DEPRESSION ACROSS EARLY, MIDDLE AND LATE ADULTHOOD

A thesis submitted for the degree of

Doctor of Psychology (Clinical)

by

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Declaration of Reprint

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Chapter 1

Introduction to Doctorate

This introduction will provide a context for, and a brief overview of, the main themes contained within the thesis. The Doctoral Programme at City University is comprised of four sections, namely, an Introduction (Chapter 1), a Case Study (Chapter 2), Research (Chapter 3), and a Critical Review (Chapter 4).

In 1999, The Clinical Standards Advisory Group for the Department of Health, UK, noted that

> The World Bank recently assessed depression as the fourth most common cause of disability days lost world wide, including the developing world, and predicted that it would rise to second place by the year 2020.... The World Health Organisation recently rated depression as one of the top three leading causes of disability... and mortality for men and women, in The Americas and Europe.' (Clinical Standards Advisory Group [CSAG], Department of Health, 1999, p. 5).

In the category of unipolar depression alone, it is estimated that as many as 15% of people commit suicide (CSAG, DoH, 1999).

Studies done in the late 1980's (Croft-Jeffreys & Wilkson, 1989; Stoudemire, Frank, Hedermak, Damlet) have estimated that the direct and indirect economic cost of depressive illness in the USA is around $16 billion per year, and in the UK neurotic illness (which includes depression) around £373 million per year. With high prevalence rates and enormous monetary costs, this disorder warrants ongoing investigation as it not only impacts so profoundly on one's quality of life (estimated to be a greater impairment than many physical conditions), but also on society in terms of its impact on significant others, cost of treatment and loss of productivity through sickness leave (CSAG, DoH, 1999; Liaison in Community Psychiatry [LiNC UP], 1995).

The advances in precision and categorisation of diagnoses brought about by the Diagnostic and Statistical Manual (DSM) of the American Psychiatric Association and the International Classification of Diseases and Related Health Problems (ICD) of the World Health Organisation, with their continual reworking and updating, has certainly helped to streamline and refine both the definition of depression and its degrees of severity, and its symptomatology (Geiselmann, Linden, & Helmchen, 2001; Zarit & Zarit, 1984; 1998).

Depression is a commonly used lay term and using it loosely can lead to much conceptual confusion. In the lay sense, 'depression' is similar to the term 'nervous breakdown', as it can refer to a range of experiences. Depressed mood, in clinical terms, is only one manifestation of a depressive illness. It is not a necessary or sufficient indication of a clinical illness, as this requires the presence of a syndrome of clinical features and a significant level of functional impairment (DSM-IV, 1994; ICD-10, 1994). The DSM-IV (APA, 1994) allows for the presence of irritability or anhedonia (particularly relevant for older adults) in the absence of low mood (Baldwin, 1998).

The area of depression has received a great deal of exposure over the past few decades whether it be through scientific, academic, clinical or popular media sources. However, in spite of the volume of work published on it, it is still an area that attracts new research and ongoing development. Perhaps one reason for this is that it is the type of condition that can exist on its own, but also frequently co-exists with, or is a result of, other conditions (such as anxiety or dementia) thus permeating into many different areas (DSM-IV, 1994; ICD-10, 1994).

The lifetime prevalence of depression has been estimated (Kaplan, Sadock, & Grebb, 1994; Weissman, 1981) to be between 8% – 12% for men and as much as 20% – 25% for women in the adult population. Reviews of community studies by Blazer (1997) in the UK and Steffens et al. (2000) in the USA, found the frequency of clinically significant depressive symptomatology to be much higher than the occurrence of Major Depressive Disorder (MDD). These community studies reflect a prevalence of between 9% and 14.7% for 'subsyndromal' or minor depressions with clinically significant depressive symptoms.

The onset of depression can begin in childhood (Reinherz, Giaconia, Hauf, Wasserman, & Silverman, 1999) and it is now thought that the risk of developing depression is highest between the ages of 15 – 19 and 25 –29 (Burke, Burke, Regier, & Rae, 1990). There is some evidence (Reinherz et al., 1999) that females are three times more likely to experience the onset of depression by the age of 14 than males. Traditionally, the emphasis in the study of depression seems to have been on the adult population i.e. those between the ages of 16 – 65. However, it is now being viewed as a disorder that can occur at any age or life period, and the more recent research (Blanchard, 1997; Jorm, 2000; Pitt, 1997; Steffens et al., 2000; Twining, 1998) suggests that the prevalence, development, manifestation, or treatment response of depression in old age does not differ in any way from depression in the general population. Therapeutic interventions with older adults is a dynamic area, and as we are an ageing population this group will require an increased amount of attention and care. Erikson's (1978) theory gives weight to the debate that age is not simply a degenerative process but a full developmental cycle that can be viewed as a stage of life.

The impact of highly stressful and arousing events on an individual's psychic and psychological state has long been considered an area of interest, and has often been linked to

depression (Bowlby, 1977, 1980; Crittende, 1990; LeDoux, 1992; Miller, Kritman, & Ingham, 1989; Paykel, 1994; Perry, 1999; van der Kolk, McFarlane, & Weisaeth, 1996), and Janet's work in the 1800's, frequently referred to in the work of van der Kolk (1996), is a key resource to address what we now commonly refer to as 'trauma'.

Cognitive theorists, such as Beck (1967), Ingram (1984) and Teasdale (1988) have considered how depression and life events influence cognitive processes and how such processes, in turn, influence our psychological state. Research by Kuyken & Brewin (1994) and others (Brewin, Hunter, Carroll, & Tata, 1996; Brewin, Reynolds, & Tata, 1999) has extended this concept and sought to investigate autobiographical memories, trauma and depression. A further dimension of this area of research that has opened up an invaluable sphere is that of the impact of trauma on our physiological state, as well as the psychological one (LeDoux, 1992; McFarlane & Yehuda, 1996; Perry, 1999; van der Kolk, 1996), and these findings are forming the start of a new collaboration between professionals across a broad range of disciplines.

The following discourse is an attempt to view depression over the life period from late adolescence and early adulthood to older adulthood. It aims to draw together some of the more recent research and views on these complex matters, and to contribute to the pool of scientific knowledge on it, and to the clinical dimension that is constantly striving to enhance its means of intervention.

Chapter 2

This section is comprised of a clinical study of treatment with a young adult whose presentation was sufficiently complex to cause a division in opinion between professionals on

diagnosis, and whose depression was overlooked, as he did not present with the more typical features and diagnostic criteria of the illness. The case also reflected the responses of professionals to ambiguous clinical features, as well as the difficulties therapists encounter in making decisions regarding continuation of treatment when the measurements of success are so ambiguous and difficult to define.

Chapter 3

The aim of the empirical research was to determine whether an adult population of clinically depressed, recovering depressed and never depressed individuals had experienced an incident that they perceived as traumatic, and whether such an experience would influence their ability to forget material, particularly negative material, when directed to do so. Some findings were contrary to those anticipated, whilst others provided insight into the cognitive processes of individuals and how these might have implications for future therapeutic interventions.

Chapter 4

This concluding section of the thesis provides a critical review of the current literature related to older age and depression. A debate ensues around the domains of whether it is worthy of recognition and diagnosis, and whether or not psychological interventions would have any impact on individuals who are, according to dominant cultural assumptions, entrenched in their ways. One particularly concerning matter discussed is how professionals themselves tend to dismiss, overlook or be pessimistic about the possibility of treating depression in older adults.

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Chapter 2

Early Intervention & the Difficulties Around Ambiguous Presentation

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1. Rationale

This case study is presented as an example of the difficulties a therapist encountered when dealing with a young adult whose clinical presentation was unclear and ambiguous and who was reluctant to engage in therapeutic interventions.

2. Reason for Referral

Abdul (1) was a nineteen year old Asian man who was in a psychiatric inpatient rehabilitation unit and was being cared for by a multi-disciplinary team, of which I was a part. It was at his Care Programme Approach (CPA) that I first met Abdul who had been readmitted to the unit. The meeting consisted of Abdul, his parents and the multi-disciplinary team. It soon became apparent that there was conflict between the parents and their son, and this was confirmed at the second CPA that took place four weeks later. He was very withdrawn, monosyllabic in his responses and unwilling to engage in much verbal contact. The team and myself were in agreement that psychological intervention may assist him in expressing his feelings and to address any concerns that he may wish to discuss, such as his low mood, family relationships and the reasons for his being in the unit.

3. Background Information

Background information on Abdul was obtained in a fragmented manner, as both he and his family were reluctant to share personal material, except around factual matters, such as his schooling. He was the middle of three children with a brother 6 years older than himself who was successful in his career, and a sister 5 years younger who was still at school. It was

(1) The patient's name and identifying details have been changed in order to protect his identity.

reported that he had good relationships with them. His father, aged 70, had a prosperous business, and his wife, aged 60, had divided her time between assisting in the business and taking care of the children and home.

At the age of three, Abdul was sent to Pakistan to live with relatives for three years during which time he had no direct contact with his parents. The reason for this separation remained unclear, and it is significant that this only emerged during the course of my intervention. It also transpired that his father had previously been married and had two children whom Abdul had never met and who were never referred to.

Abdul had attended a Roman Catholic school as both his parents were of that faith, and he described his school years as "fine". He obtained 10 GCSEs with good grades and went on to do his A Levels. However, he dropped out before completing the first year and he had never been employed. His social and interpersonal skills were appropriate, and his general functioning and understanding were of above average ability.

The team were informed that the family were attending family therapy, but neither Abdul nor his parents would provide information about it except that they had been referred two years prior because of the patient's behaviour (i.e. his parents reported that he was difficult and unmotivated), and that they discussed some family issues. The younger sister was excluded from the sessions as the parents felt "she was too young".

Five months prior to my meeting Abdul he had been admitted to an acute mental health ward under Section 2 of the Mental Health Act (1983) after displaying what was described as bizarre behaviour. In the past year he had broken his brother's computer, smashed the side

mirror on his father's car, and killed his mother's pot plants. More recently, he was agitated with poor eating and sleeping patterns, and he had put his fist through a window. It was this incident that prompted his father to take him to hospital for an assessment. According to his family, there had been a gradual increase of what they referred to as their sons "strange behaviour" over the past three years culminating in the events described above.

The admitting doctor noted that Abdul's mental state on admission was difficult to assess as he was ambiguous and unresponsive in his answers. He was orientated to time, place and person, and no evidence of delusions or hallucinations were found. However, he would smile inappropriately and make the occasional comment such as "I cannot breath through my sex organs". Abdul remained on an acute ward for a 4-week period before being transferred as an informal patient to the rehabilitation unit. He remained there for two months and was discharged. A few weeks later his father took him to Pakistan and, according to his father, he began to "act strangely" and say 'silly' things. No concrete evidence of what this meant could be ascertained only that he would be vague and laugh inappropriately, so his father returned to the UK and Abdul was re-admitted to the rehabilitation unit, which is where I met him.

4. Assessment

This assessment was based on my initial interview, my participation in CPA and team meetings, and consulting his case notes. Abdul presented as a physically well-developed and polite young man who was withdrawn and uncommunicative. During the initial assessment he would make eye contact, but offered very little information. Most questions were responded to with "I don't know", or he would wait a long period of time, ask me to repeat the question, only to say "I don't know" or "I can't agree with you". He was not impolite or

discourteous, but he would stare at me, smile and respond in a disinterested, slightly mocking and challenging manner. Surprisingly, when asked if he wanted to engage in individual psychotherapy he said that he did, but when this was pursued all he would say was "I'll try it" or "ok". On the occasions when he answered in sentences, and based on his responses in the CPAs, it was obvious that he was a young man of average or above intelligence whose language skills and comprehension were competent and who could express himself with no difficulty when he chose to.

He denied any personal or family problems but I observed in the CPAs that his father would interrupt when his son was speaking or state that if his son did not co-operate with everything we suggested then he would disown him. His parents expressed anger and frustration with him, and they frequently became openly rejecting and critical of him. His reaction to this would be to remain silent and distant in a passive way.

He reported that he had had no relationship, heterosexual or homosexual, and he noted that he had no friends. His explanation of this was that he lacked confidence, he was shy and he had nothing much to offer in conversation. Most of his interactions were with his immediate and extended family or family friends, which would not be an uncommon occurrence within his cultural context. When this was explored with him, he only partly agreed and noted that both his siblings had friends outside of the family context, and that his brother's girlfriend was not from their circle. Initially, he said this was "fine" for him, but it later emerged that having friends and a girlfriend was his greatest wish.

Abdul remarked that he had used cannabis since the age of 15 but the quantity and frequency were unclear as he was non-committal. The possibility existed that he wished to be seen as

having used a large quantity of drugs which may have been motivated by a rebellion against his parents or wanting to establish credibility with his peers.

5. Diagnostic Uncertainty

There was much debate around Abdul's diagnosis. He had originally been given a diagnosis of Simple Schizophrenia (Diagnostic and Statistical Manual [DSM-IV], 1994) and later a differential diagnosis of a Drug Induced Psychosis (DSM-IV, 1994). There was no evidence of positive psychotic phenomena, the nursing staff on the hospital ward and in the rehabilitation unit had not reported any first rank symptoms or behavioural disturbances, and his beliefs, thought processes, level of functioning and interactions were stable and coherent. However, he may have been presenting with early signs of a psychotic illness, such as depressed mood, restlessness and social withdrawal, that are often mixed and difficult to categorise (Birchwood & Tarrier, 1994; Initiative to Reduce the Impact of Schizophrenia [IRIS], 1999). These factors created a division in the team regarding his diagnosis, with some opting for the diagnosis of simple schizophrenia and others believing there was a stronger affective component coupled with personality traits mainly of the passive-aggressive, dependent and avoidant types (DSM-IV, 1994). There was agreement that a mood component was present which required intervention, and an acknowledgement that the team would be required to tolerate and work with the uncertainty until a clearer picture emerged.

The Beck Depression Inventory (Beck, Rush, Shaw, & Emery, 1979) was administered and he obtained a score of 48 that fell within the severely depressed range. (He refused to complete an end of treatment inventory so no comparative score is available). The validity of his responses on this measure was questionable as he was observed to approach the task in a

disinterested manner. However, one could not ignore his high score and he was regarded as a potentially high suicide risk for he was young, male, socially isolated, unemployed, he had no friends or peer group, his relationship with his parents was poor and he lacked confidence in himself (Centre for Clinical Outcomes, Research and Effectiveness [CORE], 1998). When asked about feelings of depression or suicidal thoughts he always denied any presence of them.

6. Formulation

Abdul showed no evidence of first rank psychotic symptoms except for his unusual comments at times (such as, "my blood is cold"), there were no observations or reports of him responding to auditory or visual hallucinations or being preoccupied or distracted, and there was no evidence of his being agitated or aggressive. His insight appeared to be limited, though at times there was evidence of a greater understanding of himself. His view of himself and his belief in his abilities were poor, he was lacking in confidence and he perceived the world to be a critical and unsupportive environment.

His expressed range of emotions was restricted, his mood appeared low and his communications were devoid of detail. His interactions with others were always controlled and appropriate, and he presented as quiet and withdrawn. He had been socially and academically primed but it appeared that his emotional and sexual development had lagged behind (Bowlby, 1977, 1980; Bretherton, 1990; Crittende, 1990; Libet & Lewinsohn, 1973).

Abdul was in a conflictual relationship with his parents that appeared to include adolescent issues, such as difficulty in individuating from his parents (Bowlby, 1977, 1980; Erikson, 1963, 1968). Observation of family interactions revealed that both parents were authoritarian

and rigid in their approach, and unrealistic in their expectations of their son. For example, they expected him to be engaged in full-time employment on discharge from the unit. He seemed unable to verbally express his thoughts and feelings around this conflict so he was using dysfunctional means by which to relate them, namely through physically damaging his family's property and assuming a passive and non-verbal stance. This, he acknowledged, evoked annoyance and frustration in his parents.

The working hypothesis was that underlying his objectively depressed and withdrawn demeanour was anger and sadness. His family system had disallowed him to individuate or to develop a firm sense of his individual identity (Erikson, 1963; 1968). He had few positive beliefs about himself, and himself in relation to others, which research has shown to be contributing factors in depression (Beck, 1976; Bowlby, 1969, 1977, 1980; Crittende, 1990; Gerlsma, Das, & Emmelkamp, 1993; Main, Kaplan, & Cassidy, 1985; Stern, 1991). The strained and critical relationship with his parents, and his social isolation, meant that he had no positive or rewarding response from his environment and he lacked a confiding relationship with another adult (Lewinsohn, Weinstein, & Alpere, 1970; Williams, 1992). This, combined with his being unemployed and having a low level of self-esteem, were all vulnerability factors for developing a severe mental illness and being regarded as a high suicide risk (Brown, 1986; Brown & Harris, 1978; CORE, 1998; Gut, 1989; Kaplan, Sadock, & Grebb, 1994; Osipow & Fitzgerald, 1993; Rippere, 1995).

Although his diagnosis was unclear, a mood component appeared to be present, whether it was within a context of a psychotic illness or an affective one. There was the possibility that the psychotic-like symptoms were transient (through the use of drugs), or were the early signs of a psychotic illness. There was also the possibility that his symptoms were being used in a

manner that would allow him to utilise the 'mental illness' label as a means of avoiding dealing with his feelings of depression and anger (Salzberger-Wittenberg, 1970). These varying hypotheses mitigated against any precise formulation, but the case did highlight the type of uncertainty and complexity that is encountered in this more specialist area of work.

7. Therapeutic Considerations

Abdul was lacking in motivation, he was unwilling to engage or communicate in a manner that was conducive to exploring issues, he denied any depressed feelings or problem areas, and he showed limited insight into his situation. An approach based on the IRIS model for early intervention in psychosis (1999) was used. This model (i.e. Initiatives to Reduce the Impact of Schizophrenia [IRIS], 1999) is a 'working' model based on the principles of a therapeutic approach rather than on a specific theory of mind (such as Beck, 1967). It focuses on developing a broad therapeutic alliance with the client (and family, if appropriate), who is presenting with prodromal or first rank symptoms of a psychotic illness either for the first time, or who has had only a few episodes of the illness in the past. The therapeutic approaches suggested include, for example, suggestions on maintaining continued contact with difficult to engage clients, supporting family members, addressing treatment compliance issues, discussions on the use and compliance with medication, and so forth, rather than on intervention techniques (such as identifying negative cognition and automatic thoughts). It was developed by clinicians and researches working in the field of psychotic illnesses and was derived from their experiences with clients, families and services. It accommodates ambiguous presentations, and it emphasises both the importance of ongoing attempts to engage the patient and the development of a gentle, non-confrontational therapeutic relationship. In addition, the principles of intervention allow the therapist to work in a less structured and more flexible manner, without the constraints of a set model of working (IRIS,

1999). Early intervention work for both psychosis and depression (whether part of a prodromal syndrome or a disorder in its own right) could also assist in reducing the negative impact of such disorders (IRIS, 1999). A further motivation for offering therapy was to use the therapeutic relationship in a positive and adaptive manner (IRIS, 1999; Linehan, 1993; Padesky, 1994; Raue & Goldfield, 1994; Safron & Muran, 1995) for his developing a relationship with someone outside of the family unit could be beneficial in its own right (Baker, 1993; Jones & Pulos, 1993).

8. Treatment Plan

The client was given weekly sessions at the rehabilitation unit. The frequency and length of the sessions were adjusted according to his needs and capacity to sustain engagement. Ongoing discussion, observation and feedback between myself and the multi-disciplinary team were required due to the nature of the unit.

9. Intervention

Each week Abdul would arrive for his session being polite and 'compliant'. However, once he sat down in the chair he assumed his non-verbal, non-committal persona. He would stare at me in silence with a face that showed no emotion, give unashamed and large yawns whilst continuing to look at me, and refuse to say anything more than "I don't know" or "fine". He never expressed a wish to terminate the sessions, nor did he refuse to attend or leave the unit prior to them. Attempts to explore issues with him were thwarted and it soon began to feel as if he was testing me at every turn. His determined and mocking passivity and his avoidance of dealing with anything was unremitting

What was particularly concerning was his unquestioning keenness to accept the label of being mentally ill for it seemed as if it gave him the unfortunate opportunity to remain passive and disengaged. It also gave him the chance to indirectly express his anger with his parents and to curtail their criticisms of him by blaming his behaviour on his 'illness'. On the occasions when I would comment on his behaviour he would look me in the eye and say "It's my illness" in order to test my response. It seemed that Abdul was engaging with me as he did with his parents, and that he perceived therapy as a battle of wills. In psychodynamic terms, his response was one of transference (Salzberger-Wittenberg, 1970) and my response to this difficult situation required self-reflection and thought (Salzberger-Wittenberg, 1970) for his passive-aggressive stance sometimes evoked enormous irritation in me. Consequently, I was cautious so as not to make sharp comments or treat him (in an unhelpful way) like a rebellious child.

At the end of every session, regardless of its content or process, he would unfailingly say something along the lines of "thanks, it was nice talking to you". At first I thought this might be indicative of positive change, but my optimism was soon replaced with a feeling of uneasiness that he might well be mocking me. I sometimes wondered whether his coming for therapy was part of a plan - he would comply with everything, assume the role of being mentally ill, enjoy being provided for and thereby not have to change.

I soon began to shift the therapeutic emphasis and to take a more reflective approach by interspersing the stares, silences and yawns with comments on what I thought might be taking place for him. This shift seemed necessary and appropriate as a means of moving away from the sessions becoming a 'battle of wills' and as a way of providing a more conducive

situation if we were to address the very thing he so much wanted but did not know how to obtain - a relationship.

This approach, over time, began to elicit a few more responses from him and he would, on occasions, tell me if he agreed or disagreed with me. I would reflect on his wanting me to do the work in the sessions, that his refusal/reluctance to engage in any communications that extended beyond a phrase or sentence was a way of stopping me from getting to know him, but also a way of protecting himself from criticism, and that his half-smiles were a way of letting me know that he was listening even if he was not responding.

On one occasion, after about three months of his remarkable ability to continue in the same non-committal, unemotional and supposedly disengaged manner, having asked a question and whilst waiting for an anticipated response I said "It's not a trick question". For the first time, in spite of his efforts to contain himself, his façade was momentarily cracked and he laughed spontaneously. For a few minutes thereafter he continued to laugh in a genuine and warm manner. I too laughed and the last few minutes of the session were spent in silence with him smiling and laughing at himself. I think it was this incident that brought about a major shift in our therapeutic relationship.

His determination to not engage in any reciprocal 'conversation' was unremitting, and his responses were, by this stage, completely predictable, but what was different was that he could now come into the sessions and spontaneously start them by giving me a few facts about what had taken place during the week. At times he would even comment on how he felt about them. His willingness to agree or disagree with my reflections improved and on occasions he would contribute to them. He occasionally expressed insight into his behaviour

and wishes, notably around his wanting a girlfriend and friends and, over time, he felt more able to discuss his increasing desire to live away from the family home. He was also more verbal in his complaints about being tired and disinterested in activities or work schemes, and he was eager to let me know that his only wishes were to be rich, to have a girlfriend, to be allowed to sleep all day and to get his own flat. These symptoms are common to depression (DSM-IV, 1994) and early signs of psychosis (IRIS, 1999) but they are also common and typical adolescent responses (Erikson, 1968).

The staffs' observation of him outside of the sessions revealed a part of him that was being more communicative and engaged than had previously been noted. He had formed a strong attachment to the unit's chef; he would chat to him, go shopping with him and help him in the kitchen (though he insisted on telling me that he only did it because he had to, and that it meant nothing).

Continuing to engage with him was not always an easy task but there did seem to be a value in my accepting and working with what seemed like adolescent defiance and opposition, and his emotional immaturity. His passivity was being discussed and challenged but was not being responded to in a critical or rejecting manner. The aim was to be able to think about it and address it. It frequently felt as if he wanted to push me to the point where I too would criticise and reject him, so that he could then make me responsible for the ending of therapy and repeat what he seemed to have experienced at home (as referred to in the theoretical formulation).

In contrast to his behaviour in the sessions, within the unit he was almost the ideal patient. He always returned from leave at the agreed time, he carried through with any tasks requested

of him, and other than needing some encouragement to attend the in-house groups, to get off his bed or to change his clothes, he never posed any specific management problem. The main issue was that even though his affect was not blunted in the usual way that it presents in people who have a psychotic illness or are more severely depressed, he still seemed sad and lonely, and he showed little motivation to move forward in his life.

The therapy continued for 5 months, at which time the team felt that he was ready to be prepared for discharge to a small partly supervised hostel, though this would take a number of months. This decision coincided with my telling him that I was to be leaving the unit in 6 weeks time. When I told him of my leaving, his immediate response was one of shock and he blurted out "I'm going to miss you. I'm going to miss our talks", and for a while thereafter he seemed uneasy but he refused to talk about it. When I later followed up on the two issues of leaving, my leaving and his leaving, things began to change. I had commented that even though it was something positive for him to be thinking of moving on, he might also be feeling a sense of loss at losing the companionship of the staff and 13 other clients and the security and the encouragement that he had received over a relatively long period of time. It would also mean that he would be leaving the chef who had become an important person in his life. In addition to this, our sessions would be coming to an end a lot sooner than his moving on. His response was that he had not thought of these things and that what I had said was true.

Over the following 6 sessions, prior to termination, his behaviour began to change. During the sessions he would ask to end early, he would look at the clock whilst I was talking to him, his tone of voice was often irritable, he began to smirk rather than smile, and it seemed that for the first time his anger was being shown in a more direct manner. Whereas in the past he

would give the impression that he was tolerating my comments and boundaries he now seemed to be more hostile and dismissive of me. He would say, with a smile/smirk on his face, "I'm so happy these sessions are ending - it's such a relief that I wouldn't have to come to any more - they're a waste of time", or "I've really got nothing to say, can I go now". I continued to comment on what I thought might be happening for him, but he mostly refused to engage in any real discussion about it, and it was only in the final session that he briefly acknowledged the sessions being of some benefit.

10. Evaluation

I have often wondered, both throughout the intervention and whilst writing it up, whether I was being as stubborn as he was by persevering as I did! Did I collude with his passive compliance by continuing the therapy? Should I have been more challenging or brought it to a close and informed him that the responsibility of it ending was a consequence of his unwillingness to engage in a more collaborative manner? However, the IRIS model (1999) of intervention comments on similar difficulties and encourages the continuation of engagement wherever possible. In the light of this, I do believe that the therapeutic relationship and the intervention was of some benefit to him evidenced by the small shifts in therapy and the greater ones in his relationships with others within the unit. Nevertheless, my leaving him (by leaving the unit) may have partly undermined my initial intention of providing him with a relationship that would not involve being rejected.

In conclusion, I feel that my ambivalence about the intervention possibly reflected his about living his life in a different way. It was about making a choice, and my choice was to not disengage because of his ambivalence even if the process of continued engagement was often frustrating and difficult. As a professional, I think that systems or services (and the

professionals within them) sometimes struggle to tolerate ambivalence and frequently draw people in or push them out as a means of coping with uncertainty.

11. Discussion

Difficult to engage clients have received more attention over the past decade through a variety of approaches, such as Linehan's Dialectic Behavioural Therapy (1993), Sainsbury Centre for Mental Health's Keys to Engagement (Sainsbury Centre for Mental Health [SCMH], 1998) and the National Service Framework (Department of Health, 1999). These initiatives place a strong emphasis on the process of engagement, with the rationalisation that one (or services) cannot work with a client who has disengaged from services, or, as in Linehan's model (1993) with someone who is no longer alive (i.e. has committed suicide).

Engaging clients covers both concepts of those who are reluctant or resistant to arrive for sessions (such as those who would be eligible for assertive outreach work), as well as those who struggle to participate in the actual process of the therapy (i.e. they attend the sessions but do not engage in the work being done), such as with the client presented. Whereas the main focus in this case study was on developing a therapeutic relationship in which he could experience a reflective and uncritical relationship, alternative models and interventions could have been implemented. For example, utilising techniques from a more structured cognitive-behavioural approach, such as asking him to keep a thought diary or diary of daily activities which could be discussed in the sessions (Beck, 1976, 1983). Another alternative intervention model could have been Linehan's Dialectical Behavioural Therapy (1993). An example of a technique from this model would have been to facilitate his learning the skill of 'mindfulness' (Linehan, 1993) which is the process of paying attention to the task at hand,

such as breathing, eating, washing and so forth, in a purposeful and non-judgemental manner, in order to develop his awareness of, and attention to, his daily life experiences.

In conclusion, there are usually a number of different models and techniques with which one can approach a clinical case, and the choice of treatment modality is frequently determined by the clinician's preferred method of intervention. However, it is often helpful to re-evaluate that choice in light of the outcome of treatment and the advantage of hindsight.

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Chapter 3

The Avoidance of Traumatic Memories and Directed Forgetting in Symptomatic, Recovering and Never Depressed Individuals.

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Abstract

The retrieval, recall and inhibition of past traumatic experiences (excluding PTSD) have been researched (Brewin, Hunter, Carroll, & Tata, 1996; Kukyen & Brewin, 1994, 1995; Myers, Brewin, & Power, 1992; Spenceley & Jerrom, 1997), and one consistent finding has been that depressed individuals score high on both intrusion and avoidance of traumatic memories, as measured on the Impact of Event Scale (Horowitz, Wilner, & Alvarez, 1979). However, as they recover, the extent of intrusion decreases but the level of avoidance remains high. This study proposed to further develop the understanding of the avoidance component of traumatic memories in symptomatic, recovering and never depressed individuals, and to examine whether those who do successfully avoid intrusive memories are also more able to inhibit negative emotional material, when directed to do so. The findings suggest that people who have never been clinically depressed exhibit significantly lower levels of intrusion and avoidance of unpleasant memories, which appears to allow for more cognitive flexibility and a greater capacity to process material, in this case to recall words, than depressed and recovering depressed individuals. However, the enhanced recall of the recovering group over the depressed group may be due to a process whereby as the severity of the depression decreases so the capacity to process trauma increases. The recovering group were not found to differ significantly from the control group on a number of dimensions and the argument that the depressed and recovering depressed groups belong on a continuum different from the never depressed group was not supported. The widespread ramifications of depression and stressful life events on the individuals' cognitive and emotional well-being were discussed, as were the clinical implications. This research emphasises the high level of co-existence between a diagnosis of depression and the occurrence of a life event that the individual perceives as traumatic, even though such experiences do not meet the criteria of PTSD. It also emphasises the value of integrating research findings from the areas of neuroscience and trauma with that of depression.

1. INTRODUCTION

In England alone, it is estimated that mental illness costs around £32 billion per year (Patel & Knapp, 1998), with depression being rated as one of the three most common mental health problems, along with anxiety and eating disorders (Goldberg & Huxley, 1992).

Depression is a multi-referential term that describes a mood disorder of sadness that is beyond the level of 'normal' low mood (Kaplan, Sadock, & Grebb, 1994), and it is one of the most common disorders encountered in Primary Care (Clinical Standards Advisory Group [CSAG], 1999). In 1987, Blacker and Clare's study estimated that around 10% of patients presenting at their GPs revealed some symptoms of depression (Blacker & Clare, 1987), but five years later it was estimated to be as high as 50% (Freeling & Tylee, 1991), though part of this difference may have been due to the use of different measures. Goldberg and Huxley's (1992) study that found that, in the UK, approximately 80% of patients diagnosed with depression were treated solely in the primary care setting.

Theorists such as Beck (1967), Ingram (1984) and Teasdale (1988) have provided various explanations of the cognitive processes underlying depression, describing how different thought processes have been developed and can be activated during the course of this disorder, suggesting how a 'vicious cycle' of negative thoughts, feelings and experiences is set up (Beck, 1967; Teasdale, 1988). Negative childhood or adult experiences and their associated memories have been shown to affect depressed individuals (Baker & Duncan, 1985; Kukyen & Brewin, 1994; Main, Kaplan, & Cassidy, 1985; Palmer, Chaloner, & Oppenheimer, 1992; Perry, 1999; Reinherz, Giaconia, Hauf, Wasserman, & Silverman, 1999; Stern, 1991) by contributing to the development of dysfunctional thought processes about self worth and value (Beck, 1967), and by the memories and images of these events intruding into

their thoughts (Gerlsma, Das, & Emmelkamp, 1993; Perry, 1999; Rothschild, 2000; van der Kolk, McFarlane, & Weisaeth,1996).

Past traumatic childhood events and the individual's capacity to retrieve, recall or inhibit these negative experiences has also been researched (Brewin, Hunter, Carroll, & Tata, 1996; Brewin, Reynolds, & Tata, 1999; Kuyken & Brewin, 1994, 1995; Parks & Balon, 1995; Perry, 1999; van der Kolk et al., 1996). The extent of intrusive memories has been measured, in some of these studies (for example, Brewin et al., 1996; Kuyken & Brewin, 1994) on the Impact of Event Scale (Horowitz, Wilner, & Alvarez, 1979). This scale measures subjective stress directly related to a negative experience on the dimensions of intrusion (i.e. the degree to which the memory of a negative life experience comes into the individual's mind) and avoidance (i.e. the extent to which the individual attempts to avoid the intruding memory). Research (Brewin et al., 1996; Kukyen & Brewin, 1994, 1995; Spenceley & Jerrom, 1997) has shown that depressed people score high on both intrusion and avoidance, and that those who are in recovery from depression reveal a decrease in the extent of intrusion, but equivalent levels of avoidance.

This study sets out to further develop the understanding of the avoidance component of traumatic memories, and to investigate whether a clinical population (i.e. depressed and recovering depressed groups) belongs on a continuum that differs from a non-clinical population (i.e. never depressed) in terms of impact of trauma and self-referential directed forgetting. The term 'trauma', in this study, was self-defined by the individual as a life event that he or she experienced as traumatic rather than the term being objectively defined (by DSM-IV criteria or by the researcher) by events of traumatic severity.

2. LITERATURE REVIEW

The literature review aims to approach the diverse, but interrelated concepts, of depression and trauma from a developmental perspective by referring to factors that can influence an individual's emotional state from foetal development, through adolescence and into adulthood. It has been structured so that the prevalence and treatment of depression, factors that contribute to its onset, the effects of depression on memory and the cognitive theories of depression are reviewed, in the first instance. Following this, the area of trauma and its effects on the physiological and emotional state of the individual is presented, as there is a high level of co-morbidity between depression, autobiographical memories and PTSD. However, some individuals develop depression whilst others are more able to deal with life events by developing a style of coping referred to as a 'repressive coping style', which will be discussed. This will lead to an overview of the literature on the mechanisms of recovery from depression and traumatic life events, which is a relatively new of area of research. One measure used in previous research on the repressive coping style, PTSD and depression is the directed forgetting task. This task is based on Bjork's directed forgetting paradigm which posits that individuals can consciously inhibit material in order to achieve a goal. The purpose of the review, by reflecting upon the emotional and cognitive (including memory) impact of negative life events and the development of depression, is to place within context the general hypotheses of the empirical research i.e. whether those who move into recovery from depression do so by inhibiting negative material, and that the depressed and recovering depressed groups belong on a continuum that is different from the never depressed group.

2.1 Depression

2.1.1 Definition of Depression

Depression is a nosological concept that has been subdivided into bipolar, endogenous, reactive, neurotic and psychotic varieties on the basis of history and symptoms (Kaplan et al., 1994). 'Depressed mood' is a term with which most people are familiar, and which involves some feelings of distress, unhappiness and apathy. 'Depression' as a clinical term is a syndrome that describes a cluster of symptoms which are generally comprised of depressed mood, loss of interest, anxiety, sleep disturbance, loss of appetite, lack of energy and sometimes suicidal thoughts (Diagnostic Statistical Manual-IV [DSM-IV], 1994; International Classification of Diseases and Health Related Problems [ICD-10], 1994). In the context of this study, the terms 'depressed' and 'depression' will refer to a clinical syndrome, or cluster of symptoms, covering changes in affect, cognition and behaviour, and which meet the diagnostic criteria for a Major Depressive Disorder according to the DSM-IV (APA, 1994).

Depression is known to be an extreme, persistent and recurrent condition that interferes significantly with the ability of the individual to function within his or her environment, and ones vulnerability to depression is determined by the interplay of multiple genes and the environment (CSAG, 1999; DSM-IV, 1994; Kaplan et al., 1994; National Institute of Mental Health [NIMH], 1999; Solomon et al., 2000). Imaging shows that the functioning of neurotransmitters, and therefore the regulation of mood, cognition, behaviour, sleep and appetite, are disrupted in depressed individuals (NIMH, 1999).

The most prominent clinical diagnosis of depression is that of Major Depressive Disorder (MDD). In addition to MDD, which may be mild, moderate or severe with or without

psychotic symptoms, the DSM-IV (APA, 1994) and ICD-10 (WHO, 1994) both refer to a further category of Dysthymic Disorder. This variant of depressive illness is characterised by less severe or fewer neuro-vegetative symptoms but of much longer duration, i.e. two years. Devenand, Nobler, and Singer (1994) and Judd, Rapaport, Paulus, and Brown (1994, 1997) have argued that Dysthymia is a clinically distinct phenomenon from MDD. The features they present to justify this include: the equal representation of men and women (as opposed to the over-representation of women with MDD), the presence of precipitating life events, and the less frequent occurrence of co-morbid disorders. This approach is helpful in so far as it recognises two forms of depressive illness that may require somewhat different approaches to treatment. It does not, however, contradict the notion that depressive phenomena occur on a continuum of severity. It underscores the point that the threshold for the recognition of clinical significance for depressive illness needs not only to be lower than the severe symptoms of MDD, but also broader in order to consider a wider range of factors (Blanchard, 1997). The Comprehensive Assessment and Referral Evaluation (CARE) (Gurland, Golden, Teresi, & Challop, 1994) has been designed to identify this broader range of depressive illness.

Adjustment Disorder with depressed mood is another manifestation of depression recognised in the DSM-IV (APA, 1994). This refers to the presence of significant levels of symptomatology, but not severe enough to reach the criteria for a diagnosis of Major Depressive Disorder, within six months of a significant life event. Given the negative influence of the notion of, as Pitt (1997) notes, 'justified' depression (i.e. one that is the obvious consequence of life events), this diagnosis may be particularly useful when considering a treatment strategy. It may also be helpful when dysphoric experiences arise in response to the presence of a medical condition, but when the causal link between the two is

not overtly identifiable as physical or biological (Pitt, 1997). The category of Minor Depression is still under debate and still requires further empirical investigation (Blazer, 1997).

2.1.2 Prevalence of Depression

Depression is one of the most common psychological conditions with a lifetime prevalence of about 15% in the adult population, possibly up to 12% amongst men and 25% amongst women in the Western World, the latter showing a twofold greater prevalence of Major Depressive Disorder than the former (DSM-IV, 1994; Kaplan et al., 1994). Paykel (1994) attributed this discrepancy to psychosocial factors, such as the low status of women in society, both socially and economically. However, a far greater percentage of individuals suffer from depression but do not meet the full DSM-IV criteria of a Major Depressive Disorder and never come to the attention of clinicians (Blazer, 1997; Steffens et al., 2000). An estimate is that at any one time between 9% and 20% of the Western population have significant symptoms of depression (Bradley, 1995; Paykel, 1994).

In terms of ethnicity, studies (Nazroo, 1997, 1998) have reported higher prevalence rates of depression amongst African-Caribbean individuals than European Whites, with African-Caribbean men being twice as likely to suffer from depression than European White men. There is also some evidence, (Commander, Sashidharan, Odell, & Surtees, 1997) that depression in those from the African-Caribbean, Asian, refugee and asylum seeking communities is less likely to be recognised, and that individuals from ethnic minorities are less likely to be referred on to secondary services or for psychological treatment.

2.1.3 Treatment of Depression

Those who suffer with depression typically experience discrete recurring episodes throughout their lifetime, but there is great variability between individuals in terms of symptoms, course of illness and response to treatment. Whilst 80% of people respond well to treatment, others with refractory depression are more difficult to treat, and even those who respond well initially, do not always continue to do so (NIMH, 1999). Untreated depression can often accelerate its course, with episodes becoming more frequent and severe over time (DSM-IV, 1994; NIMH, 1999; Solomon et al., 2000).

There are a variety of methods of treatment available. Pharmacological treatment using the available drugs such as tricyclic antidepressants, monoamine oxidase inhibitors (MAOIs) and selective serotonin re-uptake inhibitors (SSRIs) are relatively successful for some sufferers, but due to the idiosyncratic presentation of depression in different individuals, sometimes medication in certain combinations is required in order to successfully treat symptoms (Hochstrasser et al., 2000; Lima & Moncrieff, 1998; LiNC UP, 1995; NIMH, 1999; Roth & Fonagy, 1996). ECT (Electro Convulsive Therapy) has gained more popularity over the past few years as a form of treatment, and has proved to have some success despite its past reputation (American Psychiatric Association, 1996; Robertson & Eagles, 1997; Roth & Fonagy, 1996; Zornberg & Pope, 1993).

Non-pharmacological approaches to treating depression include derivatives of Cognitive Behavioural Therapy (CBT) (Beck, 1976, 1983; Foy, 1992; Gotlib & Hammen, 1992; Roth & Fonagy, 1996), Schema-Focused Therapy (Padesky, 1994; Young, 1990), and various forms of Psychotherapy, such as Interpersonal and Psychoanalytic (Parry & Richardson, 1996; Roth & Fonagy, 1996; Weissman, 1994). In a comprehensive review of the efficacy of such

treatments, carried out by Roth & Fonagy (1996), they concluded that there was no one treatment approach that could be unilaterally applied to all psychological conditions, and that part of the preference towards CBT interventions was, in part, due to the concepts on which it is based being more accessible to being operationalised than other more dynamic concepts, such as transference. In addition to this, the volume of research available for meta-analysis was far greater in the area of CBT than in any other psychological intervention, and that this raised the issue of the necessity of other forms of psychotherapy to address the need to investigate their therapeutic processes in a manner that would allow for scientific examination.

The co-occurrence of an additional disorder with depression is frequently found (DSM-IV, 1994; National Service Framework [NSF], 1999; NIMH, 1999), for example anxiety disorders such as Panic Disorder, Obsessive Compulsive Disorder (OCD), Post Traumatic Stress Disorder (PTSD), Social Phobia or Generalised Anxiety Disorder (GAD), and it is necessary and important that each condition is identified, diagnosed and treated. Depression can also be found in individuals presenting with certain physical illnesses, such as heart disease, stroke, cancer, and diabetes (Musselman, Evans, & Nemeroff, 1998; NIMH, 1999; NSF, 1999), and as such, it should not be overlooked or dismissed as something minor. It has also been found to be a major risk factor for the development of heart disease and for death after a myocardial infarction (Musselman et al., 1998).

2.1.4 Contributing Factors

There appear to be a number of different factors that have been found to contribute to the development of depression. Research has indicated that physiological/biological factors (NSF, 1999; Perry, 1994; Perry, 1999), psychological factors (Bowlby, 1988; Gilbert, 1989)

and environmental factors (Brown, Cohen, Johnson, & Smailes, 1998, 1999; Brown & Harris, 1978, 1989a; NSF, 1999) may contribute to the individual's vulnerability, onset and maintenance of depression. The psychological triggers of depression are numerous, with severe traumatic life events, negative experiences and memories of these being postulated as some of the contributing factors (Andrews, Brown, & Creasey, 1990; Brown, 1986; Brown & Harris, 1978, 1989a; Brown et al., 1999; Perry, 1999; Perry et al., 1995). A broad overview of the extensive research that has been conducted in this area will be provided in the following sections, and specific factors have been focussed on due to the extent of the research available.

2.1.4.1 Neuronal Activation Patterns & Emotional Memory

This topic has received large amounts of attention in recent years mainly due to work by LeDoux (1992) and Davis (1992) on the function of the amygdala. Associations between physiological reactions and states are formed during foetal development whereby it learns that the womb and the foetal position are safe and comforting, and this association is made by the pattern of neural activity and the corresponding state/feeling experienced. After birth, as a child is fed within a nurturing environment it will experience eye contact, social intimacy, safety, calm, touch, cooing and so forth. This positive association continues into adulthood with the experience of eating being associated with these emotions, and the consequent neuronal activation patterns. However, the development of this normal association can be severely disrupted by neglect, depression in a caregiver or trauma (Perry, 1999).

2.1.4.2 Environment & Parenting Style

Children who experience an environment of neglect and threat as they develop may learn behaviour associated with the cues they experience (Bowlby, 1977; Duarte & Thomson,

1999; Goodman & Gotlib, 1999; Oakley-Brown, 1995; Shah & Waller, 2000; Young, 1990). A child in a constant state of anxiety will be unable to effectively process information being relayed (de Zulueta, 1993; LeDoux, 1992; Perry, 1999; van der Kolk et al., 1996;). Perry (1999) argues that the social and emotional skills a distressed child learns will be atypical, and the misinterpretation of cues will be a common occurrence. For example, eye contact will be interpreted as threat, or a friendly touch as an indicator of sexual intrusion or abuse. These reactions are learned from their experiences in their immediate environment, and become generalised to other situations. However, the difficulty arises in that the person's response to such cues in a different environment is now out of context (Perry, 1999).

Significant evidence appears to be available to support the claim that environmental and parental factors have been closely linked to the development of depression (Beck, 1967; Bowlby, 1977; de Zulueta, 1993; Duarte & Thomson, 1999; Goodman & Gotlib, 1999; Oakley-Brown, 1995; Shah & Waller, 2000). Significant differences in self-reported upbringing style between depressed and control groups have been demonstrated by Rojo-Moreno, Livianos-Aldana, Cervera-Martinez, and Dominguez-Carabantes (1999) who found that depressed individuals reveal more memories of having been rejected and were shown less affection during childhood. However, no such differences were reported with respect to overprotection. They then went on to determine whether these reports were related to severity of symptoms but, interestingly, found no relationship. These findings concur with previous work by Perris, Jacobsson, Lindstrom, van Knorring, and Perris (1980) and conclusions drawn by Oakley-Brown (1995) who found that lack of emotional warmth is repeatedly indicated as a precursor for later psychopathology.

In the same vein, Duarte and Thomson (1999) have suggested that self-reporting of negative rearing circumstances may constitute an acquired vulnerability to later effects of stress. They refer to Attachment Theory (Bowlby, 1969, 1977, 1988) put forward by Bowlby. He (Bowlby, 1977) notes that depression in adults can be a result of pathogenic parenting, such as the child's parent(s) being continuously unresponsive, rejecting or disparaging, there being separations between parent and child, persistent threats by a parent that the child will stop being loved or will be abandoned if he or she doesn't behave, or by implying that something awful may happen to the parent because of the child (e.g. the parent threatens to commit suicide or abandon the family). Duarte and Thomson (1999) conclude that depressed individuals usually demonstrate an over-representation of insecure attachment experiences within their childhoods. de Zulueta (1993), in her work on pain and violence, also incorporates Bowlby's theory and work as a key element in how separation and loss can lead to long-term effects such as depression, and the connection between attachment behaviour and violence.

The factor of parental style being frequently implicated in vulnerability to depression was further investigated by Shah and Waller (2000) who focused on the potential role of core beliefs in the relationship between parenting in childhood and major depression in adulthood. Beck (1967) had theorised that the development of negative schemata and cognitions were directly attributable to a critical and disapproving parent and there are many in support of this claim (Rogers, 1996a). Shah and Waller (2000) hypothesised that the relationship between adult depression and negative parenting style would be borne out through 'unhealthy core beliefs'. Their work confirmed this hypothesis, and they reported that the depressed group had more unhealthy core beliefs than the nondepressed control group, particularly those indicating defectiveness/shame, self-sacrifice and insufficient self-control. Data suggested

that five core beliefs (dependence/incompetence, emotional inhibition, failure to achieve, unrelenting standards and vulnerability to harm) could arbitrate the relationship between maternal bonding and paternal overprotection and levels of depression.

Children of Depressed Mothers – A Developmental Model: Goodman and Gotlib (1999) have proposed a developmental model to explain the mechanisms by which the children of depressed mothers go on to experience adverse effects at later stages of their lives. They refer to their own review discussing these adverse effects, such as more difficult temperaments, less secure attachments to their mothers, more negative reactions to stress, less socially competent, lower levels of self esteem and increased behaviour difficulties, and consider both reciprocal and transactional relationships. They stress that episodes of depression are rarely isolated, i.e. they are usually recurrent, and, therefore, a child will be exposed to more than one episode of maternal depression during his or her childhood.

They propose four mechanisms by which children are adversely affected by maternal depression. Firstly, heritability of depression, i.e. the inheritance of a predisposition to develop or be vulnerable to depression, or to experience life events as more stressful. Secondly, the dysfunction of innate neuroregulatory mechanisms. Abnormal development in the womb, due to the mother's depression, can result in constricted blood flow to the foetus, increased levels of cortisol and abnormal neuroendocrine functioning, which may lead to the presence of 'behavioural inhibition or negative affectivity' in the child. The impact of such a stressful event on the neuroregulatory systems of a child in its first few years of life has been shown to have critical implications for subsequent functioning (Heim, Owens, Plotsky, & Nemeroff, 1997). However, this area of research is relatively new and requires further replication.

Thirdly, negative maternal cognitions, behaviours and affect are pivotal in the development of risk. These may result in the mother representing an inadequate 'social partner' for the child. Consequently, the child will be negatively affected in terms of his or her cognitive and social skills development as the child learns these through role modelling and observation of the mother. The lack of an adaptive role model may lead to an increased risk of developing depression. Finally, the stressful context of the child's life contributes to the risk of developing depression. Stress is highly associated with depression in adults (Gotlib & Hammen, 1992) and it is thought that the child's exposure to the stressors associated with living with the depressed mother may lead to an increased risk of developing depression themselves. It is suggested that these mechanisms will interact with each other, in varying combinations depending on individual circumstances, to produce vulnerability to depression.

Goodman and Gotlib (1999), however, also include three moderators in their model. The first is the father of the child, which includes the amount of time he is able to spend with the child and his own mental health. Secondly, the timing and course of the mother's depression. The younger the child when it is first exposed to the effects of depression, the more negative consequences it will suffer. They are of the opinion that the first year of the infant's life appears to be the most crucial period for the mother to regulate the infant's emotions. Negative effects resulting from a lack of regulation could continue throughout the child's development, leading to the establishment of negative cognitive sets and dysfunctional behaviour. A relationship has also been found between chronicity of maternal depression, and lowered adaptive functioning and higher rates of psychopathology in their adolescent children (Beardslee, Schultz, & Selman, 1987; Keller et al., 1986).

The third moderator is the characteristics of the child i.e. temperament, gender, intellectual, social and cognitive skills. These factors impact on his or her ability to develop successfully, in the presence of potential adverse influences resulting from the genetic and home environment provided by the parents. Children with calmer, more easy-going temperaments seem to be less affected by the negative influences, and exhibit less reactivity to environmental challenges. It is possible that low intelligence and poor social-cognitive skills may represent early manifestations of psychopathology, but the authors are unable to cite reliable empirical evidence for these proposals (Goodwin & Gotlib, 1999).

Children who have reached adolescence when their mothers experience an initial onset of depression are less likely to be as affected since there is less foetal or environmental exposure to negative factors (Goodwin & Gotlieb, 1999; Heim et al., 1997).

2.1.4.3 Transition From Adolescence To Adulthood

Mood swings and behaviour difficulties are widely accepted in western society as being hallmarks of this transitional period. However, major depression can be a serious, and often overlooked, concern during this period, partly due to the adolescent tendency to act out or withdraw, as well as the possible likelihood of the depression continuing into adulthood (Leader & Klein, 1996; Reinherz et al., 1999).

Reinherz et al. (1999) examined the risks within an ongoing longitudinal population, and reviewed some of the literature around a number of domains of potential risk for developing a depressive disorder. The first domain they propose is family factors. These include low socio-economic status (Rao et al., 1995), family size, parental age, marital disruption (Kessler

& Magee, 1993), remarriage (Reinherz et al., 1993), loss of a parent, especially the mother (Marton & Maharaj, 1993), family conflict, and children's poor perception of their role in the family (Reinherz et al., 1993).

Secondly, they cite health factors as contributors to risk, which include early health problems (Cohen et al., 1993) and injury. The third domain proposed is one encompassing behavioural and emotional characteristics, and it consists of an observed tendency to exhibit externalising or inhibited behaviour (Caspi, Moffitt, Newman, & Silva, 1996), low self-esteem, the presence of an anxious and dependent temperament, and a tendency for the child to describe him or her self as unpopular with peers (Reinherz et al., 1993). Finally, the appearance of academic problems including truancy and greater use of school services (Ernst, Schmidt, & Angst, 1992) seem to be indicative of greater risk.

Stressful life events experienced in adolescence have also been shown to have a significant effect on the likelihood of onset of depression. Monroe, Rohde, Seeley, and Lewinsohn (1999) conducted research into the life event of 'loss of a relationship' and tendency to depression in adolescence. There is much evidence for the importance of severe life events in precipitating first onset of depression (Monroe & McQuaid, 1994), but Monroe and colleagues (1999) note that it is not common for researchers to isolate first onset from recurrent episodes of depression in considering risk factors. They collected information about self-reported romantic break-ups for their sample of 1, 507 adolescents (average age 16.5 years), and found a recent romantic break-up to be associated with an initial onset of depression, but no such connection with a recurrent episode (almost half of first onsets were preceded by a break-up in the previous year). Girls were more likely to report such events, and demonstrated a higher incidence of first onset depression (Monroe et al., 1999). They

propose that such a break-up may be a 'marker' for future onset of depression as opposed to a 'maker' of depression.

Reinherz et al.'s (1999) overview of the research not only looked at the potential risk domains for developing a depressive disorder, cited above, but also found the following factors to be associated with major depression: poor overall functioning (Leader & Klein, 1996), emotional behavioural problems, low self-esteem, poor self-mastery (Lewinsohn, Gotlib, & Seeley, 1997), social dysfunction such as interpersonal problems with friends, family and spouses (Leader & Klein, 1996), and education and occupational difficulties (Judd, Paulus, Wells, & Rapaport, 1996). All of these factors can contribute to the formation of a vicious cycle in which the presence, or tendencies, of these characteristics may increase the likelihood that the episode of depression will continue, or that relapse becomes inevitable (Reinherz et al., 1999).

In this longitudinal study spanning two decades (data collected at 6 time points from participants, parents and teachers starting from early childhood) they also wanted to determine the risks predicting 'the most serious outcome'. It was found that female subjects were more likely to have experienced onset of depression before the age of 14, and in addition, these episodes of depression were three times as likely to be classified as 'severe', in contrast to male subjects who were three times as likely to experience a milder depression. Indicators for the development of depression in female subjects were death of a parent by the age of nine and poor teacher-evaluated academic performance at age 9, but for males, they were poor neonatal health and poor health development.

Depressed subjects reported either emotional, physical or sexual abuse in early adulthood confirming the findings of Silverman, Reinherz, and Giaconia. (1996). Family interactions, as opposed to structural factors, are more predictive of depression; for example, having divorced parents did not predict depression (Reinherz et al., 1999). Female children who were born later in the birth order, and consequently had older parents, were found to perceive them as more punitive and unsupportive. Consequently, they have been shown to be at greater risk of depression, anxiety and lowered self-concept, which supports the work of Gates and colleagues (Gates, Lineberger, Crockett, & Hubbard, 1986).

Self-reported behavioural characteristics were found to be reliable in predicting those adolescents who would experience depression in early adulthood. Subjects assessed as depressed demonstrated poorer psychosocial functioning on all but 2 measures of early adult functioning (unemployment and job attendance). The measures they scored poorly on were self-esteem, self-mastery, interpersonal relationships, need for social support, satisfaction with career and risk of suicidal behaviour (Reinherz et al., 1999).

The one limitation to be considered in interpreting the results of this work is that the sample consisted of individuals from predominantly white, working to lower middle-class backgrounds and this may narrow the generalisability of their findings to the population as a whole.

2.1.4.4 Physical & Sexual Abuse

Within the literature on the potential risk factors associated with the onset and development of depression, physical and sexual abuse experienced during childhood is prominent. Silverman et al. (1996) has noted that adults reporting childhood abuse are at increased risk

for distress and mental disorders. What is not clear, however, is whether this risk is attributable to the abuse experience alone, or if the increased risk is exacerbated by other factors, such as complex family circumstances, dysfunctional relationships therein, parental psychopathology, the temperament of the child, or other stressors like parental substance abuse, poor health and poverty. Combinations of these risks assessed independently and prospectively have been shown to be powerful predictors of abuse and neglect (Brown, Cohen, Johnson, & Salzinger, 1999). Consequently, it may be that the risk of depression is not only a result of the abuse per se, but may be compounded by the environmental and familial context in which it occurred. The relationship between childhood abuse and suicide attempts is still uncertain, with Silverman et al. (1996) being of the opinion that abuse is associated with an increased risk. However, this discrepancy may be a result of varying design and definition of abuse.

Brown and co-workers (1999) examined a large sample of children in New York longitudinally over a period of 17 years. They found that both Dysthymia and Major Depressive Disorder were over-represented in the group of subjects reporting abuse or neglect, and suicidal behaviour was shown to be linked with a history of childhood maltreatment. The authors also examined the relationship between risk of disorder, suicidal behaviour and type of abuse. It was reported that rates were elevated for all types of abuse (3 - 4 times), but that sexual abuse seemed to produce the highest risk (independent of contextual factors). In considering the confounding factors, i.e. additional environmental factors contributing to and interacting with the abuse, the authors state that although the link is strong, childhood neglect cannot unambiguously be said to cause a higher risk of disorder or suicidal behaviour. Physical abuse falls in-between neglect and sexual abuse in terms of

the degree of risk of disorder or suicidal behaviour to which it exposes an individual (Brown et al., 1999).

2.1.4.5 Age & Gender

Klein et al. (1999) carried out work looking at the effects of age of onset of depression on its course and severity. The study was carried out with outpatients so as to be representative of the 'general' depressed population. Their sample consisted of 289 outpatients, all experiencing an episode of major depression, who were taking part in a study comparing the efficacy of Sertraline and Imipramine. The majority of the patients in their sample had onset of depression after the age of 21 (57%; mean age of onset 33.5 years) while a significant number had earlier onset (43%; mean age of onset 13.7 years). Early onset was associated with more recurrent depressive episodes, high likelihood of hospitalisation, co-morbid personality disorder and substance abuse. This group was more likely to be white women with poor education, low status jobs and a family history of depression.

Blair-West et al. (1999) in their study of lifetime risk in major depression hypothesised that gender differences in suicide risk would be significant since women are more prone to develop depression but men are at a higher risk of suicide. They found that young men with depression are a particularly high risk group for suicide as 7% of men with major depression commit suicide, in comparison to only 1% of females, and that men under 25 years of age were 10 times more likely to commit suicide, whilst those aged 25 and over were 5.6 times more likely to do so. However, women over the age 25 were 58% more likely than those under 25 to commit suicide.

In suggesting explanations for their findings, they propose that major depression may be over-diagnosed in women and under-diagnosed in men, and that females may be better at accessing help. They also note that younger people may have more difficulty seeking help than older people, that those who commit suicide may be receiving inadequate or even no care, and finally, prescription of ineffective or insufficient anti-depressive medication may be an important contributing factor. They also refer to the findings of Isometsa et al. (1994b) and Tiller, Krupinski, Burrows, MacKenzie, and Hallenstein Johnstone (1997) who reported high rates of co-morbidity, such as substance misuse, in those who have major depression and commit suicide.

Nolen-Hoeksema, Grayson, and Larson's (1999) research into gender and the onset and course of depression wanted to investigate the interplay between social conditions and personality characteristics, and its contribution to the gender difference in depressive symptoms. They proposed that women were assumed to be more susceptible to depression as a result of their lower status and lack of power, leading to the experience of more negative events and a lack of control. They also concluded that women who are employed and may be paid less than men often carry out the majority of household and child rearing duties, and may also have to care for older family members. A source of stress for women in this situation may be that they feel undervalued in their roles (Nolen-Hoeksema et al., 1999).

Butler and Nolen-Hoeksema (1994) refer to cognitive theory (Beck, 1967) and the suggestion that chronic lack of control over the environment can lead to the development of depressive symptoms, decreased motivation and loss of self-esteem. These circumstances may lead to women being more likely to ruminate than men when stressed i.e. they repeatedly focus on their symptoms of distress (Butler & Nolen-Hoeksema, 1994).

Studies by Lyubomirsky, Caldwell, and Nolen-Hoeksema (1998) showed that when those in a depressed mood were asked to ruminate, they generated more negative memories from the past, their evaluations of hypothetical and real events in the present were more negative, and they were fatalistic about the future. It is thought that such negative thought patterns could add to and draw out symptoms of depression, either directly or indirectly, by negatively impacting on problem solving. In addition, ruminators may be at higher risk of unintentionally decreasing the level of social support that is available to them by isolating themselves and, consequently, alienating others. Rumination lowers motivation and persistence in dealing with events in life which result in people failing to take action that they know will improve a situation with which they are dissatisfied (Lyubomirsky & Nolen-Hoeksema, 1995; Nolen-Hoeksema & Davis, 1999).

Simpson, Nee, and Endicott (1997) found no gender differences in terms of risk of relapse, recurrence, time to recover or number of episodes. Subsequently, Kuehner (1999) has shown that women are more likely to experience a recurrent episode of depression sooner than men (more that 2.5 times as likely to relapse as men) whether these patients were experiencing an initial episode or a recurrent episode of depression. She found no increased tendency for men to underrate previous depressive experiences (period of recall - 6 months), and some authors (Nolen-Hoeksema, 1987) support the theory that women have a better memory for negative past events than do men. However, such findings should be interpreted with caution due to the differential usage of various terms, such as relapse, recurrence, remission and recovery. In addition, the length of time without relapse after which people are considered recovered tends to vary.

Jack (1991) investigated the concept of self-silencing and depression, particularly in relation to women. Self-silencing refers to the silencing of one's own needs and feelings in favour of meeting the needs of one's partner. According to Jack (1991), self-silencing can result in a woman becoming angry with herself for putting the needs of others before her own desires, in line with social expectations of women originating in history. She was the first to propose that this concept leads to an increased vulnerability to depression in women, and devised the 'Silencing the self scale' encompassing four subscales - externalised self perception, care as self sacrifice, silencing the self and divided self. Focusing on social expectations of 'female goodness', self-silencing leads to a situation wherein a woman tries to present herself as an acceptable female, and yet is simultaneously angry with herself for not expressing her true self or needs. Jack (1991) cites this as 'the core dynamic of female depression'.

Associations have been demonstrated between self-silencing and various depressive symptoms (Towill, 1998), co-dependency (Cowan, Bommersbach, & Curtis, 1995), insecure attachment and lack of intimacy (Thompson & Hart, 1996). It has not, however, been demonstrated that women engage in higher levels of self-silencing behaviour than men. Studies using college students have found that men and women engage in the behaviour to an equal degree (Cowan et al., 1995), and some have shown that men actually engage in the behaviour more so than women (Duarte & Thompson, 1999; Gratch, Bassett, & Attra, 1995). Duarte and Thompson (1999) explain their findings by suggesting they may be due to social norms. For example, men should develop characters that include being courageous, competitive and aggressive. If this is so, men engage in self-silencing for somewhat different reasons from women, and it is inevitable that their perception of their behaviour will be vastly different to the perceptions held by women. This theory is supported by the fact that 'care as self sacrifice' and 'divided self' sub-scales were not found to be correlated with depression

for men but were significantly so for women (Duarte & Thompson, 1999). An important point, however, is that the scale was developed by Jack using only a small sample of women, and may, therefore, not be valid for use with men.

In summary, it appears that there is a number of interacting contributing factors associated with the development of depression. The main thrust of the arguments discussed would seem to suggest that at the current time, the risk factors likely to lead to an individual becoming depressed include being female (Nolen-Hoeksema et al., 1999), being a child who has experienced a traumatic or erratic upbringing (Perry, 1999; Reinherz et al., 1999; Shah & Waller, 2000), whose mother has suffered from depression (Goodwin & Gotlib, 1999), having a familial history of depression and its concomitant inherited predisposition and having experienced sexual or physical abuse during childhood (Brown et al., 1999).

Once an individual has experienced an initial episode of depression, there are then numerous maintaining factors that come into force, all of which may be complexly interlinked with the factors that contributed to the initial onset. Post (1992) theorised that once a person has experienced a stressful life event that precipitates a depressive episode, then he or she will be more sensitised to future stressful events and more likely to react adversely.

The development of cognitive theories of depression furnishes us with some conceptualisations of thought patterns and schemas that have been implicated in the perpetuation and mediation of depression.

2.1.5 Cognitive Theories of Depression

Beck's (1967) theory of depression was the forerunner of the cognitive models. Later theories and models stemmed from his work, such as Ingram's Information Processing Paradigm (1984), Teasdale's Differential Activation Hypothesis (1988), the Hopelessness Theory of Depression (Abramson, Metalsky, & Alloy, 1989), and Schema Focused Theory (Padesky, 1994; Young, 1990). These will be discussed in order to contextualise the hypotheses of the empirical research.

2.1.5.1 Beck's Theory

In 1967, Beck proposed a cognitive model of depression wherein he saw depression as the consequence of particular patterns of thinking. He postulated that dysfunctional thought patterns resulted from negative childhood experiences that led to the individual forming negative core beliefs about the self, the world and the future. This cognitive triad was activated when the experience of a negative situation occurred that 'matched' the theme of that experience. This could be by experiencing the same emotions, such as an intense feeling of loss or helplessness, or by being placed in a similar physical situation. Once triggered, this would set off the cycle of negative thought and action that leads into depression.

A person's depression may be maintained by negative cognitions. Both Beck's (1967, 1976, 1983) cognitive theory and Seligman's (1974, 1975) learned helplessness theory of depression, suggest that there is a negative cognitive bias in the depressed person's appraisal system. Beck's central thesis is that depressed individuals feel as they do because they commit characteristic errors in thinking. When people get depressed, they view the world in a negative light and are unable to achieve what they would like to, or were once able to.

Their failure to achieve reinforces their negative feelings about themselves and the world around them, and they tend to interpret situations as being more negative.

They also orient their attention towards the negative aspects of their experiences, thus setting up a 'vicious cycle' of negative thoughts, feelings and experiences (Beck, 1967; Teasdale, 1988). Beck found that his depressed patients tended to distort whatever happened to them in the direction of self-blame and catastrophisation. According to his negative cognitive triad, depressed people draw illogical conclusions, and come to evaluate themselves, their immediate world and their future in negative terms, and there is a large body of literature to support this selective attention to negative material (Mathews, 1997).

Criticism of Beck's Theory: Although cognitive therapy is widely accepted as being effective, the validity of cognitive theory is received with more scepticism. Haaga, Dyck, and Ernst (1991) reviewed much of the empirical research relating to Beck's cognitive theory. They first considered the descriptive features of depression, such as automaticity and exclusivity, and then the causal hypotheses for depression, such as stability and subjective valuation. Contrary to Matthews' (1997) findings that there is a large body of evidence to support selective attention, Haaga et al. (1991) note that the research on the feature of exclusivity, which predicts that depressed individuals will report virtually no positive cognitions, were mixed, with studies showing that depressed individuals rated positive adjectives as self-descriptive as well as negative ones. Therefore, it would be more accurate to conclude that depressed individuals are negative thinkers, but to an unspecified degree. They suggest that although depression is associated with negative thoughts, positive thoughts should not be excluded, and the exclusivity hypothesis should be removed.

In general, Haaga et al. (1991) again demonstrated only mixed support for the descriptive factors of cognitive theory, and they argue that there is room for refinement. They suggest that theorists should reconsider whether thoughts about the self, world and future should be treated as separate entities in the negative cognitive triad, or as a singular dimension. They challenge the concept of necessity, i.e. that it is necessary to experience all three features of the cognitive triad in order to be depressed, and argue that having only one aspect of the triad is all that is necessary. They do note, however, that in personal communication with Beck in 1989, he was in agreement with them on this issue (Haaga et al., 1991). They concluded that the issue of automaticity of negative thought was unclear, and more research was needed in this area, as automatic negative thoughts are not automatic cognitive processes, as conventionally defined. The process that leads to the thoughts occurring may be automatic, but the actual thoughts occupy a substantial amount of attention. Finally, they concluded that although information processing differences between depressed and nondepressed individuals have only been demonstrated in recall of negative material, the possibility that differences also exist in encoding should not be dismissed.

In their review (Haaga, et al., 1991), they comment on the four components of information processing bias. Regarding biased attention deployment, they comment that research has shown that depressed individuals pay more attention to themselves than nondepressed individuals. They found that there was no compelling evidence to support a negative bias for encoding in those who were depressed, but there was evidence for biased recognition memory, in that depressed individuals showed enhanced recognition of negative words in comparison to non-depressed individuals. However, the evidence was not conclusive. With biased recall, they state that studies have shown that as severity of depression increases, so do the probability and speed of retrieval of negative autobiographical memories. However, they

note that Blaney, in his review paper of 1986, argues that this does not take into account the amount of positive and negative memories initially present, so these results cannot be seen to conclusively demonstrate biased recall.

Haaga et al. (1991) go on to consider the four causal hypotheses of depression: stability (i.e. dysfunctional beliefs remain stable before, during and after a depressive episode); subjective valuation (i.e. personality modes can be used to predict how individuals value events); onset (i.e. the interaction between dysfunctional beliefs, valuations of events and vulnerability as congruent negative events are predictive of depression onset); and recurrence (i.e. recurrence of depression is predicted in the same way as initial episodes of depression).

They (Haaga et al., 1991) argued that there was no conclusive evidence in support of the causal hypotheses. However, they stopped short of advocating its abandonment. They concluded that as it is not yet possible to test whether the negative beliefs of the individual are latent, these causal hypotheses remain viable. However, they do suggest two conceptual issues that could limit the explanatory power of cognitive theory as it stands. Firstly, cognitive theory underestimates the possibility that the vulnerability created from the valuing of a particular experience could result in satisfaction gain as well as the experience of loss. The theory tends to emphasise the pathological potential of personality features, and not their potential to create positive biases. Secondly, they foresee the need for a more dynamic relationship between the dispositional and environmental causes of depression, and argue that the concept of the environment is underdeveloped. The environment that influences the individual is itself shaped by the individual, and therefore, the lines between personal and environmental factors need to be blurred.

2.1.5.2 Schema-Focused Theory

Schema theory found its origins in the work of Piaget who was the first to use the word 'schema' to relate to the meaning of a cognitive structure (Padesky, 1994). Padesky (1994), a firm proponent of schema-focused therapy, provides a comprehensive summary of schema change processes in cognitive therapy. She notes that, using Piaget as a foundation stone, Beck initially defined the concept as follows:

> ...a schema is a structure for screening, coding, and evaluating the stimuli that impinge on the organism. It is the mode by which the environment is broken down and organised into its many psychologically relevant facets. On the basis of schemas, the individual is able to... categorise and interpret his experiences in a meaningful way (Padesky, 1994, p. 267).

However, he later elaborated on and refined this definition. In essence, the formation of schemas provides a set of rules which, in following, an individual learns how to behave in certain situations and with certain people, and they become stable as information learned sequentially confirms their validity.

Schemas encompass beliefs, both core and conditional. They are assumed to develop as part of the normal cognitive development that we all experience throughout our lives, and that as we progress through life we learn to categorise experiences in an effort to bring order into our world. The schemas most commonly relevant in the psychological domain are those associated with affect and behaviour. It is not proposed that schemas are involved in causing psychiatric and emotional problems, only that they have been shown to be pivotal in maintaining these conditions. As schemas encompass both core and conditional beliefs, the way that we think about others and ourselves is determined by the schemas we form, which

in turn are dependent on the experiences we have in our world, such as our emotional experiences with family and friends. It is clear that there are an infinite number of variables that can potentially affect our cognitive development and these variables will be different for every individual.

From the perspective of schema-focused theory (Beck, 1967, 1976; Padesky, 1994) our schemas determine what we do in life, i.e. our behaviour and, consequently, our prospects for the future, and they are crucial in determining what we hear, see and remember of all of our experiences. If people have a large number of negative schemas, it is likely that they will focus on the negative aspects of events in their lives. If they become depressed, it is unlikely that they will succeed in recovering if they continue to be influenced by such negative, self-propagating schemas. Negative schemas; however, appear to be enduring even in the face of evidence that is contradictory to the beliefs held. This information is ignored, distorted, discounted, considered to be a unique exception and then discarded or forgotten (Beck, 1967; 1983).

In some cases, people have a number of contradictory schemas in their 'repertoire'. If they are depressed, their negative schemas are activated, and then when they are recovered, the positive schemas are once again activated and they experience their environment and events in their lives in a different light. However, those who do not have access to positive schemas are particularly difficult to treat, and in such cases, it is necessary to first develop alternative, acceptable schemas before they will consider the error in their belief systems (Padesky, 1994; Young, 1990).

Bricker, Young, and Flanagan (1993) provide a review of Schema-Focused Cognitive Therapy (SFCT) as developed by Young (1990). This way of working is aimed to deal with the needs of individuals with chronic 'characterological disorders' with an integrative approach. The difference between SFCT and CBT is that, in SFCT, early childhood experiences are explored in an effort to isolate the origins of the current problems. Circumstances that can lead to, wholly or in part, the development of maladaptive thought processes or core beliefs include neglect, deprivation, rejection, instability, abuse, abandonment, or criticism.

With such a distorted image and understanding of themselves, others and the world, when these children develop into adults, they are significantly disadvantaged since they are not able to orchestrate and obtain the security, stability and regard for themselves that is necessary to survive successfully in the world (Bricker et al., 1993). Therefore, the development of schemas is very important in children, but the variables determining how these schemas develop are vast.

2.1.5.3 Teasdale's Differential Activation Hypothesis

Teasdale (1988) notes that studies carried out to support Beck's stability hypothesis show that depressed individuals do seem to have dysfunctional attitudes, as measured by high scores on the Dysfunctional Attitude Scale (DAS). However, once in remission their DAS scores return to normal levels. He argues that these findings do not support Beck's model of dysfunctional thought patterns and attitudes determining the onset of depression. This is because, according to Beck, individuals' dysfunctional attitudes should exist whether they are in depression or not. Therefore, depressed and recovered depressed individuals should both score high on the DAS scale. However, this is not the case. As Teasdale points out, once in

remission negative thinking returns to normal levels, even when no attempt is made to deal with the dysfunctional thought patterns.

In response to this, Teasdale (1988) proposed his differential activation hypothesis. When considering depression, it is necessary to distinguish between what makes a person vulnerable to the onset of depression, and what maintains it, as they are two different processes. Teasdale's differential activation hypothesis postulates that there are events that can trigger depression, but there are also patterns of thought that can only be triggered once within the depressed state. It is these latter thought patterns that render one vulnerable to the severity of depression and its persistence. Therefore, depression is not entirely dependent on the initial event or mind-state that may have triggered it, but also on those thought patterns that are activated during a depressive episode.

2.1.5.4 Ingram's Information Processing Paradigm

Ingram (1984) put forward an information processing paradigm to explain the cognitive mechanisms that were in effect during depression. He proposed that people have cognitive networks of associated concepts, which have to be activated above a certain level to attain conscious access to them. As affective and thought structures are part of these associative networks, emotions therefore affect cognition. The deeper information is processed, the more it is elaborated and understood. The individual also has a finite attention span and limited processing power, but not all information utilises the same amount of processing capacity. For example, automated tasks such as riding a bicycle require less processing capacity than learning a new skill. Complex cognitive processes can occur automatically, triggered by small stimuli that produce complicated behaviours, that at the time one cannot be seen to consciously control.

These mechanisms can be used to explain the onset and maintenance of depression. For example, an individual may have a dysfunctional network of negative associated concepts formed by negative childhood experiences (Ingram, 1984). These associated concepts could be feelings of helplessness, lack of control, feelings of physical threat and vulnerability. If an environmental experience re-evokes a specific constellation of feelings the dysfunctional cognitive network will then be activated. Thus, a complex behaviour can be triggered by an unspecified or small stimulus. Once activated, this dysfunctional cognitive network can perpetuate negative thoughts and memories that crowd individuals' consciousness (or finite processing capacity). This leaves them unable to deal with or process other information, especially that which does not reinforce the existing cognitive schema, thus maintaining the depression.

The use of these constructs facilitates the empirical analysis of depression, and they are helpful when attempting to explain its onset and maintenance. Blaney (1986), in his review paper, cites a study that looked at mood congruent memory, and showed that negative mood led to recall of more negative memories, and Bower (1981) showed that negative mood biased the interpretation of ambiguous situations. As the information processing paradigm includes the concept of activation of thought content above a certain level to achieve conscious awareness, it is necessary to know what determines the level of activation, and whether or not there is conscious awareness.

2.1.5.5 Hopelessness Theory of Depression

The hopelessness theory of depression was defined by Abramson, Metalsky, and Alloy (1989) with hypothesised cause, symptoms, course, therapy and prevention. Hopelessness is the expectation that something highly desirable will not occur and that something highly

adverse will occur, and that you are powerless to change this. Hopelessness theory defines cause as a 'chain of distal and proximal causes hypothesised to culminate in a proximal sufficient cause of the symptoms of hopelessness depression' (Abramson et al., 1989, p. 359). A proximal sufficient cause is the expectation that something highly adverse will happen, or that something highly desirable will not happen. In addition to this, is the belief that you have no control over the outcome. It is the inferences individuals make about negative life events that determine whether they contribute to hopelessness. A person makes inferences about why the event occurred (inferred cause), the consequences of the event (inferred consequences), and inferences about the self in light of the event (inferred characteristics about self). The symptoms of hopelessness depression are more likely to occur if one attributes these events to global and stable causes, i.e. ones that are enduring and likely to effect many outcomes. They suggest that the relationship between negative life events and the symptoms of depression is moderated by inferred negative consequences. Abramson et al. (1989) also suggests that distal factors (i.e. factors that occur at the beginning of the chain and are distant from the occurrence of the symptoms) can influence one's causal inferences, such as individual differences in attributional style. Therefore, if an individual has a depressogenic attributional style (i.e. the attribution of negative events to stable global factors), they are more likely to develop hopelessness depression. This is conceptualised as a diathesis-stress component. The diathesis is the depressogenic attributional style, in that it operates in the presence of a negative life event, but not in its absence.

Symptoms of hopelessness depression are similar to those in the reformulated theory of helplessness and depression (Abramson, Seligman, & Teasdale, 1978). These are retardation of voluntary responses (motivational symptom) and sad affect (emotional symptom). In

addition, suicide attempt and suicidal ideation, lack of energy and apathy, sleep disturbance, difficulty in concentration and increasingly negative cognitions are symptoms.

The course of hopelessness depression can cover maintenance, recovery, relapse and recurrence. Maintenance is determined by the duration of the feelings and expectations of hopelessness, the stability of their attributions, and the inferences individuals make about consequence, self and causes. Recovery, as modelled by Needles and Abramson (1990), postulates that the occurrence of positive events gives the hopeless individual occasion to feel hopeful, and therefore become non-depressed. In natural conclusion to this, the relapse or recurrence of hopelessness depression can be predicted by the re-appearance of hopelessness.

In many ways, hopelessness depression is similar to Beck's (1967) theory. Both stress the importance of maladaptive inference in depression, demonstrate an important role for hopelessness, and have diathesis-stress components. However, they differ importantly as well. Beck does not postulate any sub-types of depression, i.e. hopelessness depression, but focuses on cognitive processes in aetiology, maintenance and treatment, whereas hopelessness depression theory also emphasises the role of environment in these processes. A further difference is that hopelessness depression theory does not formally characterise depressive cognitions, but does specify vulnerability factors for depressive symptoms, whereas Beck's theory does characterise depressive symptoms but does not specify vulnerability factors. Abramson et al. (1989) suggest that what is interesting to the depression researcher is not why individuals develop depression when confronted with a negative life event, but why some people do not succumb. Hopelessness theory is able to explain why hope is lost, but is also able to explain how hope can be re-established or endure.

2.1.6 The Effects of Depression on Memory

Veiel (1997) notes that there is much evidence to suggest that depression leads to impaired memory performance. However, he lists some of the problems incurred in interpreting the results of many analyses as being the existence of large age differences between patient and control groups, the failure to match groups for education, the inclusion in some patient groups with structural damage, such as Parkinson's and Alzheimer's disease, and the difficulty in ensuring that older adult subjects do not have underlying degenerative brain damage. Kalska, Punamaki, Makinen-Pelli, and Saarinen (1999) note similar issues, but also mention that there is little work that demonstrates the performance of those under the age of 65 who have major depression, and very little research that includes a comparison with a control group.

Using a younger subject group with depression, Veiel (1997) utilised a particularly comprehensive assessment battery (for details, refer to Veiel, 1997). He concluded that the most significant difficulties were experienced in cognitive flexibility/control, verbal fluency, scanning and visuo-motor tracking and executive functioning as a whole. He comments that the degree of impairment in some areas of neuropsychological functioning is comparable to that exhibited by those with 'severe' brain injury (Veiel, 1997)

Although not unequivocal, there is some agreement as to which memory functions are affected in those with depression. These include poorer visual than verbal memory (Deptula, Manevitz, & Yozawitz, 1991), impaired visuospatial memory (Miller, Fujioka, Chapman, & Chapman, 1995) though Calev and Erwin (1985) do not support this, and the preservation of short term memory in comparison to long-term memory which is somewhat affected (Austin et al., 1992; Channon, Baker, & Robertson, 1993). In addition, recognition has been shown to be unaffected whereas free recall seems to be more arduous for depressed patients (Ilsley,

Moffoot, & O'Carroll, 1995) although, Golinkoff and Sweeney (1989) found otherwise, and there is the suggestion that semantic priming is preserved in those with depression (Georgieff, Dominey, Michel, Marie-Cardine, & Dalery, 1998).

Reasons to explain these potential difficulties include the increased difficulty in carrying out effortful tasks, such as recalling information from long-term memory, and that incidental and more automatic memories that do not require this effort are therefore less affected. Veiel (1997) has found that metabolic activity in frontal sub-cortical areas is decreased in depressed subjects, and he hypothesised that these areas are pivotal in carrying out effortful processes associated with memory, and that these structures are also used to enhance cognitive speed and flexibility, which have been shown to be deficient in those with depression. Complaints of poor memory have also been found to be associated with low mood (Grut et al., 1993).

It has also been shown (Roth & Rehm, 1980; Silfe & Weaver, 1992) that depressed individuals are less able to predict and rate their performance, and are also less able to alter their performance after receiving feedback. This monitoring has been referred to as metamemory, i.e. one's knowledge and judgement regarding one's own memory performance (Dixon & Hultsch, 1983). Kalska et al. (1999) set out to determine the differences, if any, between the memory awareness (metamemory) of a depressed patient (with no structural brain lesions) and a control group, in relation to general and task-specific performance. In addition, they wished to determine the differences in memory performance in depressed patients with different symptoms, and the associations between actual, and awareness of, memory performance.

The performance of the depressed group confirmed Veiel's (1997) finding of impaired cognitive speed and flexibility, and suggested that specific impairment in memory functioning may be present (Kalska et al., 1999). Immediate and delayed visual recall were most impaired whilst verbal recall, short-term memory (STM) and recognition were less affected. This data confirms previous work that free recall is more affected, while STM remains preserved (Channon et al., 1993). The observed deficits could be a consequence of reduced expenditure of effort, which is supported by neuroimaging studies showing that depression can lead to temporary changes in cerebral functioning which is associated with impairment in effortful memory processes (Veiel, 1997).

Considering variation of performance in relation to presentation of depressive symptoms, it was found that cognitive, physiological and behavioural aspects of depression were 'differentially associated with memory performance' (Kalska et al., 1999). Physiological symptoms seemed to lead to impairment in STM. Up until the present time, research has suggested that state anxiety produces such an effect, not depression. This finding may be novel, but it is possible that it may be due to underlying anxiety disorders in the depressed group that were not considered in the analysis. Cognitive symptoms of depression were found to correlate with impaired visual memory, and those with behavioural complaints showed deficits in logical memory. No relationship was observed between the severity of reported symptoms and memory performance, rather it was the 'phenomenological nature' of the depression (at least in mild to moderate cases) that predicted the type of memory deficit.

Metamemory and actual memory performance were found to be discrepant among depressed subjects. There was no suggestion of distorted metamemory accuracy in the depressed patients but they did display a tendency to underestimate their actual general memory

performance, which may have been a consequence of their negative self-schemata or mood impinging on their self-appraisals of performance (Kalska et al., 1999). However, some caution is needed in interpreting these results in the light of the sample size (30) and that the subjects were inpatients on medication that potentially could have affected their cognitive functioning.

Depressed people have been shown to have frontal lobe dysfunction in terms of the function of neurotransmitters (Baker, Frith, & Dolan, 1997). The function of the central executive (Baddeley & Hitch, 1974) is thought to be centred within the frontal lobes and consequently, this dysfunction is hypothesised to have an effect on its working. The role of the central executive is essentially to 'allocate attentional resources in the processing and manipulation of information in executive operations' (Channon & Green, 1999, p. 162), and depression is thought to lead to the decreased efficiency of the working of the central executive (Channon, 1996). It is hypothesised that the presence of negative self-related thoughts would take up some of the capacity of the central executive thus leaving it without the resources it needs to work to its full potential in an individual (Teasdale & Barnard, 1993).

Channon and Green (1999) aimed to determine whether there was an observable difference in performance of depressed patients on tasks known to rely on executive function, depending on whether or not they had been given helpful suggestions as to the way to perform the test appropriately. They hypothesised that those given hints would perform better than those who were not. In addition, they were interested in comparing the performance of the depressed group with that of the control group. The tasks were a memory for categorised words task, a response suppression task and a multiple scheduling task.

Depressed individuals performed at a lower level than controls on all three tasks. They were not inclined to utilise appropriate strategies independently in order to perform the tasks efficiently, and when a strategy was supplied, it did not enable the depressed group to perform significantly better than they did without it. The results also showed that although the depressed patients used similar performance strategies to the controls, they did so less often. The differences in the level of performance between the control and depressed groups when strategies were provided may be due to the tendency of the depressed group to make less use of them, or to use them in a less efficient manner.

Memory deficits are commonly reported in those with depression but they are neither observed in all patients, nor to the same extent in all individuals (Yozawitz, 1986). Post (1992) proposed the depressive kindling hypothesis that suggests that fundamental neurochemical changes occur as a function of successive depressive episodes. The consequence of these changes is that normal cerebral function decreases resulting in a lowered threshold for onset of subsequent depressive episodes. Therefore, it is possible that this population may exhibit diminished performance on certain tests of memory in comparison to those who have had only one episode.

Basso and Bornstein (1999) compared two inpatient adult (age range 20 – 65) groups, one consisting of those experiencing a first episode of major depression, and the other being comprised of those who had experienced recurrent episodes of depression. Subjects were matched for age, education, demographic status and medical history. They hypothesised that the recurrent episode group would exhibit a poorer performance on the California Verbal Learning Test (that taps acquisition, retrieval and retention), and found that this group did demonstrate significantly more memory dysfunction in the area of deficits in acquisition (as

opposed to retrieval or retention), whilst the single episode group displayed a normal performance on the task. They also administered the block design and vocabulary subtests of the WAIS-R, and observed no significant differences on results of the block design and vocabulary subtests, that are accepted to be highly correlated with IQ. Sheline, Wang, Gado, Csernansky, and Vannier (1996) note that functional neuroimaging studies confirm that there is hippocampal atrophy in those with major depression and suggest that the degree of it is related to the length of depressive illness.

The literature review, thus far, has focussed on a number of contributing factors to depression, the effects of depression on memory and cognitive theories of depression. The following section explores the area of trauma, and the effects of fear on the physiological structure of the brain and on the encoding and recalling of experiences.

2.2 Trauma

2.2.1 Definition of Trauma

Traumatic experiences can loosely be defined as life experiences that are both psychologically and physiologically disruptive to the individual, even when no physical or direct damage has been done to the body (DSM IV, 1994; van der Kolk et al., 1996). In addition to this, these experiences do not need to be directly life threatening (as in child abuse). The individual attempts to process experiences in order to give them a sense of context and meaning within his or her memory. However, when the experience has been sufficiently disruptive it appears that it is not necessarily processed in the same manner as ordinary material (LeDoux, 1992; Nadel & Jacobs, 1996; Perry et al., 1995). It tends to be halted at the sensory and emotional level of encoding, and its transfer to the areas of the brain

that would normally give it meaning and semantic cohesion is disrupted (Rauch et al., 1996; van der Kolk et al., 1996).

Posttraumatic Stress Disorder (PTSD) as a diagnostic category first appeared in the DSM-III (APA) in 1980 but has subsequently been reviewed in definition and criteria (DSM-IV, 1994; Rothschild, 2000). The typical symptoms of PTSD include the re-experiencing of the event (for example, through flashbacks or recurrent and intrusive thoughts, images or perceptions of it), the avoidance of reminders of the trauma and a numbing of general responsiveness (such as, feeling detached, loss of interest in significant activities), and hyperarousal of the autonomic nervous system (for example, hypervigilance, sleep problems, irritability). Such symptoms need to be of more than one-month duration coupled with a decrease in the capacity of the individual to function in areas such as work or social relationships (DSM-IV, 1994). A diagnosis of PTSD can be accompanied with the specifiers of acute (duration of symptoms is less than 3 months), chronic (duration of symptoms is 3 months or more), or with delayed onset (i.e. at least 6 months have passed between the event and the onset of symptoms) (DSM-IV, 1994).

An associated diagnosis is that of Acute Stress Disorder (DSM-IV, 1994) wherein the diagnostic criteria are very similar to those required for PTSD but the duration of symptoms should be a minimum of 2 days and a maximum of 4 weeks and occur within 4 weeks of the traumatic event. Therefore, PTSD cannot be used as a diagnosis within the first month of symptoms appearing, but can thereafter be considered should they persist beyond this time restraint.

2.2.2 Prevalence of Trauma

It is estimated that only 20% of those who experience trauma will develop PTSD (Rothschild, 2000). Mediating factors such as psychological resilience, childhood experiences, social and familial support, personality characteristics, vulnerability to stress, premorbid state, pre-existing mental disorders and so forth appear to be important in the question of why some people develop conditions in response to traumatic events and others do not (Bowlby, 1977, 1980; Crittende, 1990; de Zulueta, 1993; DSM-IV, 1994; McFarlane & de Girolamo, 1996; McFarlane & Yehuda, 1996; Rothschild, 2000).

The prevalence of Acute Stress Disorder tends to be dependent on the severity and persistence of the trauma and the degree of exposure to it. The figures for community based studies range from 1% - 14% for lifetime prevalence, but can vary from 3% - 58% in at-risk individuals, such as victims of criminal violence or combat veterans (DSM-IV, 1994).

2.2.3 The Effects of Fear on Memory

As we develop, the brain organises responses to experiences in the cognitive, motor, emotional and 'state-regulating' areas, it creates internal representations of the external environment and it stores associations between pieces of information, such as sights, sounds and smells (Perry, 1999, Rothschild, 2000; van der Kolk, 1996). Most of the organising and development of the brain takes place within the first three years of life, resulting at this age in it being 90% of the size of an adult brain. The lower parts of the brain, regulating breathing, heart rate and so forth, remain constant once developed whereas higher areas of the brain, that are responsible for more abstract processes, retain a considerable degree of plasticity throughout life and thus are susceptible to neurophysiological change according to the

experiences they register (LeDoux, 1992; Perry, 1999; Perry et al., 1995; van der Kolk., 1996).

In relation to trauma, two processes of the memory system seem to play an important part. The first is that of 'declarative' or 'explicit' memory that involves the individual being consciously aware of facts or events that have taken place in his or her life before they are stored away in memory. The second is referred to as 'nondeclarative', 'implicit' or 'procedural' memory. This mostly relates to memories that the individual is not usually fully aware of. For example, this memory system would involve reflexive actions, emotional responses, or the acquisition of skills learnt over a period of time (Squire, 1994).

Individuals tend to remember by integrating an experience or piece of information into their mental schemata so that it no longer remains as a separate or fragmented experience but becomes part of, and fits into, their cognitive schema. This in turn allows for the experience to be translated into semantic, sequential and coherent narrative (van der Kolk, 1996; Rothschild, 2000). However, once information is integrated into the schemata and greater memory pool it may be subject to distortion or alteration when associated experiences occur or by the individual's emotional state when recalling the experience (Rothschild, 2000; van der Kolk, 1996). van der Kolk (1996) cites the studies of Brown and Kulik (1977) and Reisser and Harch (1992) whose work suggested that whereas memories for significant cultural or societal events, such as the shooting of President Kennedy, are subject to some distortion and disintegration over time, other research by Bohannon (1990), Christianson (1992) and van der Kolk (1996) has shown that memories for very personal and significant events, ones that directly affect one, are particularly accurate and stable over time. However, once these memories, or fragments thereof, are verbalised and integrated into some form of

semantic narrative they too may become vulnerable to some distortion (van der Kolk & van der Hart, 1991).

In line with Ingram's Information Processing Paradigm (1984) and Teasdale's (1988) Differential Activation Hypothesis, recent research (such as, LeDoux, 1992; Squire, 1994; and van der Kolk, 1996) has helped to establish the concept that memory systems are comprised of networks of related information. When a relevant network is activated it can then lead to the retrieval of memories and information, whether stored as cognitive schemata or as fragments, which have been stored along specific associative pathways. Key triggers or cues to set off this process of activation are either cognitive in nature, or more frequently, emotions or sensations.

Memories of experiences that have been more personally traumatic and 'indigestible' tend to be stored at a sensory or emotional level rather than at the integrated cognitive level. As a result of this, sensory or emotional cues that are similar to those experienced during the trauma can trigger the re-experiencing of the index event at the same levels (i.e. emotional or sensory) and in the same fragmented manner. These emotional or sensory cues can also lead to the activation of the memory networks associated with the specific cognitive schemata attached to that experience. In other words, an emotional or sensory stimulus can trigger a memory that would otherwise not have been in consciousness. If the trigger was not there, then those memories would remain dormant or at a subconscious level. If cognitions are also activated, they will be those that are attached to or associated with the traumatic experience. Therefore, cognitive schemata associated with the traumatic experiences will be activated when associated networks are aroused, and normal or non-traumatic ones will be activated by non-threatening stimuli. Thus, cognitions are as state-dependent as emotions (LeDoux, 1992;

Perry, 1999; van der Kolk, van der Hart, & Marmar, 1996). One could therefore say, albeit it in a simplified form, that with each experience or set of events (traumatic or otherwise) an idiosyncratic emotional, sensory and cognitive memory state is developed. There is some evidence (de Zulueta, 1993; van der Kolk, Hostetler, Herron & Fisler, 1994) that supports Freud's (Storr, 1989) original view of people becoming fixated at the stage at which deprivation, or trauma, had first been experienced, and that future experiences and information tends to be processed in a manner commensurate with that phase of developmental capacity.

The brainstem is, broadly speaking, responsible for the regulating of the system. Two predominant structures of the brainstem are the thalmic and limbic systems. The thalamus is the 'relay centre' for sensory input and information that is received from the body en route to the cortex. The limbic system is thought to be the powerhouse involved in emotional and behavioural responses associated with self-preservation and survival and is comprised of two major components, the amygdala and the hippocampus (van der Kolk, 1996). The amygdala tends to be mature at birth whilst the hippocampus only matures between the second and third year of life (Nadel & Zola-Morgan, 1984). It is believed that the amygdala assists in processing and storing highly charged emotional memories and is not affected during traumatic experiences, but that sensory information goes via the hippocampus en route to the cortex and that trauma can interfere with this process (Rothschild, 2000; van der Kolk, 1996). The hippocampus is thought to assist in giving an event or experience a context of time and sequencing, and to be more dependent on language and cognitions to process information. Normally, information once having gone through the hippocampus is then further processed by the cortex, which is responsible for higher mental functions such as speech, thought,

semantic and procedural memory, and for integration and planning (Perry, 1999; Rothschild, 2000; van der Kolk, 1996).

The brain senses, perceives, processes, stores and mobilises in response to threatening information from both external and internal environments in order to promote survival. This survival 'mechanism' is fast acting and of ultimate importance. When a threat is perceived, i.e. sensory information reaches the brain stem and is not recognised or is associated with a previous threat, the brain activates warning signals via neurotransmitters (norepinephrine, serotonin, dopamine) that create a 'near-reflexive' response in the individual. It is only momentarily later, in the higher areas of the brain, that this information is interpreted as being a real threat or a false alarm. Activation of the thalamic and limbic areas creates the sensation of anxiety, which is interpreted and allocated cognitive associations by the cortex. These reactions are then stored as memories of the event that lead to the reaction (Perry, 1999). Research by Yehuda, Resnick, Kahana, & Giller (1986) has found that people who suffer from PTSD do not seem to secrete enough cortisol that assists the body in reverting back to a normal state after arousal.

The classic response to threatening cues, the fight or flight reaction, leads to the activation of the autonomic nervous system which results in physiological manifestations of alarm, such as arousal and emotion via profuse sweating, tachycardia and rapid respiration (Cannon, 1929). This leads to the storing of a 'cue-evoked state memory' that the brain stores in association with fear, and in future, it will act in response to this pattern of neuronal activity. The human brain has an extraordinary capacity to make associations between paired cues, for example it pairs the roar of a lion with potential threat. These paired associations are then rapidly generalised so that other similar threatening stimuli can be readily identified in order to

survive. However, these generalisations, and encoded responses such as physiological arousal, may lead to responses to new non-threatening cues (false alarms) and result in an ongoing 'fear' state ready for activation. For example, if an individual with a history of a traumatic experience(s) such as physical assault experienced a rise in heart rate at the time of the trauma, when he or she then goes to the gym and exercises his or her heart rate increases, due to a non-threatening cue (exercise), but this increase in heart rate leads to the triggering of a brainstem-mediated alarm response in line with the state memory (of the abuse) that has been stored (LeDoux, 1992; Perry, 1999; Solomon, Garb, Bleich, & Grupper, 1987; van der Kolk, 1996). It is also possible that some sensory cues may be genetically coded to lead to an alarm state, for example, all infants tend to become distressed following loud sudden noises. Consequently, information can be acted on by the brainstem before it has a chance to be classified as harmless by the cortex. It is through this process of paired associations, generalisation and stored memories that exposure to traumatic experiences can significantly alter the sensitivity of the brain's alarm system that can result in a state of fear or anxiety arising from non-threatening cues (Perry, 1999).

Subsequent to the initial experience of trauma, recalling the event may lead to physiological sensations associated with it. Conversely, experiencing the physiological state may lead to recall of the memory initially associated with this (Kolb, 1987; Perry, 1999; van der Kolk, 1994; van der Kolk, Greenberg, Boyd, & Krystal, 1985). However, it is not uncommon for individuals to be unaware of this connection, and to only feel, for example, the depression or fear (Perry, 1999, van der Kolk, 1996). Perry (1999) also makes reference to how the actual feeding of an infant, and the nurturing and intimate atmosphere associated with it, may affect the development of patterned neuronal activities concerned with attachment, emotional regulation, normal eating patterns and relationship formation, for it is during this infancy

period that the brain develops these specific neuronal patterns that remain for many years to come, if not for life. This concept has also found favour in the works of others such as LeDoux (1992), de Zulueta (1993), Rothschild (2000) and van der Kolk (1994).

Consequently, all areas of the brain can change in response to patterned neuronal activity and are therefore capable of storing memories. Cognitive, emotional and motor memories are a result of 'use-dependent' changes (i.e. the more a path is used the more it becomes entrenched) in neuronal patterns of activity during the process, for example. learning languages, grief/joy, riding a bike and so forth (Perry, 1999; Rawlins, 1980; van der Kolk, 1994). State memories are stored when a pathway is entrenched in state-regulating parts of the brain that are sensitising, chronic or prolonged, for example chronic abuse, traumatic stress or violence (Perry, 1999; Rawlins, 1980; van der Kolk, 1994). Patterns of activity caused by traumatic events will be vastly different from those evoked by everyday activity and will have an effect on the functioning of the brain from the brainstem (physiological state regulation) to the cortex (cognitive functioning) (Perry, 1999; Rothschild, 2000; van der Kolk, 1994; van der Kolk et al., 1985).

During a traumatic experience threatening stimuli activate the limbic system in order to prepare for flight or fight. The entire human system is in a state of arousal. If arousal is extreme, as in trauma, the flow of the sensory information that should then proceed to the hippocamus for further processing is disrupted, and these pieces of experiences are stored as fragments of memories in the form of affective states or sensorimotor modalities (van der Kolk, 1996). With trauma, there is no sense of sequencing (i.e. a beginning, a middle and an end), and there is evidence (Nadel & Jacobs, 1996; van der Kolk, 1996) that the activity of the hippocampus is suppressed during a traumatic experience so events are not contexualised

and stored, and thus can continually be re-experienced as if in the present. In addition to this, the Broca's area of the brain, which is associated with translating experience into language, has also been found to shut down during trauma (Rauch et al., 1996; van der Kolk, 1996). Hence, people who have been traumatised have described a sense of 'speechless terror' (Rauch et al., 1996). This shutting down of the Broca's area combined with the disruption between the information going through the amygdala and reaching the hippocampus, may be the reason for traumatised people having difficulty putting an emotional experience into words and formulating it as a coherent mental construct or relating it as a personal narrative.

Wills & Goodwin (1996) are of the opinion that trauma can also affect the stages of normal information processing and that chronic exposure to fear can lead to overload and the less efficient processing of incoming information. They conclude that the consequences of this can range from poor learning in the classroom to withdrawal, desensitisation and dissociative amnesia. This inefficient information processing may lead to sequences of events being stored erratically, or in such a way that it is not clear that they are associated with each other, leaving the individual unable to construct a narrative to describe and explain the events. Subsequent sensory experiences can act as triggers to these emotional and seemingly unconnected memories and thus the experience is re-experienced, as found in PTSD (Perry, 1999; Volpe, 1996). There is evidence (Reynolds & Brewin, 1998) supporting the potential co-morbidity of PTSD and depression, and that the two disorders share certain characteristics, such as intrusive memories.

Memories of events, experiences and material that are personal are referred to as autobiographical memories. Autobiographical memory has featured in a large proportion of published work associated with depression over the past two decades (for example: Moore,

Watts & Williams, 1988; Perris, Holmgren, von Knorring, & Perris, 1986; Post, 1992; Reynolds & Brewin, 1998, 1999; Spenceley & Jerrom, 1997; van der Kolk et al., 1994).

2.3 The Role of Autobiographical Memory in Depression

Autobiographical memory is defined as a "subclassification of memory within the declarative memory system and signifies memory for one's own personal life experiences in the recent and/or remote past" (Parks & Balon, 1995 , p. 199). Rubin (1996) has proposed that when people wish to recall information from their past, they initially access higher level more general descriptions and use this information as a means of directing the path towards the lower and more specific 'event representations'. Thus, autobiographical memory is like a hierarchy of personal information through which one can make one's way. Depressed individuals, however, may not be able to achieve this smooth passage through the hierarchical layers, their progress halting when they reach the level that holds the more specific information. This phenomenon has been demonstrated in suicidal patients (Williams & Dritschel, 1988), depressed individuals (Kuyken & Brewin, 1995), and those suffering from PTSD (McNally et al., 1995).

Kuyken & Brewin (1995) have suggested that this halting search at general memories may have a protective effect in that it prevents the recall of overly emotional and painful information. However, it can also have a maladaptive effect. Depressed individuals have been shown to demonstrate overgenerality for positive as well as neutral events, difficulty with problem solving and envisaging the future in any specific way, and they tend to find it harder to recover from their illness. Williams et al. (1997) have shown that this cognitive style is not mood dependent, meaning that improvement in mood does not automatically lead to an improvement in the memory of material or cognitive functioning.

In an attempt to find markers of cognitive vulnerability, Brittlebank, Scott, Williams, and Ferrier (1993) looked at the role of autobiographical memory in depression. They found that depressogenic schemas and depressive cognitions would result in a poor prognosis for depressed individuals, and that these would manifest themselves as overgeneralised recall of emotional autobiographical memories, that is, depressed people would recall general memories around a feeling or word, such as 'sad', rather than recall a specific memory about one 'event'. For example, a person may respond to the word 'sad' by saying 'I felt sad whenever I was left alone', rather than 'I felt sad when my dog died'. Moore, Watts, and Williams (1988), had previously shown that depressed individuals were more likely to respond to emotional cue words with overgeneralised autobiographical memories. Overgeneralisation of memories could be seen as a product of the depressed individual's limited working memory being predominantly occupied by intrusive memories, thus allowing little processing time for the retrieval of specific memories. The subjects completed an Autobiographical Memory test (AM) and the Dysfunctional Attitude Scale (DAS). The DAS was found to be a poor predictor of subsequent severity of symptoms, but the number of overgeneralised autobiographical memories strongly predicted the severity of depression at 3 and 7 months. Patients who demonstrated over-general recall were also less responsive to anti-depressant treatment. Importantly, however, over-generalisation was not associated with failure to recover. This, argued Moore et al. (1988), demonstrates that individual differences in processing autobiographical memories can exacerbate individuals' vulnerability to the maintenance of depression, but not overall ability to recover.

Parks and Balon (1995) investigated patterns of recall for autobiographical events in childhood in psychiatric patients with a history of alleged trauma. In comparison to the non-patient group and the patient group without alleged history of trauma, psychiatric patients

with history of trauma took longer to recall memories and failed to retrieve a memory more frequently. Unlike the two control groups, the trauma patients recounted fewer early memories, their earliest memories were of a later age, and they provided fewer memories for affective cues than did the control groups.

They maintained that in these patients, conditions of hypervigilance, anxiety and increased arousal were common and frequent characteristics of their daily existence, and, consequently, the resources available to allocate to the encoding of events into memory were somewhat diminished. Consequently, when attempts were made to recall this information, it was more difficult to remember and required more effort. Their findings supported the work of Christianson and Nilsson (1984).

Researchers such as Baker and Duncan (1985) and Palmer et al., (1992) have established that depressed individuals have experienced significantly more early childhood sexual abuse than non-depressed individuals, and that the depressive state is characterised by high levels of intrusive memories (Spenceley & Jerrom, 1997). Kuyken and Brewin (1994) looked more specifically at intrusive memories of child abuse during depressive episodes. They hypothesised that higher levels of intrusion and avoidance would be associated with repeated rather than non-repeated childhood abuse, with intercourse rather than non-intercourse sexual abuse, and with abuse from a primary care-giver rather than a non-primary care-giver. They demonstrated significantly higher levels of intrusion and a trend towards higher avoidance within all of these categories. This stresses the importance of actual specific stress experiences, not just abstract activated negative schema about the self, in intrusive memories.

Taking into consideration their previous findings and the work of Brittlebank et al., (1993), Kuyken and Brewin (1995) predicted that depressed patients reporting a history of high levels of negative emotional experiences would demonstrate faster recall of negative autobiographical memories, and more unrelated general memories. They compared depressed individuals who had experienced childhood sexual abuse (CSA) with those who had experienced childhood physical abuse (CPA) to those who had no experience of abuse. As predicted, the results showed that depressed individuals with the most adverse intrusive memories (CSA) retrieved significantly more inappropriately general memories to both positive and negative cues. Levels of avoidance were also shown to have an effect on overgeneralisation of memories, indicating that avoidance affects depressed subjects' performance on information processing tasks. However, early childhood adversity did not facilitate the faster retrieval of negative autobiographical memories. They demonstrated that intrusive memories are an index of abnormal cognitive functioning in those with depression.

Subsequently, Brewin et al. (1996) demonstrated that intrusive memories of negative experiences did not predict chronicity or recurrence of depression, nor was severity of depression correlated with number of intrusions. However, longer episodes of depression were related to a greater number of intrusive memories. They looked at a broad range of negative intrusive memories ranging from death and injury, family and relationship problems to financial worries. They found that these memories were predominantly associated with the emotions of sadness, anger, guilt and helplessness, and, to a lesser extent, with anxiety and shame. They showed that memories of assault and abuse, and current relationship problems, were also associated with particularly high levels of intrusion.

Much of the research into the role of autobiographical memories has been conducted in relation to PTSD. Reynolds and Brewin (1998, 1999) investigated intrusive cognitions, coping strategies and emotional responses in both depression and post-traumatic stress disorder as well as in a non-clinical population. They state that researchers commonly find that those with major depression experience intrusive memories of specific autobiographical events that are much like those characteristic of PTSD (Brewin et al., 1996). The authors questioned the three groups about intrusive thoughts and memories associated with traumatic life events.

Comparing these cognitions, and the emotional reactions to them among the three groups, they found that the depressed and PTSD groups were similar in their intrusive cognitions, with the most frequent form being that of intrusive thoughts. These thoughts were consistent with the work of Beck (1967), i.e. they focussed on the presence of negative evaluative thoughts about the self, the world and the future. The means of coping adopted by the clinical subjects in this study included distraction and suppression, whilst the control group reported thinking through and talking.

PTSD is generally characterised by the re-experiencing of symptoms whereas depression is not (Reynolds & Brewin, 1999). However, it has been found that depressed individuals experience intrusive memories at a level equivalent to PTSD (Brewin et al., 1996), and that co-morbidity with depression is common in PTSD (McNally, 1992). In general, it is characteristic of those with depression to be unable to retrieve a specific autobiographical memory in response to a cue word, but rather to recall a period of their life or a series of events (Williams, 1992; Brittlebank et al., 1993), and this has also been observed in PTSD (Mc Nally, Lasko, Macklin, & Pitman, 1995).

Brewin, et al. (1999) found that, once symptom levels are controlled for, the severity of depression at follow-up could be predicted by the amount of intrusion and avoidance of stressful memories, but it could not be predicted by the degree of distress associated with intrusive memories and the tendency to produce overgeneral memories on an autobiographical memory test. They confirmed previous findings that depressed individuals have memories of specific events that frequently intrude into consciousness in much the same way as is experienced by those suffering from PTSD (McNally et al., 1995). Of note is that men were found to report fewer intrusive memories than women. They also state that intrusive thoughts are constituents of normal cognition albeit at lower frequencies.

The authors suggest that the state of remission may be characterised by a decrease in intrusions of memory but not in the effort to avoid such memories. The effort taken to continue to ensure these memories are avoided may be key in the relapse process. Baseline measures taken by the authors of avoidance and intrusion were found to predict depression severity at follow-up. The authors note that as in PTSD, these memories influence the course of the illness. When subjects who had experienced assault or abuse (highly associated with the development of PTSD), were excluded from the analysis, the results remained the same.

Brewin et al. (1999) provide two suggestions as to why the intrusion of memories may lead to a poorer course for depression. Firstly, the activation of these memories may lead to the accessing of more general negative beliefs and ideas that go on to impede recovery. Secondly, the intrusive memories may monopolise resources within working memory thus decreasing resources allocated to concentration, attention, encoding and so forth, which leads to the experience of difficulty in carrying out other tasks.

Regarding the similarities between depression and PTSD and the tendency for there to be recall of autobiographical information, a difference should be noted. In PTSD the most common cognition is usually relating to a memory of a specific traumatic event whereas in depression, the recurring cognition is more likely to be an 'evaluative thought' (Brewin et al., 1999).

A number of theories have been proposed to explain the relationship between trauma and autobiographical memory. Attempts have been made to explain why a traumatic memory that a person has been unaware of for a long period of time is suddenly recalled. The first is the repression hypothesis (Davis & Schwartz, 1987) that posits that memory for traumatic events is repressed and denied in order to protect the self. Limited rehearsal of material related to the self leads to inadequate encoding of information and weak associative pathways and hence repression (Davis & Schwartz, 1987). Davis and Schwartz, (1987) demonstrated that individuals who repressed information remembered fewer negative memories from their childhood. This poor encoding may be a result of diminished attentional resources resulting from the high levels of emotional and physiological arousal experienced during traumatic times (Christianson & Nilsson, 1984).

The second hypothesis, dissociation (Parks & Balon, 1995), maintains that the individual has an 'asymmetrical pattern of amnesia for his or her personal history' (Parks & Balon, 1995, p. 200). This finds support in the work of Nissen, Ross, Willingham, Mackenzie and Schacter (1988) who found that individuals with multiple personality disorder exhibit no transfer of memory to other personalities in explicit and implicit memory tasks, hence suggesting successful dissociation.

Herman (1992) has argued that there are a large number of people who live with repressed memories of childhood abuse, and Pope and Hudson (1995) surveyed the literature to determine whether this was possible. They found only four studies meeting their strict criteria and subsequently found that none of these provided absolute confirmation of abuse, nor proof that the subjects had repressed this information. They noted, however, the difficulty that exists in determining whether abuse has occurred and in proving amnesia.

Howe and Courage (1997) claim that there is no evidence for the possibility of a 'discontinuity in memory' as the fundamental components of the information processing system are in place (the 'hardware'), and that the cause of lack of memory for events prior to this time is due to the 'software' of the memory (i.e. strategy use, metamemory, acquired knowledge and so forth). They refer to this as the development of the cognitive self that they propose coincides with self-recognition.

2.4 Repressive Coping Style

Research into coping styles of people in recovery from major depression is still in an early phase. It differs from one coping style that has been researched, namely, the "repressive coping style" (Weinberger, Schwartz, & Davidson, 1979). Research highlights how individuals with this coping style inhibit memories of childhood experience, and this could serve as a useful comparison when considering what occurs when individuals start to recover from depression. The difficulty that then ensues is how to define and measure a repressive coping style. The most widely acceptable definition and measurement were devised by Weinberger et al. (1979). They defined the repressor as an individual who scored low on self report measures of anxiety (as measured by the Taylor Manifest Anxiety Scale) (TMAS), but high on defensiveness (as measured by the Marlow-Crowne Social Desirability Scale)

(MCSDS). Within these parameters, they proposed three personality types, High Anxious, Low Anxious and Defensive High Anxious (see Table 1).

Table 1. Personality Types according to Weinberger, Schwartz & Davidson (1979)

	Defensiveness Low	Defensiveness High
Anxiety Low	**Low Anxious**	**Repressors**
Anxiety High	**High Anxious**	**Defensive High Anxious**

The repressive coping style is seen as a personality trait, and it is unlikely that a repressor would become depressed because depression implies a break down in the ability to repress. Recovering depressed individuals do not have a repressive coping style, only a seeming ability to inhibit negative material (Spenceley & Jerrom, 1997).

One of the characteristics of the repressive coping style is that unpleasant memories are kept from consciousness. Therefore, a repressor should exhibit low access to emotional memories, particularly negative emotional memories (Davis, 1987; Davis & Schwartz, 1987). Comparing repressors with low and high anxious and defensive high anxious individuals, Davis and Schwartz (1987) demonstrated that repressors had fewer negative memories. Additionally, their earliest negative memory was at a significantly older age, and surprisingly, they had significantly fewer positive memories. In extension, Davis (1987, 1990) looked at recall of childhood experience pertaining to the self or others. The prediction that repressors would recall fewer negative emotional experiences involving themselves was supported. She then looked at memories related to certain emotions, and demonstrated that repressors demonstrated longer retrieval latencies for fear and self-consciousness. This

suggests that repression is not a general processing strategy, but particular to certain experiences and situations.

2.5 Recovery from Depression

Major depression is characterised by fluctuating symptoms expressed over a period of time that fall onto a continuum of 'symptomatic severity' (Judd & Akiskal, 2000). Each phase of the illness may be represented by major, minor or dysthymic symptoms, and these symptoms are associated with psychosocial impairment that worsens in line with symptom severity. Judd and Akiskal (2000) claim that patients return to their optimal level of psychosocial functioning when they are asymptomatic and the greater the amount of time they are asymptomatic, the greater the likelihood that they will not relapse following an episode of depression. They maintain that minor, intermittent depression and major or acute depression do not represent two distinct diagnostic categories but lie on a continuum of unipolar depression. They further posit that the subsyndromal expression of depressive symptoms represents the longitudinal fluctuating course of major depression.

Subthreshold depressive (SD) symptoms, i.e. those that do not qualify for major depression, have been found on many occasions to equal the impairment criterion of clinical cases (Angst & Merikangas, 1997). They are described as two or more depressive symptoms lasting two or more weeks but otherwise falling short of the diagnostic criteria for minor or major depression. Judd et al. (1994) found the prevalence of such a situation to be present twice as often as major depression in the general population. The documentation of considerable psychosocial impairment in those suffering with SD, according to the authors, justifies the fact that this condition is not likely to be a variation of normal mood. In an investigation of data from an NIMH national sample, Judd et al. (1997) found that at one year follow-up, of

those initially diagnosed with major depression, only 28% continued with this diagnosis while others were classified as having minor depression, subthreshold depression or as having become asymptomatic. They found a similar variation among those with other diagnoses. Consequently, they concluded that over the course of time, the structure of depression within the same individual was subject to frequent change in levels of severity and symptoms.

The use of polysomnographic measurements has shown that there is no significant difference between depression at the personality, dysthymic, major depressive, minor and residual subthreshold levels. "They all appear to be part of a psychopathological continuum with the common denominator of a depressive trait which is neurophysiologically measurable as short REM latency" (Judd & Akiskal, 2000, p. 5), and this finding is consistent with that of Giles, Kupfer, Rush, and Roffwarg (1998).

In considering and attempting to predict the likelihood of relapse, it has been shown that those who experience asymptomatic remission are likely to have a longer period before a relapse than are those who experience residual subsyndromal remission (Judd et al., 1998b). In light of this, it would seem that it is beneficial to treat those with depressive symptoms until they become asymptomatic.

Marx, Williams, and Claridge (1992) have shown that people with depression have difficulties with social problem solving. However, most recovered depressed people are able to regain their ability to function socially (Stewart, Quitkin, & McGrath, 1988) although there is a minority who do not manage to do so. In a study of previous inpatients, Coryell, Endicott, and Keller (1990) found that this minority group remained substantially socially

impaired at two-year follow up. Those with more long-standing histories of depression are likely to function more poorly (Leader & Klein, 1996).

Agosti (1999) used an outpatient group all of whom had been responding to treatment for at least 6 months. It was hypothesised that those who showed greater social impairment would be suffering from intermittent depression and, in addition, would have experienced social difficulties during adolescence, and this was confirmed. Patients with a long and chronic history of depression were less likely to make a full recovery in terms of their ability to function well socially, whereas those who had a brief history were more likely to readjust. This social impairment may be the result of co-morbid pyschopathology that is undiagnosed (Agosti, 1999).

Lewinsohn, Allen, Seeley, and Gotlib (1999) used a prospective design with a sample of high school students (likely to contain more people who would experience a first onset of depression during the follow-up period than would be found in an older sample) and correctly predicted, in line with Post's kindling model (Post, 1992), that those adolescents with a prior history of major depression would show a stronger association between depressive symptoms and dysfunctional thinking.

They also examined a number of potential risk factors for first or recurrent onset of depression, namely, dysphoric mood, dysfunctional thinking and psychosocial stress. They found, again in support of Post, that the processes mediating initial and recurrent onset of depression were different. In those without a history of major depression, the combination of dysphoric mood and dysfunctional thinking did not pose a risk for the development of depression whereas in those with a history of depression, it did. Major stressors were

predictive of first episodes more so than recurrences, and the occurrence of 3 or more severe stressful life events could be considered a 'threshold' for risk of depression in those with no previous history. This supports Brown and Harris' work (1978). Importantly, with regard to the recovery from depression, there may be no relation between stress experienced and likelihood of a recurrent episode of depression, thus suggesting that those who have been depressed in the past are at risk regardless of the degree of stress they are experiencing (Lewinsohn et al., 1999). Zautra, Guarnaccia, and Reich (1989) have suggested that although severe stressors may not be related to risk of recurrence, minor events could play a significant role in increasing dysfunctional thinking or dysphoric mood in those with a history of depression.

Little research has been conducted to look at the recovery process from depression. The research (for example: Beck, 1976; Weissman, 1994; Williams, 1993) that has been carried out mainly looks at treatment and its outcomes. The formulation of hopelessness theory (Abramson et al., 1989; Abramson, Seligman, & Teasdale, 1978) has provided a theoretical framework in which to investigate recovery from depression. The process of recovery is similar to the process of onset. The experience of positive events can trigger the process to recovery, just as the experience of negative events can trigger off depression. Therefore, if a depressed individual has a tendency to attribute positive life events to global stable causes, they are likely to enter into the process of recovery.

Needles and Abramson (1990) were the first to look at the effect of positive and negative major life events on depression. They demonstrated that depressed individuals who gave global, stable attributions to positive events (enhanced attributional style) experienced significantly restored hopefulness when they had more positive life events. However,

subjects lacking an enhanced attributional style did not benefit from increased positive life events. They further demonstrated that subjects with enhanced attributional style demonstrated reduced levels of hopelessness when there was a decrease in negative life events. Therefore, a decrease in the negative functions is similar to an increase in the positive ones. This counters the ideas formed in previous research that negative life events play a larger role in depression than positive ones. It would seem that negative events have the larger role in the onset of depression, but that positive ones could play the larger role in recovery.

Negative cognitions are associated with poor recovery from depression and a high frequency of relapse following remission. Drawing on the work of Kelly's Personal Construct Theory 1955), Woolfolk et al. (1999) have demonstrated a close relationship between depression and the complex structuring of negative information within self-schema. They suggest that such people have a highly sophisticated framework of negative appraisals and evaluations that can explain almost any event. Extremely organised and discrete cognitive domains each containing negative evaluations are likely to lead to a very small chance that any positive information will be generalised across these domains. They suggest that the possession of positive means of self-evaluation that are related to internal and global factors would be beneficial in recovery from depression and this should become an ultimate goal for depressed individuals. However, they are unable to define the process by which one can instil such means of self-evaluation into those who suffer from depression.

Beck's work, and that of many others (for example, Linehan, 1993; Padesky, 1994; Safran & Segal, 1990; Williams, 1993; Young, 1990;), on methods to remediate negative and distorted cognitions and evaluative processes has a large following, but even this has its limitations and

by no means has it provided a definitive and all-encompassing theoretical or clinical structure, nor does it define the actual process of recovery.

The complex therapeutic work done with patients who have suffered trauma (de Zulueta, 1993; Michenbaum, 1994; Perry, 1999; Rothschild, 2000; van der Kolk, et al., 1996) too struggles to define the recovery process, though the work of those who marry the concepts of science and clinical practice (such as, Perry, 1999; van der Kolk et al., 1996) are providing important and vital clues to the workings of the recovery process. The field of neuroscience is by no means new, but its impact on the clinical sphere seems set to become greater and greater.

The directed forgetting paradigm (Bjork, 1989) is based on the conceptualisation that individuals are capable of consciously inhibiting unwanted material. This paradigm has been operationalised into a research measure, namely, the directed forgetting task.

2.6 The Directed Forgetting Paradigm

Myers, Brewin, and Power (1992) replicated Davis' study (1987), demonstrating that the age of the earliest negative memories in repressors was older, and that they had fewer negative memories. They concluded that repressors had limited access to negative childhood memories. However, they saw this as a product of a generalised capacity to inhibit unwanted material, not specific to autobiographical memory. This was paralleled by Bjork's (1989) conceptualisation of repression as an active inhibitory cognitive process. Information is inhibited to achieve a goal. Bjork based these ideas around the phenomenon of the directed forgetting paradigm.

In the directed forgetting task (Bjork, 1989), subjects are exposed to a list of words and directed to learn or make judgements about them. Half way through the task, half of the subjects are told that the learning/judging was only practice, and that they were to ignore what had occurred previously. The other half carried on as normal. At the end of the task, subjects were asked to recall all the words they had been exposed to, even those they had been cued to forget. The directed forgetting effect is evident in the fact that the to-be-forgotten (TBF) words are recalled significantly less than the to-be-remembered (TBR) ones. However, the TBF words are still present in memory, because in a recognition task, subjects recognise TBF words to the same level as TBR ones. Bjork (1989) proposed that a cue to forget sets up a retrieval inhibition.

If repressors have a general coping style that facilitates the inhibition of unwanted material, repressors should remember significantly less TBF words on the directed forgetting task (Myers et al., 1992). However, Myers et al. (1992) were not able to find a significant difference between recall of TBF and TBR in repressor and control groups. They did demonstrate that repressors were significantly slower in retrieval of negative cue words of anger, fear, hurt and loneliness in the autobiographical memory test. These results suggest the general repression of negative early childhood experiences. Combined with Davis' data (Davis, 1987; Davis & Schwartz, 1987), this suggests there are two cognitive processes working in repression, namely, reduced access to memories of negative childhood experiences and a slower retrieval of childhood memories. Also, the lack of significant difference between groups on the directed forgetting task would suggest that repressors do not have a general capacity to inhibit items in memory, only those related to negative emotional memories and feelings. In a further study, Myers and Brewin (1994) demonstrated that repressors had significantly slower latencies of retrieval for negative memories than

controls. To cement their previous conclusion (Myers & Brewin, 1995) that repressors have deficits in more than just autobiographical memories, Myers, et al. (1998) looked at repressors' retrieval of negatively valenced emotional words in the directed forgetting task. They predicted that repressors would recall significantly fewer negative words from the to-be-forgotten (TBF) set, and this was supported in their findings. In their second experiment, they demonstrated that repressors' incidental recall of negative TBF words was significantly worse than that of the non-repressor group.

Repressors cope with negative childhood experiences by directing their attention away from negative affective cues. Cloitre, Cancienne, Brodsky, Dulit, and Perry (1996) investigated whether all people who have suffered from childhood abuse adopt this type of strategy. They predicted that individuals who had experienced childhood abuse would show enhanced directed forgetting. They theorised that a child's limited psychological resources would result in a strategy of temporary forgetting, which would be reinforced by the abuser prompting them to forget. However, women abused in their childhood exhibited enhanced directed remembering, and remembered equal levels of TBF words as the controls. This could be because the coping strategy adopted was one of focusing attention on neutral material, as opposed to specifically ignoring negative material. It may not be that inhibition as a process of coping is the only coping style that should be considered, and that a focus on neutral or positive material may be what is occurring. The lack of attention to negative stimuli may only be a bi-product of this strategy.

2.7 The Directed Forgetting Paradigm & The Investigation of Cognitive Patterns with Major Depression

If the directed forgetting paradigm can be used to measure cognitive processing in those who have been abused, then it could also be a useful task with which to investigate the cognitive patterns within those who are depressed. Power, Daigleish, Claudio, Tata, and Kentish (2000) investigated the interaction between the inhibition process in memory and the threat content of list items. They compared depressed subjects with high levels of anxiety to depressed subjects with low levels of anxiety with a non-depressed control group. This is because most depressed individuals have high levels of anxiety (MacLeod, Byrne, & Valentine, 1996), and it was important to ensure that the behaviour was not just a bi-product of high anxiety. Subjects were asked to intentionally learn words and rate them for pleasantness, and all subjects were given the forget cue half way through the experiment. Although they were able to demonstrate a general directed forgetting effect (the recall of fewer TBF words), depressed subjects only displayed a tendency to remember more of the negative TBF words.

However, the researchers attributed their failure to find an effect to the fact that the subjects were not rating the words in relation to themselves. They then replicated the experiment, with the subjects processing the material in relation to themselves, and found an overall directed forgetting effect. Non-depressed subjects recalled significantly more positive words overall. Depressed subjects recalled significantly fewer TBF positive words, and demonstrated a tendency to recall more negative TBF words. Next, they compared the effects of forgetting and remembering separately on the depressed and non-depressed subjects, with half of each subject group being cued to forget, and the other half being cued to remember. An overall directed forgetting effect was found, and depressed subjects showed

significantly higher levels of recall of negative material on the TBF cue than when cued to remember. This was interpreted as indicating that when depressed, an attempt to forget the material actually increases the likelihood of it returning to consciousness. Therefore, the forget cue facilitates retrieval, not inhibition, of negative memories in those who are depressed. In conclusion, differential directed forgetting effects are dependent on the mood of the individual, the valence of the material that is to be remembered, and whether the material is processed in relation to the self.

One can conclude that intrusive memories affect cognitive processing in depressed individuals, but quite how this occurs in unclear. Serious life events can trigger depression (Brown & Harris, 1978), but the ramifications of the event may not be what is maintaining the depression. It may be that the intrusion of memories takes up so much processing space in the individual's working memory, that there is simply insufficient capacity for the effective functioning of other processes which may help the individual effectively deal with that memory or situation. The cognitive theories of Teasdale (1988) and Ingram (1984) receive mixed support, for although chronicity is not associated with increased activation of negative memories, the length of the episode is related to the number of intrusive memories. What is not yet clear, is what cognitive processes come into effect that facilitate depressed individuals to move into recovery. As individuals, we cope with similar experiences in different ways, and memories affect the cognitive processing style in those who are depressed, but not in uniform ways.

2.8 The Avoidance of Traumatic Memories

Spenceley & Jerrom (1997) looked at the prevalence, intensity and avoidance of intrusive memories in a depressed, a non-depressed and a recovered depressed group. Their results

showed that although there was not a significant difference in the levels of intrusive memories each group had, depressed subjects had significantly more intrusion of these memories and they were significantly more intense, than the never-depressed and recovered-depressed groups. They also demonstrated that depressed subjects showed significantly higher avoidance than the never depressed, but they were not more avoidant than the recovered depressed. Although the recovered group did not have a significantly higher avoidance score than the never depressed, the score was at an intermediate level between the two groups. Spenceley & Jerrom (1997) account for this finding by arguing that both depressed and recovered depressed individuals try hard to avoid these intrusive memories, but that there is something about the recovered depressed individuals' coping style that enables them to avoid them in such a way as to free up the cognitive process for recovery from depression.

2.9 In summation, there is an ongoing interest in obtaining knowledge regarding recovery from depression. Needles and Abramson (1990) found that a depressed individual with an enhanced attributional style could benefit from restored hopefulness with positive life events, and obtain reduced levels of hopelessness when negative life events decreased. In terms of cognitions, recovering depressed individuals have lower levels of intrusion of negative memories in comparison to those who are depressed, but still high levels of avoidance (Spenceley & Jerrom, 1997). It is proposed that this occurs because the recovering depressed develop, or access, an ability to inhibit attention towards negative self-referential information. The directed forgetting paradigm (Bjork, 1989) was initially developed to look at individuals' ability to inhibit certain types of material. It has been applied to look at inhibition abilities of individuals who have a repressive coping style (Myers, et al., 1998) and to see whether

depressed individuals pay more attention to negative words, even when directed to forget them (Power et al., 2000).

This study seeks to contribute to the small but growing body of knowledge in the area of recovery from depression. It proposes to explore why those in recovery are successful at inhibiting negative memories when depressed individuals are not. One hypothesis is that depressed individuals start to recover once they begin to inhibit their attention towards negative material, particularly material that is self-referential, and focus on positive material. In addition, it may be that both depressed and recovering depressed individuals continue to remain as a clinical population, as opposed to a never depressed non-clinical population, and that their cognitive processes will, in terms of a framework of functioning, remain much the same regardless of whether they are symptomatic or in recovery. This leads to the supposition that once a person has had at least one major depressive episode, he or she will always belong along the continuum of a clinical population that differs from a non-clinical one.

3. HYPOTHESES

1. Relative to the currently depressed group, recovering depressed individuals will report lower levels of intrusion, but equivalent levels of avoidance of their traumatic memories, but both groups will report higher levels of intrusion and avoidance than the control group.

2. Relative to the currently depressed and recovering depressed subjects, the control group will be more successful at forgetting negative material, when instructed to do so in the context of a directed forgetting task.

3. Currently depressed and recovering depressed groups will recall fewer words overall on both a directed forgetting and remembering task than controls.

4. The extent of avoidance will be correlated with the total number of words recalled.

4. METHODOLOGY

Ethical approval was obtained from the relevant committee prior to the start of the study, and the aim and process of it was discussed with the Unit Psychiatrists and Psychologists as the depressed and recovering depressed participants would be recruited from their clinical case load.

4.1 Participants

A sample of 82 people (f=52; m=30) was recruited, 40 (f=24; m=16) being currently diagnosed with a primary diagnosis of Major Depressive Disorder according to research criteria (DSM-IV, 1994), 21 (f=14; m=7) being in recovery from a primary diagnosis of a major depressive episode, and a never depressed (i.e. never been diagnosed with, or currently having sufficient symptoms to meet the criteria of, a major depressive disorder) control group of 21 (f=14; m=7). Participants with a secondary or co-morbid diagnosis of, for example, anxiety or personality disorder were included but those with Obsessive-Compulsive Disorder, PTSD or any psychotic illness were excluded. The symptomatic and recovering participants were patients currently being treated on an inpatient or outpatient basis in Acute Psychiatry

and/or in the Adult Psychology Department of the Community Mental Health Unit of a NHS Trust. Participants' initial diagnosis of clinical depression was undertaken by one of the three consultant psychiatrists or their junior doctors in the unit who made diagnoses according to DSM-IV (APA, 1994) criteria for a Major Depressive Disorder (MDD). An information sheet regarding the study and requesting participants (Appendix 1) was placed on the hospital notice boards and posted to any potential participants. The control group was selected from the same or similar socio-economic boroughs and from a range of educational backgrounds. The information sheet (Appendix 1) was displayed on hospital notice boards and also distributed to hospital staff, colleagues and associates within the catchment area who may have been willing to participate or distribute the material to relevant individuals. The same exclusion criteria applied as for the clinical population.

This thesis excluded individuals who met sufficient criteria to warrant a diagnosis of PTSD. The clinical sample was selected on the basis of a diagnosis of clinical depression (Major Depressive Disorder) only, and those who had a diagnosis of PTSD were excluded prior to interview. The reason for this was that the study aimed to look at those who suffered from depression but who may or may not have been exposed to events that they perceived as difficult or 'traumatic'.

Recovery from depression was defined according to a cut-off score on the Beck Depression Inventory (BDI) (Beck, Rush, Shaw, & Emery, 1979; Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961; Robins & Block, 1988) of 18 points or below (normal to mild depression), and a score of 19 or above as being currently depressed (moderate to severe depression). The version in which subjects are asked to report about their feelings during the past 7 days was used (Beck, et al., 1979).

They were then asked after their present state i.e. whether they were currently depressed or in recovery from depression, and their responses were checked against research criteria (DSM-IV criteria for a Major Depressive Disorder, 1994). A total of 82 participants' responses were used for the statistical analyses.

Participants from each group were randomly assigned to one of the two tasks (to-be-forgotten task or to-be-remembered task) according to their designated subject number (i.e. subject number 1 was assigned the to-be-forgotten task, and subject number 2 to the to-be-remembered task). Participants were not matched for education level, income, marital status or ethnicity, as these appear to be (DSM-IV, 1994) unrelated factors to the prevalence of major depressive disorder.

4.2 Measures & Procedures

The interviews were undertaken by two individuals, namely the primary researcher, and an assistant psychologist who was trained to conduct the interview. The aim of this was to maintain professional boundaries with participants with whom the researcher had had professional contact as the clinical population was drawn from the unit in which she worked. Each interview was approximately one hour in length, and was conducted in an office with the Unit. The office had two chairs in one part of the room, and a chair and desk with a computer placed on it in another part of the room. This was to ensure a degree of privacy for the participant whilst he or she was undertaking the computer-generated task (cf. section 4.2.4). The interviewer remained in the room throughout the interviewing schedule.

At the beginning of each interview each participants were asked if they had read the information sheet regarding the research study and if there was any that they wished to

clarify. Written informed consent (Appendix 2) was obtained from all participants. The clinical participants were then asked after their present state i.e. whether they were currently depressed or in recovery from depression, and their responses were checked against research criteria (DSM-IV criteria for a Major Depressive Disorder, 1994). Confirmation from the control participants was obtained regarding no present or past diagnosis of major depression, and none were excluded on this basis.

4.2.1 Severity of Depression

This was measured on the Beck Depression Inventory (BDI) (Beck et al., 1979), a 21-item inventory each ranked on a four-point scale to indicate degree of depression. Corrected split-half reliability was at .93, and test-retest reliability around .7 after one week (Kline, 1993). In terms of validity, the test scores are significantly related to clinical diagnoses of depression with correlations ranging from .6 to .9 (Kline, 1993).

4.2.2 Semi-Structured Interview

Each participant was then given a semi-structured interview (Appendix 3) regarding personal information and intrusive memories (as used by Brewin et al., 1996). They were questioned about the death of friends and family, and then asked about major life events, such as financial crises and unemployment, that have been consistently linked to the onset of depression (Brugha, Bebbington, Tennant, & Hurry, 1985). The clinical participants were asked if there was anything that had occurred that they thought might be linked to the onset of their depression.

The next section of the interview concerned childhood memories. Participants were asked if they had experienced any physical or sexual abuse. Questions were asked to enable the

classification of their accounts of abuse according to research criteria (Brewin et al., 1996). They were then asked whether there were other memories they had recently thought about, what their most prominent memory was, the emotion that they most strongly associated with it, such as fear, hate, anger or guilt, and whether these memories had intruded in the past 7 days. The frequency and duration of the most prominent intrusive memory were noted.

4.2.3 Impact of Event Scale

After completion of the semi-structured interview, participants completed an Impact of Event Scale (Horowitz, Wilner, & Alvarez, 1979) in relation to their most prominent intrusive memory corresponding to a specific event. The Impact of Event scale is a 15-item self-report scale that indicates the extent to which the individual has suffered from intrusion of, or has actively tried to avoid, the memory in the past week. Horowitz et al. (1979) demonstrated that the sub-scales of intrusion and avoidance of experience were empirically valid, and were sensitive to life experiences of varying magnitude. The reliability of the scale was supported by adequate test-retest results, and the scale was also shown to be a sensitive indicator of changes when re-administered and compared over time (Horowitz et al., 1979). Corcoran and Fischer (1987) note that the intrusion subscale has an alpha coefficient of .78, and the avoidance scale has one of .82 suggesting that items within subscales tap similar constructs without item redundancy.

4.2.4 Directed Forgetting Task

The participants were then administered a computer-generated version of the Directed Forgetting Task (as used by Myers et al., 1992) to measure the individual's ability to inhibit negative material. Participants were told they would be exposed to a set of words, asked to judge whether they were self-descriptive, and that they should try and remember as many of

the words as possible, as at the end of the task they would be asked which words they remembered. The set of words consisted of three neutral words (for example, 'blue-eyed'), twenty negative words (for example, 'boring') and twenty positive words (for example, 'considerate') that were matched and randomly assigned to one of two lists of words (Appendix 4, 5 and 6). A computer programme (developed on Micro-experimental Laboratory[MEL] Version 2) up to display each word individually on a PC screen for five seconds, in which time the participant had to indicate his or her self-descriptive judgement by pressing a 'yes' or 'no' key. Computer software then recorded these results. Half way through the task the computer screen displayed a message. Half of the participants (randomly assigned) in each group were told that the first half of the words was only a practice run, and they were to forget what they had seen and concentrate on remembering the second set of words. This was the 'to-be-forgotten' cue (TBF). The other half of the participants was told that they were half way through the task and reminded to try and remember all the words that they had seen. This was the 'to-be-remembered' (TBR) cue. Once they had finished the task, they were asked to write down all the words they could remember from both lists, even if they had been instructed to forget the words previously. Table 2 shows numbers of participants in each group given TBF and TBR instructions.

An important methodological consideration is the placement of the cues to remember or to forget. Directed forgetting tasks have previously been of two kinds. The 'list method', which was used in this study, has the cue given after a list of words. The 'word method' has the cue given after every word that is to be judged. Although both methods produce a directed forgetting effect, Basden, Basden and Gargano (1993) demonstrated that it was due to different processes. In the word method, the directed forgetting effect was due to differential encoding for each word. This was demonstrated by the fact that the forgotten words could

neither be recalled nor recognised, suggesting that they were no longer available. In the case of the list method, directed forgetting was a product of retrieval inhibition. This was supported by the fact that although words were not available when tested in a recall task, the subjects were able to retrieve the forgotten words in a recognition task. In the case of depressed individuals and their unwanted memories, this study sought to evaluate whether recovery was facilitated by the inhibition of negative memories, and therefore the list method was the appropriate one to use.

Table 2: Number of Participants by Group & Task

	Depressed Group	Recovering Depressed Group	Control Group
TBF Task	**19**	**9**	**10**
TBR Task	**21**	**12**	**11**
Total	**40**	**21**	**21**

TBF = To-be-forgotten task
TBR = To-be-remembered task

5. RESULTS

5.1 Types of Traumatic Memories Identified

One purpose of the semi-structured interviews was to identify a traumatic memory of a specific event that the participant recalled as a vivid image. The most reported traumatic memory from the clinical group (depressed and recovering depressed) was around death or loss of a loved one (34%), after which was sexual abuse (13%), physical abuse (11%), poor and/or dysfunctional family relationships (10%), their own 'breakdowns' (7%), relationship problems with their partners (5%), problems at school (5%), bad choices (3%), and the

remainder had individual responses, such as a motor vehicle accident and a mother's 'breakdown'. One respondent said that her memories were there but she would not allow herself to think about them. Two respondents said that they had no traumatic memories now that they were in recovery, even though they had had them during their depressive episodes.

In the control group, the most reported traumatic memory was around parental issues (38%), after which was death (19%), and the break-up in a relationship (10%). There were also individual responses to situations such as a work incident and unemployment. Three individuals (15%) said they had had no traumatic experience, and two subjects (10%) said that they had not had any intrusive thoughts of their traumatic experience within the past 7 days.

5.2 Demographic Characteristics of Groups

Table 3 shows no significant differences were found when the groups were compared on age and gender using ANOVA and Chi-Square tests respectively, nor were any significant differences found on these variables between the groups administered the TBF task and the TBR tasks. ANOVA revealed a significant main effect of group on BDI scores ((***F***=158.293; ***p***=.000; ***df***=2).

Table 3. General Characteristics of the Study Population

		DEPRESSED GROUP (n=40)	RECOVERING DEPRESSED GROUP (n=21)	CONTROL GROUP (n=21)	F=	χ^2	p=	df=
Age	mean	44.18	40.52	39.48	2.685		.074	2
	(*SD*)	10.29	12.39	10.75				
	range	22 - 62	19 - 62	19 – 59				
Gender	male	16	7	7		.392	.822	2
	female	24	14	14				
BDI	mean	31.20	12.57	3.86	158.293		.000	2
	(*SD*)	7.57	4.55	3.55				
	range	19 – 48	2 – 18	0 – 11				

5. Impact of Event Scale: Intrusion & Avoidance

To check for differences between the groups on the Impact of Event Scale (IES) on the dimensions of Avoidance and Intrusion, non-parametric tests were utilised as the scores on the Avoidance scale were not normally distributed. Using the Mann-Whitney U test results confirmed hypothesis 1. The recovering depressed groups showed significantly lower scores on Intrusion than the depressed group (z=-2.890; p=.004), and the control group scored significantly lower than the recovering depressed group (z=-2.960; p=.003) and the depressed group (z=-5.019; p<.001) (Table 4).

As hypothesised, on the Avoidance dimension, no significant difference was found between the depressed and recovering depressed groups (z=-1.353; p=.176) but the control group scored significantly lower than both the depressed group (z=-4.884; p<.001) and the recovering depressed group (z=-3.527; p<.001).

Table 4. Impact of Event Scale (IES) Intrusion & Avoidance Mean Scores & Standard Deviations

	DEPRESSED GROUP		RECOVERING DEPRESSED GROUP		CONTROL GROUP	
	Mean	(*SD*)	Mean	(*SD*)	Mean	(*SD*)
IES Intrusion	23.37	9.84	15.95	9.16	7.38	8.26
IES Avoidance	18.40	8.51	14.67	8.05	4.52	6.74

IESI = Impact of Event Scale on the Intrusion dimension for the Depressed, Recovering Depressed and Control groups.

IESA = Impact of Event Scale on the Avoidance dimension for the Depressed, Recovering Depressed and Control groups.

5.4 Recall of Words After Directed Forgetting

The Kruskal-Wallis test was used to examine the effect of diagnostic group on recall of words in the directed forgetting condition. No significant differences were found between the three groups on the recall of negative words on List 1 (TBF task) (χ=.634; ***p***=.728; ***df***=2). However, the depressed group recalled the lowest number of mean words (***M***=2.37; ***SD***=1.7), the control group at an intermediate level (***M***=2.6; ***SD***=1.43) and the recovering group the highest number of mean words (***M***=2.78; ***SD***=1.64). On the recall of positive words from List 1 (TBF task), again no significant differences were found between the groups (χ=.343; ***p***=.842; ***df***=2), with the mean number of positive words recalled showing the same trend as found on the recall of negative words with the lowest number being recalled by the depressed groups (***M***=.95; ***SD***=1.18) followed by the control group (***M***=1.2; ***SD***=1.03) and then the recovering group (***M***=1.22; ***SD***=1.3). These results, though not shown to have a statistical significance, suggest a trend towards the recovering group recalling more words when told to forget than both the depressed and control groups respectively, and the depressed group

recalling the least. In addition, Wilcoxon Signed Rank Tests revealed that all three groups recalled more negative words than positive ones on List 1 of the TBF task though the difference was only significant in the depressed (z=-2.744; p=.006) and control groups (z=-2.565; p=.010). The recovering depressed group showed a similar trend, albeit not significant (z=-1.843; p=.065).

Table 5: Means of Negative & Positive Words Recalled on List1 TBF

	Depressed Group	Recovering Depressed Group	Control Group
Negative Words	**2.37 (SD=1.7)**	**2.78 (SD=1.64)**	**2.6 (SD=1.43)**
Positive Words	**.95 (SD=1.18)**	**1.22 (SD=1.3)**	**1.2 (SD=1.03)**

TBF = To-be-forgotten task

5.5 Overall Recall of Words

Hypothesis 3 was not confirmed. When using ANOVA to test for the effect of group on overall recall including both TBF and TBR participants, a significant main effect was found (F=8.467; p<.001; df=2). T-tests for independent samples with one-tailed significance were applied to examine group differences, and showed a significant difference between the depressed and recovering depressed groups (t=-2.044; p=.023; df=59) and between the depressed and control groups (t=4.309; p<.001; df=59) but not between the recovering depressed and control groups (t=1.630; p=.111; df=40).

5.6 Recall of Words by Group & Condition

When compared by task and group, ANOVA revealed no significant effect of group on the overall recall of words in the TBF condition (F=1.708; p=.196; df=2) but a significant effect

of group in the TBR condition (*F*=8.502; *p*=.001; *df*=2) (Table 5). Post hoc t-tests to compare group in the TBR condition showed significant differences between the depressed and recovering depressed groups (*t*=-2.542; *p*=.016; *df*=31) and between the depressed and control groups (*t*=-4.321; p<.001; df=30) but not between the recovering depressed and control groups (t=-.984; *p*=.336; *df*=21).

Table 6. Recall of Words by Group & Condition

		DEPRESSED GROUP	RECOVERING DEPRESSED GROUP	CONTROL GROUP	*F*=	*p*=	*df*=
TBF	**mean**	**10.74**	**11.00**	**13.70**	**1.708**	**.196**	**2**
	(*SD*)	**4.75**	**4.30**	**2.91**			
	range	**2 – 17**	**6 - 17**	**10 – 19**			
TBR	**mean**	**9.10**	**13.75**	**15.64**	**8.502**	**.001**	**2**
	(*SD*)	**4.60**	**5.80**	**2.69**			
	range	**2 – 21**	**4 - 24**	**11 – 20**			

TBF = To-be-Forgotten Task
TBR =To-be-Remembered Task

Table 7. Recall Data (Means & Standard Deviations) List 1 & List 2 by Group & Task

		LIST 1		LIST 2	
		+ve	-ve	+ve	-ve
DEPRESSED	Remember (TBR)	.57 (.978)	1.86 (1.108)	2.76 (1.261)	2.29 (1.736)
	Forget (TBF)	.95 (1.177)	2.37 (1.641)	3.58 (2.009)	2.95 (1.508)
RECOVERING DEPRESSED	Remember (TBR)	1.83 (1.586)	2.58 (1.311)	4.42 (1.881)	3.17 (2.368)
	Forget (TBF)	1.22 (1.302)	2.78 (1.641)	3.56 (1.236)	2.22 (1.302)
CONTROL	Remember (TBR)	2.27 (.786)	2.45 (1.508)	5.36 (1.502)	3.27 (1.272)
	Forget (TBF)	1.20 (1.033)	2.60 (1.430)	5.20 (1.135)	3.10 (1.969)

TBF = To-be-Forgotten Task
TBR =To-be-Remembered Task

5.7 Directed Forgetting Effect

To determine whether an overall directed forgetting effect occurred, ANOVA was used to compare the groups by condition. There was no significant effect of condition or interaction though examination of means indicated that there was a trend towards a directed forgetting effect for both the recovering depressed and control groups. The depressed group, however, tended to remember more words on the TBF task than on the TBR one, though the difference was not significant.

5.8 Relationship Between Avoidance & Recall

To look at the relationship between avoidance and overall recall the non-parametric test Spearman Rank Order correlation co-efficient was used as avoidance scores were not normally distributed. Results indicated no significant relationship for the recovering depressed (r=.003; p=.990) and depressed groups (r=.077; p=.635) but a significant correlation was found for the control group (r=.492; p<.05), (i.e. for the controls, low levels of avoidance were associated with good recall).

A number of significant correlations were found, using Pearson correlation co-efficient, between BDI scores and other variables, namely overall recall (r=-.371, p<.01), intrusion (r=.593, p<.01), and age (r=.254, p<.05). There was a significant correlation between BDI scores and avoidance (r=.516, p<.01) using Spearman's rho correlation. Overall recall and age were also found to be significantly correlated (r=-.424, p<.01) , using Pearson correlation co-efficient, as were intrusion and avoidance (r=.606, p<.01) using Spearman's rho correlation.

5.9 Variables Associated with Intrusion & Avoidance

Linear (simple) regressions were undertaken to explore the predictors of intrusion and avoidance. BDI score was a good predictor of both intrusion and avoidance explaining 35% of the variability within intrusion scores (R^2=.351; df=1; F=43.311; p=.000) and 28% of the variability within avoidance scores (R^2=.284; df=1; F=31.787; p=.000). Intrusion scores explained 43% of the variability within the avoidance scores (R^2=.431; df=1; F=60.566; p=.000) suggesting a strong relationship between the scores on the intrusion and avoidance scales. No significant linear relationship was found between age and the scores on the avoidance and intrusion scales.

Forward stepwise selection methods were used to build the model to predict avoidance and intrusion scores. These multiple regressions revealed that avoidance explained 43% of the variability in the intrusion scores, but when the BDI scores were added to the equation 51% of the variability in the intrusion scores was explained (R^2=.513; df=2; F=41.630; p=.000). Intrusion explained 43% of the variability in the avoidance scores but when the BDI scores were added to the equation 46% of the variability in the avoidance score was explained (R^2=.463; df=2; F=34.048; p=.000). Age did not appear in the equations as it contributed so little to explaining the variability.

5.10 Variables Associated with Overall Recall

To look for the predictors of overall recall, simple regressions showed that the BDI scores explained 14% (R^2=.138; df=1; F=12.795; p=.001) of the variability within the recalling of words indicating a weak but significant linear relationship between the scores on the BDI and overall recall. Age explained 18% (R^2=.180; df=1; F=17.558; p=.000) of the variability within the recalling of words indicating a weak but significant linear relationship between age and overall recall. Therefore, both BDI scores and age, respectively, could predict overall recall.

To build the model to predict overall recall, the forward stepwise selection method was used and it revealed that age explained 18% of the variability in overall recall but when the BDI scores were added into the equation 25% of the variability in overall recall was explained (R^2=.254; df=2; F=13.467; p=.000).

6. DISCUSSION

This study aimed to independently support the findings of Spenceley & Jerrom (1997) in that the recovering depressed group showed a significantly lower score on intrusion than the depressed group, but showed similar scores on avoidance, and this was supported (Hypothesis 1). Hypothesis 2 was not confirmed, in that the control group did not recall fewer negative words than the depressed and recovering depressed groups when instructed to do so in the context of a directed forgetting task. The hypothesis that the currently depressed and recovering depressed groups would recall fewer words overall on both a directed forgetting and a remembering task than controls was not confirmed (Hypothesis 3). Hypothesis 4 was confirmed in that the extent of avoidance was correlated with the total number of words recalled, but this inverse (or negative) correlation was only found to be significant for the control group and not for depressed or recovering depressed groups.

Hypothesis 1: This study supported Spenceley & Jerrom (1997) findings in that the recovering depressed group showed a significantly lower score on intrusion than the depressed group, but showed similar scores on avoidance. The new finding of this study was that a significant finding was found between the recovering depressed and the control groups on both the intrusion and the avoidance scales, respectively, whereas no significance was found between these groups on the two dimensions in Spenceley & Jerrom's (1997) study. Again, unlike their study, this study found that the control group scored significantly lower than both the depressed and recovering depressed groups on both avoidance and intrusion. In addition, there was no significant difference within the control group between the two dimensions. One reason for the difference in findings may have been that the cut-off points on the BDI for the three groups differed, with their study using four points below that which was used in this study as a criterion of admission to the respective groups (i.e. they used a

score of 15 or above for the depressed group and 14 or below for the recovered and control groups, compared with 19 or above and 18 or below, respectively, used in this study). However, the mean BDI scores for the three groups did not signify much difference when compared with those found in this study. Additional differences between the studies is that their study was comprised of women only, as opposed to both sexes in this study, and that they asked their participants to report on traumatic memories of events that they had experienced prior to the age of 16 whilst this study did not include any age limit.

The findings for the Intrusion scale could be explained in terms of Ingram's (1984) and Teasdale's (1988) theories. However, their theories do not include any explanation of the dimension of avoidance. With reference to the findings on intrusion, Ingram's Information Processing Paradigm (Ingram, 1984) would suggest that the depressed group are likely to have higher levels of intrusion than the recovering group as they have a dysfunctional negative cognitive network that has been activated and they are therefore preoccupied with the network in such a way that they cannot process information outside of it. This in turn leads to ongoing intrusion of traumatic memories in those who are depressed. However, in recovering depressed individuals there may be less preoccupation with the dysfunctional cognitive network thereby allowing information outside of the network to be processed which could account for the decrease in the extent of intrusion of traumatic memories.

Teasdale's Differential Activation Hypothesis (Teasdale, 1988) states that there are patterns of thought that can only be activated once an individual is within the depressive state. Consequently, in the depressed group, these patterns of thought are actively in process and thus allow for greater intrusion of them. However, once depressed individuals move into

recovery the activation of these thought patterns decreases, which leads to a reduced level of access to their content and therefore less intrusion of them.

The recent research (such as, LeDoux, 1992; Squire, 1994; and van der Kolk, 1996) on trauma and its neuroscience is in agreement with both Ingram's Information Processing Paradigm (1984) and Teasdale's (1988) Differential Activation Hypothesis in as much as it has established the concept that memory systems are comprised of networks of related information. When a relevant network is activated it can then lead to the retrieval of memories and information, whether stored as cognitive schemata or as fragments, which have been stored along specific associative pathways.

In support of research findings in the non-clinical population (Berntsen, 1996; Brewin, Christodoulides, & Hutchinson, 1996; Brewin et al., 1999) the control group did report traumatic memories that intruded into their conscious thinking. However, it appears that the impact of these events did not impinge in a manner that resulted in a significant degree of activation of their dysfunctional negative cognitive networks or patterns of thought, and consequently their mood states remained sufficiently stable, i.e. they did not become clinically depressed.

Hypothesis 2: Both depressed and recovering depressed individuals try as hard to avoid their traumatic memories, but it is the recovering depressed who seem more effective at doing so. This study had hoped to explain this through the hypothesis that the recovering depressed group would be more able than the depressed group to inhibit negative material, and that the inhibition of such would facilitate their progress into recovery but the hypothesis was not supported. In fact, there was a trend for the recovering depressed group to be poorer at the

directed forgetting task than the depressed group. It may be that the words subjects recalled were influenced by the initial instruction of the task whereby they were asked to judge whether or not the word on the screen was self-descriptive. Research (Anderson, 1995; Craik & Lockhart, 1972; Forsyth & Wibberly, 1994; Hartlep & Forsyth, 2000; Symons & Johnson, 1997) has shown that material that is self-descriptive or relates to one in a personal manner, is more readily recalled than that which is neutral or distant from one's personal frame. Consequently, this may have contaminated the general process of recall. In addition to this, all groups recalled more negative words than positive ones when instructed to forget them (List 1, TBF). It would seem unlikely that subjects would describe themselves only in positive terms and perhaps the residual effect of confirming a negative description of oneself was more powerful than confirming a positive one.

An explanation for the trend of the depressed group to recall the fewest of both negative and positive words on the forget cue (List1, TBF) is more likely a result of their poorer memory for recall than their better performance in carrying out the task instructions. This explanation was confirmed by the analyses carried out for hypothesis 3. One explanation for the opposite trend of the recovering group recalling the most words on the same list might be that they found it difficult to negotiate the requirements of the task (i.e. to forget words after they were originally instructed to remember them). This rationalisation falls within the context of the work carried out on memory and depression referred to below, particularly Veiel's (1997) work on the impairment of cognitive flexibility found in depressed subjects.

Hypothesis 3: Research (Baddeley & Hitch, 1974; Baker et al., 1997; Channon, 1996; Channon & Green, 1999; Veiel, 1997) on the effects of depression on memory, namely that depressed individuals have processing difficulties and memory impairment when compared

to other groups, supports the finding of significantly greater recall of the control group over the depressed group. In addition, the study found that those who were recovering from depression appeared to have better recall performance which suggests the recovering process may be associated with an improvement in memory performance (i.e. the recovering depressed group recalled significantly more words overall than the depressed group). Therefore, those who are in recovery appear to have fewer deficits in these areas (i.e. to exhibit an improved level of information processing capacities) and to be functioning at a level that is similar to those who have never been clinically depressed.

These findings are also in keeping with those confirming hypothesis 1 whereby the information processing capacity of the recovering group is being freed up from the interference of intrusive thoughts and the activation of networks or patterns of thoughts associated with depression, thereby allowing more of the individual's capacity to be spent on the memory requirements of the task. It also implies that there is now more capacity to expend effort on the tasks that previously may have been reduced by the presence of a more severe level of depression (Veiel, 1997). The clinical symptoms of depression include poor concentration, anxiety, and a decrease in one's level of interest and energy (DSM-IV, 1994), all of which can interfere with depressed subjects' recall of material, as can limited processing capacities. However, once in recovery, clinical symptoms decrease and processing capacity increases which may allow for greater concentration, interest in, and attention to, a task. The control group, as previously suggested, do not have the same level of intrusion or interference in memory or task performance as found in depressed individuals.

However, it appears that when task instructions become more complex, such as in the to-be-forgotten task (TBF) the performance of subjects from the recovering depressed and

depressed groups are at similar levels, and the differences between the two groups may only become apparent when the task is simpler, as on the to-be-remembered task (TBR). In addition, there was no evidence to support the view that recovery from depression is associated with improved ability to forget negative material, which is a new finding that would warrant further exploration.

A further implication is the effects of trauma on memory. In a previous discussion, some evidence was put forward to account for how trauma interferes with the mechanisms of memory storage and the retrieval or the re-evocation of material (van der Kolk et al., 1996; Perry, 1999), and on the co-morbidity of depression and PTSD (Reynolds & Brewin, 1998, 1999). The enhanced recall of the recovering group, and the poorer performance of the depressed group, may be linked to a process whereby as the severity of the depression decreases so the capacity to process the trauma increases. This concept would certainly benefit from further exploration.

Power et al. (2000) found a general directed forgetting effect using a similar measure (i.e. a directed forgetting task) with a depressed, anxious and never depressed population but this study was only able to support it in as much as a similar trend was found for the control and recovering groups but not for the depressed group. Power et al.'s (2000) study found a significant difference between the recall of negative words from List 1 for the depressed group when compared on the TBF and TBR tasks (i.e. the depressed group's recall of List 1 negative words on the TBR task (M=2.63) vs the depressed group's recall of List 1 negative words on the TBF task (M=5.00)). This study found the same result (i.e. the depressed group's recall of List 1 negative words on the TBR task (M=1.86) vs the depressed group's recall of List 1 negative words on the TBF task (M=2.37), however, the difference was not

significant. This study also found a trend for the depressed group to recall more words overall on the TBF task than on the TBR task (i.e. not only on List 1 negative words but on the recall of both positive and negative words from both List 1 and List 2). Power et al.'s explanation was that the instruction on the TBF task is a paradoxical one. In order to forget something, a person must first remember what it is he or she is to forget before it can be forgotten. The forget cue, therefore, facilitates recall rather than inhibits it, and it may set up a stronger memory of the material than an instruction to remember. However, since these findings have only been evident in those who are depressed, it may be that their cognitive dexterity is still too impaired to manipulate this paradoxical situation. This suggestion would be supported by Veiel's (1997) findings of depressed individuals exhibiting decreased level of cognitive flexibility and impaired memory functioning.

The discussion thus far has postulated that the recovering depressed have fewer or less severe symptoms of depression, which leads to an increase in their processing capacities and ability to recall material, particularly if the task instructions are less complex, and hence that they do not differ significantly from the control group in their recall.

Hypothesis 4: The significant relationship found between avoidance and recall for the control group can be explained by the control group exhibiting the highest recall of overall words but also the lowest level of avoidance, when compared to the other two groups. They did not appear to require the same level of effort needed to avoid their traumatic memories and were thus able to utilise their information processing capacity to a greater extent than the other groups. The results of the regression analyses support this in that the level of depression (BDI scores) could predict the extent of avoidance, (i.e. high levels of depression correlated

with high levels of avoidance) as well as overall recall, (i.e. high levels of depression predicted poor recall), which was a new finding.

The influence of the level of depression on recall and on the extent of intrusion and avoidance were clearly evident in the correlation and regression analyses. Whereas Brewin et al. (1999) found that depression at follow-up (rated on the BDI) was predicted by the extent of intrusion and avoidance at initial contact, this study found that current levels of depression could predict intrusion and avoidance scores, and that the level of avoidance coupled with the level of depression was a slightly better predictor of intrusion than intrusion plus level of depression was of avoidance.

The findings suggest that although the recovering depressed group were less depressed and lower on intrusion but still high on avoidance, their scores were still relatively lower on all scales when compared to the depressed group. This implies that as the level of depression diminishes so does the extent of intrusion, and, in part, the extent of avoidance. In addition, the findings suggest that the greater the extent of intrusion of traumatic memories, the more severe the level of depression. This was verified by the depressed group scoring the highest on both the IES (Intrusion) and BDI scales, whilst the recovering depressed exhibited lower levels of both intrusion and depression. The control group, although scoring on both scales, revealed significantly lower scores on all scales and dimensions than the other groups, and it was only in this group that a percentage of participants scored zero on both the IES and the BDI scales.

This gives a strong indication of how widespread the ramifications of depression are on individuals' cognitive functioning as well as emphasises the extent of the impact that trauma

has on the mental and emotional well-being of individuals, particularly when a co-morbid depressive disorder is present.

Age was not an important predictor of intrusion or avoidance, but, as expected and as previously established (Anderson, 1995), it was significantly correlated with recall as was depression with recall (Channon, 1996; Channon & Green, 1999; Veiel, 1997). These inverse correlations verify that with an increase in age comes a decrease in the capacity to recall material, and that depression can interfere with the recall of material.

A significant conclusion that can be drawn from the findings of this study is that it does not support an argument that the cognitive process of recall of a non-clinical (never depressed) group differ so markedly from a clinical group (depressed and recovering depressed) as to warrant the two groups being placed on different continuums. This conclusion is substantiated by the absence of significant difference in performance between the recovering depressed and control groups on both the overall recall of words and on the recall of words on the less complex to-be-remembered tasks being found. A blurring of the boundaries between these two groups may also be, in part, due to the control group still obtaining scores on the Beck Depression Inventory and therefore indicating that those who do not have a diagnosis of depression may still have some level of depression present. This also holds for the presence of traumatic experiences in some of the control group.

It appears that having had an episode of clinical depression does not necessarily mean that the individual should continue to be categorised as belonging to a clinical population once in recovery. The implication of this is that some clinically depressed individuals, once they move into recovery, appear to regain levels of cognitive functioning and information

processing capacity that are similar to those of their never depressed counterparts. However, what the findings cannot address is to what extent the emotional reaction to the perceived traumatic experience has been resolved. It may even imply that the capacity to avoid the intrusion of memories is more dependent upon a defence mechanism or cognitive suppression rather than some skill implicating resolution. The study did not take into account whether or not the subjects had been involved in a therapeutic intervention, and for some, simply being attached to a psychiatric unit or seeing someone of any profession (not necessarily a psychotherapist) may have had some therapeutic impact that led to a process of recovery.

7. <u>CLINICAL IMPLICATIONS OF THE FINDINGS</u>. One of the most frequent presentations clinicians come across is depression. The findings of this study suggest that when an individual presents with features of depression, it may be helpful to explore the presence of intrusive memories and their origins, as they may be contributing to the onset and maintenance of the depression.

Depressed individuals appear to have more difficulty in inhibiting distressing memories than those who are in recovery from depression, even though they try as hard to avoid them. Their preoccupation with these intrusive memories utilises a good deal of processing capacity, limits their ability to take in and make use of information, and is distressing. Clinicians' awareness of this obstructing activity can help them to monitor the impact of the memories and the feelings associated with them, as well as the extent of their intrusion and the procedures that the individual utilises in order to avoid them.

Both depressed and recovering depressed clients have some degree of intrusion, but the depressed clients may feel despairing that they are unable to inhibit them. Clinicians could

be mindful of the extent of information they are providing to, or expecting from, clients as they may only be able to utilise small pieces of information at a time, and may need to be reminded of their content in order to absorb and process them. The more severe the state of depression, the more intrusion and avoidance will be present, the poorer ones recall performance and processing of information, and the less the client will be able to tolerate, concentrate on, or participate in, therapeutic interventions. This does not imply that the client should not participate in a therapeutic intervention, but rather that clinicians should be respectful of the distress that these traumatic memories can cause, and the extent of their impact on a person's emotional and cognitive functioning. Those clients who are in the process of recovery, show improved concentration, memory performance and a greater capacity for processing information. Consequently, they may be more receptive to more focussed interventions, though he or she could still have a restricted capacity to process information and integrate material, particularly if it is more complex in nature, and will still be vulnerable to relapse.

In conclusion, this study emphasises the high rate of co-morbidity between trauma and depression. All too often in clinical practice the effects of traumatic experiences and their lasting impact are underrated and, at worst, disregarded. In particular, it appears that when the traumatic experience is not the reason for referral or primary presentation, such as with victims of torture or PTSD, its presence and its long-term consequences are overlooked. However, as this study has shown, these personal events (regardless of when they occurred) have been perceived by the individual as being sufficiently traumatic to the extent of intruding into their conscious thinking, disrupting their ability to process other information, and being closely associated with their level of depression. The issue of importance is not

how difficult the clinician perceives the event to have been, but how the client has perceived it and to what extent it has impacted on him or her in a very personal manner.

8. LIMITATIONS OF THE STUDY.

Other studies do not report their effect sizes and used different methods and/or report different findings making it difficult to calculate effect size based on their standard deviations. Consequently, a power calculation was carried out on this study using a medium effect size (.50) and was calculated at .72 giving the study an adequate level of power. A limitation of this study is that the small number of subjects in each cell division, particularly in the recovering depressed and control groups, perhaps prevented more significant findings being revealed and placed a limitation on the generalisability of the results. In addition to this, nonparametric tests were required for some of the analyses as not all the variables were found to be normally distributed, and these tests are not regarded to be as powerful as parametric ones. Had the subgroups been larger in size, they may have reached normal distribution thereby meeting the requirements for the use of more powerful parametric analyses and therefore a greater chance of revealing significant results. A further consideration is that the version of the Directed Forgetting Task that was used may not necessarily have been devised for a population who were of mixed ethnicity.

9. CONCLUSION

Depressed and recovered depressed groups showed a significant difference in the extent of intrusion of their traumatic memories, when measured on the Impact of Event Scale, but revealed no significant difference on the dimension of avoidance. The control group, however, was significantly lower on both dimensions than the depressed and the recovering depressed groups. The impact of traumatic events is closely associated with depression, and

this was evidenced in the level of depression being a good predictor of the extent of intrusion and avoidance. It was found that the control group was not free from either traumatic memories or some level of depression, but that these were significantly lower than the two other groups.

No significant difference was found in the recall of negative material on a directed forgetting task between the three groups, and the only significant correlation between the extent of avoidance of intrusive memories with the ability to recall material was evident in the control group.

An argument against the depressed and recovering depressed groups belonging on one diagnostic continuum (a clinical population) that is dichotomous from that on which a never depressed group belongs (a non-clinical population) was put forward. In other words, the recovering depressed group did not reveal significantly different results from the control group in areas such as recall of negative words and recall of overall words, but did show significant differences when compared with the depressed group. This implies that those who are in recovery from depression are moving towards the same level of cognitive functioning as those who have never been clinically depressed. However, the former still tend to show some impairment in their cognitive flexibility and in their level of processing capacity when compared to the control group, and this may be linked to the cognitive deficits associated with depression but also with the impact that trauma has on the storage and integration of material. In contrast to expectation, all three groups recalled more negative words when directed to forget them than positive ones. This may imply that when material is self-referential it emphasises that all individuals have some negative bias in their appraisal systems and not only those who are, or have been, depressed.

The discussions around these findings have been linked to differences in the extent of processing capacity and the intrusion and avoidance of traumatic memories between depressed, and recovering depressed and control groups. Research into the mechanisms and processes that facilitate recovery is sparse, and more knowledge in this area is needed. In particular, prospective studies examining memory and cognitive processes during recovery and relapse in depression will help to clarify the role of these processes. The clinical implications of these findings were also discussed with particular reference being made to the need for a raised awareness in clinicians of the extent of overlap between trauma and depression, and the extensive impact that both have on clients' cognitive functioning and emotional well-being. What the client perceives as being a traumatic experience may not necessarily be what the clinician perceives as important or requiring exploration. Furthermore, the findings of this research underscore the links between neuroscience, cognitive functioning, trauma, depression and clinical symptomatology, and emphasize the importance of exploring the origins of symptoms, for what we are today is an accumulation of all our past experiences. People are complex beings and dissecting them into separate, and often disparate, sections can lead to professionals perceiving them as fragmented bits rather than as an extraordinary combination of multiple systems all combining into one highly specific entity – the individual.

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Appendix 1

HEALTH AUTHORITY
LOCAL RESEARCH ETHICS COMMITTEE

CONSENT FORM

AGREEMENT TO PARTICIPATE IN RESEARCH PROJECT

I, [name of subject]

of [Address]

agree to take part (or agree that my child/ward may take part) in the research project:

AVOIDANCE OF TRAUMATIC MEMORY AND DIRECTED FORGETTING IN SYMPTOMATIC, RECOVERED AND NEVER DEPRESSED INDIVIDUALS

I confirm that the nature and demands of the research have been explained to me and I understand and accept them. I understand that my consent is entirely voluntary, and that I may withdraw from the research project if I find that I am unable to continue for any reason and this will not affect my medical care.

Signed:

(Print Name):

Witness:
(Print Name):

Date:

Investigators' Statement:

I have explained the nature, demands and foreseeable risks of the above research to the subject:

Signature:

Date:

Appendix 2

PATIENT INFORMATION SHEET

Will you consider helping us to understand more about depression and memories?

A research study is being undertaken on depression and memories. People often attempt to avoid unpleasant or traumatic memories, but do not always manage to do so. This study is looking at why some people seem to be able to avoid unpleasant memories more so than others, and how it affects a state of depression.

Approximately 100 people who are, or have been, under treatment for a ***major episode of depression*** as well as individuals who have ***never had a diagnosis of, or treatment for, depression*** are to be interviewed. The interview will last about an hour, and will involve being asked some questions, filling in a couple of forms, and doing a short and simple task. Only one interview will be required.

All information will be kept strictly confidential, and will be kept in a locked filing cabinet. If the results are published, no personal information identifying an individual will be released.

No drugs or treatment are involved, only an information-gathering interview. No adverse effects are expected, but should any participant feel distressed by the questions, he or she has the right to terminate the interview and be given emotional support if deemed necessary .

No costs incurred by taking part in the study can be reimbursed,

Refusal to take part or withdrawal from the study will not affect the participant's medical or psychological care.

Participation is voluntary and the participant may withdraw from the study at any time.

IF YOU OR ANYONE YOU KNOW IS INTERESTED IN PARTICIPATING

IN THIS STUDY PLEASE CONTACT .....

Appendix 3

SEMI-STRUCTURED INTERVIEW SCHEDULE : -

Name:

Date of Birth:

Gender:

Address:

Tel:

Date of Interview:

Study No:

Age at leaving full-time education:

Level of Education:

Investigator:

1. Patient to fill out BDI

2. Check if patient is current or recovered stage of depression (DSM-IV Criteria)

3. General Life Events:

I would like to ask about some events you may have experienced.

		Age	Yes/No
a)	Has anyone in your immediate family died?		
b)	Has a close friend or relative died?		
c)	Have you suffered from a serious illness, injury or assault (not depression)?		
d)	Has a serious illness, injury or assault happened to a close relative?		
e)	Has your child died (miscarriage or termination)?		

f) Have you had a separation due to marital/partner difficulties?

g) Have you had a serious problem with a close friend, neighbour or relative?

h) Have you become unemployed or were seeking work unsuccessfully for > 1 month?

i) Have you had a major financial crisis?

j) Have you had problems with the police or law?

k) Have you had something you valued being stolen or lost?

l) Has anything else happened recently that you think may be connected with the beginning of your illness?

4. <u>Childhood experiences</u>

Now I would like to ask you about childhood experiences
(up to the age of 17)

a) Was there anything that stands out as being particularly disappointing or upsetting?

b) When you were young, did anybody hit you or hurt you in any way?
Did your parents punish you in anyway?
(If yes) Establish criteria for physical abuse (any episode involving kicking, punching, being hit with an implement, strangling, beating up)
(Record details of abuse, age it occurred and for how long, frequency, perpetrator, severity, hospital treatment).

Criterion abuse Yes/No

Yes/No

c) Did anybody ever approach you sexually against your wishes?

d) Have you ever had any unpleasant or threatening sexual experience?
(If yes) Establish criteria for sexual abuse (intercourse, contact with genitals of an adult, adult contact with child's genitals).

Record details of abuse, age it occurred and
for how long, frequency, perpetrator, severity, hospital treatment)

Criterion abuse Yes/No

d) Have noticed memories of any of these events coming to mind in

the past 7 days? Yes/No

5. Memories

Age Yes/No

a) Is there anything else in the recent past that you keep finding yourself thinking about'? Anything from your childhood? Anything else from any period of your life?
(Ask for details of the event)

Have you noticed memories of any of these events coming to mind in the past 7 days'?

(Must establish that these are memories of a specific event that happened at some particular time and place, and is accompanied by a clear visual image -exclude ruminations, worry etc)

(If previous episodes of depression) Do you remember any distressing memories from the past that kept coming to mind on those occasions: Record details

b) Have you noticed memories of any of these events coming to mind in the past 7 days?

(Must establish that these are memories of a specific event that happened at some particular time and place, and is accompanied by a clear visual image -exclude ruminations, worry etc)

(If previous episodes of depression) Do you remember any distressing memories from the past that kept coming to mind on those occasions': Record details

c) Of the memories you have identified so far, which is the most prominent one?

d) What are the strongest emotions that you associate with this memory'?
(How do these memories make you feel?)

(Circle one or more strongly felt emotion)

Sad Angry Guilty Anxious Ashamed Helpless Other (state)

e) In what ways, if any, does this memory remind you of your situation now?

f) Did you notice these memories coming to mind before you became depressed?

1=no hardly at all 2=yes, but less frequently 3 =as frequently as now.
4=more frequently than now

g) Are they in any way different now? Yes/No

h) Does it feel like you are there reliving the experience or is it like looking back at a period in the past?

1=reliving the experience 2=looking back at the past

i) When this memory comes to mind are the details unclear, or is it very vivid, i.e. are the images, sounds, smells or sensations ver;' clear and familiar

1=unclear 2=some detail 3=vivid

j) Approximately how often have you experience it over the past 7 days?

k) How long does it last when you start thinking about it?

6. Give Impact of Events Scale

7. Do Directed Forgetting Task

Appendix 4

DIRECTED FORGETTING TASK (To-Be-Forgotten Task) (TBF)

During this task, a list of words will be presented one at a time.
YOU HAVE TO DECIDE WHETHER THE WORD DESCRIBES YOU.
If you think that the word does describe you, then press the key marked 'YES'.
If you think that that it does not, then press the key marked 'NO'.

Try to respond as quickly as possible.
Each word will be shown for five seconds after which there will be a beep.
Do not respond to a word if the beep has already sounded.

TRY TO REMEMBER AS MANY WORDS AS POSSIBLE.
At the end of the task you will be asked to try to remember the words.
If you have any questions then ask the experimenter now.
Otherwise press the spacebar to start.

TALL
BLUE-EYED
ENGLISH
MEAN
BORING
GUILTY
LOYAL
LAZY
DISHONEST
STRONG
ALONE
UNHAPPY
POPULAR
CALM
CAREFREE
POSITIVE
UNLOVED
GOOD
CRUEL
SINCERE
MATURE
UGLY
CONFIDENT

You are now halfway through.

PLEASE TRY TO FORGET THE LIST YOU HAVE JUST SEEN BECAUSE IT WAS ONLY PRACTICE.

(To-Be-Forgotten Task continued)

The proper list of words to remember will now be presented.

Press the spacebar to continue.

UNKIND
UPTIGHT
CLEVER
THOUGHTFUL
UNRELIABLE
AMUSING
CONSIDERATE
FRAIL
USELESS
HELPFUL
WARM
LOVED
HEARTLESS
WORTHLESS
SELFISH
KIND
BOSSY
ATTRACTIVE
WISE
TIRED

This is the end of the task.

Appendix 4

DIRECTED FORGETTING TASK (To-Be-Remembered Task) (TBR)

During this task, a list of words will be presented one at a time.

YOU HAVE TO DECIDE WHETHER THE WORD DESCRIBES YOU.
If you think that the word does describe you, then press the key marked 'YES'.
If you think that that it does not, then press the key marked 'NO'.

Try to respond as quickly as possible.
Each word will be shown for five seconds after which there will be a beep.
Do not respond to a word if the beep has already sounded.

TRY TO REMEMBER AS MANY WORDS AS POSSIBLE.
At the end of the task you will be asked to try to remember the words.
If you have any questions then ask the experimenter now.
Otherwise press the spacebar to start.

BLUE-EYED
ENGLISH
TALL
POSITIVE
CRUEL
LOYAL
ALONE
POPULAR
SINCERE
CALM
UNLOVED
CAREFREE
GUILTY
GOOD
CONFIDENT
BORING
UGLY
STRONG
MATURE
UNHAPPY
MEAN
DISHONEST
LAZY

You are now halfway through.

TRY TO REMEMBER THE LIST YOU HAVE JUST SEEN AS WELL AS THE NEXT LIST

(To-Be-Remembered Task continued)

Press the spacebar to continue

HELPFUL
CLEVER
SELFISH
AMUSING
ATTRACTIVE
CONSIDERATE
HEARTLESS
WORTHLESS
UNKIND
FRAIL
USELESS
BOSSY
THOUGHTFUL
TIRED
WISE
LOVED
UPTIGHT
WARM
UNRELIABLE
KIND

This is the end of the task.

Appendix 5

DIRECTED FORGETTING TASK (TBR) LIST WORDS

LIST 1

Neutral Words
TALL
BLUE-EYED
ENGLISH

Positive Words
LOYAL
STRONG
POPULAR
CLAM
CAREFREE
POSITIVE
GOOD
SINCERE
MATURE
CONFIDENT

Negative Words
MEAN
BORING
GUILTY
LAZY
DISHONEST
ALONE
UNHAPPY
UNLOVED
CRUEL
UGLY

LIST 2

No neutral words

Positive Words
CLEVER
THOUGHTFUL
AMUSING
CONSIDERATE
HELPFUL
WARM
LOVED
KIND
ATTRACTIVE
WISE

Negative Words
UNKIND
UPTIGHT
UNRELIABLE
FRAIL
USELESS
HEARTLESS
WORTHLESS
SELFISH
BOSSY
TIRED

Appendix 5 (cont.)

DIRECTED FORGETTING TASK (TBF) LIST WORDS

LIST 1	**LIST 2**
Neutral Words	No neutral words
TALL	
BLUE-EYED	
ENGLISH	
Positive Words	**Negative Words**
POSITIVE	CRUEL
LOYAL	ALONE
POPULAR	UNLOVED
SINCERE	GUILTY
CALM	BORING
CAREFREE	UGLY
GOOD	UNHAPPY
CONFIDENT	MEAN
STRONG	DISHONEST
MATURE	LAZY

Chapter 4

Depression in Older Adults – Is It Worthy of Recognition & Treatment

CONTENTS

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1. Introduction

The scientific and academic community is split on the question of the inevitability of depression in older age. Findings by researchers such Blazer (1996a), Blazer & Koenig (1996), Popkin, Mackensie, and Callies (1984), Mather (1997) and Collins, Katona, and Orrell (1995) suggest that, on the one hand, there are those who argue that depression is a predetermined consequence of ageing. There are, however, also those who view it as a specific clinical condition that can respond to appropriate treatment interventions (Baldwin, 1997a, 1997b, 1998; Blazer & Koenig, 1996; Gerson, Belin, Kaufman, Mintz, & Jarvik, 1999; Karel & Hinrichsen, 2000; Pearson & Brown, 2000). Studies (such as Baldwin, 1997b; Blazer, 1996a; Geiselmann, Linden, & Helmchen, 2001; Gurland, 1976; Jorm, 2000; Lindesay, Briggs, & Murphy, 1989) on the prevalence rates of Major Depressive Disorder (MDD) in an elderly population have tended to vary in outcome, and authors such as Copeland et al. (1987) Geiselmann, Linden, and Helmchen (2001), Blazer & Williams (1980), and Blazer (1996a) have emphasised the discrepancy in rates between those diagnosed with MDD and those with some degree of depressive symptomatology.

In order to define the context of this review a working definition of depression is required and its prevalence in an older adult population. An overview of the theories of why depression can occur in later life will be provided, as well as an evaluation of the assessment of depression and the efficacy of current psychological treatment modalities on offer.

2. Definition

The most widely accepted classification systems for determining a case of depression are the DSM (American Psychiatric Association) and ICD (World Health Organisation) diagnostic

manuals. The definition of depression and the terminology used to describe the state of it appears to have found greater clarity, and precision of definition and categorisation, as a result of the extensive work carried out in order to produce the DSM-IV (APA, 1994) and the ICD-10 (WHO, 1994) (Geiselmann et al., 2001). Kaplan, Sadock, and Grebb (1994, p. 303) define depression as 'a psychopathological feeling of sadness'. This definition is somewhat broad and therefore lacks precision and specificity, though it does give a general sense of what the condition implies. In order to give a precise diagnosis of this psychiatric disorder one needs to look to the various symptoms of it, as it presents itself as a syndrome rather than as one specific symptom.

According to the DSM-IV (APA, 1994) and ICD-10 (WHO, 1994) depression is a syndrome that includes depressed mood, loss of interest and enjoyment, reduced energy leading to increased tiredness and a decrease in activity, reduced concentration and attention, reduced self-esteem and self-confidence, ideas of guilt and unworthiness, ideas of acts of self-harm or suicide, disturbed sleep and diminished appetite. In older adults, it may be more difficult to establish whether the presence of such symptoms, for example reduced energy, concentration, and confidence, are due to depression, changes in circumstances or to the ageing process (Blazer & Koenig, 1996; DSM-IV, 1994; Siegler, Poon, Madden, & Welsh, 1996).

3. Prevalence

There have been a number of attempts to establish the prevalence of depression in the elderly. The main difficulty in comparing these studies is the differences in methodology. Zarit and Zarit (1984) and Blazer (1996a) suggest that the method of gathering information and the case identification process used to determine estimates of prevalence of depression (or other disorders) is what tends to account for discrepancies in the statistics reported. Greater

variation occurs between studies drawn from community dwelling populations compared with those from psychiatric facilities or nursing homes for the elderly (Blazer, 1996a; Jorm, 2000; Zarit & Zarit, 1984). Arean, Hegel, and Reynolds (2001) note that recent research by Demmler (1998) shows that as people get older there is a decrease in their use of mental health services, and Badger (1998) found that depressed older adults compensate for this by seeking help from primary care services, and that this section of the population would not, therefore, be represented in studies taken from mental health services.

A further difficulty is that the instruments used to measure symptoms of depression tend to vary across studies. For example, Steffens et al. (2000) used a modified version of the Diagnostic Interview Schedule and not a clinical interview. The use of such a schedule may lead to the under diagnosing of disorders as the symptoms and their severity cannot be teased out (Steffens et al., 2000). In contrast, Geiselmann et al. (2001) carried out their study with the use of an intensive interview schedule over an extensive 14 session period allowing them to be more specific and detailed in their findings, and possibly more accurate. Knauper and Wittchen (1994) found an important factor in the use of standardised questions or statements in that older people have difficulty in understanding or interpreting the language of such methods, and that this may influence the clinician's decision regarding a diagnosis of depression.

The Guy's/Age Concern Survey (Lindesay, et al., 1989) of older adults living in a socially deprived inner city community in London found that the prevalence of depression steadily increases from 65 years of age upwards. Gurland's study (1976) found that severe depression tends to have a peak time between the ages of 45 and 55, though there are factors that often prevent the true prevalence of it in later life being measured. One factor is the reticence of

professionals to admit older adults to psychiatric units for treatment, as there is a more pessimistic view regarding their recovery. A second factor is that depression tends to be under-diagnosed as it is more often present with somatic complaints in the elderly than in younger age groups (Kaplan et al., 1994), and older people are referred less frequently for treatment compared with younger people (Gurland, 1976). In addition, Geiselmann et al. (2001) cite Gallo and colleagues (Gallo, Rabins, Lyketsos, Tien, & Anthony, 1997) who found that older individuals have a reduced tendency to complain and are more prone to deny their depressed mood, or they may conceal or refute the presence of symptoms (Blazer & Koenig, 1996).

The community prevalence of Major Depressive Disorder (MDD) in older adults ranges from between 1.8% to 3% (Baldwin, 1997b; Blazer, 1997), though Lindesay, Briggs, and Murphy (1989) found a prevalence of it in older adults to be as high as 4.3%. Helmchen, Linden, Reischies, and Wernicke (1999) refer to studies that have reported prevalence rates of moderate and severe depression to vary between 0.07% and 5.4%, and all forms and degrees of clinically relevant depressive symptoms to range from 11.5% to 26.2%. The Guy's/Age Concern Survey (Lindesay et al., 1989) found that 13.5% of the sample suffered from a depressive illness, and Copeland et al. (1987) found that 11.2% of their elderly population in Liverpool suffered from depression.

Steffens et al. (2000) suggest that a more complex picture emerges if adults over the age of 65 are not viewed as homogenous. They cite a large number of epidemiological studies that suggest that the prevalence of MDD decreases compared to the general population after the age of 65. On closer examination, the demographic profiles of these studies under-represent individuals over the age of 80. Studies that have been sensitive to this 'within group'

variation have suggested that MDD increases again after 80 years of age (Steffens et al., 2000). Helmchen et al. (1999) note that people over the age of 80, or even 90, are no longer rare exceptions, and that epidemiological estimates of prevalence rates and investigation into the age-dependence of a disorder require people over the age of 85, and a higher proportion of men, to be included. They also state that cross-sectional assessments of prevalence rates ought to be enhanced by longitudinal studies if accurate statements are to be produced on the relationship between ageing and mental disorders.

Studies (Blazer, 1996a; Blazer & Williams, 1980; Copeland et al., 1987; Kaplan et al., 1994) of elderly populations consistently demonstrate that the prevalence of depressive symptoms far exceeds that of depressive illness. Between 10% and 15% of elderly people in the community have some degree of depressive symptomatology at a given point in time, but only about 3% will be found to have a depressive illness (Copeland et al., 1987).

This discrepancy, between depression as a symptom and as a psychiatric diagnosis, was investigated by Blazer and Williams (1980). They found a prevalence of depression of 14.7% with only 3.7% of these cases being regarded as a Major Depressive Disorder. The rest were subdivided into dysphoric disorders secondary to health problems, simple dysphoria, or cognitive impairment, resulting in depressive symptoms that fall short of an actual psychiatric diagnosis of MDD being common in older age. More recently, Geiselmann et al. (2001), in a community-based random sample of 516 subjects aged between 70 and 90, found MDD to be present in only 4.5% of subjects but 16.5% to have subthreshold depression. Blazer, Hughes, and George's (1987) community survey of 1, 306 adults over the age of 60 found that 27% of the population experienced depressive symptomatology that did not always fit the DSM-III categories (the relevant classification system at the time).

Even amongst institutionalised care, such as long-term care homes and acute-care hospitals, where the prevalence rates for MDD in both is estimated at 12%, significant depressive symptomatology (not MDD) was found in 31% of those in long-term care and 23% in acute-care (Allen & Blazer, 1991).

In the general adult population, lifetime risk for major depression remains around 16% (DSM-IV, 1994; Sturt, Kumarakura, & Der, 1984). At any one time, there are some 5% of the adult population who are suffering from depression (Williams, 1997), and 9% - 20% of the population suffering from significant symptoms of depression (Bradley, 1995; Paykel, 1984). In older adults, studies (Baldwin, 1997b; Blazer, 1997; Blazer & Williams, 1980; Copeland et al., 1987; Geiselmann et al., 2001; Helmchen et al., 1999) have suggested that the prevalence of MDD ranges from 1.8% – 5.4%, and from 10% – 16.5% for depressive symptomatology. When comparisons are made, there does not appear to be a greater prevalence of depression in older adults compared to the general adult population. There is a suggestion that the prevalence is greater in younger adults (Roth & Fonagy, 1996), but that the elderly kill themselves more often (Butler & Lewis, 1982).

A number of studies (Osgood, 2001; Pearson & Brown, 2000; Pitt, 1997) have found that older adults are at greater risk from completed suicide than younger adults. Pearson and Conwell (1995) report that statistics from industrialised countries reveal that suicide rates rise progressively with age, with the highest rates for men of 75 years and older. Pearson and Brown (2000) refer to the most current rates in the US (Peters, Kochanek, & Murphy, 1998) that report the highest rates are amongst older white males (65 per 100 000) and not amongst adolescent males aged 15 – 19 (16.6 per 100 000), and that older adults commit one out of every five suicides in America. In the UK, similar results have been found. Baldwin (1998)

notes that those over the age of 65 account for 15% of the population but for 20% – 25% of all completed suicides. One of the difficulties in establishing precise figures on suicide is that it is often under-reported or misclassified, particularly if the individual used alcohol (Pearson & Brown, 2000). Another difficulty is that in the US, different states have different registry systems as well as a variety of people from diverse backgrounds who are responsible for completing death certificates.

Psychiatric disorder and/or substance use was found to be present in approximately 90% of all suicides, and affective disorders were the most common psychopathology found when the Psychological Autopsy method was used (Conwell & Brent, 1995). This method assesses mental and physical disorders, health service use, personality and life events from all sources including interviews with knowledgeable informants, clinical records and case formulations from mental health professionals with expertise in post-mortem studies (Pearson & Brown, 2000). However, the results thus far using this method for older adults, has only yielded findings on age-based comparisons and has not as yet been applied to control groups. Pearson and Brown (2000) note the work of Conwell (1994) and Conwell and Brent (1995) that suggest older suicide victims had a physical illness and suffered from depression that was not co-morbid with a substance disorder. The type of depression found in the majority of cases was an uncomplicated first episode, which they state is the most treatable of late-life depressions.

Pitt (1997) states that epidemiological research has also shown that suicide increases with age, bereavement, isolation, deteriorating health and pain, but Angst (1999) argues that suicide risk in affective disorders is linear and thus constant over the lifetime. However, it is not clear whether Angst's (1999) conclusion excludes the factors stated by Pitt (1997), and his

argument is specific to affective disorders and therefore not necessarily generalisable to other disorders or to the population in general. Blazer (1996a) argues that the trend over the past 100 years for suicide rates to increase with age has flattened due to age, generational or cohort effects and period effects. He cites an example of a marked period effect postulated by Murphy, Lindesay, and Grundy (1986) in their study of suicides in England and Wales. In their cohort analysis of recorded suicides from 1921 – 1980, successively older cohorts were found to show a fall in suicide rates that contradicted findings in the US. Murphy et al. (1986) postulated that the decline was due to the detoxification of domestic gas in the 1960's. The rate of gas poisoning in the more elderly group decreased dramatically and this decrease, they suggest, was not offset by an increase in other methods of suicide. However, there does appear to be stronger evidence for suicide rates to increase in those over 65 rather than the curve to flatten (Baldwin, 1998; Center for Disease Control and Prevention [CDC], 2000; Osgood, 2001; Pearson & Brown, 2000).

Regarding the prognosis of depression in later life, Pitt (1997) notes that 30% of older patients are still depressed after 3 years, and Livingston and Hinchliffe (1993) found complete recovery in only 20% of cases. Burvill (1993) found that a significant proportion of participants made a partial recovery (24%) or relapsed within one year (18%), and he also found a high mortality rate of 11% in his study. Angst (1999) cites a recent UK study that supports the literature showing prognosis to be no worse in late-onset depression than in the early-onset variety. However, the majority of the epidemiological studies on prognosis cited above refer to cases in which there was very limited psychotherapeutic input, as the emphasis in treatment was predominantly pharmacological.

4. Theories of Depression

Different schools of thought have produced different theories on why older adults may become depressed. Biological theorists propose that ageing critically alters the levels of neurotransmitters and the body's response to stress (Finch, 1977; Lipton, 1976) which implies an increased vulnerability to depression in the elderly. Seligman (1975) proposed the learned helplessness theory, wherein individuals perceive themselves as having no control over events, and thus feel helpless and unable to actively re-organise or alter their circumstances. Behavioural theorists view depression as a state when there has been a withdrawal from positive activities and reinforcement, and cognitive theorists, such as Beck (1967, 1976) emphasise negative beliefs about oneself, events and experiences, and the future, resulting in negative thinking and distortions of reality. The multidimensional model of depression of Akiskal and McKinney (1973) combines many factors, such as stress, biochemistry, recent losses and so forth, and perhaps offers the most holistic and interactional conceptualisation of depression in older adults.

Psychoanalytic theorists link depression to traumatic early childhood experiences that are rekindled and exacerbated by more recent events (Freud, 1905). Others (Ardern, 1997; Butler & Lewis, 1982) emphasise the extent of the losses that occur in later life, and how the individual adapts to them,, as being a component of depression. Loss and the events that occur in ones' life impact on individuals in different and idiosyncratic ways. Life changes and the onset of depression have a close link in that significant life changes were found to be widely reported by depressed elderly people as having preceded a depressive episode (Post, 1967, 1972). The elderly are commonly bereaved of their loved ones, and bereavement has often been hypothesised to be a common precipitating factor for depression that hospitalises elderly patients (Turner & Sternberg, 1978). Yet, longitudinal studies have found relatively

low rates of depression in the bereaved, and that symptoms of depression in bereaved individuals are generally less severe and fewer than those in individuals institutionalised for depression (Gallagher, Breckenridge, Thompson, Dessonville, & Amaral, 1982). Retirement has also been assumed to have a negative consequence for the person, but research does not generally support this assumption (George, 1980). The ill effects found by studies may have much to do with the poor health and low income of the retired rather than with the retirement itself (Wilson, Chen, Taylor, McCracken, & Copeland, 1999). Physical illness too appears to play a prominent part in late-life depression (Kim, Braun, & Kunik, 2001).

However, Ariskel and McKinney (1973), Butler and Lewis (1982) and Ardern (1997) emphasise that as unique entities, each older person brings to late life a developmental history, a set of coping skills and a personality style all of which combine in order to determine how he or she will react to common problems and new life events. Therefore, one should be cautious not to assume that depression, rather than adaptation, is the common reaction to loss and stress in later life (Ardern, 1997; Butler and Lewis, 1982). Nevertheless, should depressive symptoms be present, the next task would be to assess them with a view to accurate diagnosis and appropriate intervention.

5. Assessment of Depression in Later Life

5.1 Symptomatology

Depression may have similar clinical presentations across age groups, but research (Mithani & Ancill, 1997; Pulska et al., 2000) appears to be showing that there may be factors that can modify the expression of it in the elderly. Some of these arise out of the ageing process itself, whereas others are due to generational differences in the perception of psychological and physical health, or differences occurring because of the frequent overlap of depressive

symptoms and physical illness (Karel & Hinrichsen, 2000; Mithani & Ancill, 1997; Pulska et al., 2000). Some factors may accentuate certain aspects of the clinical picture, whilst others may obscure the diagnosis. Therefore, problems associated with old age, such as cognitive impairment, memory loss, physical illness, and functional disability, rather than old age per se, may contribute to differences in presentation (Baldwin, 1998).

The symptomatology of older age depression was generally thought to differ qualitatively from that seen in the general adult population in that there are certain aspects of depressive illness that are thought to be more common in, or typical of, old age. They include more frequent delusions, an increased likelihood of presentations coloured by confusion (Horden, Holt, Burt, & Gordon, 1963), and a more 'endogenous' picture (Blazer, George, & Landermark, 1986). These findings, however, have tended to create a stereotypic picture of depressive illness in old age, and Baldwin (1997b) states that surprisingly few of the 'typical' features of later-life depression have withstood the rigours of modern research; this view is increasingly being born out in current research (Baldwin, 1997b; Gallo et al., 1997; Geiselman et al., 2001; Mithani & Ancill, 1997).

Mithani and Ancill (1997) emphasise that older adults present with a much higher weighting of neuro-vegetative symptoms in depressive illness than in a younger population. Gurland (1976) found that older adults tend to report more somatic symptoms, or a higher level of anxiety (Winokur, Morrison, & Clancy, 1973), than younger individuals, but fewer thoughts of suicide (Gallagher, Breckenridge, Thompson, Dessonville & Amaral, 1982; Winokur, Behan, & Schelesser, 1980). However, it has been noted that suicidal threats by the elderly are a rarity: older adults simply kill themselves (Butler & Lewis, 1982). Baldwin (1998) notes that older adults also present with an increased incidence of somatic preoccupation,

agitation, forgetfulness and delusions, though he does warn that these features may arise out of an over-emphasis on the severe end of the depressive spectrum, as is seen in in-patient settings. Penninx et al. (1998) found that the reporting of depressive symptoms by older adults resulted in them being at a higher risk for later physical decline. Neiderehe and Schneider (1998) state that symptoms such as lack of interest in normal pursuits, deterioration in self-care and actual or perceived problems with memory or attention may frequently predominate in older depressed adults.

5.2 Memory & Dementia

Prevalence estimates for the presence of dementia in those of 65 years of age and over are at least 5% of this population, and if analysed even further, it amounts to 1% of those who are 65 years old and the percentage then doubles approximately every 5 years to reach a figure around 30 % for those who are over 90 years of age (Helmchen et al., 1999).

It seems that older adults who complain of memory loss should also be evaluated for differential diagnoses of dementia and depression. Helchem et al. (1999) found, in line with other international studies, that the Berlin Ageing Study [BASE] reported a steep increase in the age-related prevalence rates of dementia from 1% in the age group 70 – 74 to 43% in those aged 90+. Memory problems may be a product of depression or anxiety (Watts, 1995) rather than Alzheimer's disease or another form of dementia.

Whereas there is some overlap of symptoms between depression and dementia, in that depressed people often report loss of memory and other intellectual impairment, and dementia patients may have depressive symptoms especially in the early stages, there are differences. Depressed persons do not necessarily have an obvious memory loss, though they

may complain of it, and they very seldom make errors on mental status tests (Kahn, Zarit, Hilbert, & Neiderehe, 1975; Raskin & Rae, 1981; Sinclair, Lyness, King, Cox, & Caine, 2001). Subtle intellectual impairments may occur in depressed persons, but seem to be less global or profound as one would find in cases of dementia (Kahn et al., 1975; Raskin & Rae, 1981). Gurland (1976) notes that depression is typically episodic, but with dementia the onset is mostly insidious and there is a gradual progression of symptoms, and Woods (1995) remarks that patients, particularly in the early stages of dementia, are aware of cognitive decline and feel depressed about it.

The term 'depressive pseudodementia' is a controversial one that is used in a number of ways (Baldwin, 1998). Its usefulness lies in facilitating the recognition that evidence of cognitive impairments in elderly patients can be misleading if these occur within the context of a mood disorder. As mood disorders are amenable to treatment, an exclusive focus on cognitive impairments at the exclusion of the influence of the depressive component can suggest an unnecessarily poor prognosis.

5.3 Physical & Medical Conditions

Mithani and Ancill (1997), Kim, Braun, and Kunik (2001), and Salzman and Shader (1978b) cite the most common factor complicating the identification of a depressive illness as the co-morbid presence of a physical illness. They list many conditions that can give rise to depressive illness, such as cardio-vascular diseases, various carcinomas, endocrine and metabolic disorders, infections, brain disease, intercranial aneurysms and Parkinson's disease. Adverse side effects from medications such as antiparkinsonian, antihypertensive and neuroleptic drugs have been found to cause depressive symptoms in their own right (Salzman & Shader, 1978b) and should be taken into account. Kim et al. (2001) suggest that

an assessment should involve a comprehensive diagnostic interview, including a complete medical history, and a careful examination. Teasing out the effects of the physical disease in order to differentiate the secondary depressive illness can be a difficult task. Baldwin (1998) suggests that appropriate sensitivity to anhedonia and cognitive features, such as guilt and self-deprecation, and changes in symptoms when physical health status is static may be useful.

Pulska and colleagues (Pulska, Pahkala, Laippala, & Kivela, 2000) carried out a study on 169 depressed (DSM-III criteria) elderly subjects drawn from an older adult population of 1, 225 from a municipality in Finland over a six-year period. They found that the emotional symptom of dissatisfaction (confirming Blazer's 1982 study) was common and pervasive amongst the elderly. When age, sex, smoking, physical health and functional abilities were taken into account, they found that dissatisfaction, weight loss and gastrointestinal symptoms (namely anorexia and constipation) predicted mortality together with high age and physical health. They also state that people with depression have poorer compliance concerning disease and their treatments than do those who are not depressed. At the other end of the spectrum, dismissing or under-estimating physical complaints through focussing on the features of depression can have fatal consequences. The results of this study are in line with those found in a nation-wide (n=29, 173) Finnish project spanning a 20 year follow-up period of adults aged 18-64 at the start of it who were unselected for mental health status (Koivumaa-Honkanen et al., 2001). They found that life dissatisfaction had a long-term effect on the risk of suicide and that this appeared to be mediated, in part, through poor health behaviour (e.g. smoking and drinking levels) and social situation.

5.4 Environmental & Social Factors

Factors such as social environment, living conditions, family support, family background, family psychiatric history, premorbid functioning, finances, mobility, community support and coping resources have also been found to be important and contributing issues (Akiskal & McKinney, 1973; Butler & Lewis, 1982; Kim et al., 2001; Wilson et al., 1999)

5.5 Late Onset of Other Psychiatric or Behavioural Disturbances

If depression can occur later in life, then perhaps so can other psychiatric or behavioural disturbances. Baldwin (1998) refers to this as 'neurotic symptoms' of recent onset. He notes that it is extremely rare to have obsessive-compulsive, hypochondriacal and dissociative disorders begin for the first time in late adulthood, and for this not to be part of a larger clinical picture that can frequently include a depressive disorder. Similarly, anxiety can be the most prominent symptom of distress but can co-occur with a depressive illness (Baldwin, 1998; Niederehe & Schneider, 1998). Baldwin (1998) also notes that recent onset of alcohol abuse or shoplifting, food refusal and aggression in more institutional settings, can be important markers of depression, and that behavioural problems may also arise out of the exacerbation of pre-morbid personality features in the context of a mood disorder. However, if the difficulties are simply attributed to the person's 'temperament' or age, then the mood component may be missed (Baldwin, 1998).

5.6 Assessment Tools

Assessment instruments developed for the diagnosis of psychiatric disorders, such as the Structured Clinical Interview for DSM-III-R (SCID), the Research Diagnostic Criteria (RDC) and the Diagnostic Interview Schedule (DIS) are the more commonly used tools for the use in an older population (Blazer, 1996b). In general, the scales used to assist in the assessment of

depression in older adults have proven to be both valid and reliable, such as the Beck Depression Inventory, the Hamilton Anxiety and Depression Scale, and the Center for Epidemiologic Studies-Depression Scale (Karel & Hinrichsen, 2000). However, such scales were not specifically developed for use with this population, though they appear to be proving reliable and valid with older adults who have a high level of education (Kazniak & Christenson, 1994; Pachana et al., 1994 in Karel & Hinrichsen, 2000). The Geriatric Depression Scale (GDS) (Yesavage et al., 1983) which has been recommended by the Royal College of Physicians and the British Geriatric Society (1992), and the adapted version of the Brief Assessment Schedule (BASDEC) (Adshead, Cody, & Pitt, 1992) were developed specifically for use with older adults, and both have shown acceptable levels of sensitivity and specificity (Rait & Burns, 1998). Hammond, O'Keeffe, and Barer (2000) have developed and validated a brief observer-rated screening scale for depression in elderly medical patients where prevalence rates of depression have been found to be as high as 50% (Hammond, O'Keeffe, & Barer, 2000), and was devised in order to facilitate a non-verbal assessment tool that could be used by nurses. The Camberwell Assessment of Need for the Elderly (CANE), although not specifically focussing on depression, can determine the presence of psychological distress which would allow for further investigation (Walters, Iliffe, Tai, & Orrell, 2000). Walters, Iliffe, Tai, and Orrell (2000) have also validated this tool for use in a primary care setting.

Rait and Burns's (1998) commentary on the screening for depression and cognitive impairment in older adults from ethnic minority groups raises the issue of many current assessment tools not necessarily being applicable for use with people from ethnic minorities. They note that the detection and management of depression in older adults from these groups has received little attention, which may, in part, be due to the difficulties and complexities

inherent in cross-cultural research. However, they cite examples of the GDS being adapted for use in India with a rural illiterate population (Kohli, Banerjee, & Verma, 1991) and with Chinese immigrants in the USA (Mui, 1996). The BASDEC has been translated into South Asian languages, but not as yet validated in these groups, the Hospital Anxiety and Depression Scale has been translated into Urdu, and an instrument for detecting emotional distress in older African-Carribeans has been developed by Abas (Rait & Burns, 1998).

6. Professional Beliefs and its Impact on the Detection and Treatment of Depression in Later Life

Professional pessimism regarding old age, i.e. a belief that illness is a natural consequence of old age, will affect assessment and treatment provided (Blazer, 1996b; Blazer & Koenig, 1996; Mather, 1997). Karel & Hinrichsen (2000) are of the opinion that inadequate recognition is the biggest barrier to effective treatment of depression in older adults. However, they note that this poor level of recognition is not only by physicians, but also by older adults and their families. Waxman, Carner, and Klein (1984) state that older adults have had less exposure to information on mental health issues than younger adults, and therefore, this older cohort may not view depressive symptoms as a mental health problem and seek help. Elderly people with even mild symptoms of depression may frequently present themselves to the physician and express more physical complaints than the elderly without such symptoms (Allen & Blazer, 1991; Niederehe & Schneider, 1998; Pearson & Brown, 2000; Waxman et al., 1984). Karel & Hinrichsen (2000) note that a different picture may emerge when more 'psychologically-minded' younger cohorts fall into the category of 'older adults'.

It appears that depression in the elderly is often 'missed' both in the hospital setting and in the community. Elderly people occupy nearly half of all hospital beds, and during a hospital admission for assessment or treatment of medical or surgical illness, between a third and a half of these patients will have, or will develop, a psychiatric disorder that can significantly influence the outcome of the associated physical illness, and approximately a quarter of these patients will suffer from a depressive disorder (Cooper, 1987; O'Riordan, et al., 1989; Burn, Davies, McKenzie, Brothwell, & Wattis, 1993). Both Baldwin (1998) and Pitt (1997) suggest that older people tend not to report their mood or to show a reduced expression of sadness, and this has been attributed to the shame of being depressed, the cultural attitude towards expression of emotion, the stigma attached to mental illness and a tendency to stress bodily symptoms rather than emotional distress.

Despite the high numbers of old people in general hospitals, the rates of referral to consultation liaison psychiatric services for the elderly are relatively low. Popkin et al. (1984) found that there were one third fewer psychiatric referrals amongst the elderly medically ill, than for those less than 60 years of age. There may be many reasons for this low referral rate of elderly people with depression for psychiatric assessment. First, there may be a failure to recognise psychiatric illness, or the depressed mood may be masked by a presentation of somatic symptoms (Kim et al., 2001; Mithani & Ancill, 1997). There may be a belief that certain symptoms are part of the ageing process, which involves depressed mood, apathy and impaired cognitive functioning (Pearson & Brown, 2000), and thus be viewed as normal due to losses and life changes, and therefore not identify those needing mental health treatment (Blazer, 1996b; Blazer & Koenig, 1996; Gurland, 1976). Collins et al. (1995) found this to be particularly true for older practitioners. Apart from the failure to detect potentially treatable psychiatric conditions, there may also be more general reasons for failing

to refer for psychiatric assessment, which has been analysed by Steinberg, Torem, and Saravay (1980). These may be particularly pertinent for the elderly population where the physician's difficulties in assessing psychological issues may be compounded by the attitudes and beliefs of the elderly person and his or her family. The physician may have difficulty in discussing the reason for psychiatric referral; he or she may believe that it will upset the patient, that it may prolong the hospital stay, or that physical treatment for depression will be too dangerous and complicated. Furthermore, physicians may be ignorant of other effective interventions for the elderly (Steinberg et al., 1980).

The tendency to under-diagnose depression in later life was highlighted in a major cross-national study by Mather (1997) who found that the rate of detection of depression by doctors and nurses was low, that most cases went unnoticed, untreated or were inappropriately managed, and that this affected subsequent morbidity and mortality. Zarit and Zarit (1984) found that American psychiatrists made fewer diagnoses of depression than British psychiatrists, and that they were more likely to diagnose depressive symptoms as organic brain damage. Woods (1995) notes DesRosiers' finding that across 18 studies, 10% of cases initially diagnosed as organic dementia were later rediagnosed as depression. However, Rao's (2001) prospective study, albeit much smaller in sample size, found that 10 out of 12 people referred by physicians in a hospital setting to an old age psychiatric service were correctly diagnosed with dementia, and 13 out of 16 with depression.

Blazer, Hybels, Simonsick, and Hanlon (2000) cite the USA Epidemiologic Catchment Area Study by Weissman, Bruce, Leaf, Florio, and Holder (1991) who found that the frequency of MDD between African Americans and White USA groups appear to be similar. However, Blazer et al.'s (2000) community-based cohort study of older adults over a 10 year period

reveal that African Americans were significantly less likely to be prescribed SSRI antidepressants, as opposed to tricyclic ones (in spite of their reduced side-effects) than Whites. They explain this as being a consequence of relative under-diagnosis of depression as a treatable condition in elderly African Americans coupled with prescribing practices of physicians being partly determined by the race of the individual.

Twining (1998) suggests that there is almost always a psychodynamic factor that can be identified in the histories of patients presenting with depression and that a detailed history is a crucial component. He (Twining, 1998) states that care should be taken not to stereotype older people and so leave certain problems unconsidered such as substance abuse, sexual difficulties, neglect or abuse by others. These facets of assessment focus on what is frequently not addressed and that is the salient uncomfortableness (or counter-transference) that working with older adults can present for the younger clinician (Ardern, 1997; Porter, 1997).

An issue that is frequently overlooked in the elderly is that of substance abuse (Atkinson, 1999; Blazer, 1996c; Phillips & Katz, 2001; Stewart & Oslin, 2001; Twining, 1998). Phillips and Katz (2001) cite the United Nations International Drug Control Programme of 1997 wherein the increase in prevalence of substance misuse in the elderly over time is already established. Even when substance abuse is diagnosed, which is seldom (Atkinson, 1999; Phillips & Katz, 2001; Stewart & Oslin, 2001), it may not be addressed. Estimates of the prevalence of alcoholism, drug addiction, problematic use and heavy use among older adults appear to vary from 2% to 25%, with significantly higher representation among medical and psychiatric patients as well as nursing home residents (Phillips & Katz, 2001; Stewart & Oslin, 2001). Stewart & Oslin (2001) cite figures of around 10% for gambling as an

addiction, and Phillip and Katz (2001) cite the UK Department of Health's figures that 19% of men and 23% of women between the ages of 65 and 74 smoke, and that 10% of men and 9% of women over the age of 75 are smokers. They also note that smoking has been found to be a risk factor in 8 of the top 16 causes of death for those who are 65 years of age and older.

Atkinson (1999) emphasises the links between depression, alcoholism and ageing, and he cites the largest US household survey (in Grant and Harford, 1995) on the assessment of syndromal depression and alcoholism for people aged 65 or more. The survey found that amongst those with lifetime DSM-IV Major Depressive Disorder 13.3% also met criteria for a lifetime alcohol use disorder, whilst only 4.5% without a history of MDD had an alcohol use disorder. Phillips and Katz (2001) and Stewart and Oslin (2001) note that the reluctance of professionals to consider the possibility of substance misuse and addiction in older adults as real problems, and to believe that individuals may be motivated to change, are still barriers that result in the condition being under-diagnosed and treatment interventions under-utilised.

An even more difficult issue to acknowledge and assess is abuse. Osgood and Manetta (2001) review the literature in this area. They cite a number of studies that confirm that adult women survivors of childhood physical or sexual abuse, rape, and battering have an increased risk of depression and of suicidal behaviour. They refer to the US Department of Justice's 1991 study that places the incidence of rape of women 65 and older at 10 per 100 000 in the US. Their own study (Osgood & Manetta, 2001) found that women with suicidal ideation had significantly more victimisation (such as sexual abuse, rape and battering) than women without suicidal ideation, but no significant difference was found between the subtypes of abuse. Many of these women, both in their study and in the others, stated that they had never mentioned the abuse or received support around it. They note that the distress around the

incident(s) may still be present once they move into later life. They also mention that many of these women have turned to drugs or alcohol as a means of coping with the distress. In addition, Osgood and Manetta (2001) cite a study by Raymonds (1994) wherein it is estimated that each year in the US between 700 000 and 1.5 million older adults are victims of abuse by another elder.

Thus far, this review has aimed to discuss the incidence of clinical depression in an older adult population, the possible causes of it, and the complex nature of making an accurate diagnosis of it in the light of confusing clinical presentations and professional pessimism. What remains to be explored is whether psychological intervention is an appropriate form of treatment for this population.

7. Psychological Treatment Interventions

A conclusion of the 1992 National Institutes of Health [NIH] Consensus Development Conference on Diagnosis and Treatment of Depression in Late Life (Washington, DC) that psychological interventions for older adults experiencing depressive symptoms were only moderately effective and were listed third in a hierarchy of recommended treatments, behind pharmacotherapy and electroconvulsive therapy. It is uncertain on what grounds such a conclusion was drawn, though in part it appears that it may have been due to the very limited research available for scrutiny at the time. Now, a decade later, research findings (Bortz & O'Brien, 1997; Gerson et al., 1999; Karel & Hinrichsen, 2000; Knight, 1999; Niederehe & Schneider, 1998) are providing sufficient evidence to contest this view.

On the whole, psychological treatment for depression seems to be offered less often to older adults than to younger adults (Woods, 1995). The efficacy of antidepressant medication in

older adults has been proven but there are difficulties in their long-term use with some patients with specific physical problems (Blazer, 1996b, Kim et al., 2001), and there is an increased risk for adverse side effects in this population, particularly with tricyclic antidepressants (Karel & Hinrichsen, 2000; Gerson et al., 1999; Niederehe & Schneider, 1998). Relapse rates are relatively high: a significant number of patients recover only partially (Burvill, 1993; Livingston & Hinchliffe, 1993; Murphy, 1982; Pitt, 1997) with some being left with disabling symptoms; almost a fifth relapse within a year (Burvill, 1993); and as many as a tenth do not improve at all (Murphy, 1982), though Gerson, Belin, Kaufman, Mintz, and Jarvik (1999) place this at a lower estimate of a third who do not show any improvement. The scope, therefore, for psychological approaches to supplement or substitute pharmacological treatment seems apparent, particularly as this population tend to have multiple medical conditions, they are more sensitive to adverse drug effects, and they may be exposed to a number of psychological stressors such as loss of status (Gerson et al., 1999).

The reluctance to use psychological approaches in large is part of a legacy of the Freudian view that older adults are not suitable for therapy. Freud, in 1905 (p. 264) wrote that older individuals were not fit for psychoanalytic treatment because "... near or above the age of 50 the elasticity of the mental processes, on which treatment depends, is as a rule lacking - old people are no longer educable ...". Although this needs to be seen within the context of his era, Porter (1997) expresses the view that this attitude lingered for years and has often been the excuse for not treating depression in later life with psychological methods. Though age-related changes in mental functioning do necessitate modifications of the psychological techniques used for treating depression (Bortz & O'Brien, 1997; Karel & Hinrichsen, 2000; Knight, 1999) it is becoming increasingly evident that they do not exclude older adults benefiting from treatment (Gallagher-Thompson, Hanley-Peterson, & Thompson, 1990;

Karel & Hinrichsen, 2000; Katona, 1994; Knight, 1999; Niederehe & Schneider, 1998; Scogin & McElreath, 1994; Steur et al., 1984; Thompson, Gallagher, & Steinmetz-Breckenbridge, 1987).

Scogin & McElreath (1994) found comparable effect sizes when comparing the overall effect size (***d***=.78) established in their meta-analysis of psychological interventions with older adults with that found in such interventions with adults of all ages (***d***=.73) and psychotherapy efficacy in general (***d***=.85). A study by Meats, Timol, and Jolley (1991) of in-patients found a better outcome for an older compared to a younger group, and Roth and Fonagy (1996) also identify older subjects to be as responsive to psychotherapeutic interventions as younger patients but that they may require a longer period of treatment in order to show the same gains. Weiss and Lazarus (1993) point out that high drop-out rates in outcome studies are frequently attributable to transportation and physical health problems. Ardern (1997) and Twining (1998) therefore suggest that therapy be taken to the client in order to work around such difficulties and assist compliance. They also suggest that session lengths require flexibility so as to accommodate the likelihood of potential difficulties with concentration and fatigue, and that sensory impairments need to be accommodated, such as failing eyesight or hearing.

Various forms of brief, structured psychotherapeutic input have been developed in recent years for the treatment of depression in older adults. Behavioural therapy (Lewinsohn, Munoz, Youngren, & Zeiss, 1978), Beck's cognitive-behavioural therapy (Beck, 1967), Klerman and associates' (1984) interpersonal psychotherapy, a psychoanalytic approach (Ardern, 1997), and family therapy (Benhow & Marriott, 1997; Herr & Weakland, 1984) have emerged.

An overview of review studies (Bortz & O'Brien, 1997; Karel & Hinrichsen, 2000; Gerson et al., 1999; Niederehe & Schneider, 1998; Scogin & McElreath, 1994) of the efficacy of psychotherapy with older adults appear to reach similar conclusions, regardless of the differences in inclusion criteria. Gerson et al.'s (1999) meta-analysis included double blind (for the drug trials), randomised controlled studies with a quantitative documentation of depressive symptoms (MDD) utilising observer pre- and post-treatment rating scales. Niederehe and Schneider's (1998) review incorporated eight randomised controlled studies with participants diagnosed with Major Depressive Disorder, and ten controlled studies including mixtures of depressive subtypes. Scogin and McElreath's meta-analysis (1994) included seventeen studies of psychosocial treatment interventions, each of which included either a comparative control group or a second psychosocial intervention. Bortz and O'Brien (1997) and Karel and Hinrichsen (2000) studies reviewed the available literature but did not state exclusion criteria.

Across the board, any psychological intervention appeared to yield more positive results than no therapy or placebo (Gerson et al., 1999; Niederehe & Schneider, 1998; Scogin & McElreath, 1994). The comparative efficacy of one modality over another was not found, nor was there any advantage of individual therapy over group therapy, or vice versa (Gallagher-Thompson et al., 1990; Gallagher & Steffens, 1994; Gerson et al., 1999; Niederehe & Schneider, 1998; Roth & Fonagy, 1996; Scogin & McElareath, 1994; Steur et al., 1984).

The comparisons of intervention efficacy appear to be mainly between studies that utilise cognitive, behavioural, and psychodynamic modalities, with only a few studies on interpersonal therapy being included (Bortz & O'Brien, 1997; Karel & Hinrichsen, 2000;

Niederehe & Schneider, 1998; Scogin & McElreath, 1994) and even fewer on reminiscence therapy or review therapy (Bortz & O'Brien, 1997; Karel & Hinrichsen, 2000; Niederehe & Schneider, 1998). A marked absence of research with depressed older adults in the modality of family therapy was noted. Karel and Hinrichsen (2000) make reference to this and state that of the nine studies they found all except one was on the efficacy of treatment with caregivers of older adults. They cite Teri, Logsdon, Uomoto, and McCurry's (1997) study that involved two behavioural interventions taught to the family caregivers of depressed dementia patients on techniques to help reduce the patients' depressive symptoms. There does, however, appear to be a growing body of literature on the use of family therapy with older adults (Benbow & Marriot, 1997; Carpenter, 1994; Gilleard, 1996; Herr & Weakland, 1984; Qualls, 1999) but few empirical studies appear to be available.

In a systematic review on treatments (both drug and psychotherapy) for late life depression in primary care Freudenstein, Jagger, Arthur, and Donner-Banzhoff (2001) used literature searches from electronic databases for articles in French, English and German, and quality criteria for inclusion based on Cochrane Effective Practice and Organisation of Care Group. Their inclusion criteria were controlled clinical trials, randomised controlled trials, controlled before and after studies, and time series studies on cognitive/cognitive behavioural therapy, interpersonal therapy, counselling, social support, and drug treatment. The subjects needed to have been recruited from a sample of the general population or from primary care attenders, and to be 60 years of age or over. Studies that were based on those of adult age but included those of 60+ were also used. The outcome was that no studies on psychological treatment were found. Only five studies were applicable in the area of drug or other treatment, two of which were specifically with adults over the age of 60 whilst the others only included participants of that age group. The authors emphasise how research into depression has

tended to be carried out exclusively on those with severe, uncomplicated Major Depressive Disorder as there are reliable methods to identify and categorise it (such as the DSM and ICD), but that symptoms below the diagnostic level are more difficult to classify and are therefore mostly excluded. However, they note that milder forms of depression are more commonly found in primary care than MDD, and that research on MDD may only be applicable to <15% of depressed primary care patients. In addition, the findings on efficacy can therefore only be applied to this minority and not to all older patients who are depressed, and particularly not to those who are only seen in primary care settings.

Arèan et al. (2001) discuss three ongoing research programmes that are utilising specifically modified forms of psychotherapeutic modes (namely, CBT, IPT and Problem Solving therapy). These intervention models have been modified to suit their use in a primary care setting specifically with older adults. Preliminary results suggest that all three modified models are showing some level of efficacy, though a precise analysis of their efficacy can only be carried out once the results are more fully available.

Gerson et al. (1999) note that the literature comparing psychological and pharmacological treatments for depression across the age groups suggests that psychological interventions may be comparably effective as medication in milder to moderate depression as well as severe depression. Niederehe and Schneider's (1998) meta-analysis of ten psychosocial treatments with previous meta-analyses by Schneider (1994) on imipramine and nortriptyline, respectively, for the treatment of depression in late-life found comparable mean effect sizes between psychosocial interventions (***d***=.74), imipramine (***d***=.60) and nortriptyline (***d***=.62). Combined treatment (i.e. psychotherapy and medication) has been found to be more effective than only drugs or psychotherapy when treating and preventing relapse in severe depression

(Gallagher & Thompson, 1983; Gerson et al., 1999; Reynolds, 1997; Thase et al., 1997; Thompson, Gallagher, Hanser, Gantz & Steffens, 1991).

The main limitations stated in the studies are, firstly, the limited number of psychological treatment studies available, particularly in modalities other than cognitive-behavioural therapy (Freudenstein et al., 2001; Gerson et al., 1999; Karel & Hinrichsen, 2000; Niederehe & Schneider, 1998; Roth & Fonagy, 1996; Scogin & McElreath, 1994). However, there appear to be numerous studies available on the use of different antidepressant medications. Gerson et al. (1999) comment that this may be due to funding issues rather than to any reluctance to undertake research. Secondly, many of the psychological treatment studies have used participants with mixed depressive disorders whereas the drug studies have focussed predominantly on MDD making comparisons more difficult (Gerson et al., 1999; Niederehe & Schneider, 1998; Roth & Fonagy, 1996). Lastly, the generalisability of the psychological treatment studies is limited as the majority of participants were either relatively well-functioning, or came from predominantly white populations (Gerson et al., 1999; Karel & Hinrichsen, 2000). Furthermore, those of 80 years and older were seldom included in such studies, as were older adults with multiple co-morbid medical conditions, neurobiological and/or chronic psychiatric disorders (Karel & Hinrichsen, 2000).

8. Conclusion

This review has discussed and challenged what appears to be the simplistic view that depression in older adults is a predetermined reaction to the ageing process and thus does not require accurate assessment or treatment priority. It appears plain that when comparing prevalence rates, symptoms, treatment efficacy, and the implications of loss and life events in the older depressed population with that of the adult population, there were no significant

differences between the age groups. These findings led to a discussion and analysis of issues around the understanding and treatment of depression in older adults and how it is frequently 'missed' and at worst, dismissed. Whether this is a reflection of an unwillingness to treat the disorder or a lack of understanding is uncertain. What appears to be evident is that health care specialists may benefit from a greater understanding of its assessment, treatment and management. Professionals are adjusting and altering present methods, and developing new assessment and treatment strategies that are specific to older adults and not to all age groups, and different antidepressant and psychological interventions have proven to be successful as individual and combined treatment modalities. It is anticipated that the implementation of the National Service Framework for Older Adults (Department of Health, 2000) will raise the profile of the needs of older adults and establish age-appropriate facilities, both physical and interventional, for this important and ever-increasing population.

9. References

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