A WAIS-III MARKER FOR DEPRESSION IN OLDER ADULTS?

BY

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DECLARATION

"This thesis has been composed by myself and the work contained herein is my own"

Signed LORNA ANN TORRENS

ABSTRACT

Depression is a common and disabling condition in older adults associated with increased medical complaint and mortality. Depression in late-life may be accompanied by significant cognitive deficit, at least in a proportion of individuals. The basis of this deficit is unclear but permanent structural brain change and/or reversible mood-state deficiency have been suggested.

Three groups of older adults comprising currently depressed individuals, individuals previously depressed but now recovered and individuals with no previous history of depression were assessed and compared using the WAIS-III.

Difficulties relating to accurate definitions of change points across illness are highlighted and, in particular, the consistent measurement of recovery from depression. It was however tentatively proposed that recovery from depression is associated with the reversal of cognitive deficit, at least in the majority of cases.

Depressed older adults demonstrated a WAIS-III profile indicative of broad cognitive deficit across the Verbal Comprehension, Perceptual Organisation, Working Memory and Processing Speed Indices. Processing speed appeared to be particularly impaired by the presence of depression.

Results are considered in relation to explanations of such deficit previously offered in the research. Suggestions are offered for future research and, in particular, the areas of processing speed, attention and formal classification of behavioural observations on assessment.

1. REVIEW OF LITERATURE

1.1 Introduction

The application of psychological knowledge to the needs of older people provides both challenge and opportunity in a climate where the knowledge base relating to the psychological processes of both normal and abnormal ageing is continually expanding (Nettlebeck & Rabbitt, 1992; Ritchie, Touchon, Ledesert, Leibovici & Dupuy-Gorce, 1997; Stuart-Hamilton, 1999; Woodruff-Pak, 1997).

Dementia and depression constitute the major mental health problems encountered by older adults (Woods, 1999). The boundaries of dementia, particularly when cognitive deficit is mild, are ill-defined. Similarly, although considerable progress has been made in understanding the diagnosis, etiology and treatment of late-life depression, questions remain regarding its prevalence (Futterman, Thompson, Gallagher-Thompson & Ferris, 1995) and the differences between late-life and earlier onset depression (Burvill, Hall, Stampfer & Emmerson, 1989).

Traditionally, there has been an emphasis on trying to distinguish between dementia as an organically based and irreversible process and the functional, reversible deficits evident in depression. More recently, awareness is increasing of the possibility of dual diagnosis in up to 30 per cent of individuals suffering from dementia (Teri & Gallagher-Thompson 1991; Teri & Wagner, 1992) as well as the presence of cognitive impairment severe enough to be diagnosed as dementia in around 20 per cent of depressed individuals (La

Rue, D'Elia, Clark, Spar & Jarvik, 1986). This latter group have been described as having 'pseudodementia' (Kiloh, 1961). Several studies have suggested that they occupy a position midway between normal controls and individuals with dementia on a variety of indices of brain function and structure (Pearlson, Rabins, Kim, Speedie, Moberg, Burns & Bascom, 1989).

However, the extent to which cognitive deficits observed in late-life depression resemble dementia is debatable (Poon, 1992) and their reversibility has been questioned (Abas, Sahakian & Levy, 1990). Neither the nature nor the etiology of cognitive deficit observed in late-life depression has been agreed. The vast heterogeneity of the population under consideration complicates the issue still further (Stuart-Hamilton, 1999).

Depression in late-life can be a disabling condition associated with greater frequency of medical complaint and increased mortality rates both by suicide and natural causes (Ames, 1994; Burvill, Hall, Stampfer & Emmerson, 1991; Hepple & Quinton, 1997). There are clear and specific benefits to the recognition and treatment of depression in older adults which include the potential for symptom level improvement, the enhancement of general mental, physical and social functioning and the minimisation of cognitive disability.

The current study seeks to investigate further the relationship between late-life depression and cognitive deficit.

1.2 Depression in Older Adults

1.2.1 Prevalence

It is often assumed that depression in late life is common, a function of losses experienced, exclusion from positions of importance and influence in society, declining health and fears about impending death (Murphy, 1983; Roth, 1955). This is a complex assumption which contains elements of truth but also distortions.

The National Institute of Mental Health/Epidemiologic Catchment Area (NIMH/ECA) studies utilised DSM-III criteria to diagnose depression from data obtained using the Diagnostic Interview Schedule (DIS) (American Psychiatric Association, 1980; Myers, Weissman, Tischler, Holzer, Leaf, Orvaschel, Anthony, Burke, Kramer & Stoltzman, 1984; Regier, Boyd, Burke, Rae, Myers, Kramer, Robins, George, Karno & Locke, 1988; Robins, Helzer, Croughan & Ratliff, 1981). These studies indicated a prevalence rate for major depression of 3-4 per cent in adults over 65 living in the community. This rate was two to three times less than the estimate for younger adults. However, rates were doubled by the inclusion of other complaints, such as bereavement, in which symptoms of depression figured prominently (Myers *et al.*, 1984). Such an observation highlighted the difficulties relating both to defining and assessing depression in later life.

A British study reported similar results to those obtained in the NIMH/ECA studies including a 4.3 per cent prevalence rate for major depression in urban-based older adults and a further 13.5 per cent of individuals experiencing a mild to moderate degree of

depression (Lindesay, Briggs & Murphy, 1989). More recently in the US, Schneider, Reynolds, Lebowitz & Friedhoff (1994) have reported a 15 per cent prevalence rate for late-life depression .

The relationship between age and depression is not straightforward. The cross-sectional nature of most studies confounds age and cohort effects (Schaie 1965). Most studies investigating patterns of depression in older adults include only two age groups, (65-75 and 75-85 years), (Futterman *et al.*, 1995). Depression may, in fact, increase in the "oldest old" or those over 85 (Lewinsohn, Duncan, Stanton & Hautzinger, 1986). Prevalence rates for different subgroups of older adults may also vary. Depression is, for example, more common in women than men (Nolen-Hoeksema, 1987). There may also be a higher prevalence of depression amongst individuals with concurrent physical illness and those living in residential care (Katon & Sullivan, 1990). Factors such as ethnic origin, socio-economic position, existence of cognitive impairment and social support may also significantly alter the observed relationships between age and depression.

The NIMH/ECA studies have been criticised for a number of perceived flaws in methodology. Kermis (1986) concluded that the use of the DIS in the NIMH/ECA sample resulted in an underestimate of depression and an overestimate of dementia amongst older adults. Burvill (1987) commented on the much higher prevalence rate of phobia than affective disorder identified in the studies and questioned whether the DIS identified phobias where other scales would indicate anxiety or depression. Snowdon (1990) criticised the use of inexperienced raters who, he considered, may have been oblivious to the complexities of non-verbal cues, ambiguities, misunderstandings,

rationalisations or other factors involved in the interview process.

Scales of measurement and diagnostic criteria employed also affect prevalence estimates. Self-report questionnaires or "test" approaches, such as the Geriatric Depression Scale (GDS) (Yesavage, Brink, Lum, Huang, Adey & Leirer, 1983), suggest prevalence rates for depression of up to 20 per cent in community based samples (Futterman *et al.*, 1995) with rates decreasing up to the age of 55 and, thereafter, either stabilising or increasing (Frerichs, Aneshensel & Clark, 1981; Murrell, Himmelfarb & Wright, 1983). Such scales are quick and easy to administer but they presuppose an individual's ability to respond accurately, often fail to assess a broad enough range of symptoms and may not adequately discriminate between major depression versus a more general distress associated with other common, late-life problems (Baldwin, 1990). Various research has suggested both the under and over-estimation of depression using scales such as the GDS (Harper, Kotick-Harper & Kirby, 1990).

A "classification" or diagnostic approach to identifying depression on the other hand is often based on structured clinical interviews and usually results in lower overall estimated prevalence of depression in older adults which peaks in mid-adulthood before declining (Newmann, 1989). The National Institutes of Health (NIH) Consensus Statement (1991), relating to the diagnosis and treatment of depression in late life, argued that low prevalence estimates are a result of formal diagnostic criteria being inappropriate for older adults. A similar view was expressed by Caine, Lyness & King (1993). In particular, the 'presence of dysphoric mood' may be inappropriate. Depressive affect may be "masked" or de-emphasised in older adults when physical or cognitive symptoms are also present

(Barrett, Barrett, Oxman & Gerber, 1988). Diagnostic systems such as DSM may also include bias. Exclusion from diagnosis may be based on concurrent physical illness or likely effects of medication. Positive diagnosis might be made only if symptoms 'interfere with everyday functioning'. Older adults though may be less likely than their younger counterparts to experience changes in role functioning as a result of symptoms of depression and more likely to experience concurrent physical illness thereby making diagnosis less likely (Newmann, 1989).

To summarise, estimated prevalence rates of major depression in older adults vary according to samples studied and methods employed, although it is increasingly recognised that late life depression is perhaps no more common, and may be less so, than depression in earlier adulthood. The distinction between depression as a symptom and a syndrome may sometimes be difficult to make and more accurate assessment would perhaps be achieved using a combination of approaches to diagnosis (Oliver & Simmons, 1984).

1.2.2 Heterogeneity

It is important to acknowledge the enormous variability in the older adult population. Firstly, individuals collectively referred to as "older adults" may include at least two generations of the population. Each have different life experiences and historical perspectives (Zeiss & Steffer, 1996). Furthermore, whilst various cognitive abilities do decline with increasing age (Backman & Hill,1996; Rabbitt, 1993, 1996), the range of ability amongst older adults is more broadly dispersed than that of younger individuals (Morse, 1993; Rabbitt, 1993). There is considerable support for the assertion that normal,

age related change does not, in itself, necessarily involve a decline in level of cognitive function (Benton, Eslinger & Damasio, 1981). Chronological age is therefore a poor indicator of likely cognitive performance (Stewart-Hamilton, 1996).

1.2.3 Management

Identified depression in older adults is not always effectively managed (Forsell, Jorm & Winblad, 1994). There are several possible explanations for this observation.

Firstly, individuals may fail to recognise or minimise the symptoms of their own depression (which may be readily apparent to appropriate professionals) and therefore fail to seek help (Blazer, Bachar & Hughes, 1987; Zarit & Zarit, 1998). Where medication is in fact prescribed but follow-up monitoring is inadequate, depressed older adults may demonstrate compliance difficulties, either as a result of confusion or drug-induced side-effects (NIH Consensus Statement, 1991).

Health professionals themselves may also be responsible for the under-detection of depression in older adults (Bowers, Jorm, Henderson & Harris, 1990; M. Crawford, Prince, Menzes & Mann, 1998) or its misdiagnosis (Newman & Sweet, 1986). Alternatively, or in addition, they may recognise depression but fail to treat it (Blanchard, Waterreus & Mann, 1994; M. Crawford *et al.*, 1998; MacDonald, 1986).

MacDonald (1986) reported a depression detection rate by GPs in excess of 90 per cent but a concurrent failure to act on this assessment. The high detection rate may have been

due to sensitisation of the GPs who, at the time of consultation, were aware that selected patients would be subsequently interviewed by researchers. M. Crawford *et al.* (1998), in a similar study, reported a 51 per cent depression detection by GPs. The study avoided a situation whereby GPs were sensitised to the research. However, as a result, delays of up to eight weeks were introduced between collection of research data and GP assessments. Such delays may not have allowed for fluctuating symptom levels. Whatever the reason for the discrepancy in identification rates between the studies, again, only 38 per cent of individuals accurately identified as depressed were being actively treated. In particular, the frequency of specific psychological interventions, which have been demonstrated to be efficacious both in the initial remission of the symptoms of depression in older adults as well as the maintenance of gain (M. Crawford *et al.*, 1988; Dick, Gallagher-Thompson & Thompson, 1999), was very low.

Failure to act on a diagnosis of depression in late life by GPs may be a result of a belief that predisposition to depression increases as a function of ageing (Alexopoulos, 1994; Veith & Raskind, 1988). Blanchard (1996) and Woods (1996) highlight the sense of powerlessness that such a belief can create in professionals who therefore understand that depression is inevitable, unavoidable and untreatable. Professionals may also fear the possible side-effects of anti-depressant medication, particularly in those cases where concurrent pharmacological treatments for medical conditions are involved (Heeren, Derksen, Heycop & van Gent, 1997; Salzman, 1994). Alternatively, they may tend to focus on the concurrent medical complaint (NIH Consensus Statement, 1991) and, as a result, either fail to prescribe or prescribe a sub-optimum dosage of anti-depressant medication (Blazer *et al.* ,1987).

In summary, depression in older adulthood is a common, disabling condition which is often unrecognised, under-treated and even untreated. There is a clear need for appropriate instruments to detect depression in this group and accurately measure its severity.

1.2.4 Distinctive Presentation

DSM-IV (American Psychiatric Association, 1994) describes depression as consisting of clusters of symptoms which might include low mood, loss of interest or pleasure in normal activities, feelings of worthlessness, self reproach or excessive guilt, a sense of hopelessness for the future, suicidal thoughts or acts, and disordered bodily functioning particularly involving sleep, appetite or motor disturbance.

Recognition of depression in older adults may be hampered by a presentation differing from that typically observed in younger adults (Murphy, 1986; Ritchie, Toulon, Ledesert, Leibovici & Dupuy Gorce, 1997; Zarit & Zarit, 1998). Low self-esteem and reported low mood, for example, may be less diagnostically important (Bieliauskas, 1993; Wallace & Pfohl, 1995). By contrast, physical symptoms are more frequently reported by older adults (Pitt, 1986, 1995) and perceived memory loss may be more common, although not necessarily evident on formal testing (Barker, Prior & Jones, 1995; Barker, Carter & Jones, 1994; Cavenar, Maltbie & Austin, 1979; Cipolli, Neri, Andermarcher, Pinelli & Lalla, 1990; Feehan, Knight & Partridge, 1991; O'Boyle, Amadeo & Self, 1990; Popkin, Gallagher, Thompson & Moore, 1982; West, Boatwright & Schleser, 1984). Consistent variations in reported symptomatology have been used to argue the presence of a distinct,

less emotional and more structurally-based, depressive phenomenon amongst older adults (Burvill *et al.*, 1989).

One group has produced research supporting the suggestion of idiosyncratic presentation across the whole spectrum of age (Blazer, Swartz, Woodbury, Manton, Hughes & George, 1988; Blazer, Woodbury, Hughes, George, Manton, Bachar & Fowler, 1989). Five "pure" types of depression, linked to age group, were identified. Only one of these fitted standard diagnostic criteria.

Newmann, Engel & Jensen (1990) considered symptoms of depression in women living in the community aged between 52 and 92 years old and identified two factors accounting for variability in symptoms. The first was the classic 'depressive syndrome' (characterised by most of the emotional and cognitive features of depression) and the second the 'depletion syndrome' (characterised more by loss of appetite, loss of interest and thoughts about death). Later work by the same group (Newmann *et al.* 1991a,b) indicated that the depressive syndrome was more common amongst younger depressed individuals and decreased with age whereas the depletion syndrome was less common amongst younger depressed individuals but increased with age.

The presence of a distinctive, older adult, profile in depression has, however, been disputed by others. Musetti, Perugi, Soriani, Rossi, Cassano & Akiskal (1989) point out that much of the research supporting distinction has been conducted on in-patient populations. This has resulted in uncertainty as to whether symptom patterns represent depression in older adults or are derived from other conditions accompanying it. Further

confusion may have arisen due to huge individual variability and possible confounding by cohort effects including a tendency on the part of older adults to minimise feelings of sadness (Georgotas, 1983).

1.2.5 Early Versus Late Onset Depression

A distinction has been made between individuals with life-long histories of depression (early-onset) and those in whom depression appears to manifest itself for the first time in late life (late-onset). The existence of this latter group may, at least to some degree, account for observations supporting an atypical presentation of depression in older adults.

The early versus late onset distinction has become central to the debate surrounding the etiology and prognosis of depression in older adults. The distinction has been made on the basis of a number of discriminatory factors which include:

> ***Genetics.*** Early onset depression may have a more significant hereditary component (Alexopoulos, 1989; Blanchard, 1996; Hopkinson, 1964; Slater & Cowie, 1971) although some researchers have failed to find such a link (Baldwin, 1990; Jacoby & Levy, 1980b).
>
> ***Cognition.*** Late-onset depression may be associated with higher and more unremitting levels of cognitive dysfunction (Burvill *et al.*, 1989) although this too has been disputed (Greenwald & Kramer-Ginsberg, 1988).

Personality. Clinicians may be less likely to rate individuals with late-onset depression as having severe personality traits or personality disorder by comparison to individuals with early onset disorder whose presentation may be embedded in long-standing patterns of functioning (Brodaty, Peters, Boyce, Hickie, Parker, Mitchell & Wilhelm, 1991).

Outcome. The distinct nature of late-onset depression has been argued on the basis both of poor (Post, 1972) and favourable prognosis for residual symptom levels (Cole, 1983; Kay, Beamish & Roth, 1964; Magni, Palazzolo & Bianchin, 1988).

Structural Brain Change. Individuals suffering from late-onset depression may demonstrate structural abnormalities including increased ventricle size (Jacoby & Levy, 1980b), cortical atrophy (Dolan, Calloway & Mann, 1985) and increased likelihood of white matter lesions (Ebmeier, Prentice, Ryman, Halloran, Rimmington, Best & Goodwin, 1997).

Further evidence against an early/late onset distinction has been reported on the basis of electroencephalogram (EEG) investigations. No differences were detected between early and late onset cases of depression based on either sleeping or waking EEGs (Buysse, Reynolds, Houck, Stack & Kupfer, 1988; Heyman, Brenner, Reynolds, Houck & Ulrich, 1991). Clear differences were, however, noted between depressed and age-matched control groups (Brenner, Reynolds & Ulrich, 1989; Prinz & Vitiello, 1989).

1.2.6 Towards an Understanding of Conflicting Results

Conflicting findings in the area of early versus late onset depression may have arisen due to factors such as differences between populations and diagnoses, length of follow-up and assessment and treatment methods employed (Cole, 1983). Arbitrary distinctions are often made in the distinction between "late" and "early" onset. Late-onset depression may, in some cases, simply reflect late presentation to, or recognition by, services. These methodological difficulties may confound the genuine existence of a smaller distinctive group of individuals in whom biological factors, perhaps associated with the ageing process, may play a significant part.

In summary, presentation of major depression in older adults may differ subtly, but consistently, from the more usual pattern of symptoms reported by younger individuals. There is some evidence, albeit controversial, for making a distinction between early and late onset depression. The distinction has been drawn on a number of dimensions including a symptom profile more suggestive of biological disturbance in late-onset depression. Sampling and diagnostic differences prevent firm conclusions being drawn.

1.3 Cognitive Deficit in Depression

A keenly-debated area in the literature has addressed the question of whether late-life depression is inevitably accompanied by cognitive deficit. Investigators have approached this question from a variety of different stances including comparative/descriptive

approaches, cognitive/psychological approaches and experimental investigations using brain imaging. Each of these areas are discussed in more detail.

1.3.1 Comparative Approaches to Cognitive Deficit in Depression

Comparative approaches operate on the assumption that there are distinguishable cognitive boundaries between illnesses. Quantitative neuropsychological methods have increasingly been used to describe the characteristics of cognitive deficit in depression by comparison to healthy controls and groups with other clearly definable diseases.

The Evidence Against a Link Between Depression and Cognitive Deficit. Traditionally (Friedman, 1964; Granick, 1963), and even more recently (Bieliasukas, Costello & Terpenning, 1991; Bieliauaskas & Lamberty, 1991a; Bieliauskas, Lamberty & Boczar, 1991b; L. Miller, Faustman, Moses & Csernansky, 1991; Popkin *et al.*, 1982; Rohling & Scogin, 1993), a proportion of relevant research has failed to demonstrate a link between late-life depression and cognitive deficit. The research, however, has been plagued by methodological difficulties.

Taken together, the Bieliauskas studies (1991a, b) concluded that depression had 'little or no effect' on cognitive function (Bieliauskas, 1993). The studies, however, involved older medical outpatients evaluated for depressive symptoms using a continuous measure, the Hamilton Depression Scale (HDS) (Hamilton, 1967). Such a measure may have selected not only depressed individuals but also those with other psychiatric syndromes. In addition, the presence of physical symptoms, which would have been more probable in this

sample, leads to a higher depression score on the HDS giving rise to the possibility that some of those tested as "depressed" participants would not, in fact, meet more stringent diagnostic criteria for depression. When Bieliauskas & Lamberty (1991) considered individuals scoring positively in the area of loss of self-esteem and guilt separately, they did, in fact, observe slower reaction times in these individuals than in the remainder of the group.

The Popkin *et al.* (1982) study also reported no relation between depression and memory deficit but involved only 18 depressed older adults. These individuals were enrolled, at the time, in a 16-session psychotherapy programme. Some of them had already completed almost half of the course and therefore may have recovered sufficiently to perform comparably, on the memory measures, to controls. Certainly, by the time of testing, the Beck Depression Inventory (BDI) scores of some of the individuals involved had significantly decreased by comparison to scores on programme entry. The Rohling & Scogin (1993) study also focused exclusively on memory tasks using a small group (N = 15) of depressed older adults "matched" to 15 controls. Age matching, however, involved a criteria of plus or minus five years and, in fact, the mean age of the depressed group was three years younger than that of the controls who may therefore have been more subject to the effects of normal ageing.

Research selection criteria may also influence results. Specifically, in an effort to avoid the inclusion of individuals with dementia, depressed subjects have often been excluded from research on the basis of exhibiting overt memory deficit (Poon, 1992). In this way, depressed individuals demonstrating the most marked cognitive deficit can be incorrectly

excluded from study. Equally, however, where criteria are relaxed, individuals who do in fact have dementia may be unwittingly included in studies purporting to have identified cognitive deficit in "depression".

Making an accurate distinction between depression and dementia is notoriously difficult and is further complicated by a high incidence of co-occurrence (Reifler & Larson, 1990). Both groups may present with similar symptoms as illustrated in Table 1.

Table 1. Clinical Features of Dementia and Depression

	DEMENTIA	DEPRESSION
ONSET	Insidious	Gradual
DURATION	Months/years	Weeks/Months
COURSE	Stable and Progressive	Usually worst in mornings
ORIENTATION	May be normal Impaired for time and place	Usually unimpaired
MEMORY	Impaired recent and remote	Recent may be impaired Mood congruent recall
THOUGHTS	Slow and reduced interests	Slow, sad and hopeless
EMOTIONS	Shallow, apathetic, irritable	Flat, unresponsive, sad, irritable
SLEEP	Often disturbed. Nocturnal wanders/ confusion	Disturbed. Onset insomnia. Early morning wakening
PERCEPTIONS	May be normal Hallucinations in 30-40%	Mood congruent Auditory hallucinations 20%

Source: Dementia Services Development Centre, University of Stirling, UK.

Three other studies (Dannenbaum, Parkinson & Inman, 1988; E. Miller & Lewis, 1977; Vitiello, Prinz, Poon & Williams, 1990), each of which applied screening to exclude individuals with evident memory deficit, also failed to find significant cognitive differences

between depressed individuals and normal controls. E. Miller & Lewis (1977) reported that recognition memory was unaffected by depression. The study involved an outpatient sample of mildly to moderately depressed individuals, varying as to diagnostic subtype and controlling neither for intellectual level nor education. More tightly controlled studies have challenged the contention that recognition memory is unimpaired in depression. Watts, Morris & MacLeod (1987), for example, controlling for intelligence and avoiding a design which could be subject to floor effects for controls, reported a deficit in recognition memory in depression which could not be attributed to response bias.

Cognitive performance may indeed be unaffected by depression. Alternatively, cognition may be only minimally affected in the absence of dementia (Pearlson *et al.*, 1989). Poon (1992) calculated that levels of performance of depressed groups may be about 90 per cent of non-depressed individuals and, in some cases, equal or better. Boone, Lesser, Miller, Wohl, Berman, Lee, Palmer & Back (1995), reported that although depressed older adults performed more poorly than controls in visual memory and non-verbal intelligence tasks, results were of 'no clinical significance' in that they were almost within the high average range. The depressed group in this study were, however, very well educated, above average in intelligence and only mildly depressed.

A further possibility is that cognition may be affected in only a small minority of depressed individuals (Post, 1975; Speedie, Rabins, Pearlson & Moberg, 1990). This possibility, and that of minimal dysfunction, emphasises the need for adequate sample sizes to avoid overlooking significant effects. Massman, Delis, Butters, Dupont & Gillin (1992) commented:

> there is markedly greater variability within depressed groups than within normal groups... thus it is possible that only a subgroup of depressives display memory dysfunction

Cervilla & Prince (1997), in support of the proposition that only a proportion of depressed older adults exhibit cognitive impairment, suggest a failure to adequately consider the etiology of depression in older adults. They propose:

> at least two differential pathways to depression in the elderly, one via social distress factors and another mediated by cerebral deterioration clinically expressed as cognitive impairment.

Zarit & Zarit (1998) highlight a variety of processes which may be linked to the development of depression in later life. These include biological influences, early life experiences, stressful events, cognitive style and loss of reinforcement. They emphasise the importance of avoiding simple dualities in assessing causation.

To conclude, a number of studies have failed to demonstrate a link between late life depression and cognitive deficit. This may be because no such link exists. Alternatively, links may have been obscured by participant selection methods or sample sizes too small to elucidate subtle differences. Heterogeneity of presentation is also important in that a proportion of depressed older adults may show no cognitive deficit whatsoever, whilst others may perform very poorly (Speedie *et al.*,1990). A multitude of processes may influence the development of late-life depression.

The Evidence for a Link Between Depression in Old Age and Cognitive Deficit.

Considerable research evidence represents the contrasting view that late-life depression is related to cognitive deficit. McAllister & Price (1982) note:

> In elderly patients cognitive impairment associated with pure depressive illness may be extremely severe... the most prominent presenting complaint of an unrecognised depressive illness.

Hemispheric Dysfunction. Early work focused on the comparison between left and right hemispheres. Flor-Henry (1979) implicated right hemisphere dysfunction in depression inferred from a pattern of Weschler Adult Intelligence Scale (WAIS) (Weschler, 1955) test results characterised by greater deficits on Performance by comparison to Verbal scale tasks. Fromm & Schoplocher (1984); Hart, Kwentus, Wade & Hamer (1987); Pernicano (1986) and Weschler (1955) arrived at similar conclusions (although the first-mentioned study included bi-polar depressed individuals). Results were typically explained by reference to a 'mood-state' model wherein 'psychomotor slowing' and anxiety (often accompanying depression) reduced concentration and led to difficulty with timed tasks and/or tasks requiring immediate recall (Walker & Spence, 1964). By contrast, Calev, Korin, Sahpria, Kugelmass & Lere (1986) reported that when task difficulty is matched, and time constraints removed, the difference between performance and verbal competency disappears.

Kluger & Goldberg (1990), in a meta-analytic study also based on WAIS data, argue that there is bilateral hemispheric involvement in depression but that the specificity of hemispheres (and, in particular, that of the right hemisphere in dealing with novel tasks (Goldberg & Costa, 1981)) means that right hemisphere function is more severely affected than left. Similarly, Robbins, Joyce and Sahakian (1992) concluded that even if non-verbal tasks are more sensitive to depression, right hemisphere dysfunction is, at best, only

relative to other deficits that one would expect to be mediated by the left hemisphere. Boone *et al.* (1995) also highlighted various 'right-hemisphere tests' each requiring several cognitive skills tied to different brain areas.

In two relatively recent studies which controlled for intelligence, educational experience, time constraints and level of purported difficulty across left and right hemisphere tests, Deptula, Manaevitz & Yozawitz (1991) and Sackheim, Freeman, McElhiney, Coleman, Prudic & Devanand (1992), reported that performance deficits in depression could not be solely attributed to time constraint or current mood state. They proposed that discrepancies observed represented a genuine "trait" phenomenon resulting from either bilateral or right-sided dysfunction. Sackheim *et al.* (1992) reached their conclusions with reference to the observation that performance did not improve on retesting (despite marked clinical improvement), that removal of WAIS-R (Weschler, 1981) time limits did not markedly improve performance (with the exception of Digit Symbol) and that Performance IQ scores, unlike Verbal ones, were not related to severity of depression. However, individuals involved in this study were not exclusively older adults, despite including a 'high representation'; 25 per cent of them suffered from bipolar illness, 36 per cent met the criteria for probable or definite psychosis during the current episode, and all had been referred for electroconvulsive therapy (ECT), many of them having experienced this on previous occasions. It has been suggested that ECT may increase the likelihood of confusion (NIH Consensus Statement, 1991; Sackheim, 1992).

To conclude, attempts have been made to link the cognitive deficit observed in late-life depression to both "state" and "trait" related hemispheric dysfunction. Much of the work

has failed to take the issue of timed performance into account (Horn & Hofer, 1992). Hemispheric-related solutions may, in fact, simply represent decreased speed of processing. More recent work has reported performance deficits over and above those explicable by reference to time constraint or task difficulty.

A larger body of research has considered observable cognitive dysfunction more generally without necessarily trying to link this to anatomic region.

Memory Dysfunction. Many studies have focused exclusively on memory, perhaps either because of the high frequency of memory complaint amongst depressed individuals or the fact that it is a centrally defining feature of dementia (DSM-IV, American Psychiatric Association 1994).

McAllister (1981), in a review paper, concluded that depression only temporarily impairs memory and verbal learning implying a mood state rather than permanent structural deficit. Cipolli *et al.* (1990) reported an association between severity of depression and degree of memory impairment. Deficits have also been reported in the acquisition and recall of new information (Cohen, Weingartner, Smallberg, Pickar & Murphy, 1982), coding and memory strategies (Weingartner, Cohen, Murphy, Martello & Gerdt, 1981; Weingartner & Silberman, 1982) and episodic memory (Fogarty & Hemsley, 1983).

Several studies have compared memory in depressed older adults to that of individuals with progressive brain disease. Hart, Kwentus, Hamer & Taylor (1987a) used a selective reminding procedure to compare 14 depressed outpatients (mean age 70.1) to 15 age and

education matched individuals suffering from Alzheimer's disease and 16 controls. The depressed group were impaired relative to controls on total recall and proportion of information retained from one trial to the next without reminding. In addition, they were less able to benefit from the provision of imagery than controls. They did not differ from controls on a test of delayed recognition (although ceiling effects were noted). The depressed group were significantly superior to the Alzheimer group in all respects apart from their failure to benefit from imagery.

La Rue *et al.* (1986) reported even more pervasive memory deficit in depression when employing the Fuld Object Memory Evaluation. Neither the Hart *et al.* (1987a) nor the La Rue *et al.* (1986) studies controlled for intellectual level.

Hart, Kwentus, Taylor & Harkins (1987b) went on to compare older depressed to individuals suffering from Alzheimer's disease on a line drawing recognition task. Both groups had difficulty learning the drawings to criterion but only the group with Alzheimer's disease had difficulty remembering 10 minutes afterwards. The authors concluded that depressed individuals had difficulty learning new information rather than in consolidating or retaining it. Experimental groups in this study were matched for length of education but the premorbid IQ scores of the depressed group were significantly lower than controls suggesting the need for cautious interpretation of results.

Niederehe (1986) presented the results of a series of episodic, semantic and constructive memory experiments comparing "old" (mean age 64.9) and "young" (mean age 30.9) depressed individuals to age and education matched controls. The studies suggested more

significant effects of age than depression on memory function. Effects which were noted in relation to depression included conservative response style and a greater degree of subjective memory complaint.

Thus, evidence for the existence of memory deficits in depressed older adults is consistent although there is little agreement as to the basis of the deficit. There appear to be discernible differences between depressed individuals and those suffering from Alzheimer's disease in terms of memory function. This suggests either a mood-state etiology or a structural etiology distinct to that witnessed in Alzheimer's disease. Some of the memory difficulties identified may, in fact, be a feature of normal age related change.

Broader Cognitive Deficit. Broader cognitive deficits have also been identified in depressed older adults. These include cognitive, motor, perceptual and communication problems, decreased attention and reaction times and psychomotor, learning and perceptual deficits (Cohen *et al.*, 1982; W. Miller, 1975; Raskin, Friedman & Dimascio, 1982). Emery and Breslau (1989) reported reduced capacity for linguistic complexity.

La Rue, Yang & Osato (1992) referred to a 'typical neuropsychological presentation' in late life depression including pervasive loss of interest accompanied by mild memory deficit, mild to moderate visuospatial impairment and reduced cognitive abstraction and flexibility. According to this model, WAIS Verbal IQs come close to normal levels. Digit Span scores are lower than other Verbal subtests as a result of reduced concentration and Performance subtests demonstrate mild to moderate impairment as a result of slowing, carelessness or refusal to complete tests.

King, Caine, Conwell & Cox (1991a) compared the neuropsychological performance of 23 older depressed inpatients (mean age 70.8) to 20 mildly to moderately impaired individuals with Alzheimer's disease and 23 healthy controls. Whilst all groups were matched for age, sex and education, there was no control for premorbid intellectual level. Depressed individuals were superior to individuals suffering from Alzheimer's disease on language, memory and copying tasks. They were impaired by comparison to controls on word generation, immediate and delayed recall of words, delayed recall of shapes and delayed list recognition. There was also a greater negative effect of age in depressed individuals on measures involving naming and immediate recall. Low naming scores were associated with more severe depression at six month follow-up (King, Caine, Conwell & Cox, 1991b) and persisted even as depressive symptomatology remitted perhaps suggesting a "marker" for a more structural and intransigent depression in this small sub-group of individuals. Other well-controlled studies however have not observed naming difficulties in association with depression (Emery & Breslau, 1989; Hill, Stoudemire, Morris, Martino-Salzman, Markwalter & Lewison, 1992).

The issue of unmatched intellectual level arises throughout the literature and necessitates cautious interpretation of results. If cognitive test findings are to be attributed to the effects of the conditions under consideration, it is essential to establish comparable pre-morbid intelligence across the groups to be compared. Further caution in interpretation is required on the basis of patient status. Rohling & Scogin (1993) point to the increased number of studies which uncover memory deficit in hospital inpatients by comparison to outpatients. Inpatient status may represent increased depression severity making subtle

cognitive deficit easier to pick up. Additionally or alternatively, however, other variables associated with hospitalisation may affect cognitive function.

In conclusion, there is some support for the proposal that generalised and irreversible cognitive deficit exists in late-life depression. However, serious methodological flaws in many of the studies and, in particular, the issue of unmatched intellectual level, temper the credibility of results and casts doubt on acceptance of an inevitable association between late life depression and cognitive impairment.

Reversibility of Cognitive Deficit. Abas *et al.* (1990) compared depressed individuals to individuals suffering from Alzheimer's disease using a comprehensive battery of tests validated for use with older adults. They reported deficits over a broad range of cognitive function (notably speed of response and memory) in a high proportion (70 per cent) of depressed subjects. Patterns of deficit differed from those seen in Alzheimer's disease. With recovery, as assessed by 'clinical impression' and decreased Montgomery and Asberg Depression Rating Scale scores (Montgomery & Asberg, 1979), improvement was noted on most tests. However, evidence of residual slowing and a degree of memory and learning deficit persisted. Residual deficit appeared to be linked to CT scan pattern and it was therefore suggested that it may have a structural origin.

All of the depressed individuals in the study (N=20) had suffered at least one previous episode of depression, (10 of them before 50 years), and four of them had a history of manic episodes. The depression scale employed was standardised on a sample of only

106 adults aged between 18 and 69 years. It is based on 10 symptoms of depression and does not include motor retardation which was noted to occur 'in relatively few individuals'. In addition, one of the 10 items that was included ('inner tension') had the potential to be somatically perceived. The applicability of such a scale in accurately identifying depression in the older adult population has to be questioned.

Later work reported by Beats, Sahakian & Levy (1996) compared the profile of deficit seen in late-life depression and on recovery to that seen in basal ganglia disorders and frontal lobe damage. Control and depressed groups were well matched in terms of age, premorbid IQ and education. However, four individuals had a history of mania and at least five of the individuals in the depressed group had clear risk of vascular disease. Deficits in the depressed group were milder than those observed in the frontally-impaired groups but spanned a broader range of tests - specifically those indicating temporal lobe damage. In addition, it appeared that performance on the Tower of London Planning Test was adversely affected by perceived early failure. This suggested that, whilst efficiency of problem solving may be relatively intact (if slowed) in depressed older adults, it is likely to be disturbed by the perception of failure. On recovery, all deficits, apart from speed of processing, were reversed. Remaining deficit in this domain appeared to be linked to increased number of episodes of depression, increased ventricular size on scanning and clinical measures of depression. Results implied that speed of processing deficits may be trait markers of structural deficit rather than state markers of mood state.

Bulbena & Berrios (1993) considered two groups of depressed individuals. The first

group demonstrated reversibility of cognitive deficit on alleviation of the symptoms of depression and the second did not. Measures of attention, memory, visuo-spatial function and reaction time were taken. The group whose deficits were unremitting displayed more 'soft neurological signs' which, the authors concluded, represented a secondary, distinct, more 'organic' depressive disorder. An alternative interpretation is that these individuals may have been in the very early stages of dementia. Similarly Boone *et al.* (1995) reported two distinct cognitive patterns associated with depression. A relative weakness in visual memory and non-verbal intelligence was thought to be linked to structural anomalies in the right hemisphere. By contrast, decreased information processing speed and executive deficit (which appeared to be linked more reliably to depression severity) was considered to reflect temporary alterations in cerebral physiology. Positron emission tomography (PET) imaging studies have supported this view (Martinot, Hardy, Feline, Huret, Mazoyer, Attar-Levy, Pappata & Syrota, 1990).

There have been numerous reports of improved cognitive function corresponding to reduction of depressive symptomatology (Austin, Ross, Murray, O'Carroll, Ebmeier & Goodwin, 1992; Moffoot, O'Carroll, Bennie, Carroll, Dick, Ebmeier & Goodwin, 1994; Schotte, Cools & Payvar, 1990). Such observations may implicate the contribution of motivational or mood state factors to cognitive performance or, alternatively, suggest that any biological disturbance affecting performance is largely temporary and reversible.

To summarise, reversibility of cognitive deficit on recovery from depression is often reported. Decreased speed of processing may be a deficit which persists. There may be a small sub-group of individuals in whom wider cognitive deficit is irreversible, perhaps as a

result of organic abnormality.

Methodological Difficulties. The evidence for clear cognitive impairment as a consequence of late-life depression is weak resting, as it does, on studies with many obvious flaws.

Firstly, over several decades of research into late-life depression, there have been changes in the diagnostic criteria of both affective and degenerative disorders. This may have resulted in researchers making inappropriate comparisons between studies given possible fluctuations in the assessment both of cognitive deficit and depression.

Secondly, whilst controlling for education, age, sex and medication are usually achieved in research designs, there is less emphasis on controlling for either current or premorbid levels of general intelligence. This is of particular note when higher IQ has been suggested as a moderating factor in cognitive decline (Poon, 1992; Raskin, Friedman & Damasio, 1982; Ritchie *et al.*, 1997). Additionally, measured decline in IQ may be overlooked whilst it remains within the "normal" range even although it represents a decline by comparison to premorbid best. Bassett & Folstein (1991) highlighted additional factors such as geographical background, race and length of education as impacting on cognitive performance in older adults.

Poon, Krauss & Bowles (1984) considered all the papers on cognition over a two year period in a journal devoted to the study of older adults (N=41). Of these, only five provided information on education, intelligence, health and gender; factors which have all

been linked to aspects of cognition.

A third common methodological flaw is that the type of depression being studied is often not stated. In particular, 'bipolar' individuals have often been included in depressed samples. Burt, Zembar & Niederehe (1995), in reviewing 147 studies considering the link between depression and memory deficit, note that subtype of depression was not specified in 70 per cent of them.

Next, tests with unknown psychometric properties (McAllister, 1981; L. Miller *et al.*, 1991) and differing levels of difficulty (Boone *et al.*, 1995) have been employed in measuring cognitive deficit. This may affect judgement relating to severity of cognitive impairment. Anecdotal data has been employed (Lamberty & Bieliauskas, 1993) and the same individuals have often been tested and re-tested in the "depressed" and "recovered" state giving rise to the possibility of test practice contamination (Matarazzo, Carmody & Jacobs, 1980).

Age groups have often been mixed, arbitrarily defined or unstated (McAllister, 1981; W. Miller, 1975; Niederehe, 1986; Rohling & Scogin, 1993). This is significant since some of the cognitive deficits reported may reflect normal age-related change (Raskin *et al.*,1982; Parker, Hadzi-Pavlovic, Wilhelm, Hickie, Bridarty, Boyce, Mitchell & Eyers, 1994).

Specificity of target "deficits" by researchers may also influence findings. Weingartner (1986) noted:

> Investigators bring to the study of cognitive processes in depression .... their

> choices of target cognitive behaviours. If... a researcher is curious about attention, attention is what is studied, often in an isolated fashion.

Some studies have failed to employ control groups with a reliance instead on published test "norms" (Boone *et al.*, 1995). Alternatively, medical or psychiatric patients have been employed as controls. In such cases, identified deficit may be the result of disease processes other than depression and/or non-specific to depression but relating to more general psychiatric psychopathology.

The impact of medication on performance has not been given due credence. It may be that the improvement of depressive symptomatology using medication has little effect on cognitive performance (Georgotas, McCue, Reisberg, Ferris, Nagachandran, Chang & Mir, 1989). However, anti-depressants may actually cause cognitive impairment, particularly in older adults (Branconnier, Cole, Ghazvinian & Rosenthal, 1982; Moscowitz & Burns, 1986).

To conclude, methodological weaknesses have almost certainly contributed to conflicting results reported in studies investigating the presence and permanence of cognitive deficits in late-life depression. Selection and exclusion criteria forming the basis of experimental groups have not been consistently applied. A detailed focus on specific areas of interest has resulted in the utilisation of specialised and sometimes obscure tests which preclude the possibility of considering more broad cognitive function.

1.3.2 Cognitive/Psychological Approaches to Cognitive Deficit in Depression

Cognitive/psychological approaches focus on the application of theoretical models of information processing (rather than the defining features of a particular pathology) and, in particular, the distinction between automatic and effortful processing.

Automatic Versus Effortful Processing. Investigations with a focus on the idea of "automatic" versus "effortful" (attention-demanding) processing were derived from the work of Shiffrin & Schneider (1977), Schneider & Shiffrin (1977) and Posner (1978) and subsequently extended to memory by Hasher & Zacks (1979). The approach makes the assumption that the learning of well-structured material is less effortful than learning poorly organised material.

The distinction between types of processing led to the formation of hypotheses that depressed individuals could be:

a. deficient in effortful processing (which may be as a result of reversible biological change, learned helplessness (W.R. Miller & Seligman, 1975), cognitive interference or decreased motivation) or;

b. victims of the observation that effortful processing is a limited resource (Kahneman, 1973) depleted in depressed individuals due either to a tendency to ruminate on negative thoughts or an inability to filter out

relevant from irrelevant aspects of the task (Channon, Baker & Robertson, 1993).

Weingartner *et al.* (1981), employing a group of depressed individuals which included a number suffering from bipolar disorder, reported that depressed individuals do not apply effective learning strategies spontaneously despite having both the capacity to do so, if instructed, and the ability to benefit from instruction accordingly. They interpreted this finding as indicating that memory deficits witnessed in depression reflect impairment in the ability to allocate cognitive effort to increasingly effort-demanding tasks. Hasher & Zacks (1988) have proposed an age-related sensitivity to task irrelevant processing suggesting that older individuals have less control in this respect.

Effortful processing has been manipulated using paradigms such as varied task difficulty, degree of structure or specificity of retrieval demands. Research findings largely support a hypothesis involving deficient effortful processing in depression (Calev, Korin, Sahpria, Kugelmass & Lere, 1986; Golinkoff & Sweeney, 1989; Hartlage, Alloy, Vazquez & Dykman, 1993; Roy-Byrne, Weingartner, Bierer, Thompson & Post, 1986; Tancer, Brown, Evans, Ekstrom, Haggerty, Pedersen & Goldberg, 1990).

Hartlage *et al.* (1993), based on logical consideration rather than evidence, categorised WAIS-R sub-tests as effortful or automatic and, assessing individuals on this basis, reported that effortful processing was impaired in depression. The evidence would have been more credible had there been hard data to support the categorisation of subtests. By contrast, Kaufman, Grossman & Kaufman (1994) using a task designed according to the

Luria (1973) theory of cognitive complexity, were unable to demonstrate a difference between depressed individuals and normal controls on effortful and effortless tasks.

There is very little specific focus in this research field on older adults. Much of the work has methodological limitations similar to those encountered in the descriptive/comparative approaches including poorly defined depressed groups, failure to control for age, sex and education and failure to consider the specificity of the application to depressed, rather than more general psychiatric, groups. In addition, the difference between automatic and effortful processing is often not consistently defined.

The measurement of effort itself has been called into question. There has been little attempt to systematically examine the attentional demands of psychological tasks. Most tasks used in assessment have both automatic and effortful components. Furthermore, the automatic/effortful distinction may relate to psychomotor retardation which could in turn be due to impaired concentration, anxiety or low motivation (Kaufman, 1990).

In summary, deficient or limited effortful processing has been proffered as an explanation for cognitive deficit in depression. It has, however, proved difficult to make a convincing distinction between the automatic and the effortful and, indeed, to ascertain whether the origins of effort relate to conscious motivation, mood-related interference or psychomotor retardation. Composite models are also a possibility (Hartlage *et al.*, 1993).

1.3.3 Experimental Approaches to Cognitive Deficit in Depression

Experimental approaches to the investigation of cognitive deficit in late-life depression have focused on the possibility of intrinsic structural deficits and the corresponding clinical implications. This is in contrast to approaches described above stressing the inability to use fundamentally intact functions. Over 30 years ago Post (1968) commented '...subtle cerebral changes may make ageing persons increasingly liable to affective disturbance'.

Evidence of brain change in late-life depression is implied by the presence of a lower sedation threshold to barbiturates (Cawley, Post & Whitehead, 1973). In addition, depressed older adults demonstrate a latency in auditory evoked responses midway between individuals suffering from dementia and normal controls. This fails to return to baseline following recovery (Hendrickson, Levy & Post, 1979). There is also a higher than predicted prevalence of, and death rate from, vascular disease in depressed older adults (Murphy, Smith, Lindesay & Slattery, 1988).

A sophisticated body of experimental investigation has accrued quite rapidly from the use of modern imaging techniques (Baldwin & Simpson, 1997). The advent of structural techniques such as computed tomography (CT) and magnetic resonance imaging (MRI) and functional imaging methods including single photon emission tomography (SPECT) and positron emission tomography (PET) has renewed interest in linking neuropsychological theories to the anatomy and physiology of the brain.

CT. CT is the most common brain imaging modality. It is based on the measurement of

the absorption of x-rays. It shows bone well, being particularly sensitive to calcium distribution, and also ventricles and cerebrospinal fluid (CSF). It is less useful in the elucidation of non-functioning tissue, clots or detailed structures near bone.

In the first of a triad of papers (Jacoby, Levy & Dawson, 1980; Jacoby & Levy, 1980a, b), Jacoby *et al.* (1980) used CT to scan 50 "normal" older adults (mean age 73 years) and reported an increase in cortical atrophy as a function of normal ageing. Increased atrophy correlated with lower - albeit "normal" - scores on a memory and orientation test, although this observation disappeared when age was controlled. It was tentatively proposed either that some of the group may have been suffering from undetected early stage dementia or, alternatively, that the sort of non-progressive cognitive impairment sometimes witnessed in normal older adults has a structural basis.

Other groups have noted significantly increased cortical atrophy in depressed individuals by comparison to controls (Dolan *et al.*, 1985; Dolan, Calloway, Thacker & Mann, 1986). These studies however involved only individuals with depression severe enough to warrant hospitalisation. As a consequence, utilisation of ECT may have been commonplace and causal in demonstrated atrophy. Conclusions cannot therefore be extrapolated to depressed individuals as a whole.

Jacoby & Levy (1980a, b) went on to compare two 'clinical' groups (those with Alzheimer's disease and those with 'affective disorder') to the earlier identified 'normal' group (Jacoby *et al.*, 1980). Individuals with dementia had a higher degree of cortical atrophy than the other two groups (although there was considerable overlap).

Of interest, however, was the observation that lower scores on cognitive tests in individuals with dementia appeared to be linked to degree of ventricular enlargement rather than the cortical atrophy which had appeared to account for any deficits observed in "normals". This suggested the possibility of a structural contribution in this group distinct from any accounted for by normal or accelerated ageing. Similar findings have been reported by Kellner, Rubinow & Post (1986) and Abas *et al.* (1990).

In considering Jacoby & Levy's (1980b) 'affective' group as a whole (N=41), impaired cognition was reported but not linked to degree of ventricular enlargement and thought to represent the reversible phenomenon of 'pseudodementia'. However, a small subgroup (N=9) did have enlarged ventricles and were clinically characterisable from the remainder of the affective group by being late-onset cases, older, less anxious and more 'endogenous' as measured by the Newcastle Scale. This was in direct contrast to the work of Abas *et al.* (1990) and Beats *et al.* (1996) where increased ventricle size and unremitting cognitive deficit appeared to be linked to early onset depression and increased number of depressive episodes.

The sub-group with enlarged ventricles identified by Jacoby *et al.* (1980), and characterised by late-onset depression, showed higher mortality on follow-up (Jacoby, Levy & Bird, 1981) but no evidence of developing dementia. In contrast, in performing a retrospective analysis of data collected by Baldwin & Jolley (1986), Baldwin (1990) uncovered no such association between late onset depression and increased mortality.

More recent research appears to have confirmed the presence of greater enlargement of ventricles in late-onset compared with early onset depression (Alexopoulos, Young & Schindledecker, 1992; Pearlson *et al.*, 1989; Steingart & Herrmann, 1991). Alexopoulos *et al.* (1992) additionally noted the similarity between the ventricles of late-onset depressed individuals and individuals with Alzheimer's disease. They concluded that late-onset depression may have a stronger association with dementia than early-onset depression and possibly form part of a 'spectrum' model (after Malhendra, 1985). Differences did not appear to be due to severity of depression between the groups as both presented a 'similar clinical profile'. However, there was limited data regarding the ECT or medication status of the individuals involved. Tricyclics are known to cause fluid shifts (Preskorn, Hartman, Irwin & Hughes, 1982) so that water movement may have been responsible for ventricular enlargement. Another confounding issue may be the weight loss frequently observed in depression. Some anorexic patients show ventricular enlargement and sulcal widening (Yeats, Jacoby & Andreasen, 1987) which reverses on re-feeding. Ventricular enlargement in depression therefore may have been a result of starvation. Conversely, however, enlargement seen on starvation may reflect a depressive component to anorexia. Whatever the mechanics of the observed abnormality, the possibility is raised that at least a proportion of CT anomalies may be reversible.

With increasingly sophisticated methods of analysis, later work by Jacoby, Dolan, Levy & Baldy (1983) measured brain tissue density in the same three groups of control individuals, those with dementia and those with depression. Whereas the depressed group had more closely resembled control individuals than those with dementia with regard to ventricular

enlargement and cortical atrophy, when brain tissue density was considered, the group were more similar to individuals with dementia. Again, most of the variance in tissue density was accounted for by the sub-group of nine identified as having large ventricles. No relationship between enlarged ventricles and low tissue density was noted in the control group. The possibility of abnormal age related change was discounted on the basis that there did not appear to be any link between brain tissue density and ageing in any of the three groups studied.

To conclude, cortical atrophy appears to be a function of normal, age related decline (Krishnan 1993) and may be a feature in the non-malevolent 'benign senescent forgetfulness' witnessed with increasing age (Deary, 1995).

Both cortical atrophy and ventricular enlargement are increased in depressed individuals and individuals with dementia (Beats, Levy & Forstl, 1991). Cognitive decline may be more a function of ventricular enlargement than cortical atrophy in both groups although this is not to suggest that depression necessarily progresses to dementia.

Controversy surrounds attempts to separate early versus late onset cases of depression in terms of observable structural deficit. The most frequently reported finding is that of increased ventricular enlargement in late onset depression. The question of reversibility remains unresolved.

Despite confusing and conflicting findings, the possibility of a distinct, structurally-related

profile of deficits in at least a proportion of older adults suffering from depression is suggested.

Methodological Flaws in CT Studies. The Jacoby *et al.* (1980, 1980a, b) studies continue to represent one of the most important and thorough investigations of CT scanning in older adults to date (Burns, 1998). Nevertheless, they had a number of flaws.

Firstly, there were technical limitations. Despite reported high inter-rater reliability, ratings of scans were crude and made, to some degree subjectively, by neuroradiologists with no knowledge of CT history which may have included a life-long pattern of abnormality rather than representing change. Comparisons to other studies were made difficult by differing methods of CT analysis and inter-machine variations. The battery of tests used to assess cognitive change, which was rather vaguely described in the papers, may also have been insensitive to subtle cognitive change.

Entry criteria for groups was not exclusively stringent. The affective group included some individuals with manic depression and was drawn from a lower social class than the control group whom Jacoby himself later described as 'super normal' (Jacoby, 1998). Despite the reported characteristics of greater 'endogenous' features and later onset in the affective sub-group of nine, it is not specifically stated that this group did not include any of the individuals suffering from bipolar disorder. If so, this may have had a bearing on findings, particularly in such a small group.

There may have been a biased focus on a population of interest in the further analysis of the affective sub-group. In fact, eight (of 50) of the control group were also noted to have enlarged ventricles and were not separately investigated as a sub-group in the same way that the nine from the affective group were. Ventricular enlargement has more recently been reported in about 33 per cent of "unselected" individuals over 60 years of age (Fein, Van Dyke, Davenport, Turetsky, Brant-Zawadzki, Zatz, Dillon & Valk, 1990).

MRI. MRI is sensitive to water distribution and uses radio frequency radiation in the presence of a magnetic field to create cross-sectional images of the body. It has proved a more sensitive measure of structural change than CT (Baldwin, 1993). One of its main advantages is the ability to obtain multiplanar images. In addition, different types of images emphasising different structures can be obtained (Krishnan, 1993). MRI has facilitated an emphasis and expansion upon the changes noted using CT.

Utilisation of MRI has led to the reporting of more detailed and specific structural changes in older adults suffering from depression including widening of the cortical and temporal lobe sulci, the temporal horns and the Sylvian fissures and enlargement of the pituitary (Krishnan, 1991; Krishnan, Goli, Ellinwood, France, Blazer & Nemeroff,1988; Rabins, Pearlson, Aylward, Kumar & Dowell, 1991; Zubenko, Sullivan, Nelson, Belle, Huff & Wolf, 1990). It is not clear to what degree such changes are accountable for in terms of individual variation or at what point "normality" becomes "pathology".

Some of the criticisms levelled at CT research are equally applicable to MRI. In particular, a failure to quantify MRI changes is evident (Salloway, Malloy, Kohn, Gillard, Duffy,

Rogg, Tung, Richardson, Thomas & Westlake, 1996), there is a lack of normative data and a failure to use formal rating scales to assess the severity and extent of observed abnormalities is evident (Coffey, Figiel, Djang & Weiner, 1990).

White Matter Lesions. The main focus of MRI investigations has been in describing changes in the subcortical white matter and deep grey matter of older adults with depression. These changes or lesions are variously described as 'patchy white matter lesions', 'leukoencephalomalacia', 'leukoaraiosis', 'periventricular hyperintensity' or 'unidentified bright objects' (Baldwin, 1993).

It has been suggested that white matter lesions are more prevalent in individuals with depression than in either controls or individuals with Alzheimer's disease (O'Brien, Desmond, Ames, Schweitzer, Harrigan & Tress, 1996).

However, white matter lesions are not unique to late-life depression. They have been reported in other affective disturbances including depression in younger individuals, manic depression and psychotic depression as well as in other subcortical conditions that commonly include elements of depression such as Huntingdon's Chorea and Multiple Sclerosis (Coffey, Figiel, Djang, Saunders & Weiner, 1989; Krishnan *et al.*, 1988).

It seems possible that the likelihood of the presence of white matter lesions increases both as a feature of normal ageing and in the presence of çardiovascular disease (Fazekas, 1989; Sarpel, Chaudry & Hindo, 1987; Zubenko *et al.*, 1990). Fein *et al.* (1990) reported the presence of widespread white matter hyperintensities in the brains of healthy older adults

with no psychiatric illness over a seven year follow-up period and concluded that they were of no clinical significance. However, despite the observation that lesions can be widespread, this does not imply that they are necessarily non-specific. It may be, for example, that some of the lesions are always associated with the development of depression whilst others contribute little to the expression of the syndrome. Coffey *et al.* (1990) and Figiel, Krishnan, Doraiswamy, Rao, Nemeroff & Boyko (1991) point, for example, to the specific involvement of the basal ganglia, caudate and thalamus. They reported lesions in this region in up to 60 per cent of individuals suffering from late-life depression by comparison to 5-6 per cent of controls. Greenwald, Kramer-Ginsburg, Krishnan, Ashtari, Aupperle & Patel (1996) support this specificity.

Coffey *et al.* (1990) proposed a link between vascular disease and the presence of white matter lesions, an association which Rabins *et al.* (1991) and Boone, Miller, Lesser, Mehringer, Hill-Guttierrez Goldberg & Berman (1992) both failed to replicate. Howard, Beats, Forstl, Graves, Bingham & Levy (1993) controlled for vascular risk factors but still measured a higher rate of white matter lesions in depressed individuals compared to normal controls. Similar results have been reported by other groups (Greenwald, Kramer-Ginsburg, Bogerts, Ashtari, Aupperle, Wu, Allen, Zeman & Patel, 1997; Krishnan, McDonald, Doraiswamy, Tupler, Husain, Boyko, Figiel & Ellinwood, 1993; Nussbaum, Kaszniak, Allender & Rapesak, 1991).

Research with healthy older volunteers has reported strong associations between white matter lesions and increased cerebral atrophy (Kobari, Meyer & Ichijo 1990a), ventricular enlargement (Coffey, Figiel, Djang, Cress, Saunders & Weiner, 1988; Coffey *et al.*, 1989),

decreased cerebral blood flow (Kobari *et al.*, 1990a; Kobari, Meyer, Ichijo & Oravez, 1990b) and decreased cognitive function (Almkvist, Wahlund, Anderson-Lundman, Basun & Backman, 1992) making it difficult to conclude that white matter lesions in themselves are any sort of structural marker of depression in older adults. They may in fact precede and cause depression. Equally however, they could be an artefact of other structural changes, could precipitate rather than cause depression, may represent the result of chronic exposure to depression or indeed result from ECT or medication used to treat depression.

Hence, white matter lesions are not specific to depression in older adults and their role in the etiology of depression is, at best, unclear. There is a possibility of the specificity of lesions in particular areas being related to depression. Nevertheless, lesions also appear as a function of increasing age and in pathological conditions other than depression and may have corresponding cognitive effects in each of these.

White Matter Lesions and the Early Versus Late Onset Distinction. MRI investigations appear more robust than CT findings in their support of the early versus late onset distinction in depression (Conwell, Nelson, Kim & Mazure, 1989).

White matter lesions have been associated with late-onset depression and reported in 85 (Krishnan *et al.,* 1988) to 100 per cent (Coffey *et al.,* 1989, Coffey *et al.,* 1990) of such individuals. Coffey *et al.* (1989, 1990), however, included 10 (out of 51) patients with a history of dementia prior to the current onset of depression as well as individuals with stroke history and Parkinson's disease. There was no control group and it seems likely

that there may have been an over-estimation of the frequency of white-matter hyperintensity relating specifically to depression. In a later study (Coffey, Wilkinson, Weiner, Parashos, Djang, Webb, Figiel & Sprizter, 1993), a control group was employed but many of the individuals in both control and experimental groups were at high risk for cerebrovascular disease (50 per cent were hypertensive and others had histories of heart disease or diabetes) and/or had experienced abnormal neurological investigation. In all of the studies, the depressed group comprised hospitalised individuals who had been referred for ECT. A high proportion had undergone a previous, unstated, number of ECT sessions, and four bipolar individuals were included.

Figiel *et al.* (1991) noted a higher occurrence of white matter hyperintensities (60 versus 11 per cent) in late versus early onset depressed patients. Lesions were also larger in the late onset group. Similar findings have been reported by other groups (Fujikawa, Yamawaki & Touhada,1993; Krishnan *et al.*, 1993). The results from the studies of Fujikawa *et al.* (1993) were not duplicated, however, in a smaller replication (Churchill, Priolo, Nemeroff, Ranga, Krishnan & Breitner, 1995) although, in this study, "late" onset was considered to be first depressive episode after age 45 with the justification that white matter lesions are not usually observed in the normal population until after this time. The study was also retrospective, relying on file review.

Lesser, Boone, Mehringer, Wohl, Miller & Berman (1996) hypothesised, and subsequently reported, that individuals with late-life depression would show more white matter hyperintensity than those with early onset depression or controls. Furthermore, it was suggested that depressed individuals with more white matter hyperintensity would show

more cognitive deficit than depressed individuals with small amounts. The study group was not age-matched.

White Matter Lesions and Cognitive Impairment. Steingart, Hachinski, Lau, Fox, Diaz, Cape, Lee, Inzitari & Merskey (1987) reported statistically-significantly reduced test scores on the extended dementia scale of normals with white matter lesions compared to those without. Schmidt, Fazekas, Offenbacher, Dusek, Zach, Reinhart, Grieshofer, Freidl, Ebel, Schumacher, Koch & Lechner (1993) in a study of 150 'normal' older volunteers reported wide-ranging cognitive deficit in the presence of white matter lesions, vascular risk factors and increased age. When vascular risk and age were controlled, cognitive deficit remained only on Form B of the Trail Making Test. This suggested that any deficit specific to the presence of white matter lesion was subtle in nature and perhaps only evident in tasks measuring the speed of more complex processing. No link was reported between estimated total area of white matter lesion and test scores. There was no indication of lesion severity increasing over a two year period arguing against the suggestion that identified deficits represented early dementia. Ylikoski, Ylikoski, Erkinjuntti, Sulkava, Raininko & Tilvis (1993) also noted a link between white matter lesions and speed of processing in normal older adults. Study strengths included usage of a large community sample (120) and an extensive battery of tests.

Fein *et al.*, (1990), followed three subjects with white matter hyperintensities for three years and noted that, for two of them, neurological and cognitive performance remained stable. The authors themselves, however, concede that MRI scans at the two periods in time were not comparable due to advances in techniques. Furthermore, since they

presented no premorbid estimate of ability, it may have been that their (school-teacher) subjects had, in fact, suffered a decline in cognitive ability, of insignificant clinical relevance, prior to initial presentation.

Austrom, Thompson, Hendrie, Norton, Farlow, Edwards & Dean (1990) scanned 26 "normal" older, highly-educated individuals using MRI and followed them up over a two year period. Sixteen were noted to have white matter lesions. No declines in broad cognitive measures were reported and, furthermore, there was no change in health status as measured by visits to the GP, hospitalisation and perceived health satisfaction/cognitive change. These observations were consistent with the assertions made by others that white matter lesions merely represent benign age change (Awad, Spetzler, Hodak & Awad, 1986a; Brant-Zawadaski, Fein, van Dyke, Kiernan, Davenport & DeGroot, 1987; Rao, Mittenberg, Bernardin Haughton & Leo, 1989). However, in individuals in whom white matter hyperintensities had been observed, performance on the Digit Symbol Substitution Test (DSST) was significantly impaired on follow-up by comparison to their earlier scores. Although this group were older, and age is associated with slower performance on the DSST, the differences between populations with and without white matter lesions were only evident on follow-up, thereby suggesting deficit resulting from something other than mere age. The authors proposed that lesions may be associated with subtle learning and memory differences. Unfortunately, MRI scans were not repeated on follow-up. The study would also have benefited from broader and more demanding neuropsychological tests.

White Matter Lesions and Cognitive Impairment in Depression. There has been

considerable debate surrounding the relationship between observed white matter hyperintensities in late-life depression and cognition. A substantial body of evidence has failed to report a link (Bernardin, Rao, Haughton, Yetkin & Ellington, 1991; Brant-Zawadzki *et al.*,1987; Hunt, Orrison, Yeo, Haaland, Rhyne, Garry & Rosenberg, 1989; O'Brien *et al.*, 1996; Rao *et al.*,1989; Schmidt, Fazekas & Offenbacher, 1991) although the utilisation of broad and insensitive screening measures, such as the mini mental-state examination (MMSE), as an indicator of cognitive function may have influenced findings (Folstein & Folstein, 1975; O'Brien *et al.*, 1996).

Support for the view that white matter hyperintensities are associated with a variety of mild, but definite, dysfunctions in depressed older adults is reported by Boone *et al.* (1992), Breteler, van Amerongen, van Swieten, Claus, Grobbee, van Gijn, Hofman & van Harskamp (1994), Lesser *et al.* (1996) and Salloway *et al.* (1996). Executive function deficits and decreased processing speed are amongst the most frequently reported deficits (Delpla, Zatorre, Meyer, Geraud, Bes & Ethier, 1990; Junque, Pjol, Vendrell, Bruna, Jodar, Ribas, Vinas, Capdevila & Marti-Vilalta, 1990; van Swieten, Geyskes, Derix, Peeck, Ramos, van Latum & van Gijn, 1991). A link between white matter lesions in depressed older adults and motor dyspraxia has also been reported (Meyer, Kawamura & Terayama, 1992).

There has also been some support for the assertion that the most severe patterns of cognitive deficit are observed in late-onset depressed patients with more severe white matter lesions (Gupta, Naheedy, Young, Ghobrial, Rubino & Hindo, 1988; Steingart *et al.*, 1987; Steingart, Hachinski, Lau, Fox, Fox, Lee, Inzitari & Merskey, 1987). A lack of

agreement remains as to whether impairment is reversible, static or progressive (Abas *et al.*, 1990; Kral & Emery, 1989; Pearlson *et al.*, 1989). Lesser, Hill-Gutierrez, Miller & Boone (1993) report a case of a man originally assessed as a "normal" control subject by MRI and SPECT who subsequently developed a major depression that was temporarily associated with an increase in the number and size of white matter lesions. As a control participant, he had demonstrated a large area of lesion (only eight of the 100 "healthy" controls had more). However, during his illness, lesions more than tripled.

By way of explanation for the disparate findings, Boone *et al.* (1992) suggested that a threshold of hyperintensity must be exceeded before cognitive deficit, particularly frontal-type deficits, are noted. They also point out that Bernardin *et al.* (1991) and Rao *et al.* (1989) had, in fact, obtained some significant between-group differences on frontal tests but had assumed that these were chance findings due to multiple comparisons. The Rao *et al.* (1989) study was also based on a small sample who had minimal white matter lesions.

Following a similar line of argument, it may alternatively be the case that a particular combination of brain changes has to occur in order to be relevant to clinical presentation. Simpson, Jackson, Baldwin & Burns (1997) argue that total white matter load has no prognostic value (in direct contrast to Hickie, Scott, Mitchell, Wilhelm, Austin & Bennett, 1995) whilst involvement in three areas (basal ganglia, pons and frontal lobe) affects treatment response and has differential, if modest, neuropsychological relevance. In particular, hyperintensities correlated with the recollection of learned information when distracted, with planning and with tests requiring effort, attention, persistence and speed.

The study, however, did not employ a control group, disallowing the possibility of normal age-related change.

To summarise, controversy surrounds the assertion that white matter lesions are associated with cognitive decline. The wide range of reported results reflects differences in the cohorts studied including mean age, presence of associated "risk" factors, definitions of "normality" of participants, definitions of white matter lesions themselves and types of equipment used (Meyer *et al.*, 1992). The presence of so many methodological flaws calls the validity of results into question.

It may be that it is necessary for some threshold of lesion volume to be exceeded prior to an impact on cognition occurring and/or that the anatomic location of lesions is significant.

There does appear to be consistent evidence that neuroimaging findings are linked to neuropsychological impairment (Abas *et al.*, 1990; Kellner *et al.*, 1986; Lesser, Miller, Boone, Hill-Gutierrez, Mehringer, Wong & Menai, 1991; Nussbaum *et al.*, 1991; Pearlson *et al.*, 1989) although specificity to depression and, in particular, late-onset depression, is less certain. Questions persist about whether structural abnormalities are state, episode or trait findings. Studies of depressed individuals are required both during and following episodes of depression to see if imaging findings are modified.

Functional Imaging. PET and SPECT have been used in recent years as research tools. They yield information about various functions of the brain tissue as revealed by parameters such as oxygen consumption, blood flow or glucose uptake. This "dynamic"

data complements the anatomical findings of CT and MRI.

More definitive support for the involvement of the basal ganglia in late-onset depression has been reported in PET studies (Baxter, Phelps, Mazziotta, Schwartz, Gerner, Selin & Sumida, 1985). More widespread regional dysfunctions are also implicated (Sackeim, Prohovnik, Moeller, Brown, Apter, Prudic, Devanand & Mukherjee, 1990; Upadhyaya, Abou-Saleh, Wilson, Grime & Critchley, 1990).

With early exceptions (Silfverskiold & Risberg, 1989) functional imaging studies have reported both global flow deficit and abnormal regional distribution in depressed older adults, (Bench, Friston, Brown, Scott, Frackowiak & Dolan, 1992; Bench, Friston, Brown, Frackowiak & Dolan, 1993; Sackeim *et al.*, 1990). In particular, decreased blood flow to the temporal and frontal lobes has been noted (Philpot, Banerjee, Needham-Bennett, Campos-Costa & Ell, 1993; Upadhyaya *et al.*, 1990). Functional imaging research supports not only the hypothesis of structural damage in depression (Martinot *et al.*, 1990) but also that of a profile of blood flow distinct from that observed in dementia (Dolan, Bench, Brown, Scott, Friston & Frackowiak, 1992).

Bench *et al.* (1993) stressed that it was not entirely clear whether abnormalities observed were state or trait dependent. This is of particular relevance when functional imaging is often performed with individuals under different conditions and with no attention to prevailing emotional state (Kling, Metter, Riege & Kuhl, 1986; Reischies, Helle & Drochner, 1989). Researchers seldom refer to the results of contemporaneous structural imaging studies. In addition, functional imaging abnormalities have not consistently

correlated with clinical features (Caine, Lyness, King & Connors, 1994). Comparison of functional and structural imaging findings in the same individuals may enhance understanding of observations regarding apparently abnormal blood flow in depressed older adults.

In summary, results reported from functional imaging research are largely supportive of a link between late-life depression and structural abnormality. Taken in isolation, caution should be exercised in interpretation of results because of the number of variables which could contribute to determination of brain metabolism or blood flow at any one moment.

1.4 Depression and Dementia

It has been suggested that the cognitive decline sometimes witnessed in late-onset depression is merely an indicator of early stage dementia. Observed deficits may be similar to those resulting from forms of irreversible brain dysfunction and yet several early studies report high incidences of a diagnosis of dementia being made in cases where the cognitive deficit has later been, to all intents and purposes, reversed (Duckworth and Ross, 1975; Marsden and Harrison, 1972; Nott and Fleminger, 1975; Ron, Toone, Garralda & Lishman, 1979). Individual researchers have different explanations for how misdiagnosis has occurred. Nott & Fleminger (1975) emphasised personality disorder and 'neurotic' illness, whilst Ron *et al.* (1979) reported that over half of their sample were depressed and the other half had a combination of affective illness and either transient cerebral dysfunction or structural brain disease. It is of note that despite the failure of the samples

to develop dementia, they nevertheless remained, on the whole, very functionally disabled at follow-up.

The 'pseudodementia' syndrome was first described by Kiloh (1961). In an effort to counter the growing confusion with the concept of pseudodementia and underscore the validity of the cognitive impairment observed in some depressed older adults, new clinical terms have emerged (Nussbaum, 1994) including 'dementia syndrome of depression' (Folstein & McHugh, 1978), 'depression induced organic mental disorder' (McAllister, 1983) and 'depression related cognitive dysfunction' (Stoudemire, Hill, Gulley & Morris, 1989).

Confusion and uncertainty remains as to how closely the cognitive impairments observed in late life depression actually resemble those seen in dementia (Poon 1992) and the extent to which they are in fact reversible on recovery (Abas *et al.* 1990). Differences in cognitive deficit as a function of subtypes of dementia may add further confusion. Alzheimer's disease is, for example, characterised by global impairment including memory, abstract reasoning, judgement, language and other abilities. However, in frontal and subcortical dementias, some abilities are spared until later in the disease.

Zarit & Zarit (1998) attempted to provide some guidelines for discrimination between the memory difficulties reported in normal age related change and those in individuals suffering from dementia and depression. They suggested that whilst all three groups report memory difficulties, those pertaining to normal age related change do not usually interfere with everyday functioning. In depression, the focus is on subjective perception of

memory loss and its meaning whilst in dementia individuals may be aware of memory difficulties, (particularly in vascular dementia), but unable to give examples. The importance of considering carer reports is emphasised. Whereas in dementia families will refer to pronounced difficulties, an 'absent-mindedness' or 'preoccupation' is more often reported in depression.

Efforts continue to establish criteria for making an accurate distinction between depression and dementia (Huppert, 1994; Zarit & Zarit, 1998). Rabins, Merchant & Nestadt (1984) propose that the two syndromes are distinguishable on 'standard clinical observation' by reference to past and onset history, mood, appetite and weight, physical ill health, and symptoms of self-blame and hopelessness. Differentiation has also been made on the grounds of the "near miss" attempts of the demented versus the "don't know" response of the depressed on cognitive tasks. Unfortunately, studies from which such conclusions have been drawn (Post, 1975; Young, Manley & Alexopoulos, 1985) involved groups of individuals with dementia and depression who were not matched for degree of cognitive impairment so that results may in fact have confounded qualitative features of errors with overall level of performance.

1.4.1 Longitudinal Follow-up Studies

Initial follow-up studies of depressed older adults broadly suggested a risk of subsequently developing dementia no greater than that of the general population (Post, 1972; Roth, 1955). However, subsequent work from the same group did report that, on autopsy, 25 per cent of those considered to be suffering from 'functional psychoses' did in fact show

degenerative change. This overlap was considered to represent, at least in part, the coexistence of the two disorders (N. Miller, 1980). Kral (1983) estimated that at least 15 per cent of individuals with dementia have coexisting depression. More recent estimates are as high as 30 per cent (Teri & Gallagher-Thompson, 1991).

In two "long-term" follow-up studies (only 2 years), it was reported that coexisting depression and cognitive impairment did not usually presage progressive dementia (Pearlson *et al.*, 1989; Rabins, Merchant & Nestadt, 1984). Other studies have also failed to demonstrate the development of dementia following an incident of uncomplicated depression (Baldwin & Jolley, 1986; Murphy 1983).

However, there has been some suggestion of increased likelihood of pre-disease episodes of depression in individuals suffering from Alzheimer's disease (Agbayewa, 1986). It is hard to draw causal conclusions from such an observation. It may be coincidence. Alternatively, depression may represent a reaction on the part of individuals concerned to a realisation, preceding diagnosis, that all is not well (Godwin-Austen & Bendall, 1990). It may point simply to coexistence of the two disorders, may suggest that dementia is triggered by structural changes occurring in depression or may suggest that depression is a result of structural changes resulting from early dementia.

Pearlson, Ross, Lohr, Rovner, Chase & Folstein (1990) reported a link between first onset of depression in Alzheimer's disease and history of family affective disorder. This is contrary to the more usually reported pattern of late-onset depression as being less likely to have genetic links and would support the argument for a distinction between late-onset

depression with cognitive deficit and no genetic link and Alzheimer's disease in which the concurrent appearance of depression may be genetically linked (Wurthmann, Bogerts & Falkai, 1995).

By contrast, a number of studies have reported that a high proportion of depressed, non-demented patients do in fact go on to develop dementia (Alexopoulos *et al.*, 1992; Kral & Emery, 1989; McAllister & Price, 1982; Rabins *et al.*, 1984; Reding, Haycox and Blass, 1985; Wells, 1979).

Methodological difficulties may again cloud the issue and, in particular, the failure to apply stringent enough selection criteria to selected samples when studying the effects of depression on cognition. Examples have been referred to in the preceding text. Both Kral & Emery (1989) and Alexopoulos *et al.* (1992) may have included individuals suffering from dementia in their studies even although their cognitive function improved temporarily with treatment for depression. Referral bias may have resulted in an over-representation of such cases (Baldwin, 1997). It is also possible that some of the studies which report no association between late-life depression and the subsequent development of dementia may reflect inadequate length of follow-up.

1.4.2 Structural Components of Dementia

White matter lesions are extremely common in dementia with an estimated incidence of between 72 and 97 per cent (Aharon-Peretz, Cummings & Hill, 1988) and between 64 and 71 per cent (Ebmeier *et al.*, 1997). Severity of lesion has been reported as correlating with

measured cognitive impairment (Meyer, 1991).

To conclude, it would appear an oversimplification to suggest that cognitive impairment in depression necessarily presages dementia. The syndromes do share common features and can be indistinguishable in the short-term in some individuals. However, marked differences between the syndromes have also been recorded.

1.5 Normal Ageing

Many of the biologic changes associated with ageing are similar to those occurring in both depression and dementia (Alexopoulos, Meyers, Young, Abrams, & Shamoian, 1988; Alexopoulos, Young & Meyers, 1988). One of the criticisms levelled at work linking depression in older adults to cognitive deficit has been that of a failure to give due credence to the effects of normal age related change.

1.5.1 Structural Changes

Structural changes including decreased brain volume, increased cerebrospinal fluid (CSF), cortical atrophy and enlargement of ventricles are common in the normal ageing process. The likelihood of the presence of white matter lesions is increased and a decrease in a variety of subcortical structures is noted (Zubenko *et al.*, 1990).

Ageing has also been associated with decreased cerebral blood flow, possibly more so for men than women (Busse, 1989). Reductions in serotonin and noradrenaline, increased

monoamine oxidase activity and alterations in neuroendocrine functions are all features of normal ageing that have also been linked to the development of depression.

1.5.2 Age-Related Cognitive Impairment

Some degree of cognitive impairment may be a normal concomitant of ageing (Kramer, German, Anthony, VonKorf & Skinner, 1985) although the range of abilities of older versus younger people is broader and their depth of experience greater. Educational opportunity, nutritional changes, degree of competitiveness, motivation and declines relating to physical health or sensory loss may confound findings (Holland & Rabbitt, 1991).

Horn & Cattell (1967) divided intellectual skills into 'crystallised' (acquired and accumulated) and 'fluid' (adaptation to novel situations, grasping new ideas, reasoning rapidly) abilities. It seems that these latter skills, requiring the 'ability to solve problems for which there are no solutions derivable from formal training or cultural practices' (Stuart-Hamilton, 1995) are 'vulnerable abilities' (Horn, 1989), those which contribute most significantly to measured decline even when education is controlled for and time constraints removed (Wang & Kaufman, 1993). Crystallised abilities, on the other hand are 'maintained' abilities and may well even continue to improve up until the seventh decade of life (Kaufman & Horn, 1996; Kaufman, Reynolds & McLean, 1989; Rabbitt, 1988; Stuart-Hamilton, 1996).

Why fluid abilities might deteriorate with age is a matter of debate. At the most simplistic level, it may be a function of the reduction of efficiency of just about every physical process in the body. The fact that the senses are in decline means that information received by the brain is compromised and correlations have been reported between sensory functioning and intellectual ability (Lindenberger & Baltes, 1994). Metabolic and cardiovascular activity decline mean that the supply of essential nutrients to the brain wanes. In addition, the central nervous system is also in decline with decreased speed of neural transmission, irreplaceable loss of cells and an increase in neural noise (Stuart-Hamilton, 1999).

1.5.3 Depression, Ageing and Cognition

Why cognitive deficit is so frequently observed in late-life depression is also a matter of debate. Depression may affect old and young equally but prove "additive" in older adults in combination with age related decline. Alternatively, depression may have a greater cognitive effect on the old versus the young (Deptula, Singh & Pomara, 1993; Raskin, 1986) or it may be that older adults with lower intelligence are particularly and differentially affected by the cognitive effects of depression. O'Brien, Sahakian & Checkley (1993) reported broad cognitive impairment in young, unmedicated individuals suffering from Seasonal Affective Disorder (SAD) during their depressed phase. It was, however, mild by comparison to the deficit observed in older depressed individuals matched to the younger group according to depression severity. O'Brien *et al.* (1993) viewed this as support for the assertion that old age results in additive or exacerbated deficit. However, their group consisted of only 11 individuals and the inclusion of a

young, depressed (non SAD) group would have provided a more comprehensive comparison. Additional support for additive or exacerbated effects of depression in older individuals has been proposed by Niederehe (1986) and King *et al.* (1991).

By contrast, Boone, Lesser, Miller, Wohl, Berman, Lee & Palmer (1994), controlling for educational level and severity of depression, compared three groups aged 46-59, 60-69 and 70-85 and failed to find support for the assertion that age has an additive or exacerbating effect on the cognitive declines observed in depression. Participants, however, were all of above-average intelligence. This may have served as a "protective" factor either in terms of actual failure to decline or, alternatively, in performance decline that was unrecorded due to a failure to sink below a threshold for measurable deficit (Poon, 1992; Raskin *et al.* ,1982).

Overall, research efforts addressing the links between late-life depression, structural deficit and cognitive impairment have been hampered by both cognitive and medical heterogeneity inherent to the ageing process (Caine *et al.*, 1993). Variability in findings may, at least in part, be due to a confounding of this interface (Jorm, 1986).

1.6 Psychometric Measurement of Cognitive Deficit

Much of the research to date has investigated a variety of unspecified types of depression (Weingartner & Silbermann, 1982) using discrete and wide-ranging cognitive tasks (Poon, 1982). Such methodology fails to capture the assessment of the inter-relationship between, and among, tasks and an appreciation of how tasks are differentially affected by

the specific psychopathology under investigation. Methodologies would be enhanced by studying cognitive profiles constructed from performances in meaningful clusters of tasks.

1.6.1 The WAIS and the WAIS-R

The WAIS and WAIS-R have been the most commonly used measures of cognitive performance world-wide (Kluger & Goldberg, 1990) perhaps because of their ability to evaluate a range of function providing a broad-based evaluation of cognitive performance. The measures were never, however, formally standardised in the UK (J. Crawford, Gray & Allan, 1995). One of the most significant advantages of these measures has been the ability to compare an individual's performance to that of their peers.

In the case of older adults, research using the WAIS-R has been limited by the standardisation age limit of 75. Furthermore, although corrections are made at IQ level, scaled scores for individual sub-tests are calculated according to a "reference group" aged 20-34. Accordingly, WAIS-R profiles, even in "normal" older adults, are suggestive of "impairment" and do not constitute a model into which the normal features of ageing have been incorporated. This makes it impossible to make comparisons of distinctive sub-test patterns between older adults with specific disorders and their same-aged peers starting from a stance where the latter group are considered "normal".

The WAIS-R has proved largely unsuccessful as a diagnostic tool. Donnelly, Dent & Murphy (1972) using the WAIS and Halstead-Reitan battery reported that 69 per cent of their sample of depressed individuals scored in a range consistent with mild to severe brain

damage. The sample consisted however of only 13 individuals who were not re-tested on recovery. Logsdon, Teri, Williams, Vitiello & Prinz (1989) tried to make a distinction between normal controls, depressed individuals and individuals suffering from dementia using the Fuld (1984) profile based on WAIS-R subtests:

Using age-corrected scores:

$$\frac{\text{Information + Vocabulary}}{2} > \frac{\text{Similarities + Digit Span}}{2}$$

$$> \frac{\text{Digit Symbol + Block Design}}{2} < \text{(or equal to) Object Assembly}$$

and:

$$\frac{\text{Information + Vocabulary}}{2} > \text{Object Assembly}$$

They reported that the profile was unhelpful in distinguishing dementia from other common disorders. Bornstein, Termeer, Longbrake, Heger & North (1989) also employed the Fuld profile in a study of older adults (N=62) concluding that it was less common in depressed individuals (16 per cent) than those with dementia (44 per cent) individuals but similar to controls (12.8 per cent). The sample used in the study were over 50 and referred for depression although their histories of depression were not recorded.

Mood disorder has been linked to lowered Performance IQ scores (Kluger & Goldberg, 1990). The relevance of this finding is unclear (Loro & Woodward, 1976) and a simple distinction between Performance and Verbal scales is unable to elaborate. It may be that some function or contrast of verbal and performance scores other than their simple difference may emerge. Claims have been made that individuals showing this pattern have

superior verbal abilities (Robertson & Taylor, 1985). Alternatively, psychomotor retardation may impact upon successful completion of timed sub-tests (Birren & Birren, 1990; Kluger & Goldberg, 1990). Sackeim *et al.* (1992) noted a decline in Performance scores in depressed adults (mean age 55) even when time constraints were removed. Christensen, Griffiths, MacKinnon & Jacomb (1997) pointed to depressed older adults scoring poorly in 'almost every psychological test' but in particular on speeded or 'vigilance' tasks. This may have been a function of decreased motivation, impaired concentration, anxiety, structural deficit or any combination of these factors. The authors choose to view it as being support for a theory of deficit in speed or attention.

The multi-factorial nature of many of the WAIS-R subtests has made it difficult to identify specific cognitive skills which may be affected by a condition such as depression. Factor analytic studies of the WAIS and WAIS-R have resulted in the production of several "models" of intelligence which have been extensively reviewed (Cohen, 1957b; Matarazzo, 1972). Results of the various studies seem to vary according to statistical approaches employed as well as the authors' theoretical leanings. However, in summarising the various studies, Leckliter, Matarazzo & Silverstein (1986) corroborated the presence of 'g' (a general intelligence factor), a robust Verbal Comprehension Factor, a Perceptual Organisation Factor and a weaker Memory/Freedom from Distractibility Factor. The Freedom from Distractibility Factor was considered particularly important in neurological samples (Bornstein, Drake & Pakalnis, 1988; Josiassen, Curry, Roemer, DeBease & Mancall, 1982) and the refinement and expansion of this factor was considered desirable in increasing the diagnostic capability of Weschler Scales.

1.6.2 The WAIS-III

The WAIS-R has recently been revised and updated resulting in the production of the WAIS-III (Weschler, 1998). This new version of the test has been standardised up to age 89 both in the US and UK thereby allowing a far broader range of older adults to be studied systematically and with reference to published norms for same-aged, same-culture, peers. Such a modification, has removed, to a certain extent, the necessity to resolve the argument about "normal ageing" since comparisons are made to a large sample of "control" same-aged peers who, on average, would be expected to have demonstrated normal ageing effects on standardisation testing. These effects have, in turn, been incorporated into scoring systems.

A goal for the WAIS-III was to have subtests that would relate to four hypothesised Factors or 'Indices' (The Psychological Corporation, 1997). The Indices included Verbal Comprehension (measuring conceptual thought and verbal expression), Perceptual Organisation (non-verbal thinking and application of visual-spatial skill), Working Memory (working memory, sequential processing and number ability) and Processing Speed (mental and motor).

Indices are based upon more refined domains of cognitive functioning than a simple Verbal-Performance scale comparison (Kaufman & Lichtenberger, 1999). The separation of the Verbal Comprehension Index from Processing Speed and Working Memory has been shown to be important in the Weschler Intelligence Scale for Children (WISC-III).

This has led to the suggestion that specific patterns of cognitive deficit are indeed observed in particular clinical populations and, in particular, slowness of thought and motor dysfunction as well as attention/concentration difficulties (Prifitera & Dersh, 1992).

The Perceptual Organisation/Processing Speed distinction of the WAIS-III attempts to distinguish between abstract reasoning powers required to solve non-verbal tasks versus the ability to do so swiftly. Matrix Reasoning, a new, untimed, non-verbal test, and Symbol Search, a measure of processing speed, have been introduced to facilitate this comparison. Letter-Number Sequencing constitutes a third new (verbal) task devised to measure the hypothesised factor of "working memory".

Working memory and processing speed have been labelled as "mediators" in cognitive functioning because their component skills are important for learning (Kyllonen & Stephens, 1990; Woltz, 1988). Working memory is conceptualised as a set of sub-systems controlled by a central executive and capable of manipulating and transforming information and integrating information from different sources. As such, it is fundamentally involved in almost all cognitive operations occurring in everyday life. The complexity and number of components involved in such a system would suggest that individuals suffering from either depression and/or anxiety would be handicapped by comparison to same-aged peers in this modality.

The four Indices can be interpreted within Horn's (1989) theoretical framework where Verbal Comprehension measures crystallised intelligence, Perceptual Organisation visual

and fluid intelligence, Working Memory short-term acquisition and recall and Processing Speed "broad speediness".

Utilisation of the WAIS-III with older subjects has been facilitated by redrawing the artwork and enlargement of stimuli. This, in general, marks an improvement although some of the Picture Completion items remain detailed and distracting. The Object Assembly Subtest (which has low reliability with older adults) no longer contributes to the computation of IQ or factor scores. Administration and scoring rules have been made more uniform throughout the test reducing the chances of examiner error (Kaufman & Lichtenberger, 1998).

Factor analyses of the WAIS-III standardisation data support the four-factor model for four agebands (16-19, 20-34, 35-54 and 55-74) (Weschler, 1998). In the age band 75-89, however, a five factor solution is a slightly (although not significantly) better fit. Such a model is similar to those suggested by Woodcock (1990, 1997) and Flanagan & McGrew (1997) and includes Verbal Comprehension, Perceptual Organisation, Processing Speed, Memory and Quantitative Ability/Numerical Ability. The Perceptual Organisation and Processing Speed Factors do not emerge as separate constructs in this older age group as the Perceptual Organisation subtests (with the exception of Matrix Reasoning) continue to load on Processing Speed.

To summarise, Weschler scales are universally recognised, applicable and broad-ranging. The WAIS-R however has been largely unsuccessful as a diagnostic tool, perhaps, at least in part, due to a difficulty in separating the various component skills contributing to

performance on any given subtest. The modified WAIS-III may prove more successful in this respect. It measures abilities that it was not possible to assess with confidence in the WAIS-R. Modifications make it more accessible, appealing and appropriate for usage with an older adult population.

1.7 The Current Study

The focus of the current study is an attempt to further investigate the relationship between cognition, (as measured by the WAIS-III), and late-life depression by examining whether specific cognitive factors differentiate a currently depressed group from recovered depressed and normal control groups.

Given methodological difficulties referred to above, it was intended to match each group on age, sex and premorbid IQ and assess the presence or absence of depression comprehensively. Although the distinction between early and late-onset depression is controversial, it was also intended to compose both the depressed and recovered depressed groups of individuals whose depression had been of late onset (over the age of 60) for the sake of consistency.

Employment of the WAIS-III was considered to be of value and interest for several reasons. Firstly, it separates processing speed and working memory skills (hypothesised to be impaired in late life depression) from more traditional verbal (Verbal Comprehension Index) and performance (Perceptual Organisation Index) measures. This permits more detailed investigation of the specific cognitive components contributing to more general

deficit previously observed in late life depression. Furthermore, as scoring of the WAIS-III is based on comparisons between same-aged peers, rather than a younger reference group, its utilisation may further the argument relating to whether any measured declines in the depressed group simply represent normal age-related change (as demonstrated by the normal control group) or are a result of, or exacerbated by, depression.

The inclusion of a recovered depressed group was thought necessary to address the question of reversibility of cognitive deficit.

1.8 Hypotheses

It is hypothesised that deficits in cognitive performance (as assessed by the WAIS-III) in depressed older adults are mood state dependent and reversible on recovery from depression and therefore that:

1a. The depressed group will exhibit a significantly lower current full scale IQ than predicted premorbid IQ;

1b. The recovered depressed and normal control groups will exhibit no significant difference between current full scale IQ and predicted pre-morbid IQ;

2. The depressed group will demonstrate impairment, by comparison to the recovered depressed and control groups, on the Processing Speed Index;

3. The depressed group will demonstrate impairment, by comparison to the recovered depressed and control groups, on the Working Memory Index;

4. Any observed deficit in the Perceptual Organisation Index in the depressed group will be as a result of those subtests which remain, to some extent, dependent on speed of processing (the Block Design and Picture Completion subtests as opposed to the untimed Matrix Reasoning test) in so far as Perceptual Organisation in adults over 75 continues to load on the Processing Speed Index;

5. Performance on the Perceptual Organisation Index in the depressed group but not the recovered depressed or control groups, will decrease as a function of increasing age (as a result of the increased loading of processing speed on the Perceptual Organisation factor with increasing age);

6. There will be no significant difference between the three groups on the Verbal Comprehension Index.

2. METHODOLOGY

2.1 Design

A three group, between-subjects design was employed. Groups included depressed participants, recovered depressed participants and normal control participants. Participant group acted as the independent variable with three levels. WAIS-III IQ and Index scores acted as dependent variables.

It was considered that a well-controlled comparison of depressed and normal control groups, using the WAIS-III, may demonstrate specific areas of cognitive deficit related to late-life depression more clearly than had been possible with previous measures.

It was further considered that the inclusion of the recovered depressed group may facilitate investigation into the question of the reversibility of such cognitive deficit and, by extension, the debate over whether cognitive deficit observed in late-life depression is largely mood-state dependent or structural in origin.

The normal control group provided a baseline of "normal ageing" against which to evaluate the performance of the experimental groups.

It was intended that groups be matched with regard to premorbid IQ, sex, age and length of education which has been demonstrated to correlate with IQ (Kaufman, 1990).

A between-subjects design was preferred to avoid the possibility of practice effects stemming from individuals being tested and re-tested in the depressed and recovered states (Matarazzo *et al.,* 1980).

2.2 Ethical Approval

Conditional ethical approval for the project was granted by Grampian Research Ethics Committee in December 1998 and final approval in January 1999 (Appendix 1). All individuals agreeing to participate in the study signed a consent form prior to testing commencing (Appendix 1). Efforts were made, in designing appropriate consent forms and information sheets, to avoid the use of the term depression and any implication regarding cognitive function with the potential to alarm. For this reason, the project title was omitted from the consent form.

No individual received any form of payment for their participation. Participants opting to attend the Clinical Psychology Department for testing, as opposed to being tested in their homes, did not receive travel expenses.

2.3 Procedure

2.3.1 Identification of Participants

Potential participants were identified and approached via a number of sources. For the normal control group, these included psychiatry, friends and colleagues, relatives and other

volunteers. The depressed and recovered groups were identified and approached via psychology colleagues and databases, psychiatry, GPs, a day hospital and a medical physician. A description of the final sample is included in Tables 3a-3c in the Results section; participant source is evaluated in the Discussion.

2.3.2 Inclusion and Exclusion Criteria

Inclusion and exclusion criteria for the sample as a whole and by group are detailed in Table 2a and Table 2b.

Table 2a. Inclusion and Exclusion Criteria for all Participants

INCLUSION CRITERIA	Able to give written informed consent English as a native language Adequate hearing, vision and mobility to undergo testing without handicap relating to these dimensions Aged between 60 and 89 years
EXCLUSION CRITERIA	Reported history of or exhibition of psychotic features Evidence of organic pathology as assessed by observation on testing, physician report or score less than 23 on Mini-Mental State Examination (MMSE). History of alcoholism or illicit drug usage ECT within the last year Diagnosis of manic depression Significant vascular disorder

Table 2b. Inclusion Criteria by Group

DEPRESSED GROUP	Apparently "late-onset" depression determined by initial presentation at 60 years or more of age A primary diagnosis of major depression made by the referrer and supported by clinical judgement and semi-structured interview, with reference to DSM-IV criteria, at the time of testing GDS score exceeding 10 at time of testing
RECOVERED GROUP	Apparently "late-onset" depression determined by initial presentation at 60 years or more of age Previous primary diagnosis of major depression made by the referrer Failure, at time of testing, to meet criteria for the diagnosis of major depression on the basis of clinical judgement and semi-structured interview with reference to DSM-IV criteria GDS score of less than 11 at time of testing
CONTROL GROUP	No previous history of depressive illness according to participant report GDS score of less than 11 at time of testing

2.3.3 Pathways to Participation

Control Participants. Written information sheets were provided for control participants by the individuals who had identified them (Appendix 2). A week after receiving the information sheet, individuals were contacted by telephone by the researcher to ascertain

willingness to participate, answer queries and arranges test times and venues. GPs were not notified.

A number of control participants (N=4) contacted the researcher by telephone offering to participate in the study in response to an approach to the Womens' Royal Voluntary Service (WRVS) via the hospital shop. Following the telephone call, information sheets were despatched and the researcher contacted individuals a week later, by telephone, to confirm willingness to participate, answer queries and arrange test times and venues.

Currently Depressed and Recovered Participants. Individuals currently under the care of the Psychiatric Team received a brief introductory letter which included the name of the professional who had suggested involvement (Appendix 2). Information sheets, varying slightly according to depressed or recovered group allocation, were attached (Appendix 2). One week later, the individual was contacted by the researcher by telephone to ascertain willingness or not to participate. If individuals agreed to become involved, their GPs were notified (Appendix 3).

A number of individuals (tentatively allocated to the recovered group) were identified as possible participants by the psychiatric team and psychology department but were not currently receiving care from these sources. In this instance, appropriate GPs were contacted by telephone, a written outline of the study was provided by way of a portion of the original thesis proposal (Appendix 3) and GP permission was sought to approach the individual. Individuals were then approached in the same way as for the group currently under the care of the Psychiatric Team. No GP refused access to any patient.

A number of individuals attending a Day Centre were identified as potential participants following an approach to neighbouring Grampian Health Board and a project presentation to the Day Centre Manager and staff. Named key-workers at the centre provided identified individuals with an information sheet. A week later, the same staff member ascertained willingness or not to participate and mutually agreed times for testing at the Centre were arranged between the participant, the researcher and Day Centre staff.

As a result of several presentations introducing the project to GP practices, a number of individuals were identified as potentially appropriate participants by their GPs. These individuals received an introductory letter from their GP (Appendix 2) enclosing an information sheet which was slightly modified in order to take into account GP ambiguity regarding definitive group membership for some of the individuals identified. Stamped addressed envelopes were enclosed, addressed to the researcher, care of the GP surgery, and individuals were asked to return tear off slips indicating their willingness or not to participate. Affirmative responses were followed up with a telephone call from the researcher after a week to answer queries and arrange test times and venues.

2.3.4 The Final Sample

A total of 57 individuals were assessed. All participants were right handed. The sample included 39 females and 18 males. All participants in the depressed group ($N = 22$) were taking some form of anti-depressant medication. Of the recovered group, 10 (71 per cent of $N = 14$) were currently taking anti-depressant medication. None of the control group

(N = 21) were taking anti-depressant medication. Medication for complaints other than depression was frequent across the sample as a whole and most commonly included treatment for hypertension and heart complaint. Common physical ailments included hip replacements, arthritis and angina.

2.3.5 Format of the Session

Participants were assessed during a mutually agreed morning (10am) or afternoon (2pm) session either in their own homes (N = 47), the clinical psychology department (N = 8) or at a day centre (N = 2). Four control participants were assessed in evening/weekend sessions. No more than two participants were assessed in any one day and assessments spanned a period of five months. Whilst the first five participants assessed were members of the control group, (since participants for this group were more readily identified and available at the outset of the study), assessment thereafter followed order of presentation to the study and was largely random across the three groups.

Assessment was completed in a single session in all but three cases in the depressed group. In two of these, an hour long break was taken for lunch. In the third, four separate visits to the home were necessary to complete testing. Session duration ranged between two and four and a half hours per participant (inclusive of pre-assessment engagement period).

Sessions commenced with an apparently informal engagement period of approximately 15-30 minutes duration. No formal structure was imposed but information was nevertheless sought during the course of the conversation regarding age, handedness, length of

education, physical complaint, medication, alcohol and drug usage, ECT and depression history and current symptoms of clinical depression. In particular, enquiries were made regarding **P**leasure, **A**ppetite, **C**oncentration, **E**nergy, **D**eath, **S**leep, **W**orthlessness, **A**gitation and **M**ood working to an examiner mnemonic of "Paced Swam" reflecting DSM-IV criteria for major depression and with a focus on the first (P) and last (M) letters as of particular relevance. No additional information was provided as to the nature or purpose of the study at this point and none was requested. The Mini Mental State Examination (MMSE) was administered where indicated. Measures were administered in the agreed sequence in all cases.

Rapport was maintained by encouragement, conveyance of interest and enthusiasm for the participant's performance. No feedback was given as to accuracy of performance.

2.4 Measures

2.4.1 Measures Administered

Data was collected for participants on the following measures:

The Weschler Adult Intelligence Scale (WAIS-III). Twelve subtests of The Weschler Adult Intelligence Scale (WAIS-III) were administered. Object Assembly and Picture Arrangement subtests were omitted from the battery. This cut administration time. Object Assembly has been reported as being particularly unreliable when used with adults over the age of 75 and Picture Arrangement has been shown to load on two Indices simultaneously

(Kaufman & Lichtenberger 1999). The subtests are the two least reliable of the WAIS-III. Neither contributes to the calculation of Index scores and Object Assembly does not contribute to the calculation of current full scale IQ (CFSIQ). The contribution of Picture Arrangement to the calculation of CFSIQ in the current study was therefore prorated.

The WAIS-III provided an estimate of CFSIQ as well as individual measures in each of four domains (Verbal Comprehension, Perceptual Organisation, Working Memory and Processing Speed).

The National Adult Reading Test (NART). The NART consists of 50 words which participants have to read and pronounce (Appendix 3). Words are predominantly short and of irregular pronunciation so that successful performance relies more on familiarity than current cognitive capacity.

This measure provided an estimate of likely premorbid level of function by way of an estimated premorbid IQ score (PMIQ). This was an attempt to ensure the validity of comparing current functioning between individuals within the three groups on the basis that they had originally been of comparable intelligence. It also provided a "benchmark" of optimal intelligence against which to assess any individual decline.

The Geriatric Depression Scale (GDS). The GDS is a 30 item self-rating instrument requiring only "Yes/No" answers and designed specifically for rating depression in older adults (Appendix 3). It focuses almost entirely on the cognitive rather than somatic presentation of depression apart from one item which enquires about energy levels. The

measure was used to confirm referrer/researcher allocation to group based on DSM-IV criteria and clinical judgement and acted as a indicator of likely severity of depression at the time of testing.

The Beck Anxiety Inventory (BAI). The BAI is a 21 item self-rating screening instrument designed to assess subjective levels of anxiety which may have been affecting test performance either in combination with, or independently from, concurrent depression (Appendix 3).

The Mini-Mental State Examination (MMSE). The MMSE was administered only in those cases where performance during the engagement period or any of the subsequent testing suggested a possibility of the presence of dementia (Braekhus, Laake & Engedal 1992). It is a brief (30 point) mental status questionnaire tapping fundamental skills including orientation, registration, attention, calculation recall and language (Appendix 3). Three individuals were excluded from the current study on the basis of MMSE investigation. Full investigations of these individuals were nevertheless completed. Results were not discussed but reports were provided to referrers with the consent of the participant.

2.4.2 Presentation Sequence of Measures

The most appropriate order in which to administer measures was considered. It was felt that certain items on the GDS and BAI could be viewed as personal and potentially intrusive and these questionnaires were therefore administered last of all and by self-

administration rather than interview. It was further considered that the NART could be viewed as threatening, particularly to less able individuals, so that it was also positioned after the WAIS-III. The WAIS-III, the measure of main interest and potentially the least threatening measure due to its non-personal nature and variety of subtests, was administered first. Subtests were presented following the order on the record form and Picture Completion, as the first test, was considered particularly appropriate and engaging as an introductory test.

3. RESULTS

3.1 Parameters of Analysis

Parametric tests were used to analyse the data where assumptions relating to distribution, variance, scale and independence were adequately met. If assumptions were poorly met, and if more than one was not met, non-parametric methods were employed to avoid increasing the likelihood of a Type I or II error (Clark-Carter, 1997; Kinnear & Gray, 1999).

Group sizes were unequal (depressed $N = 22$, recovered depressed $N = 14$, normal control $N = 21$). Data were therefore treated as heterogeneous if the largest variance was more than two times the smallest (Clark-Carter, 1997, pp. 250).

One and two-tailed tests were employed as appropriate and available (using SPSS Base 8.0) according to whether or not hypotheses were directional. Significance levels of statistical analyses are reported only at the < 0.05, < 0.01 or non-significant (*NS*), (> 0.05,) levels. Clark-Carter recommends reporting exact probability levels (1997, pp.187). However, it was considered that the limitations of the study, and in particular the small sample size, made it potentially unreliable to express a specific degree of confidence in the results obtained. For the same reason, statistics are reported to two decimal places.

Estimates of power were made with reference to test-appropriate power tables and

large effect sizes (Clark-Carter, 1997, pp. 599-634). Effect size adjustments for paired sample *t*-tests and independent *t*-tests involving unequal group sizes were calculated according to recommendations of Clark-Carter (1997, pp. 220-221 & 599-600). Where Kruskall-Wallis ANOVAs were employed, adjustments were made to power calculations (Clark-Carter, 1997, pp. 256-257). Power is referred to only where, with $\alpha = 0.05$, it was found to be less that the recommended minimum, (0.8), (J. Cohen, 1992).

Where analysis subsequent to ANOVA was planned (a priori) Bonferroni's *t* was used. Where unplanned (post hoc) Scheffe's *t* was adopted, being more conservative and appropriate for unequal group sizes.

Where correlations were being investigated, scatterplots were produced to avoid the possibility of interpreting spurious correlations or failing to detect significant relationships which existed.

3.2 The Sample

A description of the sample is provided in Tables 3a to 3c.

3.2.1 Demographics

A summary of demographic measures is provided at Table 4.

Table 3a. Composition of Depressed Group

SOURCE	APPROACHED (A)	OBTAINED (O)	A-O DISCREPANCY ANALYSIS
PSYCHIATRY AND/OR CPNs	11	5	3 Refused 2 Hospitalised (Medical) 1 Excluded (MMSE<23)
GPs	18	8	9 Failed to Respond 1 Excluded (Hearing Loss Affecting Validity of Results)
MEDICAL WARD	4	0	1 Too Ill for Length of Session 3 Significant Vascular Disease/Stroke
PSYCHOLOGY DEPT	4	2	2 Excluded (MMSE<23)
DAY HOSPITAL	3	2	1 Refused
TOTAL	40	17	23
FROM "RECOVERED" GROUP	-	5	-
TOTAL	40	22	23

Table 3b. Composition of Recovered Depressed Group

SOURCE	APPROACHED (A)	OBTAINED (O)	A-O DISCREPANCY
PSYCHIATRY AND/OR CPNs	10	2	8 Refused
IDENTIFIED BY COLLEAGUES	1	1	-
PSYCHOLOGY	3	3	-
GPs	32	8	5 Transferred to "Depressed" 19 Did not respond/refused
DAY HOSPITAL	1	0	1 Did not Respond
TOTAL	47	14	28 (33 minus 5 who were transferred).

Table 3c. Composition of Normal Control Group

SOURCE	APPROACHED (A)	OBTAINED (O)	A-O DISCREPANCY
PSYCHIATRIST	1	1	Partner of Patient not Regarded as Suitable (Manic-Depressive)
RELATIVES OF RESEARCHER	3	3	-
IDENTIFIED BY ACQUAINTANCES OR COLLEAGUES	18	13	4 Refused 1 Excluded (Stroke)
TOTAL	22	17	5
WRVS	?	4	Responded to Posted Notice
TOTAL	?	21	5

Table 4. Summary of Demographic Measures.

Measure	Group	Mean	SD	Min	Max
Age	Depressed	71.32	7.41	63	88
	Recovered Depressed	71.93	7.28	60	85
	Normal Control	71.24	5.57	60	80
Years Education	Depressed	8.60	1.26	6	11
	Recovered Depressed	9.50	1.29	8	12
	Normal Control	8.80	1.44	6	12
PMIQ	Depressed	106.00	12.47	86	122
	Recovered Depressed	109.00	15.13	84	126
	Normal Control	112.00	9.81	94	124

Age. A one-way, between-subjects ANOVA was used to investigate the possibility of differences between the mean age of the three sample groups. There was no significant effect for age between groups ($F(2,54) = 0.05$, $p = NS$, $MS\ Age = 2.26$); power = 0.76. A mathematical average of 22 per group (as opposed to the 19 in the present sample) would have been required to achieve recommended minimum power.

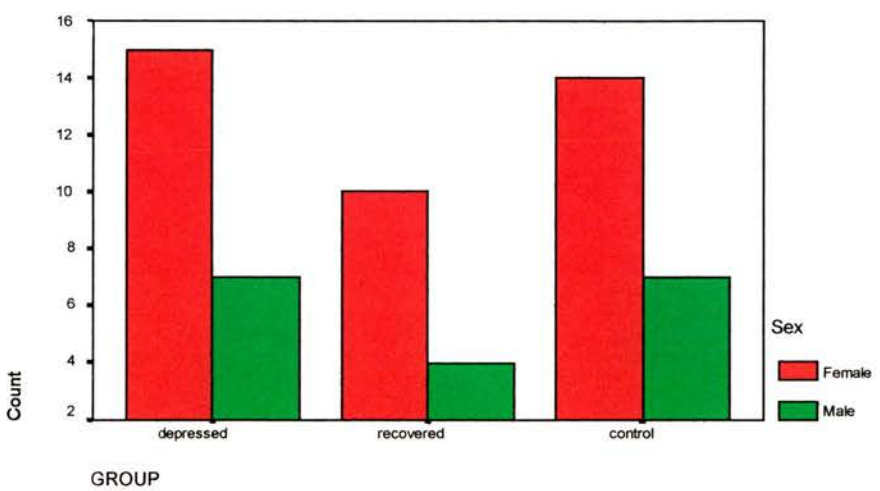
Education. A one-way, between-subjects ANOVA was used to investigate the possibility of differences in the mean number of years spent in education between the three sample groups. There was no significant effect of years education between groups ($F(2,54) = 2.06$, $p = NS$, MS Education = 3.66); power = 0.76.

Premorbid IQ. Due to heterogeneity of variance for unequal group sizes and borderline normality ($p = 0.052$), a non-parametric test, Kruskal Wallace ANOVA, was used to investigate the possibility of differences between the mean premorbid IQs (PMIQ) of the three groups. No significant difference in PMIQ was detected between

the groups ($H = 2.62$, $df = 2$, $p = NS$); power = 0.72. Twenty-five participants per group would have been required to achieve recommended minimum power.

Sex. Figure 1 illustrates that there were considerably more females than males in the sample (Females $N = 39$, Males $N = 18$).

Figure 1. Proportion of Females to Males by Group



Although one cell had an expected frequency count less than five, (4.4), it did not contravene the critical criteria proscribing the use of Chi-square (Kinnear & Gray, 1999). A three by two X^2 test was therefore conducted to compare the proportions of males and females between groups. There was not a significantly higher proportion of females than males in any group ($X^2 = 0.09$, $df = 2$, $p = NS$, $N = 57$).

3.2.2 Mood State Measures

A summary of mood state measures is provided at Table 5.

Table 5. Summary of Mood State Measures.

Measure	Group	Mean	SD	Min	Max
GDS Score	Depressed	22.18	3.22	14	26
	Recovered Depressed	6.29	3.22	0	10
	Normal Control	4.95	3.67	0	10
BAI Score	Depressed	12.18	7.44	3	29
	Recovered Depressed	8.29	5.58	0	18
	Normal Control	5.67	5.41	0	20

Geriatric Depression Scale (GDS). The research hypothesis was that the depressed group would demonstrate significantly higher scores on the GDS than the recovered depressed or normal control groups.

A one-way, between-subjects ANOVA was used to investigate the possibility of differences in mean GDS score between the three groups. A significant difference between mean GDS score was demonstrated (F (2,54) = 153.10, $p < 0.01$, MS GDS = 1976.21); power = 0.76.

A priori comparisons were conducted using Bonferroni's t to investigate the source of the significant difference in mean GDS score. The mean GDS score of both the recovered depressed ($p < 0.01$) and normal control ($p < 0.01$) groups differed significantly from the depressed group.

The research hypothesis was accepted.

Beck Anxiety Inventory (BAI). It was considered likely that the depressed group may demonstrate significant levels of concurrent anxiety.

A one-way, between-subjects ANOVA was used to compare the mean BAI scores between the three groups. A significant effect for BAI score was observed ($F(2,54) = 5.79$, $p < 0.01$, MS BAI = 230.73); power = 0.76.

Scheffe's t was used to investigate the source of the significant difference in mean BAI score. A significant difference was noted in BAI score between the means of the depressed and normal control groups ($p < 0.01$).

It was concluded that the depressed group demonstrated significant levels of concurrent anxiety.

3.2.3 Summary of Results for Demographic Variables and Mood State Measures

There was no significant difference between groups in the proportion of female to male participants.

There were no significant differences between the means of the three groups on

measures of age, education and PMIQ.

The depressed group reported significantly more symptoms of depression (as measured by the GDS) than either the recovered depressed or normal control groups.

The depressed group reported significantly more anxiety symptoms (as measured by the BAI) than the normal control group.

3.3 Testing the Hypotheses

3.3.1 Hypothesis 1a and 1b

The research hypotheses were:

1a. That the depressed group would exhibit a significantly lower current full scale IQ (CFSIQ) than predicted PMIQ.

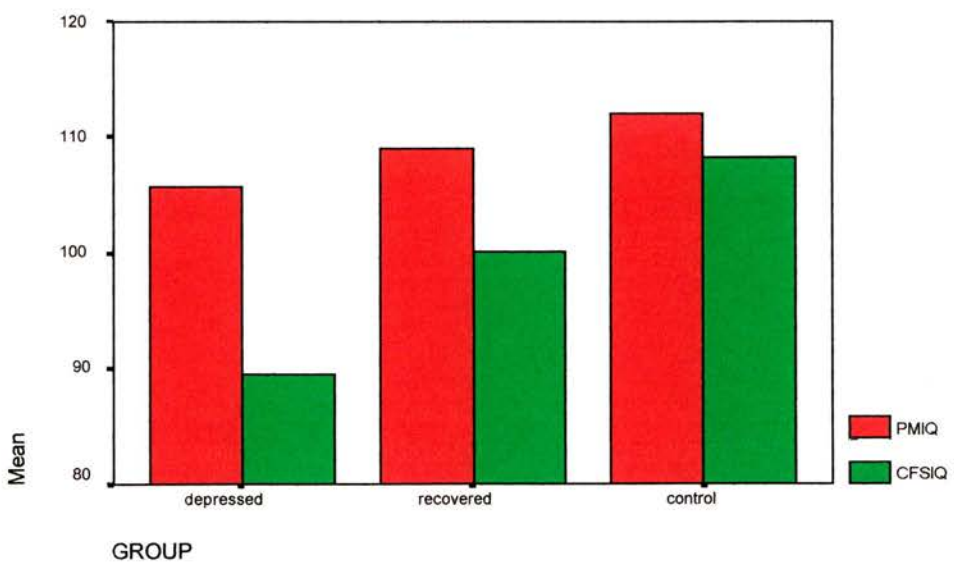
1b. That the recovered depressed and normal control groups would exhibit no significant difference between CFSIQ and PMIQ.

Exploration and description of the data.

Table 6. Summary Statistics for PMIQ and CFSIQ by Group.

Measure	Group	Mean	SD	Min	Max
PMIQ	Depressed	105.64	12.47	86.0	122.0
	Recovered Depressed	109.00	15.13	84.0	126.0
	Normal Control	112.00	9.81	94.0	124.0
CFSIQ	Depressed	89.59	13.29	66.0	108.0
	Recovered Depressed	100.07	17.26	77.0	128.0
	Normal Control	108.19	14.28	91.0	131.0

Figure 2. PMIQ Versus CFSIQ by Group



Testing Hypothesis 1a. A paired samples *t*-test was used to compare PMIQ to CFSIQ in the depressed group.

A significant difference between mean PMIQ and mean CFSIQ was detected in the depressed group ($t = 7.71$, $df = 21$, $p < 0.01$; two-tailed test).

The research hypothesis was accepted.

Testing Hypothesis 1b. Paired samples *t*-tests were used to compare mean PMIQ and CFSIQ scores in the recovered depressed and normal control groups.

A significant difference between mean PMIQ and mean CFSIQ was detected in the recovered depressed group ($t = 3.70$, $df = 13$, $p < 0.01$; two-tailed test).
A significant difference between mean PMIQ and mean CFSIQ was detected in the normal control group ($t = 2.10$, $df = 20$, $p < 0.05$; two-tailed test).

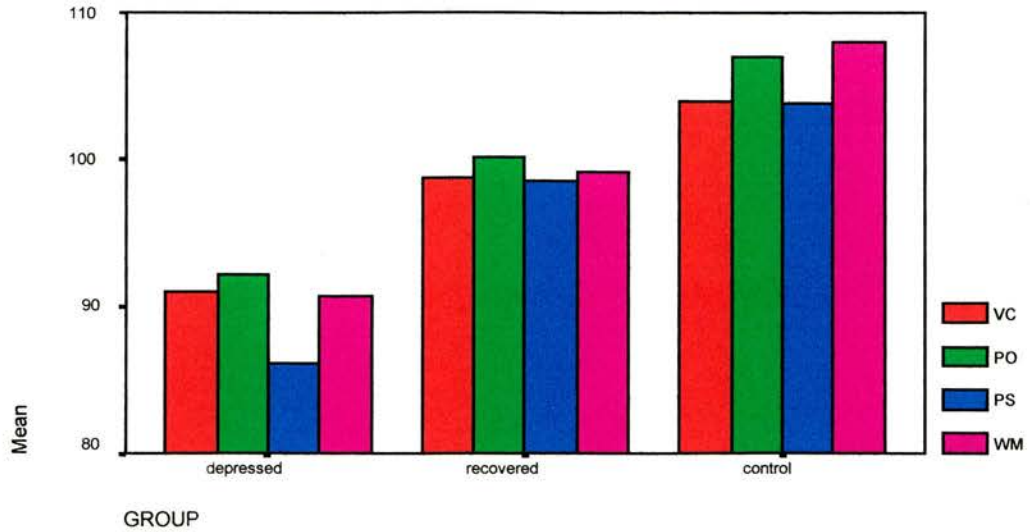
The research hypothesis was rejected.

3.3.2 Hypothesis 2

The research hypothesis was that the depressed group would demonstrate impairment, by comparison to the recovered depressed and normal control groups, on the Processing Speed Index.

Performance on all four WAIS-III Indices (Verbal Comprehension (VC), Perceptual Organisation (PO), Processing Speed (PS), and Working Memory (WM)) by group is illustrated in Figure 3.

Figure 3. Performance on WAIS-III Indices by Group



Summary and Exploration of the Data. Figure 4 illustrates the identification of one outlier (PS = 140) which was removed from the recovered depressed group.

Figure 4. Mean Processing Speed Index Score by Group

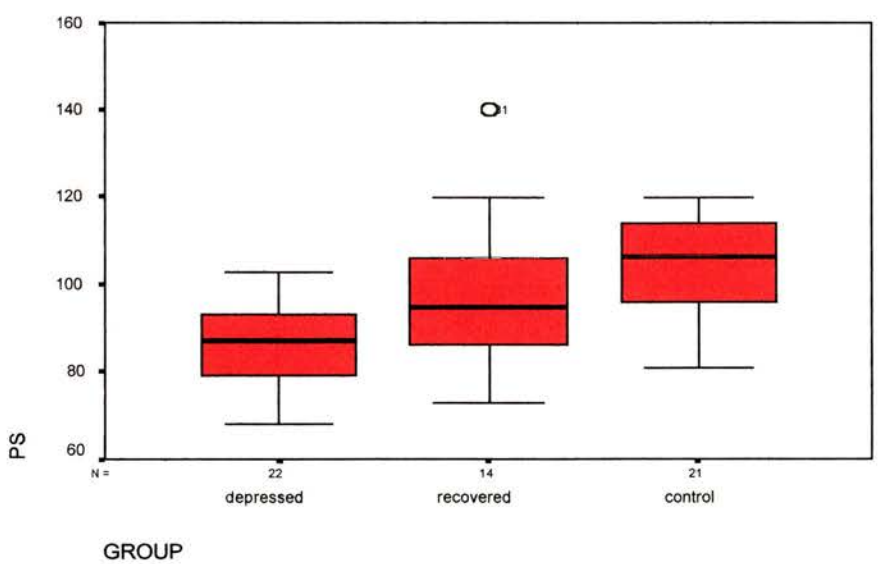


Table 7. Summary Statistics for Processing Speed Index by Group

Group	Mean	SD	Min	Max
Depressed	86.14	10.32	68.0	103.0
Recovered Depressed (including outlier)	98.57	17.53	73.0	140.0
Recovered Depressed (excluding outlier)	95.38	13.37	73.0	120.0
Normal Control	103.86	11.52	81.0	120.0

A one-way, between-subjects ANOVA was used to investigate the possibility of differences in mean Processing Speed Index score between the three groups. Results suggested a significant difference between group means on the Index (F (2,53) = 12.72, $p < 0.01$, *MS* Processing Speed = 1688.74); power = 0.73.

Scheffe's t was used to investigate the source of the significant effect. A significant difference in mean score on the Processing Speed Index was observed between the depressed and normal control groups ($p < 0.01$).

The research hypothesis was accepted.

3.3.3 Hypothesis 3

The research hypothesis was that the depressed group would demonstrate impairment, by comparison to the recovered depressed and normal control groups, on the Working Memory Index.

A one-way, between-subjects ANOVA was used to investigate the possibility of differences in mean Working Memory Index score between the three groups. Results suggested a significant difference between mean group score on the Working Memory Index (F, (2,54) = 6.28, $p < 0.01$, MS Working Memory = 1620.07); power = 0.76.

Scheffe's t was used to investigate the source of the significant effect. A significant difference in mean score on the Working Memory Index was observed between the depressed and normal control groups ($p < 0.01$).

The research hypothesis was accepted.

3.3.4 Hypothesis 4

The research hypothesis was that any deficit on the Perceptual Organisation Index observed in the depressed group would be as a result of those subtests which remain, to some extent, dependent on speed of processing (the Block Design and, possibly, Picture Completion subtests, as opposed to the untimed Matrix Reasoning subtest).

Summary and Exploration of the Data. Figure 5 illustrates the identification of one outlier (PO = 145) which was removed from the normal control group.

Figure 5. Mean Perceptual Organisation Score by Group.

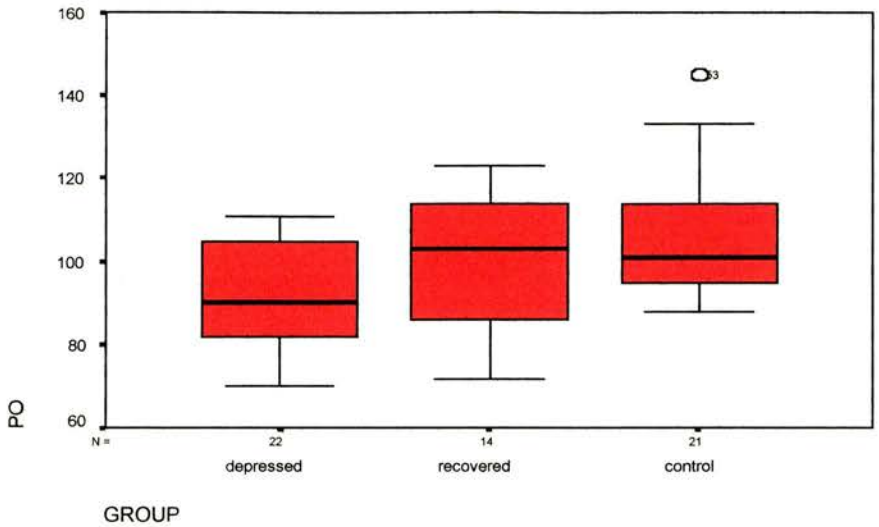


Table 8. Summary Statistics for Perceptual Organisation Index by Group

Group	Mean	SD	Min	Max
Depressed	92	12.55	70	111
Recovered Depressed	100	16.49	72	123
Normal Control (including outlier)	107	16.19	88	145
Normal Control (excluding outlier)	105	14.01	88	133

Investigating the Possibility of Difference in Mean Perceptual Organisation Score Between Groups. A one-way, between-subjects ANOVA was used to investigate the possibility of differences in mean Perceptual Organisation Index score between the three groups. Results suggested a significant difference between mean group scores on the Perceptual Organisation Index (F (2,53) = 4.51, $p < 0.05$, MS Perceptual Organisation = 899.02); power = 0.73.

Scheffe's t was used to investigate the source of the significant effect. A significant difference in mean score on the Perceptual Organisation Index was observed between the depressed and normal control groups ($p < 0.05$).

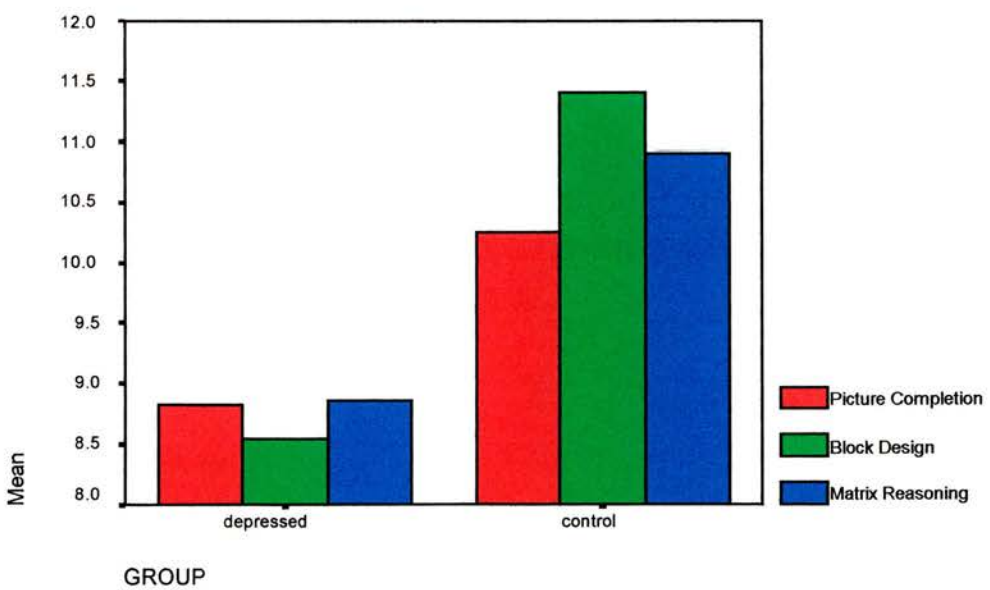
With additional reference to Table 8, it was concluded that the mean Perceptual Organisation Index score for the depressed group was significantly lower than that of the recovered depressed or normal control groups.

Investigating Age-Scaled Mean Scores on Subtests Contributing to the Perceptual Organisation Index.

Figure 6 illustrates age-scaled mean scores on the Perceptual Organisation Index by Group.

Independent *t*-tests were employed to compare the mean age-scaled scores of the depressed and normal control groups on subtests contributing to the Perceptual Organisation Index.

Figure 6. Mean Age Scaled Scores for Perceptual Organisation Subtests in the Depressed and Control Groups



Picture Completion. There was no significant difference between the mean age-scaled scores of the depressed and normal control groups on the Picture Completion subtest (t = -1.51, df = 40, p = *NS*; two-tailed test); power = 0.69. It would have been necessary to have had 26 individuals in each group to achieve recommended minimum power.

Block Design. There was a significant difference between the mean age-scaled scores of the depressed and normal control groups on the Block Design subtest (t = -3.62, df = 40, $p < 0.01$; two-tailed test); power = 0.69.

Matrix Reasoning. There was a significant difference between the mean age-scaled scores of the depressed and normal control groups on the Matrix Reasoning subtest (t = -2.33, df = 40, $p < 0.05$; two-tailed test); power = 0.69.

The proposition that any deficit in mean Perceptual Organisation Index score for the depressed group would be due to subdued performance on the Block Design and, possibly, Picture Completion subtests, as opposed to the Matrix Reasoning subtest, was rejected.

3.3.5 Hypothesis 5

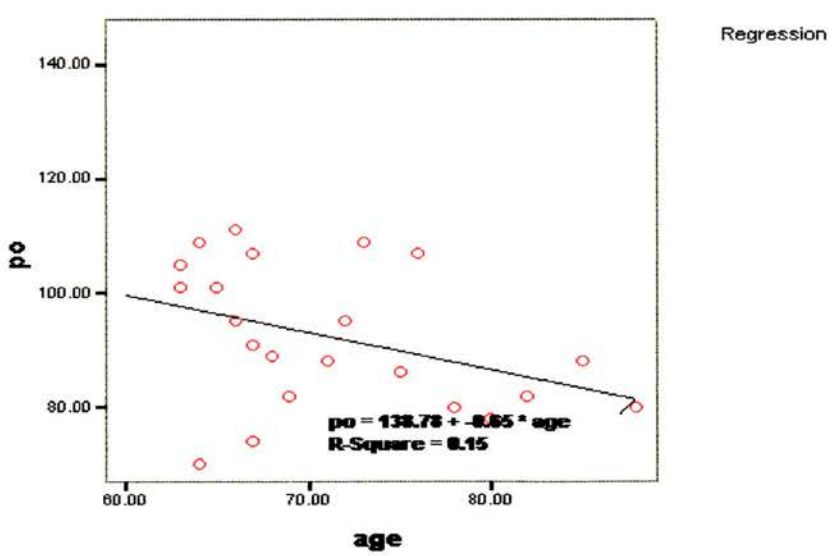
The research hypothesis proposed that decreasing performance on the Perceptual Organisation Index would be associated with increasing age in the depressed group.

Figure 7 illustrates mean age-scaled score on the Perceptual Organisation Index in the depressed group.

A significant negative correlation between age and mean Perceptual Organisation score in the depressed group was indicated (r = -0.39, df = 20, p < 0.05; one-tailed test); power = 0.78. It would have been necessary to have 23 participants in the depressed group to achieve recommended minimum power.

No significant correlations between age and age-scaled scores were indicated on the remaining three WAIS-III Indices in the depressed group (Processing Speed: r = 0.20, df = 20, p = *NS*. Working Memory: r = -0.08, df = 20, p = *NS*. Verbal Comprehension: r = 0.11, df = 20, p = *NS*; all two-tailed tests).

Figure 7. Mean Age-scaled Perceptual Organisation Score by Age in the Depressed Group



Recovered Depressed Group. In the recovered depressed group, no significant correlations between age and any age-scaled Index score were detected (Perceptual Organisation r = -0.02, df = 12, p = *NS*; Processing Speed r = -0.16, df = 11, p = *NS*; Working Memory r = 0.21, df = 12, p = *NS*; Verbal Comprehension r = 0.11, df = 12, p = *NS*, all two-tailed tests). Power however was low (0.44 for Working Memory, Perceptual Organisation and Verbal Comprehension indices and 0.41 for the Processing Speed Index).

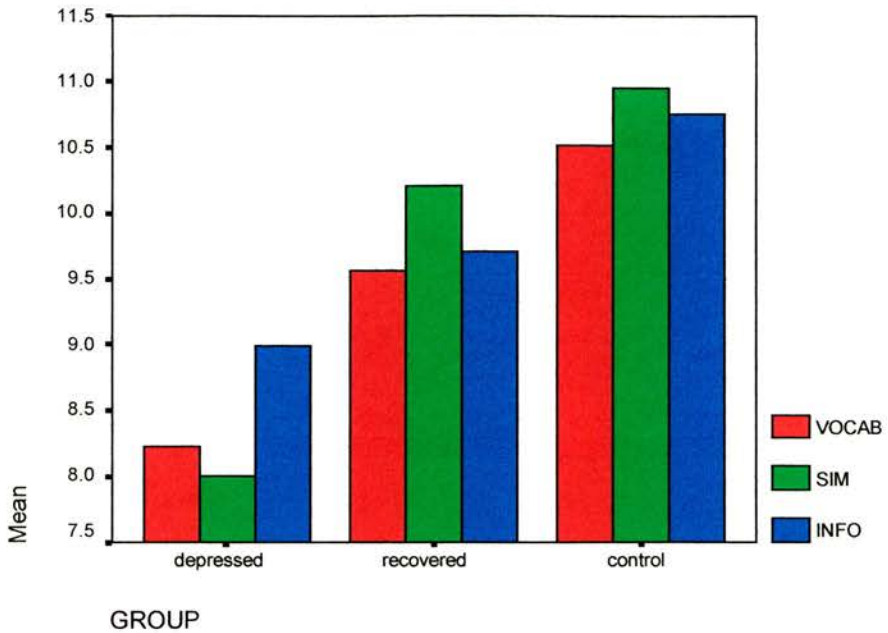
Normal Control Group. In the normal control group, no significant correlations between age and any age-scaled Index score were detected (Perceptual Organisation r = 0.43, df = 18, p = *NS*; Processing Speed r = 0.24, df = 19, p = *NS*; Working Memory r = 0.29, df = 19, p = *NS*; Verbal Comprehension r = 0.42, df = 19, p = *NS*, all two-tailed tests). Power however was low (0.66 for the Processing Speed, Working Memory and Verbal Comprehension indices and 0.62 for the Perceptual Organisation index).

The research hypothesis was accepted.

3.3.6 Hypothesis 6

The research hypothesis proposed that there would be no significant difference between the three groups on the Verbal Comprehension Index.

Figure 8. Difference in Mean Age-scaled Scores on Subtests Contributing to Verbal Comprehension Index by Group



A one-way, between-subjects ANOVA was used to investigate the possibility of differences in mean score on the Verbal Comprehension Index between the three groups. Results suggested a significant difference between mean group scores on the Verbal Comprehension Index (F (2,54) = 5.20, $p < 0.01$, MS Verbal Comprehension = 905.83).

Scheffe's t was used to investigate the source of the significant effect. A significant difference in mean score on the Verbal Comprehension Index was observed between the depressed and normal control groups ($p < 0.01$).

The research hypothesis was rejected.

Independent samples t-tests were employed to make comparisons between mean

scores of the depressed and normal control groups on each of the three subtests contributing to the Verbal Comprehension Index.

Vocabulary Subtest. There was a significant difference between the mean age-scaled scores for the Vocabulary subtest between the depressed and normal control groups ($t = -2.63$, $df = 41$, $p < 0.05$; two-tailed test).

Similarities Subtest. There was a significant difference between the mean age-scaled scores on the Similarities subtest between the depressed and normal control groups ($t = -3.56$, $df = 41$, $p < 0.01$; two-tailed test).

Information Subtest. There was a significant difference between the mean age-scaled scores on the Information subtest between the depressed and normal control groups ($t = -2.10$, $df = 41$, $p < 0.05$; two-tailed test).

4. DISCUSSION

4.1 Summary of Results

No significant differences were detected between the depressed, recovered depressed and normal control groups with regard to age, sex, length of education or estimated premorbid IQ (PMIQ). It was therefore considered appropriate to discuss the cognitive performance of the three groups in relation to the presence or absence of depression as assessed by the Geriatric Depression Scale (GDS) and clinical impression on observation with reference to DSM-IV criteria.

Individuals in the depressed group demonstrated a significantly higher level of anxiety than the normal control group. All three groups demonstrated a significantly lower current full scale IQ (CFSIQ) (as assessed by the WAIS-III) than PMIQ (as assessed by the NART). The depressed group demonstrated significant impairment on all four indices of the WAIS-III by comparison to the normal control group. The recovered depressed group were not significantly impaired by comparison to the normal control group on any of the four indices of the WAIS-III. There was a tendency, however, for scores on all indices in the recovered depressed group to be lower than in the normal control group even taking into account the suggestion of a slight difference in this same direction between the two groups with respect to PMIQ (Figures 2 & 3).

Discussion of these findings in relation to stated hypotheses and the context of

previous research follows a critique of sample characteristics and demographics.

4.2 Sample Characteristics

Following J. Cohen's (1992) recommendations relating to statistical power, the study design ideally required a minimum of 21 individuals in each of the three groups when data would largely have been analysed using ANOVAs. Considerable difficulty was experienced in obtaining the final sample comprising three groups of 22, 14 and 21 as a result of geographical location, organisation of services, confusion over group allocation and time constraint. Perhaps because of the fact that the recovered depressed group was particularly small ($N = 14$), significant differences in means were mainly only detected between the depressed and normal control groups. Further comparisons between these two groups employed *t*-tests and therefore difficulties relating to a lack of sufficient numbers to meet minimum power recommendations were emphasised.

The area in which the research was being carried out was scarcely populated and geographically vast. Altogether, 47 individuals were tested in their own, variously located, homes and a further two participants were tested in a day centre 70 miles distant. Only eight individuals attended the psychology department for testing.

The NHS Trust area involved did not offer a psychology service for older adults. Britton & Woods (1999), discussing "ageism" in clinical psychology, point to the lack of psychology posts existing to specifically meet the needs of older adults and an

additional lack of trained personnel to fill posts which do exist. GPs have suggested that older adults are less likely to request psychological intervention and are increasingly likely to offer medication to older adults (Sullivan, Copeland, Dewey, Davidson, McWilliam, Saunders, Sharma & Voruganti, 1988) although Skelton-Robinson (1995) reported that, where a service is available, referral patterns change to take advantage of it.

It had been planned to recruit the entire sample from a defined level of care in the form of outpatient psychiatry services. This would have maximised consistency. Carney, Reynolds & Sheffield (1987) point to the fact that qualitatively different episodes of depression have different prognoses. Possible advantages of such an approach would include increased confidence in diagnosis of depression according to formal criteria by comparison to a primary care population and the avoidance of confounding factors associated with inpatient status as a result of concurrent physical illness where medical samples are used (Gurland, Wilder, & Berkman, 1988).

Initial approaches and presentations describing the project were made to six psychiatrists in the Trust area and their associated CPNs. These individuals made up four teams (three adult mental health and one specifically for older adults). Depressed individuals identified in Table 3a as originating in the Psychology Department were referred initially to the older adult psychiatry team and then on to the researcher for cognitive assessment as part of a core placement in older adults being undertaken in the area with supervision from a consultant in old age psychiatry and a psychologist working in adult mental health.

It was originally intended to set a minimum age limit of 65 for participation. However, as concern grew regarding the possibility of obtaining an adequate number of participants, the age limit for participation was dropped to 60 years. Cut off criteria based on chronological age are essentially arbitrary but, by convention, "old age" is usually considered to start at 65 years. Both 60 and 65 have been used as cut-off points in psychological research (Britton & Woods, 1996).

In addition, a consultant in the Psychiatry of Old Age in a neighbouring Trust was approached as ethical approval applied to both areas. Access was granted to an older adult assessment ward and a day centre in this trust. Liaison with staff here was not facilitated by the distances involved and, in the event, only two participants were successfully recruited from this source. A consultant physician responsible for an inpatient medical ward for older adults was also approached. Individuals from this source, identified as being suitable for the study on the basis of their depression status, were considered and excluded prior to approach on the basis of disqualifying concurrent medical conditions and in particular stroke and significant vascular disease. As a result of reviewing Psychology Department databases, two recovered depressed individuals were identified. These individuals had been treated in the Psychology Department, as a concession to their referrers, some time previously on the basis that they had only just exceeded age criteria for adult mental health.

It was with some reluctance that GP practices were approached, for the reasons referred to above but also since it was considered potentially ethically unsound to ask

for their assistance in identifying depressed older adults willing to participate in the study when there was no psychological service available within the Trust to offer these individuals for the management of their condition. Three GP practices were finally approached with an accompanying offer of a limited amount of psychology time (Appendix 4). Practice meetings were attended and project presentations made. These resulted in identification of 55.6 per cent of the individuals who finally made up the depressed and recovered depressed groups (Tables 3 a,b).

Normal control participants were obtained from a variety of sources (Table 3c). The majority were acquaintances of the father of one of the department psychologists and friends and relatives of two of the researcher's local acquaintances. They were of varying occupational and socio-economic backgrounds. Four individuals were identified as a result of an approach to the Womens' Royal Voluntary Service (WRVS). The possibility that such individuals represent a non-random selection is not discounted although it should be noted that only two of the individuals obtained in this way were directly involved with the organisation. They, in turn, identified the remaining two participants who were acquaintances of theirs.

4.3 Allocation to Experimental Group (Depressed or Recovered Depressed)

Amongst individuals identified by psychiatrists a clear indication was made as to appropriate group allocation, by the involved psychiatrist, in advance of the researcher approaching the individual. Following identification, approach and agreement to participate, individuals were interviewed, with DSM-IV criteria in mind, observed

over a minimum period of two hours during testing and completed the GDS. Group allocation was confirmed by all of these means. On no occasion did a psychiatrist allocate an individual to one group where GDS score, observation and interview suggested membership of another.

However, the situation was more ambiguous amongst those participants identified by GPs. All three GP practices initially identified participants by searching their databases for prescription of anti-depressant medication. Whereas psychiatrists had a clear and accurate advance idea as to appropriate group membership for any given individual, this was not the case with participants identified by GPs.

There could be a number of explanations for this observation. It may be a reflection of differences in caseload so that psychiatrists, with a smaller load, are more able to call particular individuals and their current status to mind. Alternatively, or additionally, it may reflect more genuine ambiguity regarding diagnosis and management of those individuals whose depression is assessed as being less severe, intransigent or complicated by concurrent medication for physical illness (as indicated by their management in primary care). There may be a lack of emphasis on the establishment of boundaries accurately delineating the presence of clinical depression and its alleviation. Finally, GPs may simply have been unaware of the current status of their patients. Anti-depressant medication had been prescribed in all cases and, where follow-up had not been eventuated, the current status of these individuals may not have been stringently monitored. This would not be the case in outpatient psychiatry where follow-up for discharge back to primary care would be an essential

and frequent part of the process.

As it happened, five individuals identified by GPs as recovered depressed prior to approach were noted, according to interview, observation and GDS score to be significantly depressed and were, as such, transferred to the depressed group. Measures indicated that depression in these misallocated individuals was "severe" (mean GDS score = 22.6; > 21 on the GDS is classified as "severe") but cognitive impairment less (mean PMIQ/CFSIQ discrepancy = 5.8 points) than that in the remaining seven individuals accurately allocated to depressed group by GPs (mean PMIQ/CFSIQ discrepancy = 18.1 points). Thus it may be that individuals whose depression is accompanied by more marked cognitive deficit are "noticed" more by GPs. Perhaps this is because the GPs are alerted to the possibility of dementia developing. No individual identified as depressed was subsequently found to be more appropriately allocated to the recovered depressed group.

A comparison was made between individuals in the depressed and recovered depressed groups in terms of their "origin" (stemming from primary or secondary care) and their PMIQ/CFSIQ discrepancy. A Pearson's point-biserial correlation suggested a significant effect for origin of referral on IQ discrepancy ($r_{pb} = 0.34$, $df = 34$, $p < 0.05$; two-tailed test). Referrals from primary care exhibited less PMIQ/CFSIQ discrepancy (as measured by the NART and WAIS-III) than those from secondary care. Christensen *et al.* (1997) have also reported varying effect sizes for particular cognitive deficits according to source of diagnostic criteria.

As noted above with regard to prevalence studies, there is a fundamental question regarding the generalisability and relevance of studies of depression in older adults where symptoms are inconsistently assessed and the course of illness variously monitored. This lack of consistency leads to difficulty both in making comparisons across studies and interpreting findings. Frank, Prien, Jarrett, Keller, Kupfer, Lavori, Rush & Weissman (1991) proposed that depression research would be greatly enhanced by increasing consistency in the definition of change points across the course of the illness. They suggested an internally consistent, empirically defined, conceptual scheme for the terms remission, recovery, relapse and recurrence.

The meaning of a term like recovery should be unambiguous and based on observable and measurable phenomena such as symptom severity, signs or functional status. There should be a clear consensus between clinicians, researchers and individuals involved as to what constitutes illness and recovery. Recovery should be deemed to have occurred only when symptoms have been absent for a pre-determined length of time. Frank *et al.* (1991) propose attempting to establish a "point of rarity" meaning a period after which very few individuals experience a return of the syndrome or when withdrawal of effective treatment no longer results in return of the full blown syndrome.

In research to date, (including the present study for reasons beyond the researcher's control), the term recovery appears to have been used variably according to the specific context in which resolution of symptoms occurs and the theoretical

orientation of the observer. In the current study therefore, assertions pertaining to features which discriminate the recovered depressed group from the other two groups must be considered very tentative. Even in those cases where recovery was accurately specified in advance, thereby increasing confidence about diagnostic accuracy, no time criteria for length of recovery was imposed. Furthermore, the fact that five individuals identified in advance as recovered depressed were found to be significantly depressed on interview made group sizes uneven and the recovered depressed group particularly small.

Every attempt was made to assess the presence or absence of depression according to the number of criteria discussed. However, it was not possible to have access to any individual in the recovered depressed group in their previously depressed state and this was a weakness in the study, particularly given the awareness of a tendency to mis-assess. Specifically, it was impossible to be sure if depression had been accurately diagnosed at the outset in every case. The difficulties encountered highlight the questions of when and why GPs refer depressed older adults to secondary care and how they assess, treat and monitor recovery from depression amongst their primary care population.

The dialogue above had a number of implications for the present research. The recovered depressed group should have constituted a rich source of data with which to address the continuing controversy regarding the reversibility of cognitive deficit in late-life depression. It had been intended, for example, to consider data relating to length of recovery in the recovered depressed group. On the basis of this data, if the

cognitive function of the recovered depressed group was found to be midway between that of the depressed and normal control groups, (as indeed it was), it may have been possible to make tentative suggestions regarding the reversibility of deficit. Specifically, was increased length of recovery related to continuing improvement, (largely supporting a mood-state interpretation), or was there a point beyond which, irrespective of length of recovery time, end-state residual damage was still observed, (thereby supporting a theory relating to structural damage)? Alternatively, was there a suggestion that some combination of these explanations was possible? Given a failure to accurately allocate individuals to group at the outset according to depressed or recovered depressed status, it became meaningless to begin to attempt to accurately measure "length of recovery". Recovery was also a concept which it appeared difficult for participants themselves to monitor. Asked how long they had been better, a typical participant response included "I can't really put my finger on it. I'm not sure that I'll ever be the same as I was but I'm much better than I was this time last year". Clients themselves then may be similarly encouraged by the imposition of more formal and observable guidelines for measuring mood-state deficit and recovery. Such methods are routinely employed in cognitive behavioural treatments.

Although not a major focus of investigation in the study, attempts were made, again for the sake of consistency and meaningful comparison, to limit participation to individuals suffering from "late-onset" depression. Similar difficulties were encountered. From the point of view of the referring physician, it was simple to make a distinction between someone they had treated for depression throughout the course

of their life versus someone else who had first presented with depression after the age of 60. However, in asking the individuals involved when and how they had first become depressed, the picture was less clear. A typical response included "I felt like this after I had my second child and again when the children left home but I thought it was normal then. Now that my husband is dead, I don't think I can cope with it alone". Thus it is proposed that, at least in a primary care setting, "late-onset" depression can often be reclassified as "late-presentation" depression.

4.4 Refusal To Participate

A high proportion (60 per cent) of individuals identified as recovered depressed either directly refused to participate in the study or failed to respond to an invitation to do so (Table 3b). By contrast, the discrepancy between numbers approached and obtained in the depressed group was a function more of exclusion rather than refusal (Table 3a). This has still further implications relating to the recovered depressed group which was in fact assessed. It was not possible to know the basis for refusal of those individuals who were approached via GPs since agreement to participate was on the basis of an "opt in" system using a tear off return slip. It has already been proposed that these individuals, in their depressed state, may constitute a less impaired group than the secondary care referrals so that the lack of take-up on recovery may be due to apathy or lack of interest. However, it has also been suggested that categorisation of individual status can be inaccurate. In this case, individuals may have been too depressed or angry at the services' failure to help them to consider participation.

Psychiatry referrals were rung following their receipt of an introductory letter. Responses of some of the individuals who were spoken to and refused to participate appeared to confirm that recovery had been accurately assessed but also revealed that participation was viewed as aversive - "No, I'm really not interested. That was a part of my life which I just want to put behind me. I'm very grateful for the help I did have when I needed it but I just want to forget all about it now. Sorry" and "I'm far too busy now that I'm better".

If refusal to participate is based on genuine and complete recovery, as indicated by the secondary care group, this may imply that those individuals who did agree to participate are those who remain unwell to a degree sufficient to make them unwilling to sever contact with services. As such, their cognitive performance and anxiety scores, midway between those of the depressed and normal control groups, would support a mood-state model of cognitive deficit in depression. Alternatively, if there is no single explanation for the cognitive deficit observed in depression, perhaps fully recovered (and non-participating) individuals conform more consistently to a "mood-state", fully reversible, model whilst the experimental group assessed is more likely to include some individuals with permanent structural deficit. Figure 9 shows the variation in Index score spread for the recovered depressed group and suggests at least five individuals (case numbers 23, 24, 28, 29 and 35, or 36 per cent of the recovered depressed group) with some significant, persisting deficit on at least one WAIS-III Index.

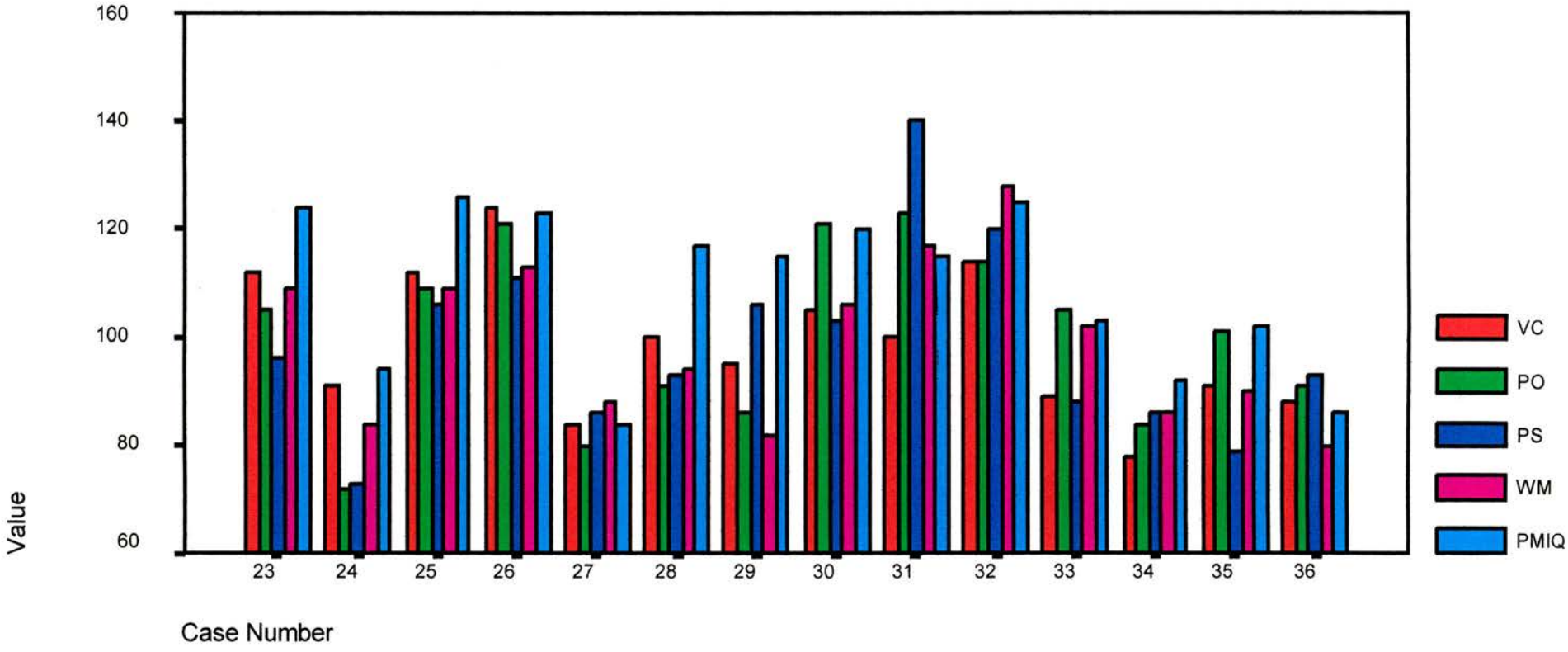
The cognitive performance of recovered depressed individuals constitutes a vital source of data in the quest to assess the cause or causes of cognitive deficit observed in late life depression. The group has not been studied systematically with sufficient consideration accorded to accurate and consistent definitions of illness and recovery. The present study is no exception to this criticism. Further rigorous research should scrutinise the performance of larger groups of individuals whose illness and recovery do meet such criteria.

4.5 Demographics

Although there was a preponderance of female participants in the sample, the ratio of females to males between groups did not differ and therefore sex was not considered as a potentially confounding factor for any of the calculations.

Increasing age has often been reported as exacerbating the cognitive deficit observed in depression (Christensen *et al.*, 1997). Whilst declining skills as a function of increasing age in a normal population have already been discussed, the present study had two apparent strengths with respect to the investigation of a potential additive effect of age and depression on cognitive function. Firstly, it employed a formal premorbid measure of intelligence so that age could be compared to *discrepancy*

Figure 9. Index Scores by Individual Case for the Recovered Depressed Group



between PMIQ and CFSIQ rather than a simple level of current function. There appeared to be no significant relationship between increasing age and IQ discrepancy either in the sample as a whole (r = 0.05, df = 55, p = *NS*; two tailed-test) or the depressed group alone (r = 0.25, df = 20, p = *NS*; two-tailed test).

Secondly, the WAIS-III is scored in such a way as to compare individuals to their same-aged peers rather than a standard, younger, reference group. In so doing, no relationship was detected between current level of function and increasing age either in the sample as a whole (r = 0.02, df = 55, p = *NS*; two-tailed test) or the depressed group alone (r = -0.22, df = 20, p = *NS*; two-tailed test) . This may imply that an additional factor or factors over and above increasing age are responsible for the cognitive deficit observed in the depressed group. However, power pertaining to the depressed group alone was insufficient in these calculations. The group would have needed to comprise 29 individuals in order to increase confidence relating to such an assertion.

There were no significant differences between groups in terms of length of education. It was important however to be aware of some of the education-related effects potentially influencing such a population. Older adults may be less accustomed to the type of "formal" testing that a WAIS-III sitting might mimic. The greatest of care was taken to promote as non-threatening a situation as possible (in particular, the word "test" was avoided). This was aided, it was felt, by carrying out the session in the participant's own home. Where refreshment was offered it was taken and time

was spent sitting chatting prior to commencing work where this was obviously welcomed. It was made clear at the outset that the battery being used was meant for "everyone from people with learning difficulties to geniuses" and that individuals would not be able to complete every stage of it in its entirety. Non-directive encouragement was offered at every opportunity and time scheduled, following administration, to discuss the various facets and anomalies of the measures and the types of skills they purported to assess. Individuals were often keen to point out, for example, that they would have been able to succeed in arithmetic tasks they had had to abandon if paper had been provided. It was possible, in this instance, to reinforce such an assessment and suggest that it was memory rather than mathematical skills which was under consideration. Participants then seemed, spontaneously, to label such an explanation as an understandable and acceptable facet of increasing age which appeared to sit more comfortably than a suggestion of being "bad at maths".

Older adults may also be less inclined to see test situations as relevant. This was most apparent in expressions made by the control group "I don't know what this is going to tell you anyway. It doesn't seem very relevant" (in relation to Picture Completion). Tasks such as Digit Span and Letter-Number Sequencing are especially open to this criticism.

Certain items did appear to have an educational (or perhaps cohort) bias. It was observed, for instance, that a large number of participating older adults were unaware what the "average" was when asked to calculate it as part of an Arithmetic question. In addition, in the Picture Completion item illustrating a train running on one rail, it

was common for participants to remark that the train had no wheels (Appendix 4).

It should also be noted that no credence was given, in the current study, to the effects of medication. Any attempt to do so would have been confounded by the complex combinations of anti-depressants, tranquilizers and hypnotics administered in a wide range of dosage throughout the sample. The vast majority of individuals in the depressed and recovered depressed groups were on varying types of anti-depressant medication. Heaton & Crowley (1981) argue that if doses are stable, the effects of anti-depressant drugs on cognitive function are negligible. McNair, Kaln, Frankenthaler & Faldetta (1984) report that anti-depressant drugs can actually enhance cognitive performance whilst Wolkowitz, Tinklenberg & Weingartner (1985a,b) point to the detrimental effects on cognitive performance of the anticholinergic activity in anti-depressants. The overall impact of anti-depressant treatment on the cognitive performance of the participants in the current study remained an important and uncontrolled variable.

4.6 Mood State Measures

4.6.1 Depression

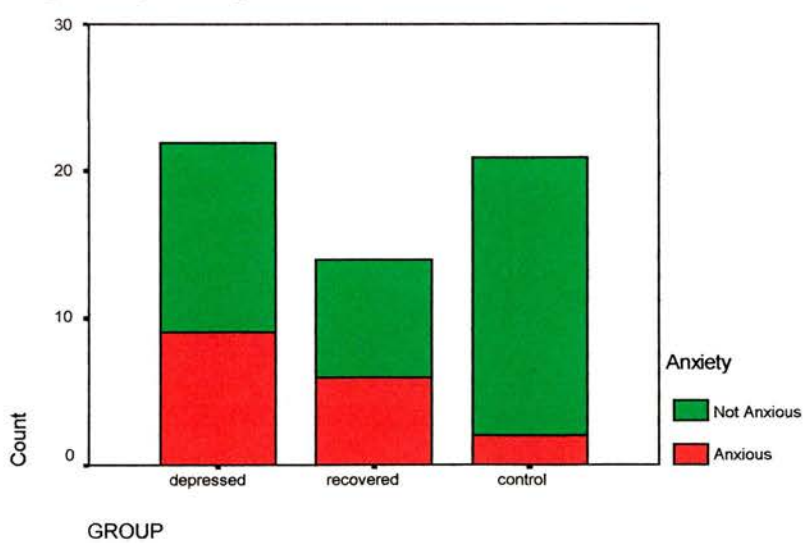
Several studies have reported increasing cognitive deficit as a function of increasing depression severity (Christensen *et al.*, 1997; Lichtenberg *et al.*, 1995). In the present study, a significant association was observed between PMIQ/CFSIQ discrepancy and GDS score (as an indicator of depression severity) for the sample as a whole (r =

0.44, $df = 55$, $p < 0.01$, two-tailed test). This would be anticipated on the basis of a fundamental link between depression and cognitive deficit. No association was observed however between increasing depression severity and PMIQ/CFSIQ discrepancy within the depressed group alone ($r = 0.001$, $df = 20$ $p = NS$, two-tailed test). Brown, Scott, Bench & Dolan (1994) reported similar findings. A lack of association could suggest that symptoms of depression and cognitive function proceed independently. St Kuny (1995) reported that identified cognitive deficits including slowness of thought, poor concentration, indecision and memory problems persisted much longer than the acute depression.

4.6.2 Anxiety

A significantly higher number of depressed than recovered depressed or control participants were assessed as anxious using the Beck Anxiety Inventory (BAI > 10).

Figure 10. Proportion of Anxious (BAI>10) and Non-Anxious (BAI<11) Participants by Group



Criticisms of self-report scales referred to in relation to the identification of depression similarly apply to the utilisation of the BAI in the current study. Two further weaknesses include the fact that the BAI was the sole measurement taken of anxiety and that the scale includes six somatic items which may give rise to older adults (and in particular depressed older adults) being "over-assessed" as anxious. What follows should be considered with this in mind.

Anxiety and depression frequently co-occur (Zarit & Zarit, 1998). It is not clear how far such an observation may confound the results of cognitive assessment. Vingerhoets, DeSoete & Jannes (1995) investigated the effect of anxiety on cognitive performance in individuals undergoing open heart surgery and reported that their significantly-elevated emotional arousal had no effect on cognitive performance. They concluded that neuropsychological abilities are robust, even under conditions of striking personal stress and anxiety. Such a "time-limited" anxiety however, with a specific cause, may not be directly comparable to the more open-ended state accompanying depression. Eysenk (1982) suggested that the presence of anxiety can exert an influence on cognitive capacity by draining attentional resources via worry but that, in addition, it can promote increased motivation or effort.

In terms of the WAIS-III measure, Kaufman & Lichtenberger (1999) identified 5 subtests on which performance was likely to be undermined by the presence of anxiety. These included Digit Symbol, Symbol Search, Arithmetic, Digit Span and Letter-Number Sequencing (or the subtests which comprise the Processing Speed and Working Memory Indices). The apparent effect of the presence of anxiety on these

Indices for the current sample was considered. Taking account of the fact that Eysenck's (1982) observations might predict *enhanced* performance in the presence of anxiety, two-tailed tests were employed to reflect a degree of uncertainty relating to the direction of the hypotheses.

It was proposed that the presence (as assessed by BAI cut-off indicators) and severity (as assessed by BAI score) of anxiety would be associated with mean score on the Processing Speed and Working Memory Indices.

Processing Speed. One participant from the recovered depressed group was removed from the analysis having been identified as a high-functioning outlier on this index.

For the sample as a whole, there was no significant association between mean Processing Speed Index score and the presence or absence of anxiety (r_{pb} = 0.12; df = 54, p = NS; two-tailed test). Neither was there any association between BAI score and mean Processing Speed Index score (r = -0.15, df = 54, p = NS; two-tailed test).

Working Memory. For the sample as a whole, there was no significant association between mean Working Memory Index score and the presence or absence of anxiety (r_{pb} = 0.12, df = 55, p = NS; two-tailed test). Neither was there any association between BAI score and mean Working Memory Index score (r = -0.15, df = 55, p = NS; two-tailed test).

The possibility was then considered that the presence of anxiety exerts an influence on cognitive performance only in combination with depression. The depressed group was divided into anxious (N =12) and non-anxious participants (N = 10) and mean performance on Processing Speed and Working Memory Indices compared in the depressed/anxious and depressed/not anxious groups using independent sample t-tests.

It was proposed that concurrent anxiety (as assessed by BAI cut-off criteria) and depression (according to experimental group allocation) would be correlated with mean scores on the Processing Speed and Working Memory Indices.

Processing Speed. Despite low power (0.42), a significant difference was detected in mean score on the Processing Speed Index according to whether or not depressed participants reported concurrent anxiety ($t = 2.35$, $df = 20$, $p < 0.05$; two-tailed test). Anxious depressed individuals performed better (mean score 90.42) than non-anxious depressed individuals (mean score 81).

Working Memory. A significant difference was also detected in mean score on the Working Memory Index according to whether or not depressed participants reported concurrent anxiety ($t = 2.33$, $df = 20$, $p < 0.05$; two-tailed test). Anxious depressed individuals performed better (mean score 97.75) than non-anxious depressed individuals (mean score 82.2).

It occurred, however, that these findings may have been a simple reflection of

premorbid status and that, for example, individuals of higher intelligence may be more likely to be anxious (perhaps as a result of increased potential for insight). If this was the case, enhanced performance may be a facet of intellectual capability, rather than a function of the presence of anxiety. However, no association between PMIQ and presence or absence of anxiety was detected (r_{pb} = -0.23, df = 20, p = NS; two-tailed test) although power was only 0.33.

If the findings reported above represent something more than chance and turning again to Eysenck's (1982) observations, it could be that attentional resources are not taxed on WAIS-III subtests to a degree where worry becomes counterproductive. Instead, the increased effort that comes about from being anxious pays off in terms of improving performance in depressed individuals where it would otherwise be more impaired.

In the sample as a whole, no relationship was detected between the presence of anxiety and enhanced performance on Processing Speed and Working Memory Indices. Power levels however were adequate only for the detection of large effect sizes and this may be significant if, for example, anxiety exerts only a small effect on cognitive performance. In addition, the insufficient number of anxious individuals in the recovered and control groups may account for the lack of significance. (The depressed group included 60 per cent of the total number of individuals identified as anxious). However, if the finding is robust and concurrent anxiety does enhance the performance only of depressed older adults, it may suggest that, when processing

speed and working memory skills are not impaired by concurrent depression (as in the normal control group) and attentional resources are not being stretched to a maximum by the test being employed, the increased effort, which is a function of anxiety, has no impact upon "personal bests" which are already being achieved.

Whatever the explanation for such findings, it would seem insufficient for researchers to simply continue to observe in passing that anxiety and depression frequently co-occur without considering the effects which each may be having individually on performance. Failure to control for anxiety may have been a further source of confusion in previous research particularly since measures varying so widely in attentional demand have been employed. Future research might address the question of whether measures more demanding of attentional resources result in a breakdown of the enhanced functioning which anxiety may facilitate in depression as a result of increased effort. A further point of interest would be in the extent to which more attentionally-demanding measures might uncover measurable deficit in recovered depressed individuals. However, whilst the detection of any such subtle residual deficits may aid the delineation of the mood-state/structural argument, one would have to question their clinical and functional relevance.

4.7 Hypotheses 1a and 1b

It has been reported that the National Adult Reading Test (NART) is largely resistant to the effects of neurological and psychiatric disorder (J. Crawford, Besson, Parker, Sutherland & Keen, 1987). It is considered robust in the face of poor motivation or

concentration and one of the most reliable tests currently used in psychological practice (Nelson, 1982). All three groups in the present study however demonstrated significantly lower CFSIQ (as assessed by the WAIS-III) than predicted PMIQ (as assessed by the NART). Median predicted PMIQ was 112 (where 100 would be anticipated if the NART was performing as a true measure of IQ). Several points deserve further investigation.

Firstly, NART estimates may be accurate and the present sample may represent a group of higher than average intelligence. In this case, further investigation of the PMIQ/CFSIQ discrepancy becomes necessary. A second possibility is that PMIQ may have been over-estimated in the sample as a result of examiner error. However, it has been suggested that the NART has high inter-rater reliability even in individuals with no previous experience in administering it (J. Crawford, Parker, Stewart, Besson & DeLacey, 1989). In addition, the researcher was aware that some words had been shown to demonstrate lower inter-rater reliability than others (aeon, puerperal, aver, sidereal and prelate) and took particular care to score these accurately.

The NART has not yet been restandardised to function reliably by comparison to the WAIS-III. It was originally conceived to work alongside the WAIS (Nelson, 1982; Weschler, 1955). When the WAIS-R was introduced, obtained IQ scores were reported to be seven and half points lower, on average, than had been the case with the WAIS (Weschler, 1981). The NART was restandardised accordingly (Nelson & Willison, 1991). In a similar way, the relationship between the WAIS-III and its predecessor was examined in a sample of 192 adults (aged 16-72) (Weschler,1997).

It was reported that obtained full scale IQ on the WAIS-III was 2.9 points lower than that obtained on the WAIS-R. Table 9 illustrates the point that the discrepancy scores, calculated, by necessity, with reference to data relating to the WAIS-R rather than the WAIS-III, become far less unusual if 2.9 (or 3) points are deducted from obtained WAIS-III CFSIQs to "transform" them, as accurately as is presently possible, into approximate WAIS-R scores.

Based on such a "transformation", the differences in PMIQ and CFSIQ become insignificant in the control group (paired sample *t*-test, $t = 0.45$, $df = 20$, $p = NS$; two-tailed test) but remain significant in both the depressed and recovered depressed groups (paired sample *t*-tests $t = 6.27$, $df = 21$, $p < 0.01$; $t = 2.44$, $df = 13$, $p < 0.05$; two-tailed tests).

Another source of the discrepancy may relate to the use of the NART with a sample of older adults. Standardisation of the test included individuals aged up to only 70 years (Nelson, 1982). However, the measure should be assumed to be accurate in older individuals to the extent that reading ability is not thought to deteriorate with increasing age until after the age of approximately 85 (Nelson, 1982). Studies have demonstrated the validity of the NART in individuals up to age 84 (Beardsall & Brayne, 1990; Brayne & Beardsall, 1990). However, education and sociodemographic information can also predict NART performance (J. Crawford, Stewart, Garthwaite, Parker & Besson, 1988). On this basis, estimates of full-scale IQ made according to the NART might be expected to be lowered in a population of

Table 9. "Transforming" Group WAIS-III Scores into WAIS-R Scores and Assessing the WAIS-R NART Discrepancy Probability

GROUP	MEAN PREDICTED FSIQ (NART)	MEAN OBTAINED CFSIQ (WAIS-III) TO NEAREST POINT	% OF INDIVIDUALS HAVING THIS SIZE OF DISCREPANCY OR LARGER (BASED ON WAIS-R DATA)	"TRANSFORMED" MEAN WAIS-III SCORE OR ASSUMED WAIS-R SCORE	% OF INDIVIDUALS HAVING THIS SIZE OF DISCREPANCY OR LARGER
ENTIRE SAMPLE	108	99	14	102	26
DEPRESSED	105	90	3	93	8
RECOVERED DEPRESSED	109	100	14	103	26
NORMAL CONTROL	112	108	35	111	49

older adults many of whom would have left school at the minimum statutory age. In fact, slightly less than half of this sample (47 per cent) received eight or less years of schooling. This may add some support to the assertion, referred to above, that the sample could have been non-significantly skewed towards higher achievement. In addition, the issue of high probability of less education in older samples has to be matched against the observation that "crystallised" skills (such as vocabulary) can improve up to the seventh decade of life (Stuart-Hamilton 1999).

A further consideration is the disproportionately higher number of females making up the sample. Females have better reading skills than males with equivalent IQs (Nelson & Willison, 1991) and a bigger discrepancy between PMIQ (as assessed using a "reading" measure) and CFSIQ (assessed using a range of measures) might therefore be predicted for females and thus this sample as a whole. There was a trend in this direction (mean discrepancy between PMIQ and CFSIQ = 10.23 for females and 8.83 for males) but this was found to be non-significant when an independent *t*-test was applied ($t = 0.47$, df = 55, $p = NS$; two-tailed test); power = 0.69.

It is further possible that predicted IQ scores obtained using the NART were accurate and that the WAIS-III scores were in fact depressed. The WAIS-III was found to be a very lengthy test to use with older adults. Administration time in the current study varied between one and a half and four hours. Only one of the sample took the opportunity to split testing over several sessions. Hayslip, Kennelly & Malloy (1990) reported that depressed older adults frequently report fatigue. Unfortunately, it is not

possible to obtain an objective measure of this or to corroborate or refute a complaint of fatigue. Where fatigue exists, it may suggest concurrent physical illness such as vascular disease which would itself, in turn, impact upon cognitive performance. By contrast, a subjective perception of fatigue may exist in some individuals which is unrelated to cognitive performance (or, at least, related only to the extent that motivation is decreased by depression and the accompanying perception of fatigue). To the extent that older individuals might be assumed to tire more readily than their younger counterparts, the presence of fatigue in any of the sample may have contributed to a lower assessed CFSIQ than PMIQ. It would have been of interest and potential value to accurately record the time taken to complete the tests for each individual as well as seeking a subjective rating of degree of fatigue before considering any relationship between such measures and WAIS-III performance.

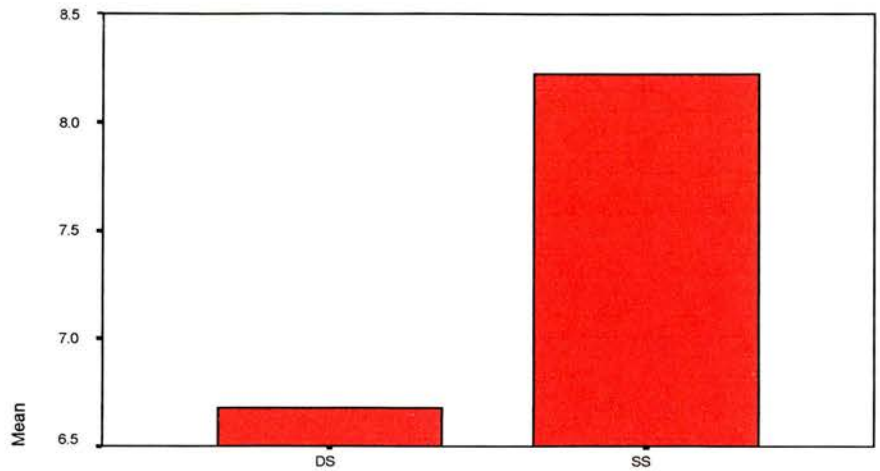
Despite the presence of significant differences between PMIQ and CFSIQ in all three groups, the deficit in the depressed group was very much greater (Figure 2) and, in addition, the "pattern" of scores for the recovered and control groups did not suggest the presence of specific deficits in these groups warranting further consideration (Figure 3). Investigations therefore proceeded on the basis that the depressed group were demonstrating considerably more decline than any which may exist in either of the other two groups.

4.8 Hypothesis 2

Processing Speed has been the most consistently reported deficit relating to depression (W.R.Miller, 1975; Nebes, Brady & Reynolds, 1992; St Kuny, 1995). Figure 3 indicates a deficit in the WAIS-III Processing Speed Index in the depressed group which appears to exceed the more general cognitive deficit identified. Interpretation of this observation is not straightforward since it involves the integration of multiple data sources. Poor performance on this Index may be a function of any one or combination of a number of skills including processing speed, visual motor coordination, motivation, reflectiveness, compulsiveness, visual memory and planning ability (Kaufman & Lichtenberger, 1999).

The Processing Speed Index of the WAIS-III comprises only two tasks, Digit Symbol Coding and Symbol Search. Symbol Search is a new task involving the presentation of 60 items consisting of paired groups of symbols. The participant is required to indicate whether or not either of the symbols in the target pair appear in a search group of symbols. To the extent that Symbol Search might be expected to tap "mental speed" whereas Digit Symbol may rely more on "motor speed", there was some suggestion that participants in the depressed group were more impaired on motor speed (Figure 11), although mean scaled scores on both tasks were impaired by comparison to normal controls.

Figure 11. Mean Age-Scaled Performance of Depressed Group on Digit Symbol (DS) and Symbol Search (SS) Subtests.



The term "psychomotor" implies both cognitive and behavioural processes flowing together in a single act. Cornell, Suarez & Berent (1984) made an attempt to systematically vary cognitive and motor demands and concluded that 'endogenous' depression resulted in the retardation of both cognitive and motor skills whilst 'non-melancholic' individuals exhibited only motor deficit. The research was weak in that it included only 26 experimental participants of non-specified age divided into two groups and two individuals (in the endogenous group) who had a 'reported history of hypomanic periods'. It is nevertheless an interesting line of enquiry. Further investigations might address the extent to which observed psychomotor slowing always represents the same underlying cause. Nebes *et al.* (1992) suggested that motor retardation is most likely to be motivational in origin whereas cognitive slowing may be more representative of structural deficit.

That it was speed rather than memory which was primarily responsible for the poor

scores was reinforced by the observation that on the optional "Symbol Copy" procedure, (requiring, as the name suggests, simple copy of individually presented symbols and thereby lessening the requirement for working memory by comparison to the Digit Symbol Coding task), scores in the depressed group appeared to be as impaired as those on Digit Symbol Coding. Only 14 per cent of 22 individuals scored above the 50th percentile, a further 27 per cent between the 25th and 50th percentiles, 32 per cent on or between the 10th and 25th percentiles and the remaining 27 per cent scoring below the 10th percentile. In addition, to the extent that a degree of working memory and planning might be required in the assessment of two abstract symbols simultaneously in Symbol Search, the group performed comparatively well. It would be of value if WAIS-III scoring procedures permitted more formal comparison between the Digit Symbol Coding and Copy procedures.

The WAIS-III also promotes an optional measure of Incidental Memory which involves assessing an individual's ability to recall, following the Digit Symbol Coding task, both the symbols (Free Recall) and the numbers they were paired with (Pairing). It had been intended to implement this procedure in the current study. The first depressed participant who was asked to attempt the procedure simply remarked "I don't think I can remember any of them. I wasn't thinking about that" and refused to carry out the task. It was considered that such a refusal did not necessarily constitute the reality of what could have been achieved had the participant been willing to try. The second depressed participant asked to attempt to the task was so concerned by their failure to complete the Pairing task, ("I don't know any of them that went together. Did you ask me to do that? *Should* I be able to do it?"), that it was

concluded that persisting in the implementation of this procedure with following participants may well unsettle them to a degree which might affect their performance on subsequent subtests. The procedure was therefore omitted from the battery.

It may have been that the researcher was unduly sensitive to these initial responses coming, as they did, close to the outset of the project. If participant assertions are taken at face value, however, they did not support the observations of Hart *et al.* (1987, 1987a, 1987b) who reported that incidental memory was unaffected by depression. Perhaps future research focusing on processing speed might consider including the Digit Symbol Coding, Copy and Incidental Learning procedures as an experimental study in their own right. Certainly decreased processing speed appears to feature so prominently in the cognitive deficit observed in depression that it would seem appropriate to recommend that future studies utilise broader and more intricate measures of processing speed than can be afforded by a two-subtest Index of the WAIS-III.

4.9 Hypothesis 3

Mean scores on the Working Memory Index in the depressed group were significantly lower than those obtained by the normal control group. The Index comprises the Arithmetic, Digit Span and Letter-Number Sequencing subtests of the WAIS-III. Letter-Number Sequencing is one of three new subtests involving 21 trials which require the participant to sequentially order a series of letters and numbers which have been orally presented in a mixed up order.

Like the Processing Speed Index, the Working Memory Index can be assigned diverse interpretations including attention, concentration, anxiety, sequencing ability, sequential processing, number and planning ability, short-term memory, executive processes and event visualisation (Kaufman & Lichtenberger, 1999). The Index spans both cognitive and behavioural domains.

There did not appear to be undue scatter in the depressed group amongst the mean, age-scaled scores of any of the three subtests comprising the Working Memory Index (Arithmetic = 8.3, Digit Span = 9.2, Letter Number Sequencing = 8.3). This might suggest that depleted overall Index scores were not directly related to subtest- specific deficits such as acquired knowledge, long-term memory, working under time pressure and reasoning, (specific to the Arithmetic subtest), negativism (more a function of Digit Span and Letter-Number Sequencing) or persistence (a feature of Letter-Number Sequencing). The mean forward/backward Digit Span Discrepancy in the depressed group differed little from that obtained by the control group and range of discrepancy was in fact less (Digit Span discrepancy mean for depressed group = 3.2, range 0 to 7; Digit Span Discrepancy for control group = 2.9, range -1 to +8.). Such an observation does not support the idea of a deficit in effortful processing in depression if, as is suggested, "Digits Backwards" is viewed as an effortful process and "Digits Forwards" as more automatic.

Anxiety was expressed across the sample as a whole at the prospect of a mental arithmetic test. Similarly, the researcher came to dread the explanation preceding the

Letter-Number Sequencing subtest since it met with such horror (which was not necessarily matched to subsequent performance). However, in view of the response, quoted above, of one depressed participant to presentation of the Digit Symbol Incidental Learning task, it is important to consider the possible impact of motivation on the Letter-Number Sequencing task. It is well positioned in this respect as the final test of the battery although may also be, for this very reason, subject to the effects of fatigue. Perhaps it is of some relevance that mean scores on the Arithmetic and Letter-Number Sequencing tasks, which seemed to be perceived as potentially difficult and threatening, were slightly lower than those on the less threatening Digit Span.

Referring to the Arithmetic subtest, it has already been mentioned that many of the participants, irrespective of group, were unaware of what an "average" was. The depressed group did come across as more "careless" (four out of eight who reached the question doubled rather than halved the sum in responding to the question "Chris has two times as much money as Robert. Chris has £99; how much does Robert have?"). It was also relatively common to receive the answer £11.50 to the question "If you have £18 and spend £7.50 pence, how much will you have left?" There was a tendency in the depressed group to ask for repetition and/or to fail on the longer questions, but this did not appear to be a function of insufficient time being allowed for response. Rather there was a tendency to declare an inability to answer or to answer incorrectly in advance of time being up. This is suggestive of a fundamental deficit in working memory or, in other words, an inability to hold increasing amounts of information in mind whilst simultaneously manipulating such information.

In summary, depleted scores on the Working Memory Index amongst the depressed group appeared to represent just that. The group had a "feel" about them of being unwilling or unable to attempt questions which they perceived as overwhelming in terms of either volume or complexity of information. It will be necessary in the future to more formally record and measure some of the more anecdotal and behavioural features of WAIS-III assessment in depressed individuals. These might include requests for repetition, performance as a function as length of question, "near misses" or inferred causes of error, refusal to attempt particular tasks and individual questions within them and learning across trials in tasks such as Letter-Number Sequencing.

4.10 Hypothesis 4

It had been hypothesised that any deficit observed on the Perceptual Organisation Index would be more a function of tests remaining dependent on processing speed (specifically Block Design since time limits for Picture Completion appeared to be relatively generous) than the untimed Matrix Reasoning test. Matrix Reasoning is the third new subtest in the WAIS-III, added to enhance the measurement of abstract fluid reasoning (The Psychological Corporation, 1997). It involves the presentation of 26 gridded patterns each with a portion missing. The participant is required to complete the pattern by choosing from five possible options.

Investigation indicated that there were significant differences in mean score on both the Block Design and Matrix Reasoning subtests between the depressed and normal

control groups but not the Picture Completion test. Both Block Design and Matrix Reasoning are good measures of general ability (Kaufman & Lichtenberger, 1999). Sackeim (1992) observed that all five performance subtests contributing to Performance IQ using the WAIS-R impacted on the overall deficit exhibited by depressed individuals. Possible explanations for these discrepancies are considered.

Although the depressed group performed worse than the normal control group on the Picture Completion test, the difference was not significant. Figure 6 suggests that the discrepancy could have been more a function of a comparatively poor performance by the normal control group rather than less impaired performance by the depressed group. The normal control group did demonstrate a tendency to over-analyse and overlook the obvious. For example, in the picture of a fire, one individual referred to the fact that the wood in the fire did not appear to be charred and a second to the lack of chimney pot. Several normal control participants pointed out that, in a picture of a bread knife cutting bread, the knife was "the wrong way round". In the train picture, one participant referred to a perceived lack of name on the side of the train and another commented "Well it could be the rail but then you do have mono-rail trains..." (Appendix 5). Whilst the Picture Completion task then is a fairly non-threatening start to the test session for more vulnerable individuals, it could be that it is too "easy" for normal controls who are starting from a stance of thinking that attempts are being made to "catch them out".

Depressed participants had a tendency to offer "don't know" responses relatively quickly and move on with little or no attempt to pursue the matter. Normal control

participants, on the other hand, were more inclined to scrutinise the pictures and confidently proffer incorrect answers. The tendencies of depressed individuals to take less risks, be more likely to offer "don't know" responses and less likely to give themselves the benefit of the doubt have been previously reported (Alloy & Abramson, 1979; E. Miller & Lewis, 1977; Young, Manley & Alexopoulos, 1985).

The significant discrepancy between the normal control and depressed groups on the Block Design subtest was predicted. However, the discrepancy did not seem to stem entirely from the source predicted, (speed of processing). Again, depressed individuals appeared to make a clear decision as to whether or not they were able to achieve what was required of them and either got on and did it within the time limit or pushed the blocks back towards the researcher with a clear expression of inability to complete the task. Concept formation on design number 11 (narrow stripes) was of particular interest. Depressed individuals demonstrated an apparent difficulty in switching from the wide stripes of Pattern 7 to the narrower version required in this design (Appendix 4). They often attempted to apply Design 7 strategies and, when this proved unsuccessful, they frequently gave up.

The discrepancy between the normal control and depressed groups on the Matrix Reasoning subtest was not predicted in advance. The subtest includes four types of items: continuous and discrete pattern completion, classification, analogy reasoning and serial reasoning. Some degree of flexibility is required to switch between these appropriately. It may have been of interest to analyse the responses given by the depressed group more closely to see if there was any indication that they were

attempting to apply any one strategy consistently rather than appreciating the necessity to switch.

Depressed individuals seemed less likely to give "don't know" responses in this subtest than in some of the others. "Don't know" responses were most frequent when faced with some of the overwhelmingly colourful and "busy" items requiring detailed visual analysis. The apparent novelty of such a task may make it more acceptable to try and fail than on the more traditionally education-based tasks such as Arithmetic and Vocabulary where participants might feel that they ought to know the answer. Perhaps thought processes are less "conspicuous" in a test such as Matrix Reasoning and it is this which contributes to an ability to offer incorrect rather than "don't know" responses.

The fact that the depressed group did no better on this test despite their increased likelihood of responding might suggest that "don't know" responses in other subtests are in fact accurate assessments of the situation rather than lack of motivation or unwillingness to try. As in Picture Completion, depressed participants often gave the impression of failing to give the task adequate attention. They flicked through the book offering incorrect responses until the task was halted. Normal controls seized on the advice that the task was untimed and seemed to use this information to check and re-check responses before replying. It may be interesting to consider, in the future, whether "don't know" responses increase as a function of being asked to explain how incorrect conclusions have been reached. Similarly, it may be enlightening to assess any change in response pattern following examiner indication

that an answer was wrong. This type of anecdotal observation clearly requires more formal and systematic measure and consideration, particularly in view of the fact that Matrix Reasoning is a new subtest. This cannot be validly conducted in a study in which an emphasis has been placed on obtaining accurate formal scores.

4.11 Hypothesis 5

Perceptual Organisation scores did significantly decrease amongst depressed individuals as a function of increasing age. Such additional decline might be thought to represent decreased processing speed. However, as has been noted above, this was not a wholly satisfactory explanation and no similar association was noted on any of the other three Indices. Furthermore, there was no relationship between increasing age and any of the Index scores in the sample as a whole. That depressed individuals often gave up prior to the expiry of time allowed on Block Design and refused to continue suggested a genuine inability, either real or perceived, to complete the task, irrespective of time constraint. There is a possibility that the type of tasks included in the Index are the most unfamiliar to increasingly older adults or that sensory limitations such as failing eyesight would be significant. However, in either of these cases, it would be expected that a similar effect would be observed on the Processing Speed Index and this was not the case. It is an area worthy of further consideration.

4.12 Hypothesis 6

It has often been reported that depressed individuals show no deficit in verbal skills, particularly by those researchers whose theoretical stance involves a hemispheric explanation for cognitive deficit in depression (Sackeim *et al.*, 1992). Contrary to prediction, the depressed group in the current study demonstrated deficit on all three subtests which make up the Verbal Comprehension Index (Vocabulary, Similarities and Information) by comparison to the normal control Group. Reference to Figure 8 comparing the pattern of mean, age-scaled scores for the three groups across the Verbal Comprehension Index, suggests that a component of the Similarities subtest is particularly influenced by the presence of depression. It is also suggested that the pattern of scores in the recovered depressed group mirror those of the normal controls far more closely than that of the depressed participants indicating reversal of such a deficit with increasing recovery.

Successful performance on the Similarities subtest requires fluid, flexible, abstract and categorical thinking. Whilst inflexible, rigid behaviour might beg a structural explanation, the apparent reversibility of such a deficit would not confirm such a stance apart from the extent to which structural damage was also considered reversible. Formal learning is less emphasised on the Similarities subtest than in Vocabulary or Information and more fluid, "problem solving" of simple verbal constructs is required. To the extent that scores on this subtest were particularly low amongst the depressed group, it might be supposed that these types of skills have been affected. The sample as a whole did express negativism by way of comments

such as "Well they're not alike are they?" but the normal control group proferred this as no more than a "starting point", going on to pursue the question with humour and imagination. For example, asked how work and play were alike, one normal control participant responded "Well you get paid to work and play costs you. Mind you, now I don't work I haven't got any money to play with but they're just things that most people do - both good fun in moderation". The depressed group, by contrast, offered no more than an initial denial that the pair were in any way alike. In addition, they demonstrated a persisting tendency to be stuck in a mindset of explaining how pairs were *different.*

Vocabulary has traditionally been viewed as an automatic process, perhaps the most so of the three "automatic" subtests (Vocabulary, Picture Completion and Information). It requires the activation of the meaning of words stored before becoming depressed (Hartlage *et al.*, 1993). However, Kennelly, Hayslip & Richardson (1985) suggested that older adults may actually perceive the task as requiring more effort than would necessarily be appreciated.

Paucity of response was noted amongst the depressed group. They were more likely to give examples of one-point answers such as "Mend" when asked "What does repair mean?" and "Sorry" when asked "What does remorse mean?" When ratios of two to one point answers were considered between the depressed and normal control groups on those items where a score was obtained, it was noted that normal control participants had a ratio of 350:119 (or 3:1) and the depressed group 244:144 (or 1.7:1). This suggests not only that the depressed group scored positively on less

items but also that the quality of their response was poorer. In terms of their failure to respond, negativism was again apparent. Thus, depressed individuals would be inclined to comment " I know what it means but I can't explain it" whilst the normal control group would make this same statement as a starting point but then continue to try until they had found an adequate way of expressing their understanding.

In the Information subtest, the depressed group again demonstrated their unwillingness to guess or give themselves the benefit of the doubt. A common response to the question "Who painted the Sistine Chapel?", irrespective of group, was "It was either Leonardo DaVinci or Michaelangelo". However, whereas the normal control group would then hazard a guess when pressed "Which one are you going to go for?", the depressed group tended to respond "I don't know".

4.13 Summary of Findings and Possible Interpretations

Cognitive deficit in late life depression (as assessed by the WAIS-III) appears to be broad ranging and not specifically confined to those tasks which load heavily on speed or attention (Processing Speed and Working Memory) although Processing Speed would indeed appear to be the most severely affected WAIS-III Index. Previous research has also reported generalised or global deficit (Siegfried, 1985; Caine *et al.*, 1993; Christensen *et al.*, 1997). Raskin *et al.* (1982) identified general deficit across measures requiring sustained effort or concentration, perceptual flexibility, abstract thinking, motivation and accuracy. Lamberty & Bieliauskas (1993) concluded that the bulk of the research supports the idea of general cognitive inefficiency and

attention rather than a lack of ability due to structural deficit. Beats *et al.* (1996), considering 'executive function', noted impairments in a depressed group relative to both controls and themselves on recovery across all domains examined. A deficit however which appeared to be specific to depression was the 'catastrophic response to failure' or suggestion that if initial attempts to solve a problem failed, thinking efficiency subsequently deteriorated. Such an observation may be highly relevant to a test such as the WAIS-III since it is quite "generous" in subtest discontinue criteria. This might mean that individuals could easily be aware of and affected by perceived failure.

The suggestion that thinking efficiency deteriorates only as a result of perceived failure strongly indicates an inability or unwillingness to use fundamentally intact systems. It is an explanation which could also account for many of the behavioural differences in WAIS-III assessment performance observed between groups in the current study. Perhaps it is easier for participants to assess their performance on some subtests that others. Individuals may be clear, for example, that they either know the meaning of a word or do not (Vocabulary) and tend, as a result, to "opt out" and offer "don't know" responses. On the Comprehension subtest however, they may be less likely to be aware of having failed. Such an "attitudinal deficit" may either combine with, or even account for, additional difficulties on Processing Speed and Working Memory Indices. The idea would require further investigation specifically in relation to WAIS-III subtests.

The cognitive deficit witnessed in late-life depression did not appear either to be as straightforward as a distinction between automatic and effortful processing. Hartlage *et al.* (1993) proposed that search, imagery, organisation and problem solving skills were required to master 'effortful' tasks and that it was these skills which were impaired in depressed individuals. They classified Vocabulary, Picture Completion and Information subtests as 'automatic' tasks and Block Design and Comprehension as 'effortful'. Several other studies have supported the suggestion of deficits in Block Design and Comprehension but not Vocabulary amongst depressed individuals (Hart *et al.*,1987; Robertson & Taylor, 1985). The evidence has not been so convincing for Picture Completion and Information (Robertson & Taylor, 1985). However, when subtests in the present study were divided according to the Hartlage *et al.* (1993) distinction, no significant difference was detected in the depressed group between automatic and effortful tasks using a paired sample *t*-test (t = -0.58, df = 21, p = *NS*; two-tailed test). Furthermore, ranking each applicable mean subtest score for the group further disconfirmed the distinction. Individuals scored best overall on Comprehension (an effortful task) then alternately well on each of the remaining automatic and effortful tasks. Hayslip *et al.* (1990) suggest that the automatic/effortful distinction is more relevant to memory tasks. To the extent that the WAIS-III and revised Weschler Memory Scale (WMS-III) are now interlinked in terms of scoring, this theory could be pursued in future research. Christensen et al (1997) purported that effort may simply be a function of processing speed. Even if this made the distinction more relevant, it has already been suggested above that the deficit observed above was a function of more than a deficit in speed.

It was not possible either to explain results entirely by reference to a simple 'fluid' versus 'crystallised' distinction. Paired sample *t*-tests detected no significant differences in mean score on tests of 'fluid' ability (Block Design, Arithmetic and Matrix Reasoning) versus those tapping 'crystallised' ability (Vocabulary, Information and Comprehension) in either the depressed (t = 0.63, df = 21, p = *NS*; two-tailed test), recovered depressed (t = -0.35, df = 13, p = *NS*; two-tailed test) or normal control (t = -0.65, df = 20, p = *NS*; two-tailed test) groups. The test relating to the recovered depressed group fell just short of recommended minimum power level (0.79). However, results imply that, whilst the fluid/crystallised distinction may indeed exist as a function of "normal ageing" (although "neutralised" in the current control group by the age-scaled scoring system of the WAIS-III), it would not appear that an additive decline in fluid abilities is solely responsible for deficit observed in late-life depression.

In summary, no single framework or "theory" has proved to be of general value in understanding the nature of cognitive impairment in depression. An alternative possibility is that individual explanations are valid but obscured, on assessment, by the presence of more than one type, or profile, of depression in late life. Such a possibility has already been fleetingly referred to in relation to the recovered depressed group. Marcos, Salamero, Gutierrez, Cacalan, Gasto & Lazaro (1994), observing an adult population, found some support for the idea of heterogeneity of profile. The team identified three profiles of cognitive deficit in depression. The first comprised "attentional" difficulties (which may correspond to the profile anticipated

in the present study and observable as a function of depressed Working Memory and Processing Speed scores), a second demonstrated memory difficulties (difficult to assess in this study from the tests administered) and the third involved widespread neuropsychological dysfunction. Figure 12 indicates the heterogeneity of WAIS-III Index profile within the depressed group in the current study in relation to likely level of premorbid function. The most commonly occurring deficits are, as predicted, in the Processing Speed and Working Memory Indices but there is considerable variation and a suggestion that some individuals display generalised deficit. Such a pattern may reflect individuals with "uncomplicated depression" versus those likely to develop dementia. Only one of the 22 individuals appeared to have no deficit whatsoever.

4.14 Attention

A recurring theme in much of the research relates to the concept of "attention" and attentional deficit in depression (Brown *et al.*, 1994; Caine *et al.*, 1994; Christensen *et al.*, 1997; Lamberty & Bieliauskas, 1993) but there has been no systematic examination of the attentional demands required by experimental tasks used to assess psychological deficit in depression (Hartlage *et al.*, 1993).

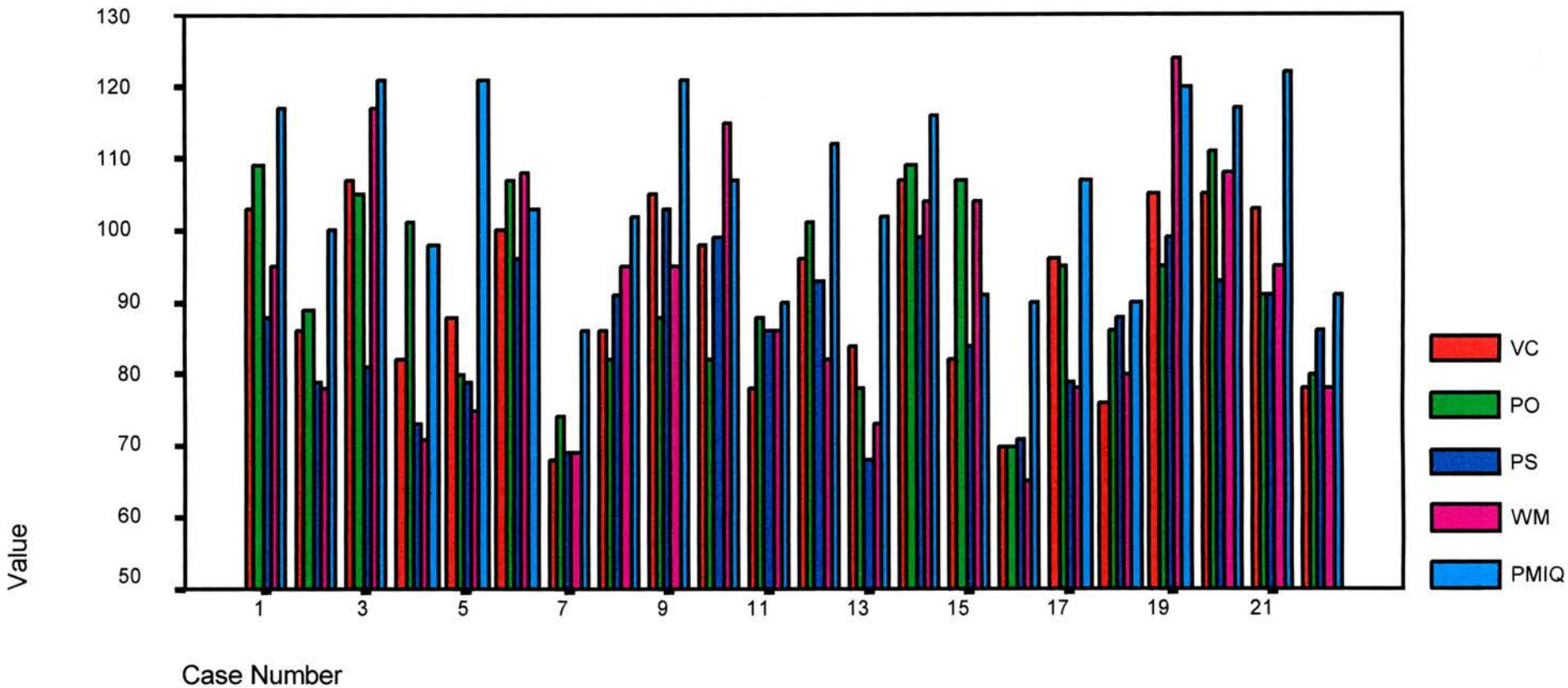
Attention remains an ill-defined concept encompassing the processes of selection, sustaining and dividing. Van Zomeren & Bower (1992) commented 'One can only assess certain aspects of human behaviour with special interest for its attentional component". Traditionally, there has been no "pure" measure of attention and it is

hard to test a priori hypotheses. An interesting development in this respect is the 'Test of Everyday Attention' (Robertson, Ward, Ridgeway & Nimmo-Smith, 1994) standardised to age 80 and designed to assess independent attentional systems serving different functions in everyday behaviour. Whilst the automatic/effortful distinction could be argued in attentional terms, it is also possible that attention has other components which have yet to be systematically researched. Anticipation is one such possibility and 'anticipatory cells' have recently been identified (Mialet, Pope & Yurgelun-Todd, 1996).

Impaired concentration is a recognised feature for the DSM-IV diagnosis of depression (American Psychiatric Association 1994) . A primary attentional deficit may have important implications for a wide range of tests and represent the effect of a single common factor producing cognitive deficit in depression. It is hard however to reconcile the extent of the decreased speed entirely in attentional terms.

The role of attention in relation to cognitive function in depression needs to be the subject of more detailed investigation. PET studies support this assertion. Significantly reduced regional blood flow in the depressed patients has been detected in the anterior cingulate cortex and the left dorsolateral prefrontal cortex using PET. These same areas have been implicated in behavioural and attentional regulation (Bench *et al.*, 1992, 1993; Philpot *et al.*, 1993; Sackeim *et al.*, 1990; Upadhyaya *et al.*, 1990).

Figure 12. Individual Index Profiles against PMIQ for Depressed Group



4.15 Conclusions and Future Directions

Research investigating the relationship between late-life depression and cognitive impairment has been hampered by a lack of widely accepted and utilised diagnostic criteria for illness and recovery, inadequate follow-up and diverse test procedures. An emphasis on attempting to distinguish between structural and motivational explanations of cognitive deficit has encouraged narrow focus and a body of research which is largely fundamentally flawed.

The current study employed a revised and cognitively broad-ranging measure which is nevertheless amenable to specific focus in its revised form and destined to become as widely used as its predecessors. Depressed older adults were clearly distinguishable from their normal control counterparts by a WAIS-III profile demonstrating diminished performance on all four Indices, notably the Processing Speed Index. This was contrary to the anticipation that deficit would be largely confined to the Processing Speed and Working Memory Indices. No single framework or theory seems able, in entirety, to account for the nature of such a profile. It is possible that progress may be made in this area by future research with a focus on aspects of attention and more systematic investigation of the effects which factors such as self-esteem or negativism might have on response patterns, particularly in the face of perceived failure.

It was not possible to offer firm conclusions from the present study as to whether cognitive deficit accompanying late-life depression is entirely reversible. Preliminary indications would suggest optimism in the majority of cases. More definitive conclusions were hampered both by sample size and the extent to which the recovered depressed group were considered representative. Some time was devoted to discussing the very salient difficulties inherent in reliably assessing the presence of illness and recovery and the delineation between them. Efforts pursuing more formal and multidimensional classification of the definition of change points according to pre-determined time-scales are essential. These would facilitate investigation of questions relating not only to reversibility but also the possibility that symptoms of depression and cognition may fluctuate and change independently.

The extent to which such a generalised profile of deficit could be depression-specific requires further investigation. Most particularly , from an older adult perspective, further research must investigate the possibility of any reliable differences in profile between depression and dementia. More fruitful however may be a focus on the formalisation and consistent measurement of some of the more anecdotal or behavioural aspects of WAIS-III test performance to enhance more traditional scores. The refining and development of diagnostic profiles may also be facilitated by future work linking WAIS-III and WMS-III data.

The ongoing interest in the issues of specific profiles and etiology of deficit have

tended to result in the broader benefit of cognitive testing being overlooked. Cognitive symptoms are not necessarily the most dominant features of depression. However, to the extent that all of the individuals participating in the current study continued to be treated on an outpatient basis and were therefore potential victims of the handicap that the extent of their cognitive deficit might place on their everyday function and adaptive behaviour, they merit greater focus. Specifically, cognitive testing is more reliable than more subjective attempts to monitor some of the other symptoms of depression. It allows predictions to made, on theoretical grounds, and confidently verified or rejected. Since it was noted that individuals found it hard themselves to monitor their progress, perhaps formal measurement should be more routinely applied for the mutual benefit and information of both client and clinician.

Another area worthy of more detailed investigation is the presence of concurrent anxiety and depression. Is the presence of concurrent anxiety simply chance yet motivationally beneficial in late-life depression, reflected in increased effort and enhanced results on cognitive testing? Alternatively, does the presence of anxiety indicate the first stages of recovery, an insight, concern and shift from passive helplessness? In either case, how much anxiety is enough and when does it become detrimental?

Finally, it is highly probable that depression in late-life does not represent a single entity. Perusal of individual profiles in the current study supported such a view. This

is hardly surprising given the heterogeneity that exists within "normality". Late-life depression might include any combination of biology, genetics, life-events, personality and temperament and the complex interaction and feedback between them. All of these indicators might be expected to reach their full maturity and potential for influence with increasing age. What is less clear is the extent to which specific combinations of factors may give rise to specific profiles of depression and the particular treatment modes which are more or less likely to be effective in defined circumstances? For the present, there are no answers, only more questions.

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APPENDIX 1

GRAMPIAN HEALTH BOARD
AND
UNIVERSITY OF ABERDEEN

GRAMPIAN RESEARCH ETHICS COMMITTEE

Chairman

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Law Lecturer
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Clerk to the Committee

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Dept of Public Health
Grampian Health Board
Summerfield House
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ABERDEEN, AB15 6RE

Tel: (01224) 663456 Ext 75225
Fax: (01224) 404014

Project No: 98/0322

23rd December 1998

Mrs L Torrens
Trainee Clinical Psychologist
'Drumtennant'
Station Road
Lhanbryde
Moray, IV30 8PY

Dear Mrs Torrens

A wais-III marker for depression in older adults

The above project was considered at the Grampian Research Ethics Sub-Committee meeting of 21st December 1998, and I am pleased to confirm that ethical approval for this project has now been granted subject to the following amendments:

1. Please give the Committee details of your supervisor.

2. Please change the Patient Information Sheets so they read;
Heading 1 "What would I have to do if I take part" and paragraph 1 should be more specific and say for example how the study will help other people.

With regards to medical indemnity, I enclose a form which should be completed and returned to either; (i) Dr J Broom, Research & Development Director, Research & Development Offices, Aberdeen Royal Hospitals NHS Trust, Aberdeen Royal Infirmary, Foresterhill, Aberdeen, (ii) Medical Director, Grampian Healthcare NHS Trust, Westholme, Woodend Hospital, Aberdeen, or (iii) Medical Director, Moray Health Services NHS Trust, 317 High Street, Elgin, as appropriate, if you wish one of the above Trusts to accept liability for medical indemnity for this project.

We would be very glad to receive, in due course, copies of any publications arising from this research. Thank you for bringing this study to the Committee's attention.

Yours sincerely

Mrs Diane Murray
Clerk to the Grampian Research Ethics Committee

GRAMPIAN HEALTH BOARD
AND
UNIVERSITY OF ABERDEEN

GRAMPIAN RESEARCH ETHICS COMMITTEE

Chairman

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Project No: 98/0322

14th January 1999

Mrs L Torrens
Trainee Clinical Psychologist
'Drumtennant'
Station Road
Lhanbryde
Moray IV30 8PY

Dear Mrs Torrens

A wais-III market for depression in older adults

Thank you for your letter of 5th January 1999. I am pleased to confirm that full ethical approval has now been granted for the above project.

Yours sincerely

Mrs Murray

Mrs Diane Murray
Clerk to Grampian Research Ethics Committee

CONSENT FORM

PARTICIPANT:.....

PRINCIPAL INVESTIGATOR: LORNA TORRENS, DEPT OF CLINICAL PSYCHOLOGY, PLUSCARDEN CLINIC, DR GRAY'S HOSPITAL, ELGIN. TEL: 01343-543131 EXTENSION 77499.

I have read the participant information sheet supplied and have had the opportunity to ask questions and discuss details with Lorna Torrens. I understand fully what is proposed to be done.

I have agreed to take part in the study as it has been outlined to me, but I understand that I am completely free to withdraw from the study or any part of the study at any time I wish and that this will not affect any continuing or future treatment in any way.

I understand that this study is part of a research project, approved by Grampian Ethics Committee, designed to promote knowledge and that it may be of no benefit to me personally. Details and data relating to the project may be inspected by the committee at any time.

I also understand that my General Practitioner may be informed that I am taking part in this study although this will be agreed with me in advance.

I hereby fully and freely consent to participate in the study which has been fully explained to me.

Signature: .....

Date: .....

I confirm that I have explained to the participant named above, the nature and purpose of the tests to be undertaken.

Signature: .....

Date: .....

APPENDIX 2

Moray
HEALTH SERVICES

Dr Gray's Hospital
Elgin, Morayshire, IV30 1SN
Tel: 01343 543131
Fax: 01343 552612

Dear

I would like to invite you to take part in a piece of research looking at how mood affects understandings and ways of thinking. The study requires the participation of healthy volunteers willing to give some time to complete questionnaires and a series of different "puzzles".

This sheet gives you a little more information about what would be involved if you decided to take part.

What would I have to do if I take part?

The study aims to look at some of the ways in which mood affects the functioning of older adults. Increasing understanding in this area has obvious value for those who are trying to help people manage and/or recover from low mood.

If you agree to take part, you will be asked to attempt a varied series of puzzles and questions with the guidance of the researcher, Lorna Torrens. This is the main part of the study and takes around an hour and a half. Most people enjoy the puzzles. It is not a test or a competition and you will not be asked to attempt tasks which you feel unable to tackle.

You will then be asked to complete 2 short questionnaires (with the help of the researcher if required) as well as answering some straightforward, non-personal questions (ie whether you are right or left handed, how many years you were at school).

The procedure will take about two hours. It may be convenient for you to call in to the Psychology Department or, alternatively, if it suits better or you would prefer it, arrangements can be made to visit you at home at your convenience.

The information I obtain will be unique to you and show various strengths and weaknesses in problem solving. Only if you wish, I will be very happy to discuss your individual results with you.

There is some possibility that you may find a lengthy session tiring. If this is the case, it will be possible to break it up. No-one would be expecting or wanting you to continue if you were tired or in any way uncomfortable.

Do I have to take part?

No. Taking part is voluntary. If you choose not to, you do not have to provide a reason and your decision not to do so will not be recorded. You may decide to take part and then

change your mind and this too is perfectly ok at any point.

What do I do now?

I will contact you within the next week or so. I will be able to answer any questions you might have and you can let me know whether or not you are interested in participating.

Thank you very much for considering taking part. Please discuss this project with any of your friends, family or GP if you want to.

Lorna Torrens
Trainee Clinical Psychologist
Clinical Psychology Dept
Dr Grays Hospital
Elgin

Moray
HEALTH SERVICES

Dr Gray's Hospital
Elgin, Morayshire, IV30 1SN
Tel: 01343 543131
Fax: 01343 552612

Dear

I am a Trainee Clinical Psychologist based at Dr Gray's Hospital, Elgin. I am currently undertaking some research investigating the effects of mood on thinking patterns in older adults. Dr ..... has suggested your name as someone who may be willing to participate in the study.

I have enclosed an information sheet so that you are able to see what is involved and I will contact you by 'phone within the next week or so to see if you are willing to participate and, if so, arrange a time to visit you.

I am very grateful for your interest.

Yours sincerely

Lorna Torrens
Trainee Clinical Psychologist

Moray
HEALTH SERVICES

Dr Gray's Hospital
Elgin, Morayshire, IV30 1SN
Tel: 01343 543131
Fax: 01343 552612

Dear

I would like to invite you to take part in a piece of research looking at your present condition and how it might be affecting your understandings and way of thinking.

This sheet gives you a little more information about what would be involved if you decided to take part.

What would I have to do if I take part?

The study aims to look at some of the ways in which mood affects the functioning of older adults. Increasing understanding in this area has obvious value for those who are trying to help people manage and/or recover from low mood.

If you agree to take part, you will be asked to attempt a varied series of puzzles and questions with the guidance of the researcher, Lorna Torrens. This is the main part of the study. Most people enjoy the puzzles. It is not a test or a competition and you will not be asked to attempt tasks which you feel unable to tackle.

You will be asked to complete 2 short questionnaires (with the help of the researcher if required) as well as answering some straightforward, non-personal questions (ie whether you are right or left handed, how many years you were at school).

All in all the procedure will take around two to three hours. You will not be expected to attend a clinic at your own expense. Appropriate arrangements will be agreed with you which may, if this suits you best, include visiting you at home at your convenience.

I would normally, as a matter of courtesy, inform your GP that you are taking part in the study. I will however seek your permission before doing so.

Are there any benefits?

The information I obtain will be unique to you and show various strengths and weaknesses in problem solving. Only if you wish, I will be very happy to discuss your individual results with you. If you are having therapy, these results may help in designing programmes most suited to your individual needs. However, information will not be discussed with or provided to any other person unless you have given your express written consent for this to happen.

Is there a down side?

There is some possibility that you may find a lengthy session tiring. If this is the case, it will be possible to break it up. No-one would be expecting or wanting you to continue if you were tired or in any way uncomfortable.

Do I have to take part?

No. Taking part is voluntary. If you choose not to, you do not have to provide a reason and your decision will not affect any treatment you might be having in any way. You may decide to take part and then change your mind and this too is perfectly ok at any point.

What do I do now?

I will contact you within the next week or so. I will be able to answer any questions you might have and you can let me know whether or not you are interested in participating.

Thank you very much for considering taking part. Please discuss this project with any of your friends, family or GP if you want to.

Lorna Torrens
Trainee Clinical Psychologist
Clinical Psychology Dept
Dr Grays Hospital
Elgin

Moray
HEALTH SERVICES

Dr Gray's Hospital
Elgin, Morayshire, IV30 1SN
Tel: 01343 543131
Fax: 01343 552612

Dear

I would like to invite you to take part in a piece of research looking at your understandings and ways of thinking.

This sheet gives you a little more information about what would be involved if you decided to take part.

What would I have to do if I take part?

The study aims to look at some of the ways in which mood affects the functioning of older adults. Increasing understanding in this area has obvious value for those who are trying to help people manage and/or recover from low mood.

If you agree to take part, you will be asked to attempt a varied series of puzzles and questions with the guidance of the researcher, Lorna Torrens. This is the main part of the study and takes about an hour and a half. Most people enjoy the puzzles. It is not a test or a competition and you will not be asked to attempt tasks which you feel unable to tackle.

You will then be asked to complete 2 short questionnaires (with the help of the researcher if required) as well as answering some straightforward, non-personal questions (ie whether you are right or left handed, how many years you were at school).

The procedure will take around two hours in total. You will not be expected to attend a clinic at your own expense. Appropriate arrangements will be agreed with you which may, if this suits you best, include visiting you at home at your convenience.

Are there any benefits?

The information I obtain will be unique to you and show various strengths and weaknesses in problem solving. Only if you wish, I will be very happy to discuss your individual results with you. Information will not be discussed with or provided to any other person unless you have given your express written consent for this to happen.

Is there a down side?

There is some possibility that you may find a lengthy session tiring. If this is the case, it will be possible to break it up. No-one would be expecting or wanting you to continue if you were tired or in any way uncomfortable.

Do I have to take part?

No. Taking part is voluntary. If you choose not to, you do not have to provide a reason and your decision will not affect any current or future treatment you might require in any way. You may decide to take part and then change your mind and this too is perfectly ok at any point.

What do I do now?

I will contact you within the next week or so. I will be able to answer any questions you might have and you can let me know whether or not you are interested in participating.

Thank you very much for considering taking part. Please discuss this project with any of your friends, family or GP if you want to.

Lorna Torrens
Trainee Clinical Psychologist
Clinical Psychology Dept
Dr Grays Hospital
Elgin

Moray
HEALTH SERVICES

Dr Gray's Hospital
Elgin, Morayshire, IV30 1SN
Tel: 01343 543131
Fax: 01343 552612

Dear

I have been approached by Lorna Torrens of the Psychology Department in Elgin who has an interest in studying low mood in older adults. She is undertaking a piece of research to look at this in more detail. She wishes to talk to a variety of people including both those who are low at present but also those who have suffered from low mood in the past but are now recovered.

I have agreed, on Lorna's behalf, to approach patients of mine who might be willing to contribute to the study. The enclosed information sheet gives you a better idea of what would be involved. Perhaps you could let me know whether you would be interested in participating using the form at the bottom of this letter. I have enclosed a stamped addressed envelope for you to return it. If you are prepared to be involved, I will pass on your contact details to Lorna so that she can get in touch with you to answer any questions you might have and arrange a time to meet you either at the surgery or in your own home.

Thank you for your help.

Yours sincerely

Dr

NAME

ADDRESS

TELEPHONE

Please delete as appropriate:

* I would be willing to be involved.

* I would prefer not to be involved.

Moray
HEALTH SERVICES

Dr Gray's Hospital
Elgin, Morayshire, IV30 1SN
Tel: 01343 543131
Fax: 01343 552612

Dear

I would like to invite you to take part in a piece of research looking at how mood affects understandings and ways of thinking. It involves the participation of both individuals who are currently experiencing low mood as well as those who have previously suffered from episodes of low mood and are now recovered.

This sheet gives you a little more information about what would be involved if you decided to take part.

What would I have to do if I take part?

The study aims to look at some of the ways in which mood affects the functioning of older adults. Increasing understanding in this area has obvious value for those who are trying to help people manage and/or recover from low mood.

If you agree to take part, you will be asked to attempt a varied series of puzzles and questions with the guidance of the researcher. This is the main part of the study. Most people enjoy the puzzles. It is not a test or a competition and you will not be asked to attempt tasks which you feel unable to tackle.

You will also be asked to complete 2 short questionnaires (with the help of the researcher if required) as well as answering some straightforward, non-personal questions (ie whether you are right or left handed, how many years you were at school).

The whole procedure will take up to two to three hours. It may be convenient for you to call in to the Surgery or, alternatively, if it suits better or you would prefer it, arrangements can be made to visit you at home at your convenience.

The information I obtain will be unique to you and show various strengths and weaknesses in problem solving. Only if you wish, I will be very happy to discuss your individual results with you.

There is some possibility that you may find a lengthy session tiring. If this is the case, it will be possible to break it up. No-one would be expecting or wanting you to continue if you were tired or in any way uncomfortable.

Do I have to take part?

No. Taking part is entirely voluntary. If you choose not to, you do not have to provide a reason and your decision not to do so will not be recorded. You may decide to take part and then change your mind and this too is perfectly ok at any point.

What do I do now?

Please return the enclosed form in the envelope provided. If you have indicated that you would be prepared to participate, you will be contacted within the next week or so. You will be able to ask any questions you might have and an appointment time, convenient to you, can be arranged.

Thank you very much for considering taking part. Please discuss this project with any of your friends, family or your GP if you want to.

Lorna Torrens
Trainee Clinical Psychologist
Clinical Psychology Dept
Dr Grays Hospital
Elgin

APPENDIX 3

National Adult Reading Test (NART)

SECOND EDITION

Answer/Record Sheet

Name:..... Date of test:.....

	Errors		Errors
CHORD		SUPERFLUOUS	
ACHE		SIMILE	
DEPOT		BANAL	
AISLE		QUADRUPED	
BOUQUET		CELLIST	
PSALM		FACADE	
CAPON		ZEALOT	
DENY		DRACHM	
NAUSEA		AEON	
DEBT		PLACEBO	
COURTEOUS		ABSTEMIOUS	
RAREFY		DETENTE	
EQUIVOCAL		IDYLL	
NAIVE		PUERPERAL	
CATACOMB		AVER	
GAOLED		GAUCHE	
THYME		TOPIARY	
HEIR		LEVIATHAN	
RADIX		BEATIFY	
ASSIGNATE		PRELATE	
HIATUS		SIDEREAL	
SUBTLE		DEMESNE	
PROCREATE		SYNCOPE	
GIST		LABILE	
GOUGE		CAMPANILE	

Obtained WAIS/WAIS-R results*:

Full scale IQ | Verbal IQ | Performance IQ

NART error score

	Predicted IQ	Predicted-obtained IQ	Abnormality (%)
Full scale IQ			
Verbal IQ			
Performance IQ			

NART + Schonell error score

	Predicted IQ	Predicted-obtained IQ	Abnormality (%)
Full scale IQ			

*** Delete as appropriate**

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NFER-NELSON
INFORMING YOUR DECISIONS

GERIATRIC DEPRESSION SCALE, YESAVAGE, BRINK, ROSE & LUM 1983

CHOOSE THE BEST ANSWER FOR HOW YOU FELT OVER THE LAST WEEK

1. Are you basically satisfied with your life?	YES/NO
2. Have you dropped out of many of your activities and interests?	YES/NO
3. Do you feel that your life is empty?	YES/NO
4. Do you often get bored?	YES/NO
5. Are you hopeful about the future?	YES/NO
6. Are you bothered by thoughts you can't get out of your head?	YES/NO
7. Are you in good spirits most of the time?	YES/NO
8. Are you afraid that something bad is going to happen to you?	YES/NO
9. Do you feel happy most of the time?	YES/NO
10. Do you often feel helpless?	YES/NO
11. Do you often get restless and fidgety?	YES/NO
12. Do you prefer to stay at home rather than going out and doing new things?	YES/NO
13. Do you frequently worry about the future?	YES/NO
14. Do you feel that you have problems with memory more than most?	YES/NO
15. Do you think its wonderful to be alive now?	YES/NO
16. Do you feel downhearted and blue?	YES/NO
17. Do you feel pretty worthless the way you are now?	YES/NO
18. Do you worry a lot about the past?	YES/NO
19. Do you find life very exciting?	YES/NO
20. Is it hard for you to get started on new projects?	YES/NO
21. Do you feel full of energy?	YES/NO
22. Do you feel that your situation is hopeless?	YES/NO
23. Do you think that most people are better off than you are?	YES/NO
24. Do you frequently get upset over little things?	YES/NO
25. Do you frequently feel like crying?	YES/NO
26. Do you have trouble concentrating?	YES/NO
27. Do you enjoy getting up in the morning?	YES/NO
28. Do you prefer to avoid social gatherings?	YES/NO
29. Is it easy for you to make decisions?	YES/NO
30. Is your mind as clear as it used to be?	YES/NO

BAI®

ME _____ DATE _____

ow is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by each ptom during the PAST WEEK, INCLUDING TODAY, by placing an X in the corresponding space in the column next to each symptom.

	NOT AT ALL	MILDLY It did not bother me much.	MODERATELY It was very unpleasant, but I could stand it.	SEVERELY I could barely stand it.
1. Numbness or tingling.				
2. Feeling hot.				
3. Wobbliness in legs.				
4. Unable to relax.				
5. Fear of the worst happening.				
6. Dizzy or lightheaded.				
7. Heart pounding or racing.				
8. Unsteady.				
9. Terrified.				
10. Nervous.				
11. Feelings of choking.				
12. Hands trembling.				
13. Shaky.				
14. Fear of losing control.				
15. Difficulty breathing.				
16. Fear of dying.				
17. Scared.				
18. Indigestion or discomfort in abdomen.				
19. Faint.				
20. Face flushed.				
21. Sweating (not due to heat).				

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18 19 20 21 22 23 24 25 A B C D E
09018425

NAME:_____ DATE:_____ 202

THE MINI-MENTAL STATE EXAMINATION

ENTATION

e one point for correct answers to each of the following questions:

t is the time?_____ date?_____ day?_____ month?_____ year?_____ 5 points ( )

t is the name of this ward?_____ the hospital?_____ the town?_____
listrict?_____ the country?_____ 5 points ( )

ISTRATION

e 3 objects. Score up to 3 points if at the first attempt, the patient repeats, in order, the 3 objects you have randomly ed. Score 2 or one if this is the number of objects he repeats correctly. Endeavour by further attempts and prompting ve all 3 repeated, so as to test recall later.

3 points ( )

ENTION AND CALCULATION

he patient to subtract 7 from 100 and then 7 from the result - repeat this 5 times, scoring one for each time a correct action is performed.

5 points ( )

ALL

or the 3 objects repeated in the registration test, scoring one for each correctly recalled.

3 points ( )

GUAGE

one point for 2 objects (a pencil and a watch) correctly named.

2 points ( )

one point if the following sentence is correctly repeated:
fs, ands or buts" 1 point ( )

3 if a 3-stage command is correctly executed, score one for each stage; for example "with the index finger of your right touch the tip of your nose and then your left ear". or "take this piece of paper in you right hand, fold it in half, and it on the floor".

3 points ( )

blank piece of paper, write: "close your eyes" and ask the patient to obey what is written. Score one point if he closes es.

1 point ( )

he patient to write a sentence. Score one if the sentence is sensible and has a verb and a subject.

1 point ( )

ruct a pair of intersecting pentagons, each side one inch long. Score one point if this is correctly copied.

1 point ( )

TOTAL SCORE (=30) ( )

Dick et al. (1984) J. Neurol. Neurosurg. Psychiatr. 47.496-9
uthors suggest that a score of 23 or less is "Suggestive of cognitive impairment". However, this is based on WAIS ores and is therefore likely to be fairly insensitive. The "Mini Mental" is also insensitive to right hemisphere damage.

APPENDIX 4

Moray
HEALTH SERVICES

Dr Gray's Hospital
Elgin, Morayshire, IV30 1SN
Tel: 01343 543131
Fax: 01343 552612

Dear

I am a final year trainee clinical psychologist. I worked as an Assistant Psychologist in the area prior to taking up my training course and will be returning to take up a currently vacant adult psychology post covering the Speyside Area when I qualify in October 1999.

Prior to this, I am required to submit a thesis. My chosen topic considers the effects of depression in older adults on cognitive processes. The project involves the administration of the "WAIS-III" - a revised and updated version of the more familiar Weschler Adult Intelligence Scale, ("WAIS-R"). Results will be interpreted by reference to measures of estimated pre-morbid IQ (National Adult Reading Test), depression, (Geriatric Depression Scale) and anxiety (Beck Anxiety Inventory). I would be happy to attend a practice meeting to explain the project in more detail if this would be of interest to you.

I would welcome your help in referring any individuals who may be willing to participate in my research as experimental subjects. Such individuals, all over 60 and preferably over 65, will fall broadly into two groups:

1. Currently depressed with initial episode of "late onset" depression ie onset after age 60 rather than lifelong history of depression.

2. "Recovered" depressed ie on the verge of discharge or on longer term review as medication is reduced. Again, onset of depression should have been in late life.

Individuals who are known to suffering from dementia or have a history of serious vascular problems would not be suitable. The extent of sensory loss should not be to a degree where it might interfere with the obtaining of valid results on paper and pencil, listening and speaking tasks.

Whilst the study is ethically approved, there is clearly some dilemma around the issue of expecting participation in a psychological research project from currently depressed subjects to whom psychological treatment has not been offered. It may, in this instance, be possible to offer a limited number of treatment sessions to a few individuals for whom a psychological approach might be considered suitable and welcome.

I very much appreciate any help you are able to give with the referral of suitable individuals. Obviously I have information sheets explaining what participation would involve for any individuals who might be considering taking part. Please do not hesitate to contact me if you require any further information.

Yours sincerely

Lorna Torrens
Trainee Clinical Psychologist

Juliana C MacLeod
Consultant Clinical Psychologist

Moray
HEALTH SERVICES

Dr Gray's Hospital
Elgin, Morayshire, IV30 1SN
Tel: 01343 543131
Fax: 01343 552612

Dear Dr

This is simply to inform you that ..... has agreed to participate in a research project investigating the cognitive effects of depression in older adults.

The project focuses on the administration of the "WAIS-III" - a revised and updated version of the more familiar Weschler Adult Intelligence Scale ("WAIS-R"). Results will be interpreted by reference to measures of estimated pre-morbid IQ (National Adult Reading Test), depression (Geriatric Depression Scale) and anxiety (Beck Anxiety Inventory).

The process is clearly non-invasive and should take about 2 hours in total. ..... has agreed to your being informed of their participation. Please contact me if you have any queries regarding the project.

Yours sincerely

Lorna Torrens
Trainee Clinical Psychologist

Moray
HEALTH SERVICES

Dr Gray's Hospital
Elgin, Morayshire, IV30 1SN
Tel: 01343 543131
Fax: 01343 552612

Dear

Further to our telephone conversation, I enclose, as discussed, a brief outline of the research project I am currently undertaking. As you know, ..., has indicated that your patient, ..., may be a suitable participant in the ..... experimental group. However, since they are not currently under .....'s care, I would be grateful for your confirmation that it is appropriate to approach ..... and that you have no objection to my doing so. I will telephone you within the next week to confirm this.

If you have any further queries regarding the project, I would be very happy to discuss them with you and am willing to attend practice meetings, if required, to describe the project in more detail. I would also be grateful if you would bear the study in mind and, should you consider any of your other patients suitable to participate, I would be very happy to receive their contact details.

With many thanks for your interest.

Yours sincerely

Lorna Torrens
Trainee Clinical Psychologist

Enc

A WAIS-III MARKER FOR DEPRESSION IN OLDER ADULTS?

INTRODUCTION

Depression occurs in up to 15% of individuals aged 65 and over resident in the community (1). It is a disabling condition associated with increased mortality both by suicide (2) and natural causes (3). The proportion of the population aged over 65 is steadily increasing (4). As it does, so too will the size of the clinical problem of depression in older adulthood.

Depression in older adults is not always optimally managed. It is under-detected, misdiagnosed and sometimes mistreated (5,6). Traditionally, there has been a reluctance to intervene. This may stem from the misconception that depression is an inevitable consequence of ageing, (7), a fear of the side-effects from anti-depressant medication in older people and low expectations regarding treatment success (8).

There is general agreement that impaired cognitive performance is demonstrable in depressed older adults (9). The origins of such deficits have traditionally been considered motivational, representative of decreased attention and a narrowing of attention specific to depressive content (10).

More recently, researchers have begun to investigate the possibility that impaired cognitive performance in depression may represent structural changes in the brain. Early work implicated right, then non-dominant hemispheric dysfunction (11). The advent of scanning procedures has leant increased sophistication to this work and abnormalities including ventricular enlargement, (12) cortical atrophy, (13), white and grey matter abnormalities (14, 15) and reduced cerebral blood flow (16) have been reported in the brains of depressed older adults.

Methodologies however have been poorly controlled and variable (17). The extent to which identified deficits are depression specific and whether and at what point they impact on cognitive performance remains unclear. If structural changes are in fact responsible for the cognitive deficit noted in late-life depression, it may be that impaired cognition persists even when symptoms of depression resolve. Evidence on this point is also ambiguous (18, 19).

Furthermore, there has been wide variation in the measures used to identify cognitive impairment in depression (20) and some debate regarding the relevance of a distinction between "late-onset" and life-long depression (21).

The WAIS-R has been the most commonly used measure of cognitive performance (22). However, the fact that most of the subtests in the WAIS-R are multi-factorial adds to the difficulty in partialing out any specific cognitive skills which may be affected by structural

changes accompanying depression in older adults.

The WAIS-R has recently been revised and updated resulting in the production of the WAIS-III (23). This new test has been standardised in the US to age 89 and its usage with older subjects has been facilitated. A four-factor model including Perceptual Organisation, Verbal Comprehension, Processing Speed and Working Memory has emerged. These last two factors may prove diagnostically important in late-life depression by their relation to learning acquisition and the possibility of separating them from Verbal Comprehension and Perceptual Organisation.

The current study seeks to investigate the utility of the WAIS-III in the measurement of cognitive deficits in older adults who are depressed by comparing their cognitive profiles to a group of individuals who have never been depressed and a further group who were previously depressed but now recovered.

It is hypothesised that the cognitive deficit observed in depression is mood-state dependent and therefore reversible on recovery.

The present study will comprise two experimental groups (depressed and recovered depressed) and a control group. All participants will satisfy the following inclusion and exclusion criteria.

INCLUSION CRITERIA	Able to give written informed consent English as a native language Adequate hearing, vision and mobility to undergo testing without handicap relating to these dimensions Aged between 60 and 89 years
EXCLUSION CRITERIA	Reported history of or exhibition of psychotic features Evidence of organic pathology as assessed on observation, testing, physician report or score <23 on Mini-Mental State Examination History of Alcoholism or illicit drug usage ECT within the last year Diagnosis of manic depression Significant vascular disorder

Individuals in the experimental groups will satisfy the following inclusion criteria

DEPRESSED GROUP	Apparently "late-onset" depression determined by initial presentation at 60 years or more of age A primary diagnosis of major depression made by the referrer and supported by clinical judgement and semi-structured interview, with reference to DSM-IV criteria at time of testing GDS score exceeding 10 at time of testing
RECOVERED DEPRESSED GROUP	Apparently "late-onset" depression determined by initial presentation at 60 years or more of age Previous primary diagnosis of major depression made by the referrer Failure, at time of testing, to meet criteria for the diagnosis of major depression on the basis of clinical judgement and semi-structured interview with reference to DSM-IV criteria GDS score less than 11 at time of testing

Participants will be recruited according to the criteria above. The assessment procedure will comprise:

1. A short engagement period during which subjective views will be sought regarding the presence of the symptoms of depression and demographic data will be established.

2. Administration of measures of cognition as follows:

a) The Mini-Mental State Examination (24) will be administered only in those cases where impression during interview or subsequent test performance suggests the possibility of the presence of dementia. The measure comprises a brief, simple, mental status questionnaire addressing areas of cognition such as orientation, personal and current information, memory, apraxia and language.

b) The WAIS-III as described above.

c) The National Adult Reading Test (NART) (25). The NART consists of 50 words which subjects have to read aloud and pronounce. The stimulus words are largely short and selected for their irregular pronunciation so that success is attributed more to familiarity than current cognitive function. The NART has proved a valid measure of previous intelligence in depressed patients (26). It will be utilised in the current study to match, if possible, participating groups in terms of their likely premorbid level of function and also to facilitate an estimate of the likely magnitude of any decline observed in individual cases.

3. Administration of measures of affect as follows:

a) The Geriatric Depression Scale (GDS) (27). The measure comprises a self-completed rating scale, specifically for elderly subjects, which can also be completed by interview. The GDS consists of 30 Yes/No response Questions. A score of 11 or above is indicative of the presence of depression.

b) The Beck Anxiety Inventory (BAI) (28). The measure consists of 21 descriptive statements of anxiety symptoms which are rated on a four point scale from "Not at all" through to "Severe". It measures the severity of self-reported anxiety. It has been suggested that anxiety is associated with low mood, that it can have specific effects on attention and that particular personality factors can make individuals more prone to anxiety resulting in apparent cognitive impairment (29). The BAI is therefore employed in this study as a broad screen for presence of high anxiety which may be confounding the attribution of results to depression.

Written consent will be obtained from participants indicating their willingness to contribute to the study. Individual results will not be discussed with participants and assessment reports will be produced for GPs only where requested and with participant agreement. Confidentiality of participants is assured.

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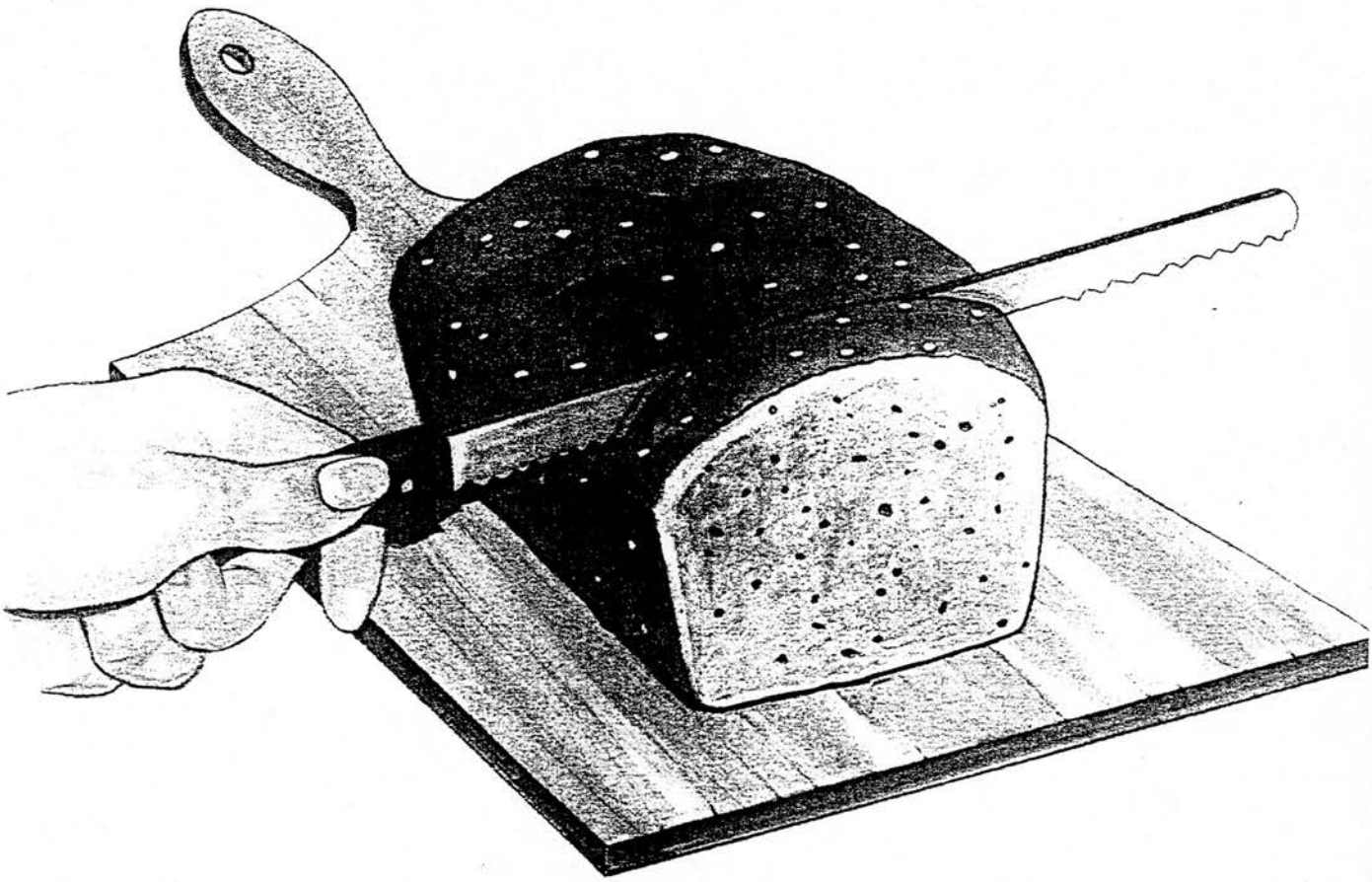
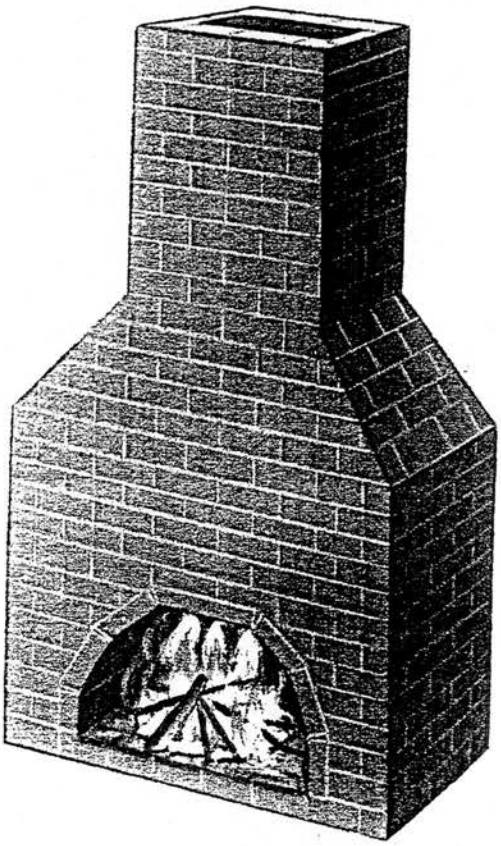
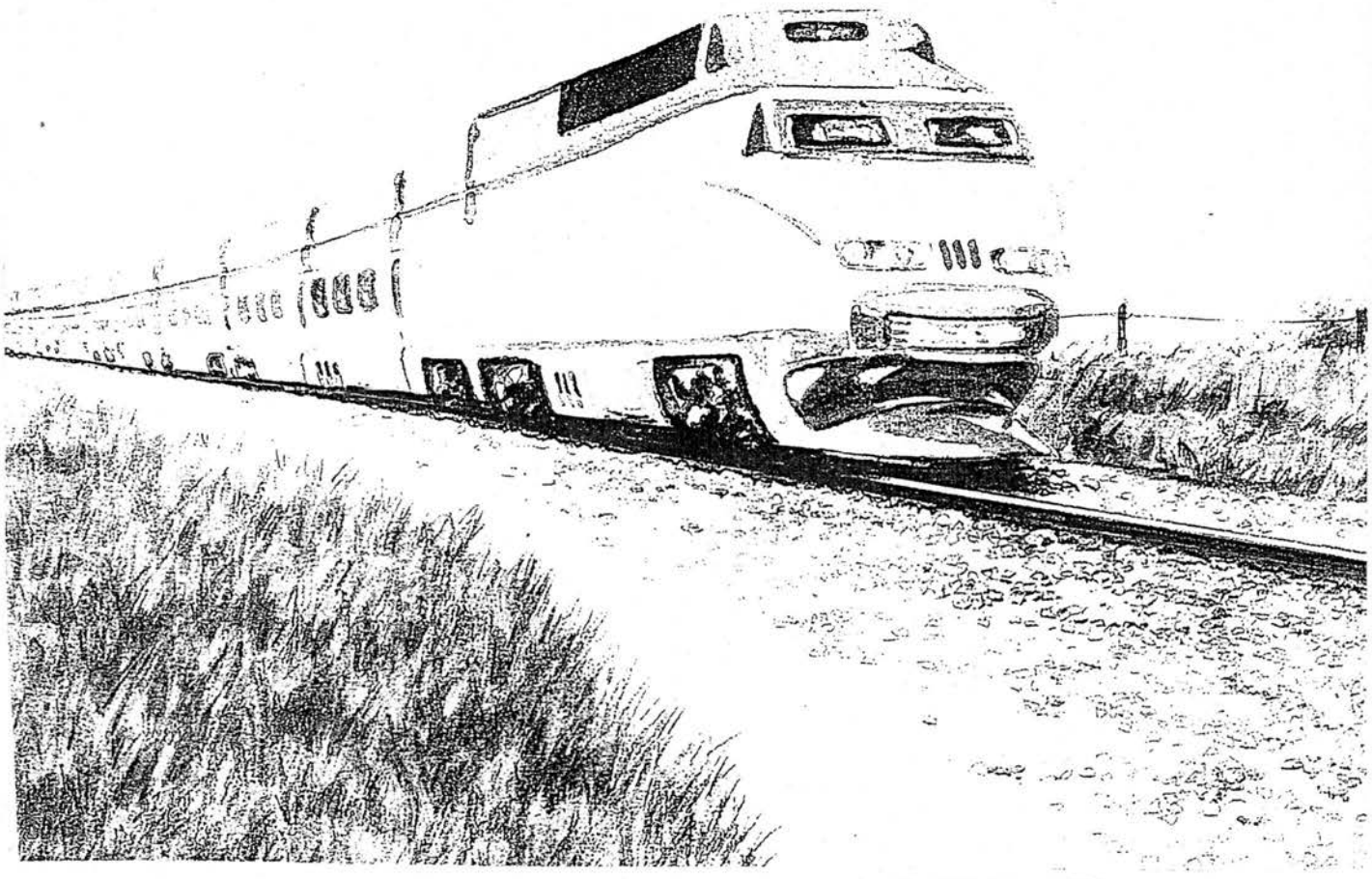
APPENDIX 5







DESIGN 11

DESIGN 7