinical Psychology, Vol 64, 1996.

Franulic, A., Horta, E., Maturana, R., Scherpenisse, J., Carbonell, C. (2000). Organic personality disorder after traumatic brain injury: cognitive, anatomic and psychosocial factors: A 6 month follow-up. Brain Injury. Vol 14(5), 431-439

Fraser, K, Gallop, R (1993). Nurses' confirming/disconfirming responses to patients diagnosed with borderline personality disorder. Archives of Psychiatric Nursing. Vol 7(6), Dec 1993, 336-341.

Frith, C.D. (1992). The cognitive neuropsychology of schizophrenia. Hillsdale: Lawrence Earlbaum.

Fyer, M. R., Frances, A.J., Sullivan T., Hurt, S.W., Clarkin, J. (1998). Comorbidity of borderline personality disorder. Archives of General Psychiatry, Vol 45, 348-352.

Gardner, D.L., Lucas, P.B., Cowdry, R.W. (1987) Soft sign neurological abnormalities in borderline personality disorder and normal control subjects. Journal of Nervous & Mental Disease. Vol 175(3), Mar 1987, 177-180.

George, C., Kaplan, N., Main, M. (1985). The Adult Attachment Interview. Unpublished manuscript, Department of Psychology, Berkeley CA, 1985.

Green, A.H., Voeller, K., Gaines, R., et al. (1981). Neurological impairment in maltreated children. Child Abuse and Neglect Vol 5, 129-134.

Golomb, A., Ludolph, P., Westen, D., Block, M.J., et al. (1994) Maternal empathy, family chaos, and the etiology of borderline personality disorder. Journal of the American Psychoanalytic Association. Vol 42(2), 1994, 525-548.

Goyer, P.F., Andreason, P.J., Semple, W.E., Clayton, A.H., et al. (1994) Positron-emission tomography and personality disorders. Neuropsychopharmacology. Vol 10(1), Feb 1994, 21-28.

Grant, D.A., Berg, E. (1948) A behavioral analysis of degree of reinforcement and ease of shifting to new responses in a Weigl-type card-sorting problem. Journal of Experimental Psychology. 38, 1948, 404-411.

Guralnik, O. Schmeidler, J., Simeon, D. (2000). Feeling Unreal: Cognitive Processes in Depersonalization. American Journal of Psychiatry. Vol 157(1) January 2000 pp 103-109

Gunderson, J.G. (1994) Building structure for the borderline construct. Acta Psychiatrica Scandinavica. Vol 89 (suppl. 379).

Gurtvis, I.G., Koenigsberg, H.W., Siever, L.J. (2000). Neurotransmitter dysfunction in patients with borderline personality disorder. Psychiatric Clinics of North America Vol 23(1) 27-40.

Halstead, W.C. (1947). Brain and Intelligence. Chicago: University of Chicago Press.

Happe, F., Brownell, H., Winner, E., (1999) Acquired "theory of mind"impairments following stroke. Cognition. Vol 70(3), Apr 1999, 211-240.

Happe, F., Malhi, G.S., Checkley, S., (2000) Acquired mind-blindness following frontal lobe surgery? A single case study of impaired 'theory of mind' in a patient treated with stereotactic anterior capsulotomy. Neuropsychologia. Vol 39(1)

Happe, F. (1994). An advanced test of theory of mind: understanding of story character's thoughts and feelings by able autistic, mentally handicapped, and normal children and adults. Journal of Autism and Developmental Disorders. Vol 24, 129-154.

Hawton, K., Kirk, J. (1989) Problem solving. In: Hawton, K., et al (Eds) Cognitive behaviour therapy for psychiatric problems: a practical guide. Oxford: OUP.

Janes, K.L., Barron, D.S., Ensink, K., Schneider, T., Crosse, J.L. (2000) Coding manual for the strange stories. Unpublished Manuscript, Anna Freud Centre, University College London.

Jones, B., Heard, H., Startup, M., Swales, M., Williams, J.M.G., Jones, R.S.P. (1999) Autobiographical memory and dissociation in borderline personality disorder. Psychological Medicine. Vol 29(6), Nov 1999, 1397-1404

Judd, P.H., Ruff, R.M., (1993). Neuropsychological dysfunction in borderline personality disorder. Journal of personality disorders. Vol 7, 275-284.

Karmiloff-Smith, A., Klima, E., Bellugi, U., Grant, J., et al. (1995) Is there a social module? Language, face processing, and theory of mind in individuals with Williams syndrome. Journal of Cognitive Neuroscience. Vol 7(2), 196-208.

Kernberg, O. (1976). Object relations theory and clinical psycho-analysis. Northvale NJ: Jason Aronson.

Klein, S.B., (2001). A self to remember: A cognitive neuropsychological perspective on how self creates memory and memory creates self. In: Sedikides, Constantine (Ed); Brewer, Marilynn B. (Ed) Individual self, relational self, collective self. (pp. 25-46). Philadelphia, PA, US.

Lafleche, G., Albert, M,S. (1995) Executive function deficits in mild Alzheimer's disease. Neuropsychology. Vol 9(3), Jul 1995, 313-320.

Layden, M.A., Newman, C.F., Freeman, A, Morse, S.B. (1993). Cognitive therapy of borderline personality disorder. Boston, MA, USA: Allyn & Bacon, Inc. 1993

Leekam, S. & Perner, J. (1991) Does the autistic child have a metarepresentational deficit? Cognition. Vol 40, 203-218.

Levine, D., Marziali, E., Hood, J. (1997). Emotion processing in borderline personality disorders. Journal of nervous and mental disease. Vol 185(4), 240-246.

Lezak, M. (1995) Neuropsychological Assessment – 3rd edition. New York: Oxford University Press.

Lhermitte, F. (1983). "Utilization behaviour" and its relation to lesions of the frontal lobes. Brain. Vol 106, 237-255.

Linehan, M. (1993), Cognitive Behavioural Treatment of Borderline Personality Disorder. The Guilford Press, New York.

Linnoila, M., Virkkunen, M., Scheinin, M., Nuutila, A., Rimon, R., Goodwin F.K. (1983) Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from nonimpulsive violent behavior. Life Sciences. 33(26):2609-14, 1983

Lucas, P.B., Gardener, D.L., Cowdry, R.W., Pickar, D. (1989). Cerebral structure in borderline personality disorder. Psychiatry Research. Vol. 27, 111-115.

Lyoo, K., Han, M.H., Cho, Y.C. (1998). A brain MRI study in subjects with borderline personality disorder. Journal of Affective Disorders. Vol 50, 235-243.

Maffie, C., Fossati, A. (1999). The structure of DSM-IV borderline personality disorder and its implications for treatment. In: Derksen (Ed) Treatment of personality disorders. New York: Plenum.

Maher, M., Kaplan, L. (1966) Developmental aspects in the assessment of narcissistic and so-called borderline personalities. In: Hartocollis (Ed) Borderline personality disorders: the concept, the syndrome, the patient. New York: International Universities Press.

Mateer, C.A. (1999) The rehabilitation of executive disorders. In: Stuss, D.T., Winocur, G., et al. (Eds) Cognitive neurorehabilitation. (pp 314-332). New York, NY, US: Cambridge University Press.

Meares, R., Stevenson, J., Gordon, G., (1999). A Jacksonian and biospsychosocial hypothesis concerning borderline and related phenomena. Australian and New Zealand Journal of Psychiatry. Vol 33, 831-840.

Miceli, G., Caltagirone, C., Gainotto, G., et al., (1981). Neuropsychological correlates of localized cerebral lesions in nonaphasic brain-damaged patients. Journal of Clinical Neuropsychology Vol 3, 53-63.

Miller, L.A., Lai, R., Munoz, D.G. (1998). Contributions of the entorhinal cortex, amygdala and hippocampus to human memory. Neuropsychologia. Vol 36(11), Nov 1998, 1247-1256.

Na, C., Doraiswamy, P.M. Lee, K.H., Krishnan, K.R. (1991) Magnetic resonance imaging in biological psychiatry. Progress in Neuropsychopharmacology and Biological Psychiatry. Vol 15., 581-593.

Nehls, N. (1998). Borderline personality disorder: Gender stereotypes, stigma, and limited system of care. Issues in Mental Health Nursing. Vol 19(2), Mar-Apr 1998, 97-112.

Nelson, H.E., Willison, J.R. (1991) The National Adult Reading Test (revised). Windsor: NFER-Nelson.

Norman, D., Shallice, T. (1986) Attention to action: Willed and automatic control of behaviour. In: Consciousness and self regulation, Vol 4. New York: Plenum.

O'Leary, K.M., Brouwers, P., Gardener, D.L., et al. (1991). Neuropsychological testing of patients with borderline personality disorder. American Journal of Psychiatry. Vol 148, 106-111.

Osterrieth, P. A., (1944) Le test de copie d'une figure complexe; contribution a l'etude de la perception et de la memoire. Archives de Psychologie. Vol 30, 206-356.

Ozonoff, S., Pennington, B.F., Rogers, S.J., (1991). Executive function deficits in high-functioning autistic individuals: Relationship to theory of mind. Journal of Child Psychology & Psychiatry & Allied Disciplines. Vol 32(7), 1081-1105.

Ozonoff, S., Jensen, J. (1999) Specific executive function profiles in three neurodevelopmental disorders. Journal of Autism & Developmental Disorders. Vol 29(2), Apr 1999, 171-177

Patton., J.H., Stanford, M.S., Barratt, E.S. (1995). Factor structure of the Barratt Impulsiveness Scale. Journal of Clinical Psychology. Vol 51(6), 768-744.

Phillips, L.H. (1997) Do "frontal tests" measure executive function?: issues of assessment and evidence from fluency tests. In: Rabbitt, P. (Ed) Methodology of frontal and executive function Hove: Psychology Press.

Pilkonis, P.A., Kim, Y., Proietti, J.M., Barkham, M. (1996). Scales for personality disorders developed from the inventory of interpersonal problems. Journal of Personality Disorders. Vol 10(4), Win 1996, 355-369.

Porteus, S.D. (1955). The maze test: recent advances. Palo Alto: Pacific books.

Premack, D., Woodruff, G. (1978) Does the chimpanzee have a theory of mind? The Behavioural and Brain Sciences. Vol 4, 515-526.

Rabbitt, P. (1997) Introduction: methodologies and models in the study of executive function. In: Rabbitt, P (Ed) Methodology of frontal and executive function Hove: Psychology Press.

Reitan, R.M. (1958). Validity of the trail making test as an indicator of organic brain damage. Perceptual and motor skills. Vol 8, 271-276.

Risch, S.C., Siever, L.J., Gillin, J.C., et al., (1983). Differential mood effects of arecoline in depressed patients and normal volunteers. Psychopharmocology Bulletin. Vol 19, 696-698.

Russell, J (Ed) (1997). Autism as an executive disorder. New York, NY, USA: Oxford University Press. 1997,

Russell, J. (Ed) (2000). Autism as an executive disorder. New York, NY, USA: Oxford University Press.

Russell, J., Saltmarsh, R., Hill, E., (1999) What do executive factors contribute to the failure on false belief tasks by children with autism? Journal of Child Psychology & Psychiatry & Allied Disciplines. Vol 40(6), Sep 1999, 859-868.

Rolls, E.T. (1990) Functions of neuronal networks in the hippocampus and of back projections in the cerebral cortex in memory. In: McGaugh, J.L. et al. (Eds) Brain organization and memory: Cells, systems and circuits. New York: Oxford University Press.

Sass, K.J., Spencer, D.D., Kim, J.H., Westerveld, M., et al. (1990). Verbal memory impairment correlates with hippocampal pyramidal cell density. Neurology. Vol 40(11), Nov 1990, 1694-1697.

Scarpa, A., Luscher, K.A., Smalley, K.J., Pilkonis, P.A., Kim, Y., Williams, W.C. (1999) Screening for personality disorders in a nonclinical population. Journal of Personality Disorders. Vol 13(4), Win 1999, 345-360.

Schacter, D.L., Wang, P.L., Tulving, E., Freedman, M., (1982). Functional retrograde amnesia: A quantitative case study. Neuropsychologia. Vol 20(5), 1982, 523-532.

Shah, A., Frith, U. (1993). Why do autistic individuals show superior performance on the Block Design task? Journal of Child Psychology and Psychiatry, 34

Shah, A, Frith, U. (1983). An islet of ability in autistic children: A research note. Journal of Child Psychology & Psychiatry & Allied Disciplines. Vol 24(4), Oct 1983, 613-620.

Shaffer, D., Schonfield, I., O'Connor, P.A. (1985). Neurological soft signs. Archives of General Psychiatry, Vol 42, p342-351.

Shallice, Tim (1998). From neuropsychology to mental structure. New York, NY, USA: Cambridge University Press. 1988

Shallice, T, Burgess, P. (1991). Deficits in strategy application following frontal lobe damage in man. Brain. Vol 114, 727-741.

Shearin, E.N., Linehan, M. (1994). Dialectical behavior therapy for borderline personality disorder: theoretical and empirical foundations. Acta Psychiatrica Scandinavica. Vol 89 (Suppl 379) 61-68.

Silk, K. (2000) Overview of Biologic Factors. In Silk, K. (Ed). (1994): Biological and neurobehavioral studies of borderline personality disorder. Progress in psychiatry, No. 45. (pp. 23-40). Washington, DC, USA: American Psychiatric Press, Inc.

Snyder, S., Pitts, W.M. (1984). Electroencephalography of DSM-III borderline personality disorder. Acta Psychiatrica Scandinavica Vol 69, 129-134.

Snyder, S., Pitts, W.M, Gustin, Q. (1983). CT scans of patients with borderline personality disorder. American Journal of Psychiatry. Vol 140, 272.

Spreen, O., Strauss, E. (1998) A compendium of neuropsychological tests: Adminstration, norms and commentary – 2nd edition. New York: Oxford University Press.

Sprock, J., Rader, T.J., Kendall, J.P., Yoder, C.Y. (2000) Neuropsychological functioning in patients with borderline personality disorder. Journal of Clinical Psychology. Vol 56(12), Dec , US http://www.wiley.com: John Wiley & Sons Inc. 2000, 1587-1600.

Stern, A. (1938) Borderline group of neuroses. Psychoanalytic Quarterly. Vol 7, 1938, 467-489

Stein D.J., Hollander, E., Cohen, L., et al. (1993). Neuropsychiatric impairment in impulsive personality disorders. Psychiatry Research. Vol 48, 257-266.

Stein, M.B., Koverola, C., Hanna C., Torchia, M.G., McClarty, B. (1997). Hippocampal volume in women victimized by childhood sexual abuse. Psychological Medicine. Vol 27, 951-959.

Steinberg, B.L., Trestman, R., Mitropoulou, V. et al. (1997). Depressive response to physostigmine challenge in borderline personality disorder patients. Neuropsychopharmacology. Vol 17, 264-273.

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

Streeter, C.C., van Reekum, R., Shorr, R.I. Bachman, D.L. et al. (1995). Prior head injury in male veterans with borderline personality disorder. Journal of Nervous & Mental Disease. Vol 183(9), Sep 1995, 577-581

Stroop, J.R. (1935) Studies of interference in serial verbal reactions. Journal of Experimental Psychology. 18, 1935, 643-662.

Stuss, D.T., Gow, C.A., Hetherington, C.R. (1992). "No longer gage": Frontal lobe dysfunction and emotional changes. Journal of Consulting & Clinical Psychology. Vol 60(3), Jun 1992, 349-359.

Stuss, D.T. (1991) Self, awareness and the frontal lobes: a neuropsychological perspective. In: Goethaals, G.R., Strauss, J. (Eds) The self: an interdisciplinary approach. New York: Springer-Verlang.

Swirsky-Sacchetti, T., Gorton, G.E., Samuel, S., et al. (1993). Neuropsychological function in borderline personality disorder. Journal of Clinical Psychology. Vol 49, 385-396.

Tager-Flusberg, H, Sullivan, K. (2000). A componential view of theory of mind: Evidence from Williams syndrome. Cognition. Vol 76(1), Jul 2000, 59-89.

Teicher, M.H., Yutaka, I., Glod, C.A., et al. (1994). Early abuse, limbic system dysfunction, and borderline personality disorder. In: Silk, K. (Ed). (1994): Biological and neurobehavioral studies of borderline personality disorder. Progress in psychiatry, No. 45. (pp. 177-207). Washington, DC, USA: American Psychiatric Press, Inc.

Teicher, M.H., Glod, C.A., Surrey, J., Swett, C. (1993) Early childhood abuse and limbic system ratings in adult psychiatric outpatients. Source Journal of Neuropsychiatry & Clinical Neurosciences. Vol 5(3), 301-306.

Torgersen, S., (2000). Genetics of patients with borderline personality disorder. Psychiatric Clinics of North America. Vol 23(1), 1-9.

Torgersen, S., (1984). Genentic and nosological aspects of schizotypal and borderline personality disorders: A twin study. Archives of General Psychiatry. Vol 41, 546-554,

Tyrer, P. (1994). What are the borders of borderline personality disorder? Acta Psychiatrica Scandinavica. Vol 89 (379, Suppl).

Tyrer, P., Alexander, J. (1988). Personality Asssesement Schedule. In: Tyrer, P. (Ed) Personality disorders: diagnosis management and course. London: Wright.

Uno H., Tarara R., Else J.G., Suleman M.A., Sapolsky R.M., (1989). Hippocampal damage associated with prolonged and fatal stress in primates. Journal of Neuroscience. 9(5):1705-11.

Van den Hout, M., Merckelbach, H., Pool, K. (1996) Dissociation, reality monitoring, trauma, and thought suppression. Behavioural & Cognitive Psychotherapy. Vol 24(2), 1996, 97-108.

Van der Kolk, B.A., Greenberg, M.S. (1987) The psychobiology of the trauma response: hyperarousal, constriction and addiction to traumatic re-exposure. In van der Kolk (Ed) Psychological Trauma. Washington DC, American Psychiatric Press, 1987.

Van Reekum, R.L., Paul, S., Finlayson, M., Alan J., Boyle, M., et al. (1996) Repeat neurobehavioral study of borderline personality disorder. Journal of Psychiatry & Neuroscience. Vol 21(1), Jan 1996, 13-20.

Van Reekum, R., Conway, C.A., Gansler, D., White, R., et al. (1993). Neurobehavioral study of borderline personality disorder. Journal of Psychiatry & Neuroscience. Vol 18(3), May 1993, 121-129.

Vigil, W. (1996). Continuous Performance Test: User and Technical Manual. ForThought.

Wagner, A.W., Linehan, M.M. (1999). Facial expression recognition ability among women with borderline personality disorder: Implications for emotion regulation. Journal of Personality Disorders. Vol 13(4), Win 1999, 329-344.

Wechsler, D. (1981) Wechsler Adult Intelligence Scales - Revised. New York: The Psychological Corporation.

Wechsler, D. (1987) Wechsler memory scale – revised.New York: Psychological Corporation.

Wechsler, D., Stone, C.P. (1945) Wechsler memory scale manual. New York: Psychological Corporation.

Williams, J. M., Scott, J. (1988) Autobiographical memory in depression. Psychological Medicine. Vol 18(3), 689-695.

Williams, J.M.G., Broadbent, K. (1986). Autobiographical memory in suicide attempters. Journal or Abnormal Psychology. Vol 95, 144-149.

Wilson, B.A., Alderman, N., Burgess, P.W. et al., (1996). Behavioural Assessment of the Dysexecutive Syndrome. Bury St Edmonds: Thames Valley Test Company.

Wimmer, H., Perner, J. (1983) Beliefs about beliefs: Representation and the constraining function of wrong beliefs in young children's understanding of deception. Cognition. Vol 13, 103-128.

Young, A.W., Hellawell, D.J., Van De Wal, C., Johnson, M. (1996). Facial expression processing after amygdalotomy. Neuropsychologia. Vol 34(1), Jan 1996, 31-39.

Zaitchik, D. (1990). When representations conflict with reality: the preschooler's problem with false belief and "false" photographss. Cognition. Vol 35, 41-68.

Zanarini, M.C., Gunderson, J.G., Frankenburg, F.R., et al. (1989). The revised diagnostic interview for borderlines: discriminating borderline personality disorder from other Axis-II disorders. Journal of Personality. Vol 3, 10-18.

Zanarini, M.C., Williams, A.A., Lewis, R.E. (1997). Reported pathological experiences associated with the development of borderline personality disorder. American Journal of Psychiatry. Vol 148, 648-651.

Appendix

Material in the appendix is compiled in the following order:

- Ethical permission
- Consent form
- Information sheet (BPD participants)
- Information sheet (healthy control participants)
- Recruitment advert (for notice boards)
- Recruitment advert (newspaper)

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Monday, 17 July 2000

Dr Janet Feigenbaum
Sub Department of Clinical Health Psychology
University College London
Torrington Place
LONDON WC1 6BT

Dear Dr Feigenbaum

Ref: 00/57 (please quote in all further correspondence)
Title: Executive function and theory of mind in borderline personality disorder

This project has been considered by the Ethics Committee, and I am pleased to inform you that they were able to give their approval for it to proceed. Please would you write and inform **Angela Williams** of the start date of your project, at the above address.

Please note that the following conditions of approval apply:

- It is the responsibility of the investigators to ensure that all associated staff including nursing staff are informed of research projects and are told that they have the approval of the Ethics Committee.

- If data are to be stored on a computer in such a way as to make it possible to identify individuals then the project must be registered under the Data Protection Act 1984. Please consult your department data protection officer for advice.

- The Committee must receive immediate notification of any adverse or unforeseen circumstances arising out of the project.

- The Committee must receive notification: a) when the study is complete; b) if it fails to start or is abandoned; c) if the investigator/s change and d) if any amendments to the study are made.

- The Committee will request details of the progress of the research project periodically (i.e. annually), and require a copy of the report on completion of the project.

Please forward any additional information/amendments regarding your study to Michael Peat or myself at the above address. If you have any queries, please do not hesitate to contact Michael Peat at the Research office.

Yours sincerely

Stephanie Ellis
Committee Chair

Consent form

"Executive function and Theory of Mind in Borderline Personality Disorder"

Nick Stokes and Dr. Janet Feigenbaum
Sub Department of Clinical Health Psychology
University College London.

To be completed by the participant.

1.	I have read the information sheet about this study	YES/NO
2.	I have had an opportunity to ask questions and discuss this study	YES/NO
3.	I have received satisfactory answers to all my questions	YES/NO
4.	I have received sufficient information about this study	YES/NO
5.	Which health professional have you spoken to about this study?	
6.	I understand that I am free to withdraw from this study at any time without giving a reason, and without it affecting my future care:-	YES/NO
7.	Do you agree to take part in this study?	YES/NO

Signed.....Date.....

Name in Block Letters

Signature of investigator .....

Information sheet

"Executive function and Theory of Mind in Borderline Personality Disorder"

Nick Stokes and Dr. Janet Feigenbaum
Sub Department of Clinical Health Psychology
University College London.
Torrington Place
London WC1 6BT

- We are conducting a study which is looking at various aspects of thinking in people who have been diagnosed with Borderline Personality Disorder.

- The study will test two hypotheses (or ideas). The first is that people with Borderline Personality Disorder find it harder to carry out tasks which involve such things as planning and shifting from one idea to another (often called "Executive Function"). The second hypothesis is that people who have Borderline Personality Disorder find it harder to appreciate what other people might be thinking (often called "Theory of Mind").

- The results of this study will add to our knowledge of Borderline Personality Disorder, and may in the long term help to improve treatment.

- If you decide to take part, you will be asked to complete a number of different psychological tests. These aim to assess Executive Function and Theory of Mind. There will also be some questionnaires about mood and behaviour to fill out.

- The tests should not be distressing in any way, and many people find them interesting and fun to complete.

- All data will be labelled with a number in order to preserve anonymity. Any publications which arise form this study will not identify individuals in any way.

- Participants will be paid £10 for taking part.

- Testing will take place at the hostpital or health centre where you receive your care, or at the UCL Department of Clinical Psychology, Torrington Place (off Tottenham Court Road). It will last about 2 to 3 hours.

You do not have to take part in this study if you do not want to. If you decide to take part you may withdraw at any time without having to give a reason. Your decision whether to take part or not will not affect your care in any way.

All proposals for research using human subjects are reviewed by an ethics committee before they can proceed. This proposai was reviewed by Camden and Islington Locai Research Ethics Committe.

Please address any queries to:

Nick Stokes or **Dr. Janet Feigenbaum**,

tel: **020 7679 5964**
email: **ucjtnas@ucl.ac.uk**

Information sheet

"Executive function and Theory of Mind in Borderline Personality Disorder"

Nick Stokes and Dr. Janet Feigenbaum
Sub Department of Clinical Health Psychology
University College London.
Torrington Place
London WC1 6BT

- We are conducting a study for which we need to compare a group of healthy volunteers to a group of people who have a personality disorder on a variety of psychological measures.

- The study will test two hypotheses (or ideas). The first is that people with Borderline Personality Disorder find it harder to carry out tasks which involve such things as planning and shifting from one idea to another (often called "Executive Function"). The second hypothesis is that people who have Borderline Personality Disorder find it harder to appreciate what other people might be thinking (often called "Theory of Mind").

- The results of this study will add to our knowledge of Borderline Personality Disorder, and may in the long term help to improve treatment.

- If you decide to take part, you will be asked to complete a number of different paper and pencil tasks. These aim to assess Executive Functioning and Theory of Mind. There will also be some questionnaires about mood and behaviour to fill out.

- The tests should not be distressing in any way, and many people find them interesting and fun to complete.

- All data will be labelled with a number in order to preserve anonymity. Any publications which arise form this study will not identify individuals in any way.

- Participants will be paid £10 to cover costs.

- The study will take place at the Sub Department of Clinical Health Psychology, Torrington Place (off Totenham Court road). It will last about 2 hours.

You do not have to take part in this study if you do not want to. If you decide to take part you may withdraw at any time without having to give a reason. Your decision whether to take part or not will not affect your care in any way.

All proposals for research using human subjects are reviewed by an ethics committee before they can proceed. This proposal was reviewed by Camden and Islington Local Research Ethics Committe.

Please address any queries to:

Nick Stokes tel: 020 7502 7576, email: ucjtnas@ucl.ac.uk

or **Dr. Janet Feigenbaum**, tel: 020 7504 5964,
email: j.feigenbaum@ucl.ac.uk

Nick Stokes and Dr. Janet Feigenbaum
UCL Department of Clinical Health Psychology
Torrington Place
London WC1 6BT

Volunteers Needed for Psychological Study!

Payment of £10

Healthy female volunteers are needed for a psychological study run by researchers at University College London. The study will involve filling out some questionnaires, and completing a series of paper and pencil tasks. The tests will last approximately 2 hours.

By comparing the test results of healthy volunteers to the results of patients with a personality disorder, we hope to gain a greater understanding of the condition which may in the long term help to improve treatment.

Before asking you to come to the testing session we will ask a number of routine questions over the phone to check if we are able to include you in the study.

If you are interested in taking part, please call 020 7502 7576, *during office hours*, leaving your name and contact telephone number. Alternatively e-mail ucjtnas@ucl.ac.uk

UCL Psychological Study
Tel: 020 7502 7576
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PSYCHOLOGICAL STUDY

Female Volunteers needed for a psychological study run by the Dept. of Clinical Psychology at UCL.

All participants will receive £10!

Please call Nick Stokes

020 7502 7576

Leaving your name & telephone number, and we will call you back to discuss the study further. Non-graduates particularly required as we already have a large pool of graduates