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Negative self-schemata in depression: The role of
automatic self-evaluation.

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ABSTRACT
FACULTY OF MEDICINE, HEALTH AND SOCIAL SCIENCES
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NEGATIVE SELF-SCHEMATA IN DEPRESSION: THE ROLE OF AUTOMATIC SELF-EVALUATION

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Research into schemata in depression has found little evidence for schematic activity in the absence of a depressed or low mood. This has led to a widespread view that schemata in depression are latent and only influence information processing in the presence of a low or depressed mood (Segal, 1988). The lack of evidence of schematic activity in the absence of a depressed mood may be due to traditional conceptualisations of schemata and the methodological difficulties inherent in the implicit tasks that are used. The aim of this thesis was to investigate schematic activity using two relatively new automatic self-evaluative tasks (the IAT and EAST). It was found that positive automatic self-evaluation was weaker in analogue depressed individuals, high-trait depressives, and recovered clinical depressives compared to non-depressed individuals and low-trait depressives. More importantly, these differences in automatic self-evaluation were not affected by mood or levels of depression. This thesis provides some support that vulnerability to depression or schematic activity can be measured in the absence of a depressed mood. These results also provide support for the growing evidence that automatic self-evaluation may be implicated as a vulnerability factor related to affective disorders (De Raedt, Schacht, Franck, & De Houwer, 2006; de Jong, 2000, Tanner, Stopa, & De Houwer, in press), and why SSRI antidepressant treatment may not be effective in preventing relapse in depression (Hensley, Nadiga, & Uhlenhuth, 2004). Suggestions for further research into schemata include further examination into the role of positive automatic self-evaluation in healthy individuals, the ratio of and different kinds of positive and negative schematic content in individuals who are, and who are not, vulnerable to depression, and investigating schemata from the ontological and neuroscientific perspectives.

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Chapter 1

Cognition and depression

Black days.....I know you well. - Jan Brokel

1.1. Introduction

Over the last twenty years there has been a burgeoning of research in the development and refinement of theoretical accounts of depression (Segal Williams, & Teasdale, 2002). Cognitive theories of depression concerning the origins and maintenance of depression have been very influential in recent years with the development of a successful treatment for depression (e.g., Beckian Cognitive Behavioural Therapy; CBT; Beck, 1967, 1976) as a result of empirical research into cognition in depression. From this success other related treatments focusing on depression have also been developed (e.g., Mindfulness Based Cognitive Therapy; MBCT; Segal et al., 2002; Schema Focused Therapy; SFT; Young, 1990).

It is now recognised that depression is a multifaceted phenomenon, involving a complex and dynamic interaction of biological, social, developmental, affective, behavioural, as well as cognitive factors (Segal & Dobson, 1992). Cognitively orientated psychologists, whilst recognising this complexity, have focused on the role of cognition in vulnerability, onset, and maintenance of depression. Cognitive psychopathologists work upon the assumption that changes at a cognitive level can effect concomitant change at other levels (e.g., biological, affective, behavioural, etc.). Conversely, cognitive variables may be affected by biological, behavioural, affective, or social changes. In other words, cognition can affect the transmission of serotonin and change a person's behaviour (e.g., increased serotonin and positive behaviours), and serotonin transmission can affect how one thinks and behaves (e.g., happier mood and positive behaviour) (Le Doux, 1998; Bucci, 2000; Stern, 1992).

The main advantage of studying depression from a cognitive perspective, as opposed to biological, psychoanalytical, or behaviourally based formulations,¹ is that the cognitive perspective can inform researchers about the phenomenological experience of depression; the rules, representations of the world, the self and others, the past, present, and the future, that guide an individual who is afflicted by depression or who is vulnerable to depression. The cognitive model also provides an etiological and explanatory account of depression, unlike other models (Beck, 1967, 1976) (see footnote 2 & Appendix I, & II). Thus, the cognitive perspective therefore is not as reductionist as some other perspectives. The behaviourist or biological models focus only upon a very small portion of the available facts (e.g., brain chemicals or observable behaviour). These models therefore neglect the cognitive-

¹ See Appendix I for a full discussion of the biological, psychoanalytical, and behavioural based models of depression. This Appendix also provides an overview of the phenomenology and epidemiology of depression.

experiential aspects of depression that have become recognised as fundamental to the disorder, its onset, and maintenance. Further, the biological approach is unfortunately a long way off from providing a definitive explanation of the role of biological factors in depression (James, 1998). The neurotransmitter and biological abnormalities hypothesised to be linked to depression have also been implicated in a myriad of other human behaviours such as aggression (O'Keane, 1992; Linnoila, de Jong, & Virkkunen, 1990; Von Knorring, 1987; Schukit, 1986), substance misuse (Boyer, McFadden, & Feighner, 1996), compulsive disorders e.g., gambling (Coccaro, Siever, Owen, & Davis, 1990; Zuckerman, 1984), anorexia (Brewerton, Brandt, Lesem, Murphy, & Jimersen 1990), and social phobia (Bouwer & Stein, 1998). Equally, behavioural and psychoanalytical theories can be applied to other mental disorders and thus it is far from clear if the postulates of these models are only applicable to depression (Lavelle, Metalsky, & Coyne, 1979). With the cognitive model of depression, however, there are certain aspects inherent in the model that are unique to depression (e.g., negative thoughts about the self, world and future), which do not relate to other cognitive models of other disorders (e.g., anxiety) (Hagga, Dyck, & Ernst, 1991; Beck, 1967, 1976).

There are three main ways to conceptualise research on cognition in depression, each corresponding to a different level of analysis in the cognitive system. The first level can be described as cognitive products. These may take the form of negative automatic thoughts, which are of a self-critical nature, self-indicting attributions, pessimistic or hopeless expectations for the future, and/or perceptions of personal failure and inferiority. This aspect of the cognitive system is largely accessible and can be measured in a relatively direct and non-inferential way; from the result of explicit and conscious deliberation as obtained from self-report measures (Beck, 1967, 1976).

The second level, cognitive processes, operate at a less manifest level, and represent the cognitive and social mechanisms by which individuals formulate judgments, evaluations, expectations, perceptions, self-focused attention or faulty cognitive operations. These lead to errors in thinking in the cognitive products part of the system, such as jumping to negative conclusions (Beck, 1967, 1976).

Thirdly, cognitive structures or schemata are thought to operate at a structural level in order to store, organise, integrate, and direct the processing of personally important information. This level of cognition in depression is hypothesised to operate at an implicit level, i.e., it is not available to conscious introspection, yet it is implicated in the etiology, maintenance, and relapse of depressive disorders (Beck, 1967, 1976). Much of the past and current research into cognition in depression has been influenced by Beck's (1967, 1976) cognitive theory of depression and the treatment that he subsequently developed.

Beck's theory suggests that all three levels of cognition, cognitive products, process and cognitive schemata of cognition, are integral to the etiology and maintenance of

depression. However, cognitive schemata that are hypothesised to underlie vulnerability to develop depression have not been adequately addressed by empirical research. There is a preponderance of research related to cognition in depression that has either focused on the products of cognition (e.g., negative thoughts) via self-report measures, or on cognitive processes (e.g., biased judgments of depressed individuals on hypothetical social vignettes; see Ingram, Miranda, & Segal, 1998 for a review).

Researchers are now beginning to attempt to measure cognitive structure or schemata in affective disorders with new and innovative information processing tasks such as the Implicit Association Task (IAT; Greenwald, McGhee, & Schwartz, 1998) and the Extrinsic Affective Simon Task (EAST; De Houwer, 2003) (see De Raedt, Schacht, Franck, & De Houwer, 2006; Gamar, Segal, Sagrati, & Kennedy, 2001; de Jong, 2000). However, there is considerable debate over the nature of schemata in depression, and little prior research that has investigated the interface between cognitive products, processes, and schemata. Therefore, this chapter will focus on Beck's cognitive model of depression and the levels of cognitive products and cognitive processes proposed in that model. Empirical evidence for these two parts of the model will be evaluated in this chapter. The schema concept will be introduced in this chapter, but will be dealt with in a more comprehensive way in Chapter 2. This is because cognitive products and cognitive processes are two aspects of Beck's model that are well established in the literature, in contrast to the schema concept where there lies much theoretical confusion.²

1.2. Beck's cognitive model of depression

Beck's cognitive theory of depression (Beck, 1967, 1976) has generated a vast body of empirical work. One of the prime reasons for the importance of studying Beck's model is that in studies comparing pharmacotherapy and cognitive therapy, cognitive therapy seems to be as effective in reducing depressive symptoms, and more effective at reducing relapse rates (Segal, Gamar, & Williams, 1999). Secondly, in cognitive therapy, the alteration of dysfunctional cognitions may produce a concomitant change at other levels (e.g. biological, affective, behavioural; Jacobson & Gortner, 2000). Therefore, by looking at this model in more detail, it may shed light on the mechanisms involved in depressive cognition, which in turn will help depression researchers to develop improved treatments, and improve the understanding of the role of cognition in depression (Brewin, 1988).

² This chapter will not discuss other cognitive theories that have evolved from Beck's theories, which examine cognitive products and cognitive processes associated with depression. Despite having been influential in the development of cognitive theories of depression alongside Beck's theory, these other theories only focus on one small part of the cognitive system and therefore this leads to an overly-descriptive rather than an explanatory accounts of depression. This highlights the impact and comprehensiveness of Beck's model in influencing cognition research in depression. For more details on these other theories see Appendix II.

The cognitive model of depression has evolved from systematic clinical observations and experimental research (Beck, 1967; 1976; 1987). The cognitive model proposes three key concepts to explain the phenomena of depression: (1) the cognitive triad or contents of consciousness, (2) cognitive errors or faulty cognitive processes, and (3) cognitive schemata or cognitive structures. These are described in more detail below.

1.2.1. The cognitive triad

The cognitive triad consists of three major cognitive patterns, which describe the way in which depressed individuals regard themselves, their future, and their world. The first part of the triad, a negative view of the self, involves the person seeing him/herself as defective, inadequate, diseased, or deprived. Depressed people attribute negative experiences to a psychological, moral, or physical defect in themselves. Depressed people believe that, because of their presumed defects, they are undesirable and worthless. They will tend to underestimate or criticise themselves. These negative thoughts contribute to, and facilitate a spiral into a low or depressed mood (Beck, Rush, Shaw, Emery, 1979).

The second part of the triad involves depressed people's tendencies to interpret their present experiences in negative ways. They see the world as making insurmountable demands and obstacles which prevent fulfillment of, or achievement of, life's goals. They misinterpret their interactions with the environment as representing defeat or deprivation. These negative misinterpretations occur even when there are more plausible, alternative explanations available. Depressed people may realise that their interpretations are negatively biased if they are shown less negative alternative explanations (Beck et al., 1979).

The third part of the triad involves a negative view of the future. Depressed people make long-range projections and because of this they anticipate that present difficulties will continue into the future. Depressed people expect hardship, frustration, and deprivation. When they consider a specific task in the future, they expect to fail (Beck et al, 1979).

In effect, the negative cognitive triad encompasses the range of negative thoughts that make up the content of the depressed person's consciousness, and which are available to introspection (Hollon & Kendall, 1980).

1.2.2. Evidence of negativity & the cognitive triad

There is much empirical research that has investigated whether depressed people think more negatively than non-depressed people about themselves, the future, and the world. The nature of the thought content is one factor that differentiates individuals suffering from depression from individuals suffering from other psychological disorders, such as anxiety or phobias (Hagga et al., 1991). The greater negativity of depressed patients' cognitions is routinely confirmed with measures of negative thinking such as the Crandell Cognitions Inventory (CCI; Crandell & Chambless, 1986) and the Automatic Thoughts Questionnaire (ATQ; Hollon & Kendall, 1980). Depressed individuals consistently score higher on these

scales than non-depressed individuals (Blackburn, Jones, & Lewin, 1986; Crandell & Chambless, 1986; Dobson & Shaw, 1986). Also, compared to non-psychiatric patients and remitted depressed patients, depressed people score higher on these measures (Blackburn, Jones et al., 1986). Furthermore, when depressed patients are tested during remission, their scores are significantly lower than during an episode of depression (Dobson & Shaw, 1986).

Compared to non-depressed controls, depressed people are more self-critical (Blatt, Quinlan, Chevron, McDonald, & Zuroff, 1982); report lower self-esteem (Lewinsohn, Steinmetz, Larson, & Franklin, 1981); endorse more negative and fewer positive adjectives as self-descriptive (Hedlund & Rude, 1995; Bradley & Mathews, 1988); and rate themselves as more discrepant from their ideal selves (e.g., depressed people feel they are more defective in comparison to a more successful ideal they have of themselves; see Blatt et al., 1982). Depressed individuals also view themselves more negatively than individuals in remission from depression (Blatt et al., 1982).

Depressed individuals are also more negative or hopeless about the future than non-depressed or remitted depressed people (Abramson, Garber, Edwards, & Seligman, 1978; Blackburn, Jones et al., 1986; Dohr, Rush, & Bernstein, 1989). Depressed patients had higher scores than non-distressed controls and individuals who had recovered from depression on the world subscale (negative thoughts about the world, e.g. the world being a hard and unforgiving place) on the Cognitive Style Test (CST; Blackburn, Jones et al., 1986).

In agreement with the cognitive model of depression (Beck, 1967, 1976), research confirms the existence of specific content in the negative thoughts of depressed people. Empirical work has routinely observed more negative thinking regarding the self, the world and the future in depressed individuals.

1.2.3. Cognitive errors or faulty cognitive processes

Beck (1967) proposes that the thinking style of those suffering from depression is encapsulated by characteristic logical errors in cognition. Examples of this are: arbitrary inference (jumping to conclusions), selective abstraction (only taking the negative things out of experience), overgeneralisation (if one thing goes wrong then everything is wrong), magnification/minimisation (magnifying one's mistakes and minimising one's achievements), catastrophising (making more out of a negative experience than there actually is), personalisation (attributing negative things to one's own fault), and absolutist/dichotomous thinking (looking at things in black and white rather than shades of grey). Depressed individuals also engage in what Beck calls primitive thinking: non-dimensional & global thinking ("I am without hope"), absolutist and moralistic ("I am a terrible person"), invariant ("I am useless and always will be"), character diagnosis ("I have a character defect"), and irreversibility ("nothing can be done for me").

1.2.4. Evidence for cognitive errors or faulty cognitive processes

There is a whole plethora of empirical research that supports Beck's (1967, 1976) claims that faulty cognitive processing is a fundamental aspect of depression (Segal, 1988; Hedlund & Rude, 1995; Ingram et al., 1998). In a study by Gotlib (1983) investigating cognitive processes in depression, depressed patients recalled feedback that they had received from another person in a social interaction task, as significantly more negative than it actually had been compared to psychiatric and non-distressed controls. The psychiatric and non-distressed controls were more accurate regarding the feedback they received. Further, depressed patients overestimated self-punishments administered during a learning task and underestimated self-reinforcements compared to the control group who were more accurate (Gotlib, 1983). Lastly, highly symptomatic depressed individuals, unlike controls, underestimated high rates of positive performance on a task, while instead focused on the negative performance they thought they had produced (De Monbreun & Craighead, 1977).

Depressed patients are also more likely than non-depressed people or psychiatric controls to draw strong negative conclusions that go beyond the information depicted in hypothetical scenarios. In one study by Dobson and Shaw (1986), participants had to read hypothetical social vignettes and make judgments about how they would have felt or responded in that situation. The depressed people were more likely to reach negative conclusions and/or report that they would have been put in a low mood as a result of the situation.

Similarly, Watkins & Rush (1983) used the Cognitive Response Test (CRT), which is a 36-item open-ended sentence completion format and assesses dysfunctional cognitive processes. They found differences between depressed, non-depressed, and psychiatric controls on the type of sentence completion provided. A sample CRT item "My employer says he will be making some major staff changes. I immediately think: ___". They found that the depressed group made more irrational and negative sentence endings. Further, CRT scores were related to severity of depression.

Overall, the research suggests, in support of Beck's (1967, 1976) claims, that depressed people's thinking processes are distinguishable by the presence of characteristic logical errors. Depressed individuals are more likely to make negative inferences in a variety of different circumstances and contexts.

1.2.5. Schemata or structure in depression

A key postulate of the cognitive model of depression is that schemata are enduring and stable cognitive structures, which serve as conceptual filters for coding, screening, and the evaluation of impinging stimuli. Furthermore, schemata are argued to be a template which, in relation to the self in depression, are dysfunctional, and are structures that when activated produce depressive affect. Schema theorists argue that dysfunctional schemata are implicated in the vulnerability, maintenance, and relapse of depressive disorders, although

they may differ in the precise definition of a schema, and in the exact definition of how the schema operates in these aspects of depression (Beck, 1967, 1976).

It is important at this point in the discussion of schemata to highlight the distinction between negative self-schemata/cognitive vulnerability to depression and the risk factors associated with an individual's increased probability of developing depression. This is because there are important differences between the terms vulnerability to depression and risk factors associated with increased probability of developing the disorder. What is apparent is that the development of depressogenic negative self-schemata is facilitated in individuals who have been exposed to certain kinds of antecedent risks identified by research (e.g., negative childhood experience; Beck, 1967, 1976). Several antecedent risk factors for depression have been put forward as being responsible for the development of vulnerability to depression; childhood onset of major depression or dysthymia, a family history of affective disorders, alcoholism, a social learning factor (e.g., negative reinforcement), adverse early life experience involving parental loss, sexual or physical abuse, attention-deficit disorder (Hersen & Ammerman, 1995), temperament (Thase, 1990), being a female (e.g., hormone, social, environmental, and rumination style factors associated with being female; Radloff & Rae, 1979; James, 1998; Akiskal et al., 1987; Schmidt, Nieman, Grover, Muller, Merriam, & Rubinow, 1991; Nolen-Hoeksema, 1987). There are also additional antecedent risk factors such as a person's genetic heritage and biological makeup (see Downing-Orr, 1998, for a review).³ What is not clear regarding the occurrence of any of the above antecedent risk factors is how and in what way they may contribute to the development of negative self-schemata associated with the biased information processing style characteristic of depression (Ingram et al., 1998; James, 1998). For some people the loss of a parent may be enough for the development of negative self-schemata, whereas for others it is likely to be a combination of several risk factors that leads to the development of depression (Beck, 1967, 1976; Downing-Orr, 1998). Therefore, for the purposes of this thesis, vulnerability to depression refers primarily to the presence of negative-self schemata and associated negative information processing biases, not to the antecedent risk factors of depression that have been put forward as possible factors for schematic development. The extent of any discussion of risk factors to depression in this thesis will be limited to the discussions of certain negative early maladaptive schemata or negative life themes that have been argued to be an important aspect of childhood experience associated with the contribution of negative self-schemata and future adult depression (e.g., a feeling of being abandoned; Young, 1990), and aspects pertaining to traits of depression that have been argued to be associated with the increased likelihood of developing or vulnerability to depression (e.g., trait depression in studies 3 & 4; Zemore et

³ Please see Appendix I for a full discussion of the risk factors associated with depression. This appendix also includes a discussion on biological and genetic aspects of depression.

al., 1990).⁴ It is suggested that certain cognitive and behavioural traits confer an increased risk for the subsequent development and increased vulnerability of clinical depression (Zemore et al., 1990). Therefore, one may assume that the development of these traits is related to antecedent risk factors and the subsequent development of negative self-schemata and thus, theoretically, increased vulnerability to depression (Beck, 1967, 1976).⁵

As discussed earlier, both the cognitive products and cognitive processes of depression are considered as products of an underlying structure or schema. This hypothetical structure contains information about the self (self-representations) that is coded in early childhood and carried forward to the present. Beck (1967) proposed that negative schemata develop as a result of negative early life experiences, are encoded, and become templates that influence subsequent experience. Schemata comprise unconditional beliefs (“I am a bad person”) and dysfunctional assumptions (“I would not be bad if I passed the test”). Therefore, schemata consist of organised elements of past reactions and experiences that form a relatively cohesive and persistent body of knowledge that are considered to represent the basic units of personality (Kovacs & Beck, 1978). Schemata influence cognitive processes to produce distortion or errors in thinking, which then influence cognitive products (e.g., negative automatic thoughts). If these structures are activated, more and more of an individual’s processing of information is channeled according to the principles described in Beck’s schema theory (selection, abstraction, interpretation, and integration; Alba & Hasher, 1983; Taylor & Crocker, 1981). A negative construal of reality therefore may be the result of a negatively biased filtering of schema congruent information. Schemata are hypothesised to store, integrate, and direct processing of information outside the awareness of an individual at an implicit level (Beck, 1967, 1976). This means that an individual, whose negative schemata are active, is not aware of how his/her cohesive body of knowledge, based on early experience, is affecting the current processing of information to produce cognitive distortions and negative thoughts, leading to negative affect.

The main problem with schema theories of depression and the problem that poses a difficulty for Beck’s etiological hypothesis of depression is how to measure schemata outside an episode of depression. It is not clear whether schemata resemble traits and as such fuel enduring information processing biases, or remain latent until activated by a negative life

⁴ Where the term “risk” is referred to out with reference to antecedent risk factors, this will refer to themes pertaining to an increased or decreased probability of developing depression.

⁵ The reason this distinction between “risk” and “vulnerability” is being made is due to the potential misunderstandings and misuse of these two words when it comes to describing depression (see Skeat, 1993). To clarify, “risk”, pertains to themes of hazard, chance of injury, harm or loss. “Vulnerability” on the other hand pertains to themes of the consequence of being injured (e.g., of having a wound) and liable to subsequent injury. Therefore, from the perspective of depression, antecedent risk factors (negative childhood experience) associated with depression, injure an individual and to give him/her a wound (schema) that is liable to injure the person at a later date (e.g., vulnerability for the development of depression as a result of an activated schema).

event and then begin to bias processing. The evidence from much longitudinal research on depressive cognition does seem to support the etiological hypotheses advanced by Beck's theory (Beck, 1967, 1976). In other words, there is little evidence of negative schemata and associated information processing biases in the absence of a depressed mood (Lewinsohn, Steinmetz, Larson, & Franklin, 1981). This leaves the possibility of either a latent structure that exists in individuals vulnerable to depression, ready to be activated in certain circumstances. Alternatively, perhaps a depressive episode is necessary for the formation of negative schema constructs, which then confer vulnerability for future episodes of depression via negative information processing biases (Segal et al., 1999, 1988; Hammen, Marks, Mayol, & DeMayo, 1985). Thus, there is much debate about the problematic issues regarding the measurement of schemata in the absence of a depressed mood, the definition of schematic structure, the development, content and architecture of a schema, and how schemata confer vulnerability to depression (Segal, 1988; Segal & Swallow, 1994; Rector, Segal, & Gemar, 1998). However, there may be methodological reasons why the evidence of schematic activity in the absence of a low or depressed is not strong. These methodological issues have been argued to be related to the potential confounds associated with the over-reliance of self-report measures (Nisbett & Wilson, 1977), a failure to recognise error rate data from cognitive processing tasks as a useful measure of information processing biases (e.g., Bargh & Tota, 1988; Kirsch & Lynn, 1999), insufficient consideration of theoretical content of schemata (Alloy et al., 1999), and the lack of a consideration of the association of the self and stimuli processed on implicit cognitive tasks (e.g., Hedlund & Rude, 1995) and/or a disregard for the potentially important role of automatic self-evaluation in vulnerability to affective disorders. It is this latter point in particular which some researchers are only now starting to realise (e.g., De Raedt et al., 2006; Tanner, Stopa, & De Houwer, in press; de Jong, 2000).

1.3. Summary

Beck's cognitive model (Beck, 1967, 1976) is one of the most influential cognitive models of depression. The model's three variables, cognitive products, cognitive processes, and cognitive schemata provide a comprehensive description of depressive cognition, but also provide an etiological and explanatory account of depression. There is a considerable amount of research that supports the cognitive products (e.g., Hagga et al., 1991) and cognitive processes (e.g., Hedlund & Rude, 1995; Ingram et al., 1998) aspects of the model. However, cognitive schemata postulated to underlie depressive products and processes have not been adequately tested or investigated, and there is much debate in psychology on the nature and role of schemata in depression (Ingram, 1990; Segal, & Ingram, 1994; Hedlund & Rude, 1995). However, there are a plethora of methodological issues that may be implicated as to why the evidence for schematic activity in the absence of a low or depressed mood is not strong. This thesis is an exploration of negative self-schemata in depression and the role of

automatic or implicit self-evaluation. Through a series of studies that consider these methodological issues, it is hoped that the empirical evidence contained within this thesis can successfully elaborate for the reader why these issues may contribute to the lack of strong evidence of schematic activity in the absence of a low or a depressed mood. The next chapter provides a detailed examination of the schema concept including a discussion of some researchers who have used the cognitive schema or structure concept and integrated this into their theories of depression. The results of empirical research into schemata in depression will then be discussed in Chapter 3.

Chapter 2

The schema concept

2.1. Introduction

The term *schema* is derived from Latin. Etymologically the term schema means “form”, “appearance”, “to hold”, “to bear”. Present day usage of the term schema has its roots in meanings related to the ideas of “design”, “plan”, “project”, or ideas of “order”, of being “arranged” or a “program of action”. These are just some of the ways that the concept of a schema can be conceptualised in the English language. The etymological roots of psychology are related to ancient Greek *psyche*, meaning breath, spirit, or soul, and *logia* meaning the “study of” (Skeat, 1993). Therefore, it does not take too great a leap of imagination to see how the study of schemata (someone’s form, appearance, or program of action) can be linked to psychology’s interest in the study of an individual, be that the mind or behaviour.

One of the most widely known constructs used within cognitive and clinical science is that of schemata. Schema theories have proved important in accounting for a wide variety of psychological phenomena. Schema theory has been used in contemporary studies of memory (Schacter, 1992), concept representation (Smith, 1989), problem solving (Van Lehn, 1987), movement (Jordan & Rosenbaum, 1989), and language (Arib, Conklin, & Hill, 1987). It has been argued that schemata are a useful heuristic for describing the human mind (Fiske & Linville, 1980). The philosopher Kant (1963) employed the concept of a schema to discuss the possibility of human knowledge. Kant argued that knowledge has its origins in the external world and argued that schemata interdigitate between properties of the mind (a priori experiences) and raw sensory data (a posteriori experience), creating representations of experience in subjective consciousness. Representations of the self are hypothetically created by this process as a result of the experiences that one has in the world (Stein, 1992). The totality of memories, representations of the world and self cannot occupy one’s consciousness at the same time. Therefore, if schemata are the result of prior experience and memories, and if schemata are latent or hypovalent as Beck describes (Beck 1967, 1976), then they may exist at some level (e.g., unconscious) and/or be integrated biologically into the human organism.

This chapter is intended to provide the reader with an overview of how the schema concept is used within different disciplines and to show that it is a valid and useful concept in the study of depression. The first section gives a summary how the schema concept is used in contemporary models of cognition in depression. The following sections will discuss the schema concept, from the neuroscientific, developmental, psychoanalytical, and existential-phenomenological theoretical perspectives, respectively. The last section will provide an overview of how some researchers have incorporated the cognitive schema or structure concept into their theories of depression.

2.2. *Summary of schemata in contemporary cognitive models of depression*

Cognitive schemata or structures are hypothesised to store, integrate, and direct the processing of information outside the awareness of an individual, or at an implicit level. They influence cognitive processes, which then bring about the depressive products of consciousness, e.g. negative automatic thoughts. Beck (1967, 1976) proposed that negative schemata develop as a result of early life experience, are encoded and become templates that influence experience.

Young (1990) has adapted Beck's original ideas and introduced the concept of "early maladaptive schemata" (EMS), which are postulated to be stable and enduring *themes* that develop during an individual's childhood. These EMS become rigid guiding principles that interfere with appropriate adaptation to life and subsequent experience. In this context, schemata are thought to develop in childhood but fail to adapt and evolve in response to changing circumstances. Thus, the beliefs that were appropriate for a child can interfere with the individual's ability to function and form healthy adult relationships. Young describes schemata as the deepest level of cognition and as stable cognitive structures that organise knowledge about the world. He argues that schemata influence perception, processing, storage, and retrieval of information. More importantly, Young, in contrast to Beck's formulation of schemata, argues that schemata are solely unconditional in nature (e.g. "I am a failure"). By comparison, Beck (1967, 1976) argues that schemata comprise both unconditional and conditional beliefs (e.g., "I am a failure if I do not win"). A maladaptive schema is thought to play a causal role in the development of depressive episodes.

Numerous studies have found evidence of dysfunctional cognitive structures in currently depressed individuals via the use of questionnaires (Ingram et al., 1998) and implicit information processing tasks (Gemar et al., 2001), but not in non-depressed individuals (Blaney, Begar, & Head, 1980; Hamilton & Abramson, 1983; Hollon & Kendall, 1980; Teasdale & Dent, 1987). It seems that the detection of dysfunctional cognitive structures becomes undetectable as an episode of depression remits (Gladstone & Parker, 2002; Eaves & Rush, 1984). However Beck (1967, 1987) does argue that accessibility to dysfunctional cognitive structures only occurs under conditions of stress where schematic structures are activated. This tenet of his theory has come under much scrutiny in recent years due to the problematic nature of measuring schemata (Rude, Covich, Jarrold, Hedlund, & Zenter, 2001). Questionnaire measures of schemata are subject to limitations inherent with the use of self-report measures (Nisbett & Wilson, 1977), and there is also doubt over whether questionnaires can measure depressive schemata as schemata theoretically operate at implicit level (Bargh & Tota, 1988). This is because self-report measures of depression are susceptible to explicit processes, self-presentation, and demand effects (Nisbett & Wilson, 1977) and increased scores on questionnaires usually occur only in the presence of a depressed mood

(Alloy et al., 1999). Therefore, it is difficult whether to attribute high scores on these measures to vulnerability to depression and the implicit nature of schemata, explicit processes, and/or to the effects of a low mood. Further, inappropriate implicit information tasks to measure schemata use stimuli that are either, not appropriate to depression, and/or tasks that measure negative material but do not measure the implicit association of negative stimuli with the self. This raises questions about the validity of these information tasks and what exactly is being measured (Greenwald, Banaji, & Schwartz 1998). These problems will be discussed in Chapters 3 and 4.

Schema detection in depression from Young's (1990) perspective of EMS is beginning to show some promise as key themes are starting to emerge showing an association between depression and early childhood. Harris and Curtin (2002) studied the relationship between reports of retrospective parenting, EMS, and symptoms of depression. They found that EMS of defectiveness/shame, vulnerability and incompetence/inferiority partially mediated the relationship between depression and parental perceptions. However, the distorting effects of memory associated with a low mood may have confounded these results as a depressed mood biases the recall of memories in a negative way (Gemar et al., 2001). In other words, there may have been positive memories of parenting, but these were not recalled because of the low mood. However, Gladstone & Parker (2002), who also assessed EMS, found evidence that a general belief in victimisation and/or abuse was highly associated with reacting in a negative way in response to a stressful event. These individuals were more likely to develop depression when confronted with events that were perceived as unsafe or abusive. This core belief of victimisation and/or abuse involves beliefs about being unsafe or vulnerable in the world. This finding gives some support for the idea that early negative experiences may leave an individual with some degree of cognitive residue in the form of a core belief about the self or world, which creates a cognitive vulnerability to depression. This idea of individuals, who are vulnerable to depression, possessing some form of cognitive residue before the onset of depression, has also found some support from a study conducted by Alloy et al. (1999). They found that individuals who were more likely to develop depression had a more negative implicit self-concept compared to non-vulnerable individuals.

The idea that depression is associated with negative thinking that encompasses the world, future and the self, and involves cognitive distortion and biased manipulation of information, is supported in many research findings (see Chapter 1). However, with regards to the role of schemata within cognitive theories of depression, the evidence is far less clear-cut. Beck's (1967, 1976) proposal that schemata are latent diathesis to depression raises potential measurement problems. To be considered causal, schemata must predate actual depression. However, if schemata are unavailable to awareness before the onset of depression, they are difficult to measure and therefore it is difficult to establish whether they are present prior to

an episode of depression. Segal (1988) suggests that by defining schemata as latent, the model assumes that they are inaccessible except during depressive episodes. Thus causality can never be empirically determined. This claim poses a challenge to the cognitive theory of depression. If schemata do have some kind of causal status in depression, they must be capable of measurement and demonstrably present before an episode of depression (or after an episode of depression has remitted). Young (1990) argues that early maladaptive schemata developed from early childhood exist as enduring components of one's core self-knowledge or self-concept. Young states that individuals may not be aware of their core schemata due to a lack of self-reflection or awareness. However, schemata may be accessed through guided discovery or other methods. According to this theory, vulnerability to depression should be measurable in the absence of a depressed mood.

Consequently, the challenge of schema theory at the moment is to ascertain the nature and role of schemata in vulnerability to depression and how they can be measured in the absence of a depressed episode. However, the measurement of schemata depends on how it is conceptualised and therefore it is useful to consider the concept from a number of different theoretical approaches. Drawing on other perspectives may allow us to conceptualise or re-conceptualise the schema concept in a more coherent way and provide fresh ways to think about the schema concept and its role in depression and vulnerability to depression.

2.3. The neuroscientific basis of schemata

Cognitive science acknowledges that structures of the mind may have a biological basis (Le Doux, 1998). Therefore, looking at schemata from a neuroscientific perspective may provide a clearer conceptualisation of how a schema involved in information processing could be implicated in the genesis of depression. Le Doux (1998) argues that consciousness may be the result of unconsciously processed information, but that unconscious affectively guided information processing occurs outside awareness or focal attention. Therefore, from the perspective of a person vulnerable to depression, the processing of information regarding the self, the world and the future, may occur outside awareness much of the time.

With regards to how schemata (implicated in negatively biased information in processing) might operate from a neuroscientific perspective, one can investigate memory systems within the brain. Slapp (2000) argues that there are two different memory systems: declarative and emotional memory. Evidence suggests that these two memory systems operate independently. Declarative memory refers to conscious recollection of past events and experiences. This memory system has been attributed to the hippocampus. Le Doux (1998) notes that hippocampal circuits establish memories in which many events are bound together in space and time. The main sources of input to the hippocampus are the major sensory processing systems of the neocortex. After processing data, the hippocampus projects back to the neocortex and makes the information available to the prefrontal cortex.

Emotional memory on the other hand is attributed to the amygdala. The amygdala makes evaluations of perceptual data prior to the processing of that data by the prefrontal cortex. Raw perceptual data arrives at the thalamus and is transmitted to the amygdala, the neocortex and the autonomic nervous system. This transmitted data arrives at the amygdala earlier than at the prefrontal cortex because the data going to the neocortex must be processed by sensory and association portions of the neocortex, before being available to the prefrontal cortex. Therefore, the record of emotionally charged events is unconscious, but the affects generated are consciously experienced and provide the emotional tone for conscious experience (Slapp, 2000).

Further, the amygdala is thought to mature earlier than the hippocampus (Le Doux, 1998; Slapp, 2000). Le Doux (1998) argues that the failure of retrieval of an infant's experience is better explained by the immaturity of the hippocampus, rather than psychoanalytic formulations of repression. Infants do have memory, not in a declarative context, but in more of an emotional context. Therefore, the development of a depressogenic schema could theoretically occur during early childhood if the primary caregivers treat the infant in a negative way. This may instill in the infant the feeling of being unlovable, thus creating a core self-schema of being unlovable. This impression may theoretically be laid down as an emotional memory in the amygdala as a "feeling" of being unlovable. Years later, the child now an adult, if exposed to a romantic rejection, may process aspects of this situation very quickly over potentiated pathways to the amygdala releasing the emotion of years earlier. In other words, the amygdala would preferentially process this event (e.g., being rejected and thus being completely unlovable) more effectively than other aspects of the event (e.g., the partner who is doing the rejecting has to leave to look after a sick relative in a far-away country). The amygdala has been implicated in depression because it has been shown to be hyperactive (in depressed individuals) to stressful events and preferentially reacts to certain stimuli rather than others. This has been shown by preferential processing of negatively toned words and increased neuronal and blood flow activity during stressful events (Le Doux, 1998).

In essence, the neuroscientific perspective of negative early experience affecting later adult life and associated information processing, is in accord with Beck's (1967, 1976) model of depression. More specifically, the neuroscientific perspective is similar to the cognitive view that negative early experiences affect an individual in later life and create negatively biasing information processing, thus creating vulnerability to depression. Beck argues that negative schemata are also created by negative early life experience. Further, he argues that negative experiences are bound together in emotional memory, downloaded so to speak at a very deep cognitive level that is unavailable to consciousness. The jilted lover in the aforementioned example may experience a low mood at being rejected in a romantic

situation, but may well be very unaware of why he/she feels like they do (a consequence of having had a negative childhood experience). Even though rational thought may be possible (e.g., “I will find another lover”), the potentiated pathways to the amygdala may be more dominant, negatively bias information processing, override rational thought, and produce faulty cognitive processing or cognitive errors. The result of this sequence would yield Beck’s postulated negative cognitive triad; negative thoughts regarding the self, the world, and the future.

2.4. A developmental perspective of schemata

In Piagetian terms, dysfunctional emotional memory or schemata (of being unlovable) assimilate current experience in accordance with past events (Piaget, 1926). That is, a schema functions throughout life by interpreting current events in terms of templates of past experience. Therefore, an individual who is vulnerable to depression goes through life remaking the same scenarios with current people from current life cast into roles originally created by the caregivers; being made to feel unloved by parents becomes being unloved by potential partners, which in turn can potentially depress the individual. This is very much in line with the psychoanalytic idea of transference whereby one would treat the therapist (or significant other) like a primary caregiver (Laing, 1967). This mode of cognition of the pathogenic schema is consistent with what Piaget called preoperational thought (Piaget, 1926); primitive modes of reasoning control current reasoning without recognition of their influence. Preoperational thought prevails over logical thought and deduction. This is in accord with Le Doux’s (1998) ideas of the neuroscientific basis of depression, whereby the thalamo-amygdala projections and the conscious results of the processing that arise, are relatively crude and based on fragments of a situation, rather than on an accurate perception of the objects and events in an individual’s current experience.

In developmental psychopathology research it has been suggested from over 20 years of accumulating evidence, that it is not just the absence of positive developmental events that occur in childhood (mirroring, love, warmth, etc from caregivers) that are implicated in psychopathology. Actual aversive, harming, and threatening early experiences with primary caregivers are also of importance. These aversive events have a major impact on the vulnerability to develop psychopathology (Kendall-Tackett, William, & Finkelhor, 1993; Malinosky-Rummell & Hansen, 1993). The pathogenic nature of early aversive experience ranges from the extremes of sexual and physical abuse, through to overprotection, criticism, and tense threatening home environments (Bryer, Nelson, Miller, & Kroll, 1987; Brown & Anderson, 1991). It is now recognised that adverse events during infancy and childhood can significantly affect psychobiological maturation and functioning (Rosenblum, Coplan, Friedman, Bassoff, Gorman, & Andrews, 1994). Children who are very stressed in their early environments, compared to low stressed children, have increased sensitivities in their

hypothalamic-pituitary-adrenal axis stress systems (see Appendix I, section on hormones, p.201). This makes individuals prone to higher levels of circulating stress hormones, more reactive to stressors, and more likely to suffer from poor affect regulation (Perry, Pollard, Blakely, Baker, & Vigilante, 1995). The amount, type, timing, and quality of these negative adverse experiences implicated in the development of depression are difficult to judge. In an interesting study by Murray, Fiori-Cowley, Hooper, & Cooper (1996), they found that when comparing face to face interactions of depressed and non-depressed mothers with their two-month old babies, depressed mothers were less well attuned to their infants. The depressed mothers rarely gave comments that seemed to acknowledge and support their infants' current experience, and they often made negative remarks. Furthermore, depressed mothers were rated as hostile and intrusive, withdrawn, or showing negative feelings. The development of the children of the depressed mothers was significantly poorer (at 5 years) than that of children of non-depressed mothers. This was reflected by poorer IQ, and poorer capacity to attend and process information in the wider social and non-social environment. The damaging and lasting effects of adverse childhood environments have also been detected in adulthood. Parker, Gladstone, Mitchell, Wilhelm, & Roy (2000) found significant associations between early adverse events and adult depression. Notably the depressed participants in this study endorsed schemata involving themes of emotional deprivation and being worthless and/or inadequate. These endorsed themes were strongly associated with early aversive experiences and vulnerability to depression. This theoretical perspective is consistent with the cognitive model of depression where negative early experiences are laid down as maladaptive schemata and confer vulnerability to develop depression (Beck, 1967, 1976).

2.5. The psychoanalytical perspective of schemata

Psychoanalytical theories of depression have much in common with the cognitive theory of depression and the neuroscientific concept of the maladaptive schemata, despite psychoanalytic theory being presently out of fashion with cognitive and neuroscientific formulations of psychopathology. Ende (1999) argues that ongoing emotional experiences are continually active in relation to one's memory systems. That is, one's present emotional life is integrated into one's biographical past and shaped by one's past. If an individual's past has been maladaptive, this can lead to present difficulties in relationships (e.g., intimate relationships, and relatedness with others) as present experience is coloured negatively in line with past experience.

Bucci (2000) also uses the concept of schemata from a psychoanalytical perspective. She argues that schemata of the self develop from sub-symbolic input (i.e., a non-verbal form or pre-language stage of the infant; sensory, visceral etc). Further, she adds that in therapy these schemata are very resistant to change due to the sub-symbolic nature of them. She also argues that these self-schemata become the basis for the organisation of the self, that

schemata are dynamically unconscious, and are automatic-like structures that can, when activated sufficiently enough, produce conscious affect (e.g., low mood).

Stern's (1985) formulations from a psychoanalytic perspective of schemata complement not only the cognitive model of depression, but also the developmental and neuroscientific theoretical perspectives of schematic organisation. He argues that the self is constructed through interactions with primary caregivers. These constructions from early life experiences become generalised and guide expectations about subsequent behaviour, and intra-psychic affect. These early experiences are categorised in prototypic form to be used in ongoing life as templates that guide one's orientation within an interpersonal context and phenomenological context.

As the reader hopefully can see, the link between modern psychoanalytic formulations of depression has much in common with cognitive ideas of depression. The idea of templates being constructed in early life (Stern, 1985), which are dynamically unconscious and when sufficiently activated, produce low mood (Bucci, 2000), are compatible with Beck's (1967, 1976) ideas on the nature of schemata and how they are implicated in depression.

2.6. The existential-phenomenological or ontological perspective of schemata

The existential-phenomenological school of thought, in particular the ideas of ontology expressed by Wheeler (2006), Merleau-Ponty (1962), and Heidegger (2001) provide a useful insight into the concept of a schema as used within cognitive theories of depression. Their approach supports the concept of a schema and its contributory role in the etiology and maintenance of emotional disorders such as depression. More importantly, however, an ontological approach provides a way of thinking about schemata other than a latent activated structure containing negative self-representations affecting information processing. This perspective thus provides a way of thinking about observing or measuring schemata in the absence of a depressed mood.

The main difference between an ontological approach to schemata (Merleau-Ponty, 1962; Heidegger, 2001) and a more epistemological approach is the way these two different approaches conceptualise the mind (Dreyfus, 1989). The epistemological approach involves the notion that the mind contains ideas, which might (or might not) correspond with the outside world, or has neural structures containing self-representations that become activated when confronted with certain stimuli (e.g., Le Doux, 1998; Beck, 1967, 1976; Freud, 1973). For example, Beck, Freud, and Le Doux argue that the unconscious is where representations (ideas of the self and world) are buried and exert a causal role in the development of psychopathology.

Wheeler (2006), Merleau-Ponty (1962), and Heidegger (2001) posit that one's comportment or ontology acquired through negative childhood experiences is not represented in the mind by self-representations as such, but by an atheoretical unreflective embodied and

intuitive way of being, much the same way one learns a complex skill (e.g., tennis). One relates to the world and makes intuitive judgments based on embodied expectations of the world. The world for the individual is not so much a belief system or a range of stimuli impinging upon the schema system, but is given instead to an individual through an atheoretical and intuitive perception and coping. The way one copes therefore is of a nonlinguistic and a non-conceptual nature. In the non-depressed state an individual may not differ in any significant way compared to a person who is not vulnerable to develop depression. However, the ontological comportment of a vulnerable person's "being in the world" (Heidegger, 2001) or "atmosphere" (Merleau-Ponty, 1962) may fundamentally differ in some small and insidious ways. Existentially, the person may act as if the world is a bleak place, and feel quite nihilistic, although this is so slight and embedded that the vulnerable person functions well in life. Examples of insidious and subtle ontological insecurity may be difficulties in forming relationships, trepidation of the future, a preference for isolation, or not having an overly high self-regard for achievement in life. On reflection, this kind of person may not feel or think he/she sees the world in an uninviting way. This person may feel that the way he/she sees the world is ordinary. This kind of comportment is embodied and is very difficult to become aware of (Merleau-Ponty, 1962). It may take a significant event in a person's life to become aware of one's ontological insecurity. Being induced into depression via a negative event (e.g., failing an exam) may facilitate the ability to engage in reflection (e.g., psychoanalysis or CBT) about one's existential comportment and be the catalyst to create a coherent narrative of the past, present and future, which up to that point has not been reflected upon.

Theorists like Young (1990), who have recently extended Beck's (1967, 1976) cognitive model of depression, concur to a certain extent with the ontological approach to depression. The main difference between Beck's theory of a schema and Young's is that Young's idea of schemata reflect unconditional themes or core beliefs that a person has adopted or identified with due to negative childhood experiences (e.g., being defective). This is very much like Merleau-Ponty's (1962) and Heidegger's ideas of insecure ontology. Beck's formulation of schemata, however, as described earlier, comprises beliefs that are conditional (e.g., "I must be liked by everyone or I will be a failure"), and unconditional (e.g., "I am a failure"). However, Beck argues that schemata have to be activated to have any effect upon an individual. Contrary to Beck's ideas, Young (1990) argues that these themes are stable themes that colour an individual's life, but the individual may or may not be aware of the nature of his or her theme or themes. In other words, schemata are enduring in the absence of a low or depressed mood and thus potentially measurable without the need for activation of any kind.

The ontological perspective does however have similarities with a cognitive epistemological perspective (i.e., Beck, 1967, 1976). The two perspectives both concur with

the idea of negative early experiences conferring vulnerability to develop depression. Secondly, they are both in accordance with the idea that emotional processing can occur at an unconscious level or at a level which individuals are unaware of the biased way in which they see the world. Lastly, the notion that schemata serve as templates for on-going experience is essential to both perspectives.

The crucial differentiating features of the epistemological and ontological perspectives highlight important issues for schema research. In other words, do individuals vulnerable to depression possess schemata that preferentially process negative information when activated but which are impossible to detect outside of an episode of depression or mood activation, making etiological postulates difficult? Or do individuals vulnerable to depression always harbour a negative ontology (or core self-beliefs) that constantly affects the way they view the world and is detectable and potentially measurable at all times? It is the nature of the schema in depression (activation versus always available) that has sparked a great deal of research on these topics. Discussed below is an overview of how some researchers have tried to incorporate aspects of schema content and/or schema activation into their theories of depression and the problems that have resulted with regard to the etiological postulates of depression.

2.7. Sociotropic & autonomous cognitive structural subtypes

Beck (1987) refined his theory of depression by arguing that there were two categories of individuals who were prone to depression: sociotropic and autonomous individuals. In other words, there are specific concepts represented in sociotropic and autonomous individuals' cognitive structures that make them prone to depression. Sociotropic individuals are hypothesised to have cognitive structures, which place emphasis and value on positive interchange with others, and focus on acceptance, support, and guidance from others. Autonomous individuals have cognitive structures that represent concepts, which emphasise independence, mobility, and achievement. Therefore stressors congruent with these themes are expected to activate dysfunctional cognitive structures and precipitate depression. Investigation into these two sub-types has provided confirmatory results. However, problems arise with this theory in that many individuals score high on both subtypes, which is counter to the theory. Furthermore, individuals who score high on one or both of the subtypes have been identified as suffering from psychological disorders other than depression. Therefore, the subtypes may not be specific for the development of depression (Coyne & Whiffen, 1995).

2.8. Anacletic & introjective cognitive structural subtypes

Blatt (1974) developed a cognitive structure theory of depression that has similarities to Beck's depressive typologies. The model is developmental in nature and is grounded in object relations and attachment theory (Bowlby, 1969). Blatt suggests that depression is better

understood by the use of the anaclitic and introjective subtype categories. Anaclitic depression is characterised by feelings of helplessness and weakness, depletion, and being unloved. The individual suffering from anaclitic depression fears abandonment and struggles to maintain direct physical contact with a need-gratifying object. These individuals need to be soothed and cared for, helped, fed, and protected. There is a sense of hopelessness in being unable to find gratification, and others are valued only for their capacity to provide gratification. When these needs are not met, feelings of being unloved and helplessness evolve into depression.

Introjective depression is characterised by feelings of being unworthy, unlovable, guilty, and having failed to live up to expectations and standards. An individual with introjective cognitive structures has high moral standards and is involved in constant self-scrutiny. Individuals like these have high demands for perfection, a tendency to assume blame and responsibility, and feelings of helplessness to achieve approval, acceptance, and recognition. An introjective individual overachieves in order to win approval, which he/she feels that they do not have. Therefore, a sense of failure, or lack of approval from important others due to a stressful event can activate the cognitive structures and may precipitate depression.

Blatt (1974) argues that the formation of introjective and anaclitic cognitive structures resides in impairments in object relations. For example, anaclitic depression is proposed to be the result of frustration or failure to have learned how to tolerate and manage frustration, and is related to early childhood trauma (e.g., a disturbance of the basic bond with a care-giver which occurs at the early stage of separation and individuation). This trauma can take the form of loss, deprivation or overindulgence. An association between a deprived childhood experience and adult depression has been found in research (Blatt, Quinlan, Chevron, McDonald, & Zuroff, 1982). On the other hand, according to Blatt (1974), introjective depression and the formation of depressogenic cognitive structures develop at a later stage. He argues that this type of depression is the result of negative conscious and unconscious parental feelings and/or ambivalent or hostile parental behaviours occurring around the development of sexual identity, superego formation, and oedipal conflict. These feelings and behaviours give rise to feelings in the individual of guilt, worthlessness, and vulnerability to introjective depression.

Blatt's argument concerning the etiology of anaclitic and introjective depression has some support from research (McCranie & Bass, 1984). However, one shortfall is that individuals in these studies had to make judgments about their parents' behaviours earlier in their lives, confounding the results due to the retrospective methodology. Also, the anaclitic and introjective sub-types of Blatt's model seem to be highly correlated; i.e. individuals can be equally categorised on measurements of anaclitic and introjective sub-types. The

specificity of the theory also raises potential problems. Many people develop depression without necessarily having ambivalence toward their parents and/or having had to deal with frustration during childhood (James, 1998).

2.9. The self-worth contingency model of cognitive structure

Kuiper, Olinger, & MacDonald (1988) proposed a model of cognitive structure, which is similar to Beck's schema model. They suggest that although the content of the cognitive structure (negative self-referent information) is similar to Beck's model, the consolidation of this content is an important aspect of how the cognitive structures/schemata function. In other words, consolidated schemata have very strong associated links in stored knowledge. This results in the efficient processing of information that is congruent with the content of the schema.

A key aspect of Kuiper et al.'s argument is that the content of depressive self-schemata are quite similar to the types of dysfunctional attitudes suggested by Beck (e.g., "I am useless if I do not pass this exam"). This content serves as a contingency that determines whether or not the depressogenic process will be activated. Therefore, if the contingency is not met, depressive cognitions are activated, affecting self-worth, and bringing on depression (Ingram et al., 1998).

The similarity to Beck's model is evident. However, as with Beck's model, measurement of dysfunctional cognitive structure/schemata presents difficulties due to the confounding factor of mood: negative self-referent processing of information congruent with schema content is only evident when individuals are in a depressed mood. Therefore it becomes difficult to link depression and etiological causality with this model.

2.10. Cognitive networks

Theories and concepts taken from experimental cognitive psychology have fuelled cognitive network theories of depression and the notion of cognitive structure and schemata in depression. This theoretical stance places importance on the role of information processing as a key factor in depression. Historically, this is related to Bower's (1981) model of mood and memory. Bower argued that associative networks are developed between mood nodules and memory nodules. The result is that mood can precipitate changes in thinking and accessibility to memory, and changes in thinking can precipitate changes in mood.

Ingram (1984) developed an information-processing model of depression based on Bower's (1981) model. Ingram argues that the experience of depression is a result of the activation of an affective structure. Once this structure is activated, cognitions are theorised to recycle through cognitive networks that have previously become associated with low mood and depression. This process initiates the depressive episode, but when the cycle is fully activated, it perpetuates depression until the cognitive-affective activity decays. Vulnerability to depression is conceptualised in this model as the availability of well developed and

elaborated cognitive networks that are associated with low mood. If some negative event or stressor activates the network, the resultant low mood allows access to more extensive processing of information associated with depression. The result is that depression-prone individuals, who possess depressogenic cognitive networks, will spiral from a normal low mood (e.g., a mood that everyone experiences at some point) into a more pathological depressed state. These networks evolve over time so that activations become easier in response to depression-triggering events or stressful events. This process is labeled “cognitive scarring” whereby after the initial episode of depression, the cognitive network will react by “kindling” more easily to negative stressors (Segal et al, 1999).

Teasdale & Barnard (1993) have developed a very comprehensive information-processing model of depression called the Interacting Cognitive Subsystems (ICS) model. This model proposes that different aspects of experience are represented by patterns of different kinds of information or mental codes. At a superficial level, experience is coded in auditory, visual, and proprioceptive inputs. At a deeper level, patterns of sensory codes are represented by intermediate codes. An example of how this works is that the effect of visual input from objects is represented in the object code, but at a deeper level, the object may have an affective meaning. Therefore, a word at the superficial level is a string of letters; at a deeper level it may have an affective quality (e.g., death). Thus, a string of letters is explicit, whereas the implicit meaning is the fearful end of life. Patterns of implicational codes represent deeper holistic meanings, which are linked to emotions. This level does not map directly onto language. In the ICS model, depression is produced when patterns of low level meanings and sensory input produce depression-related schematic models. Depressed mood is maintained when depressogenic schematic models are continually produced.

The main problem with a cognitive network approach is that it does not address the idea of vulnerability to depression in the absence of a depressed mood. For example, how is one meant to measure vulnerability to depression if information-processing biases are dependent on a low mood? This does not fit with an ontological approach to depression, nor Young’s (1990) conceptualisation of schemata being a pervasive theme that are in-built character dispositions that lead to depression. Considering the evidence discussed earlier regarding the association of early maladaptive schemata (EMS) and later pathology and the stability of EMS, it does not fit easily with a cognitive network account of depression.

2.11. Summary and discussion

The schema concept is a widely used and useful heuristic concept for research in depression. Much of the neuroscientific (e.g., Le Doux, 1998), developmental (e.g., Piaget), psychoanalytical (e.g., Ende, 1999), and existential-phenomenological thought (e.g., Heidegger, 2001) regarding depression give credence to the use of a schema focused approach in the study of the etiology of depression. In essence, these four approaches have

much in common with schema accounts in cognitive models of depression (e.g., Beck, 1967, 1976; Young, 1990). Firstly, entwined in all four perspectives is the notion of negative early experiences affecting an individual later in life and conferring vulnerability to develop depression. Secondly, they are all in accordance with the notion that emotional processing can occur at an unconscious level or at a level which individuals are unaware of the biased way in which they view the world. Thirdly, the notion that schemata serve as templates for on-going experience is essential to all four perspectives.

However, the ontological and epistemological viewpoints propose different ways to look at vulnerability to depression. The epistemological perspective (e.g., Le Doux, 1998; Beck, 1967, 1976) views that depression is caused by activated structures or schemata containing negative self-representations which preferentially process negative aspects of an individual's world to produce depression. The ontological view espoused by the philosophers Wheeler (2006), Merleau-Ponty (1962) and Heidegger (2001) propose that one's comportment or ontological insecurity acquired through experience is not represented in the mind by self-representations as such, but by an atheoretical unreflective embodied and intuitive way of being, much the same way one learns a complex skill (e.g., tennis). From an ontological perspective, an individual's ontological insecurity will be present before an episode of depression and will affect the individual's behaviour in insidious ways. This idea is partly in accordance with Young's (1990) ideas of early maladaptive schemata that are available as a cognitive residue in the absence of a low or depressed mood.

Differences of opinion concerning the content of schemata, if and how schemata are activated, the role of childhood experiences in the development of schemata and their relation to treatment, is also present in a number of other theoretical perspectives of schemata. This is evident in Beck's (1987) refined theory identifying sociotropic and autonomous individuals, Blatt's (1974) anaclitic versus introjective theory, Kuiper et al.'s self-worth model (1988), and Ingram's (1984) and Teasdale & Barnard's (1993) theories based on networks. The main problem with both Beck's (1987) and Blatt's (1974) theories is that the schema subtypes that they each assign to depressed individuals in their respective models show weak validity. It may be that the difference between a person vulnerable to depression and a person not vulnerable to depression is not the specific postulates of being a certain subtype (e.g., anaclitic), but the general difference of the amount of positive tint with which each type of person experiences the world or the degree to which one feels safe in the world. This fits with Merleau-Ponty's (1962) notion of 'atmosphere', or Heidegger's (2001) idea of 'being in the world' where an individual may be generally negative and/or unsure or unsafe rather than positive and sure/safe. The specifics of a sub-type are not needed (explicitly known) for a negative atmosphere to envelop an individual. Further, the theories of Kuiper et al (1988), Ingram (1984), and Teasdale & Barnard (1993) to a great degree invalidate etiological

theories of depression. All three theories rely on low mood determinants to demonstrate their arguments.

It may well be that vulnerability to depression is only detectable in a low or depressed mood (or in individuals who have recovered from depression who are induced into a low mood indicating neural scarring and schematic organisation as a result of an initial episode of depression (Segal et al., 1999)). Here the argument becomes increasingly circular and unsatisfying - one can never prove vulnerability to depression in the absence of a low or depressed mood. Heidegger and Merleau-Ponty, and more recently Young, subscribe to the notion that schemata/core beliefs or an individual's colouring or atmosphere of the world are potentially available to the individual - it is just that the individual may have difficulty seeing his or her particular schema or atmosphere (Heidegger, 2001; Merleau-Ponty, 1962; Young, 1990). Therefore, the problem of schema detection in the absence of a low or depressed mood may have something to do with certain problems of measuring schemata (e.g., information processing tasks versus self-report measures and/or themes/core beliefs that are being measured). It may well be that the interface of cognitive products, processes, and structure, and aspects of implicit versus explicit information processing have not been adequately addressed. The next chapter (Chapter 3) will now discuss the empirical research into schemata and depression. For the most part, findings have confirmed schemata as potentially activated structures affecting information processing in depression (Ingram et al., 1998). However, there are problems with the methodology used to measure schemata and the conceptualisation of schemata. Chapter 3 will introduce these problems inherent in the methodology and conceptualisation of schemata adopted in schema research. Chapter 4 will expand on these problems and why they may prevent the adequate measurement of schemata in the absence of a depressed or low mood.

Chapter 3

Empirical research into schemata in depression

3.1. Introduction

Schemata have been given a hypothetical causative role in the onset of depression and associated cognitive aspects of a depressive episode (Beck, 1967, 1976). This undoubtedly raises potential measurement problems. To be considered causal, schematic activity (e.g., a negative processing bias) must pre-date actual depression or depressive mood. Evidence of schematic activity in the absence of a depressed mood has proved elusive, confusing and conflicting (Segal, 1988). Methodological approaches to testing the role of schemata in depression have traditionally compared individuals in remission from depression, individuals currently suffering a depressive episode, and samples of high risk individuals presumed to be at an increased risk of developing depression. These samples have been used along with mood induction techniques or priming procedures to activate latent schemas and looked at the effects of different treatments for depression (e.g., drugs versus cognitive therapy). These methodological approaches can assess how an episode of depression, induced low mood, hypothetical vulnerability to depression, and different treatment modalities affect schematic activity (Ingram et al., 1998).

There are two principal approaches to assess or measure schematic functioning in depression: one examines explicit cognitive processes (e.g., self-report measures), while the other attempts to measure implicit cognitive processes (e.g., tasks assessing automatic information processing). The majority of research suggests that schemata are “hypovalent” or latent structures that are activated in response to a low mood or depressive episode. In other words, evidence of depressive implicit and explicit cognitive processes seems contingent on the presence of a low or depressed mood or priming procedures (Ingram et al., 1998; Gemar et al., 2001). However, there are methodological flaws with the existing measures of schemata that may prevent measurement of schematic functioning in the absence of a depressed mood or priming procedures. This chapter will provide an overview of research that has used samples of depressed versus recovered depressed individuals, mood induction techniques, studies investigating the effects of treatments on depression, and individuals who are hypothesised to be at risk of developing depression. The reasons why evidence for schematic activity in the absence of low mood or a depressive episode has not been conclusive will be introduced and briefly discussed. The following chapter will provide a detailed critique of the research and expand upon the reasons why evidence of schematic activity in the absence of priming or a depressed or low mood has been elusive in depression research.

3.2. Studies comparing recovered and currently depressed individuals without mood induction/priming techniques

For measures of depressive schemata (cognition) in depression to be considered as a marker for vulnerability for depression, evidence of schematic processing must be present in depressed persons, and in vulnerable individuals (e.g. individuals in remission from depression). Of the studies outlined below, very few provide evidence that allows one to come to any conclusions about the specificity, sensitivity, and stability of schemas in depression. Most studies seem to be able to answer the question that scores on a questionnaire hypothetically measuring depressive cognition and underlying schemas in depression (e.g., Segal et al., 1999) are higher during an episode of depression. Thus, the findings seem to indicate consistently that schematic activity is a state dependent phenomenon.

In the studies reviewed below, scores on self-report measures of depression of remitted depressed individuals are generally no different from non-depressed controls. Blackburn, Jones, et al. (1986) found that depressed participants when compared to recovered depressed participants scored higher on the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978), which is argued to measure conditional dysfunctional attitudes or schemas (Segal et al., 1999), and is also hypothesised to measure vulnerability to depression. Dohr, Rush, & Bernstein (1989), who also used the DAS, found that individuals suffering from depression scored higher on the DAS than individuals in remission. Further, the remitted depressed individuals were no different on DAS scores in comparison to normal controls. Fennell & Campbell (1984) had similar results using a depressive cognitions questionnaire that measured negative attributions to hypothetical social contexts. They found that currently depressed participants endorsed more negative cognitions related to these hypothetical social contexts compared to remitted depressed and never depressed participants. However, the scores on this measure were significantly lower when the depressed patients were tested in remission.

Dobson & Shaw (1987) confirmed a mood contingent theory of schematic activity using a self-referent encoding task in which participants rated depressed words as either self-descriptive or not self-descriptive. This study measured the time it took participants to rate the words, and after the rating task, participants completed a recall task for the depressed words. They found that currently depressed participants rated more depressed-content words as self-descriptive, were quicker to rate depressed-content words as self-descriptive, and recalled more depressed-content words in the recall test compared to remitted depressed, psychiatric control, and normal controls. Therefore, this study confirms that schematic activity seems to be dependent on low mood or a depressive episode.

However, evidence of schematic activity showing stability in the absence of a depressed mood was found in a study by Eaves & Rush (1984). They found that the scores on

the DAS by individuals in remission from depression were similar when compared to individuals during a depressive episode. The same pattern was observed on the Attributional Style Questionnaire negative sub-scale (ASQ; Seligman, Semmel, & von Baeyer, 1979) which measures individuals' attributions to hypothetical scenarios, which were more negative. It is far from clear, however, whether the group in this study was genuinely in remission and, if they were not, the extent to which residual depression affected scores on the DAS and ASQ.

The influence of residual symptoms on the results of questionnaires was apparent in a study by Dobson & Shaw (1986), who observed similar results to Eaves & Rush's (1984) study. They found non-significant reductions (on a trend to becoming significant) on the DAS and on the Cognitive Response Test (CRT; Watkins & Rush, 1983) by individuals in remission from depression compared to currently depressed individuals. The CRT measures attributions to hypothetical scenarios, similar to the ASQ. But the power of this test is suspect. Many of the participants in this study still exhibited residual symptoms of depression after treatment. Only seven cases met both diagnostic and symptom severity for remission, and re-analysis showed a clear non-significant difference between individuals in remission and currently depressed individuals. Gotlib, Mount, Cordy, & Whiffen (1988) found that remitted depressed patients' scores on the Overprotection subscale of the Parental Bonding Instrument (PBI; Parker, Tupling, & Brown, 1979; as cited in Gotlib et al., 1988) were higher than those of normal controls. However, this result should be treated with caution as the depressed sample consisted of only eight participants.

Although the evidence for schematic activity is weak in individuals who have recovered or who are in remission from depression, there may be other more helpful ways to look at the data. If a measure of depressive cognition returns to normal with remission, there may exist a subgroup of patients for whom high scores on cognitive measures indicate stable vulnerability to develop depression again. Therefore, comparisons of group means, as used in many studies, may not be useful. Rather, estimating the predictive relationship between cognitive variables in remission and the return of depressive symptoms over a follow-up period may be more informative in participants who score in the upper quartile of a depression questionnaire. Indeed, Hollon, Evans, & De Rubeis (1990) and Thase et al. (1992) found that patients who relapsed in a follow-up period had higher ASQ scores and higher DAS scores compared to those who did not relapse. Klein, Harding, Taylor, & Dickstein (1988) found that depressed patients who had recovered from depression, had lower scores on questionnaires measuring dependency and self-criticism than those who were still experiencing significant depressive symptoms at six months follow-up.

Lewinsohn, Steinmetz, Larson, & Franklin (1981) in a prospective longitudinal design, uncovered a similar pattern to Klein et al. (1988). In their study, depressive cognition

was not a stable feature of an episode of depression and depressive cognition also failed to predict the onset of a depressive episode. However, the depressed patients who showed the highest negative cognitions on the ASQ were the least likely to improve (symptom reduction) over the course of the study. This is consistent with other studies (e.g., Nolen-Hoeksema, Girgus, & Seligman's (1992) study. They found that individuals with high depression scores (an analogue depression sample of children) were not significantly different from non-depressed individuals on a measure of explanatory style before the onset of a depressed mood, but did show a more negative explanatory style once in a depressed mood. When a decrease in depressive symptomatology did occur, this group's explanatory style returned to normal and was stable over a two-year follow-up.

Of the research studies that have been discussed so far, it is clear that depressive cognition is often mood-state dependent. That is, scores on questionnaires measuring cognition in depression are higher in a low or depressed mood, and that negative cognitive processing (e.g., ASQ or recall tasks) is more negatively biased in the presence of depression. However, there is some evidence that suggests that post-depression scores may be good predictors of future depressions (e.g., Hollon et al., 1990) and that extreme scores on attribution measures may be indicative of vulnerability for later depressive episodes (Lewinsohn et al., 1981). However, these results are contradicted by other studies (e.g. Dohr et al., 1989). These results that find no hard evidence of schematic activity in the absence of a depressed mood represent a challenge to the cognitive theory of depression, which proposes that negative schemas represent a causal diathesis for depression.

One way to investigate the schema concept in depression and test the hypothesis that negative events "activate" schemata thus affecting cognition, is to look at depressive cognition before and after priming procedures and negative mood inductions. This research will now be discussed.

3.3. Studies using priming or mood induction techniques in schema research

In comparison with studies involving investigations of depressive cognition in depressed patients in remission, there are fewer studies using priming methods to see if depressive schemata are stable. The use of priming techniques and mood induction procedures are used in order to activate latent schemas. Blackburn & Smyth (1985) completed one of the first research studies using priming techniques. This study looked at post-depression scores on the DAS and Cognitive Style Test (CST; Abramson, Metalskey, & Alloy, 1998) after a mood induction in remitted depressed patients. There was no difference between remitted depressed individuals and a non-depressed control group following a mood induction. However, there was evidence that the remitted depressed individuals did not respond to the mood induction adequately, implying some extra motivation on the part of these individuals to avoid a low mood.

Similar results were observed in a study using the Stroop Colour Naming Test (Stroop, 1935), conducted by Gotlib & Cane (1987). They looked at depressed patients during an episode of depression and then again in remission. The participants were asked to name the colour of depressed, manic, and neutral words from the Stroop Task. The priming phase consisted of presenting a list of positive or negative words, which the participants had to listen to and repeat. The Stroop task came after the priming procedure. The results showed that the depressed participants, while experiencing significant depression, were slower in colour naming depressed words compared to non-depressed words. However, at discharge this difference was no longer evident. Again the result was not conclusive, as there was evidence that the priming procedure was not adequate in inducing a low enough mood in those individuals in remission from depression.

Teasdale & Dent (1987) did employ a mood induction that was adequate in producing a low mood in the participants. Their study showed that recovered depressed individuals did not differ from never depressed individuals while in a normal mood state on a task of adjective recall. However, after a negative mood induction, the recovered depressed patients recalled more negative adjectives that had been rated as self-descriptive compared to the never depressed group. In a subsequent study using the same methodology, Dent & Teasdale (1988) looked at depressed females twice over a 5-month interval. They found that differences in the endorsement of negative adjectives as self-descriptive predicted who remained depressed and who recovered.

Similar results have also been found in studies by Miranda & Pearsons (1988), Miranda, Pearsons, & Byers (1990), and Roberts & Kassel (1996). These studies used the DAS and examined the endorsement of dysfunctional attitudes following a negative mood induction. Their results showed that mood predicted the occurrence of dysfunctional attitudes only in people who had a history of depression, but not in people without a history of depression. Therefore, people who were vulnerable to depression (as shown by previous depressive episodes) did seem to possess, or endorse more dysfunctional attitudes, but these dysfunctional attitudes were inaccessible when they were in a non-depressed mood. One possibility is that the activation of dysfunctional attitudes (or schemas) could have been the result of a psychological “scar” from previous episodes of depression as, Lewinsohn et al. (1981) suggest.

Williams (1988), disconfirming the scar hypothesis, used a longitudinal design, to examine vulnerability to depression in an analogue student sample using a mood induction procedure. Participants’ recall of negative and positive adjectives was measured in two conditions: a neutral mood and after a negative mood induction. The participants were then followed up for one year to see who experienced a significant depressive episode. The results showed that differential recall of positive versus negative adjectives in a neutral mood did not

predict whether someone would experience a depressive episode. However, recall under a negative mood induction showed that recall predicted subsequent depression. The participants who recalled more negative self-descriptors compared to positive self-descriptors after a mood induction were more likely to become depressed during the one year follow-up. Therefore, schematic activation was congruent with a negative mood, but not dependent on previous episodes of depression and did not result from a psychological “scar”.

Confirmation of the relationship between low mood and schematic activation was obtained in a study by Hartledge, Alloy, Vazquez, & Dykman (1993). They used a semantic priming paradigm to examine automaticity of attributional inferences in response to life events in groups of never depressed and depression-prone students. They used students with high, but non-clinical levels, of depressive symptoms who also had high scores on negative attributional style as measured by the Cognitive Style Test. The participants were asked to decide whether a cause for an event (e.g., failing an exam) was due to internal (e.g., incompetent) or external (e.g., difficult) factors as fast as possible. The prime was presented first, which was a description of an event (e.g., fail exam) just before the presentation of the cause (incompetent or difficult). The results showed that there was a priming effect for the depression-prone students. This group showed increased automaticity for internal attributions for negative events and external attributions for positive events. The non-depressed group showed no evidence of automaticity in decision making of internal or external events.

Ingram, Brenet, & McLaughlin (1994) obtained curious results in a study looking at attentional processing in recovered and never-depressed participants in a dichotic listening paradigm that had two conditions: completing a dichotic listening task in a neutral mood and after a negative mood induction. The task involved the participants listening to a story in one ear and repeating what they heard, while in the other ear they heard distractor words, which they were instructed to ignore. The distractor words contained both positive and negative stimuli. The study measured tracking errors, which used an index of how much the distractor stimuli were affecting individuals’ attentional capacities. There were no differences between the groups in the neutral mood condition, but in the sad mood condition the formerly depressed individuals made more tracking errors with both positive and negative distractors. The number of errors made by never-depressed group was similar in both the sad mood and neutral mood conditions. The results of this suggest that a negative mood does activate schematic processes in depression vulnerable individuals, but that the allocation of attention is unaffected by the nature of the emotional cues. Rather, this study showed that those who had recovered from depression when induced into a low mood have a bias towards processing negative and positive emotional stimuli of any kind. Therefore, unlike negative biases on information processing tasks and high scores on depression questionnaires in other studies being linked to schematic activity (e.g., Miranda & Pearsons, 1988; Hartledge et al., 1993),

this study shows that schematic activity and low mood are linked to the biases in response to all emotional material.

There were similar, but also contradictory, findings in a subsequent study conducted by Ingram & Ritter (2000). This study compared individuals in remission from depression remitted to individuals who had never suffered from depression following a mood induction using a similar dichotic listening task to the one used in the previous study. With negative stimuli, the results of tracking errors were similar to the earlier study, in that the remitted depressed group made more errors when tracking negative stimuli. However, there was a different pattern of errors with positive stimuli: the remitted depressed were not distracted by positive stimuli. The reason for this discrepancy between the two studies is unclear. It may represent that those in remission from depression in the earlier study were more fully recovered from depression and had recovered a greater degree of interconnectedness of positive self-schematic constructs (Taylor & Brown, 1988), but still had some access to negative constructs. Ingram & Ritter's study provides some support for the specific accessibility of negative schematic structures in vulnerability to depression. This is in accord with Beck (1967, 1976), who argues that when schemas become activated in response to a low mood, this facilitates the processing of negative information, but not positive information.

The results from studies using priming and mood induction procedures support the idea that latent schemata in depression produce negative information processing biases, and high scores on questionnaires measuring depressive cognition when they are activated. This is in accord with contemporary theories of the role of schemata in depression (e.g., Gemar et al., 2001; Segal, 1988). However, priming and mood induction studies are also problematic for causal theories about the role of schemata in the onset of depression. This is because evidence of schematic activity in the absence of a depressed mood and without using priming and mood induction strategies is very weak, thus causality and the role of schemata is difficult to establish (Segal, 1988). Indeed, research into schematic activity and treatment for depression also supports a latent schematic model of depression. This research will now be discussed.

3.4. Negative schemas and the effects of treatment in depression

This section will now consider some research that has investigated the effects of cognitive therapy and the use of SSRI's and pharmacotherapy for depression, with regards to schemas and depression. Investigating schematic change and treatment of depression provides further valuable information to ascertain the nature of schemas in depression and whether the latency concept in schemas is a valid heuristic.

There is encouraging evidence that cognitive therapy reduces relapse and recurrence of depression (Jarrett, Kraft, Doyle, Foster, & Eaves, 2001; Beck, Rush, Shaw, Emery, 1979). Patients who recover following treatment of depression with cognitive therapy show lower relapse rates and less need for further treatment than patients who recover with

the use of pharmacotherapy (Blackburn, Eunson, & Bishop, 1986; Evans et al., 1992; Simons Murphy, Levine, & Wetzel, 1986). Cognitive therapy following recovery with pharmacotherapy can also reduce relapse levels and recurrence of depression (Fava, Grandi, Zielezny, Rafanelli, & Canestrari, 1996). In patients who only respond partially to pharmacotherapy, the addition of cognitive therapy to treatment and the continuation of pharmacotherapy significantly reduces rates of depressive relapse (Paykel et al., 1999). These studies are argued to provide evidence that negative self-schemata are altered in individuals treated with cognitive therapy (e.g., the development of positive self-schemata), as vulnerability for further episodes is reduced compared to pharmacotherapy treated individuals (Gemer et al., 2001).

Teasdale & Barnard (1993), and Teasdale, Segal, & Williams (1995) have argued that in successfully completed cognitive therapy, patients approach depressive symptoms (post depression episode) and stressful situations with different, more functional, cognitive sets in place. The creation and storage of representations encoding such alternative cognitive sets is suggested to mediate the therapeutic effects of cognitive therapy. In other words, Teasdale et al. (1995) have suggested that the perception of depression (by the patient) as highly aversive and uncontrollable leads to “depression about depression”, and that cognitive therapy reduces this by increasing perceived controllability and reducing the perceived aversiveness of depression. Further, cognitive therapy involves a shift in the cognitive set with which negative thoughts are approached; rather than being approached as “truths or aspects of the self”; thoughts are approached as “events in the mind” that may or may not correspond to reality. This in turn theoretically leads to a restructuring of the dysfunctional schemata. This facet of schematic change via the use of cognitive techniques has been examined in studies comparing cognitive oriented therapies and pharmacotherapies for depression.

Studies have shown that the use of pharmacotherapy for depression has indeed lifted the depressed mood and reduced symptoms of depression, but still left an increased vulnerability to relapse compared to cognitive therapy shown by other markers other than depressive symptomatology. In a study by Segal et al. (1999), patients treated with selective serotonin reuptake inhibitors (SSRI's) had higher post-treatment scores on the DAS but also had an increased cognitive reactivity to a mood induction as measured by elevated scores on the DAS, compared to patients treated with cognitive behavioural therapy. Further, the SSRI treated patients were also more likely to experience a depressive relapse. In effect, post-treatment DAS scores and levels of reactivity to the mood induction predicted subsequent relapse. This implies that patients treated with SSRI have an increased cognitive reactivity following treatment and increased levels of dysfunctional attitudes. Cognitive reactivity to a negative mood induction, as already discussed in the previous section, is argued to be indicative of schematic activation (Teasdale & Barnard, 1993; Beck, 1967, 1976). The results

of Segal et al.'s study are consistent with Zurroff, Pilkonis, Blatt, Sainslow, & Biondi's (1999) study in which they found that patients treated with antidepressants still had relatively stable scores on the DAS, despite decreases in depressive symptomatology, and Hensley, Nadiga, & Uhlenhuth's (2004) suggestion that schematic change is facilitated with cognitive therapy by reducing cognitive reactivity but not by pharmacotherapy.

A further study concurs with Segal et al.'s (1999) findings. Hedlund & Rude (1995) compared individuals who had recovered from depression and had been treated with pharmacotherapy to individuals who had never suffered from depression on a self-focus manipulation procedure (participants had to focus on their own inner experience and thoughts). Those who had recovered from depression scored similarly on self-report measures of depression, including the DAS compared to those who had never suffered depression. However, on measures of implicit processing the formerly depressed individuals performed with a more negative bias compared to those who had never suffered. The tasks used were the scrambled sentences task (Wenzlaff, 1988), which contains forty scrambled sentences (e.g. "winner born I am a loser"), each of which permitted a positive or a negative solution. These are mixed with 20 distractor sentences ("beverage a hot drank cool"), each of which permitted a neutral solution to obscure the purpose of the measure. The sentences were displayed on the screen for 12 seconds and participants had to make a sentence not necessarily using all the words. The second task was the incidental recall task (Hertel & Rude, 1991) where participants were asked to recall 10 minimum or 15 maximum words from a modified Stroop test performed as part of the experimental procedure. On these tasks those who had recovered from depression were biased towards recalling more negative words and making more negative sentence solutions.

The research into treatment for depression and schematic activity, like the mood induction and priming research, supports a latent schema model of depression (Gemar et al., 2001). One other way to assess if schematic activity is measurable in the absence of a depressed mood is to investigate individuals who are thought to be "at risk" of developing depression, but who currently are not depressed or have ever suffered from a depressive episode. One can then investigate precisely the hypothetical causal role that schemata are given in depression.

3.5. Schema research using high-risk individuals

This section deals with research that has investigated samples of people who were deemed to be at high-risk of developing depression. By sampling these individuals, one can ascertain if schematic activity is present before the onset of a depressive episode. Some ways in which individuals can be identified as being at high-risk to developing depression in various ways are: measuring cognitive style (Alloy et al., 1999), assessing the presence of cognitive and behavioural traits associated with increased risk of developing depression

(Zemore et al., 1990); and whether one has a parent who is depressed, as this hypothetically puts the child at a greater risk of developing depression (Hersen & Ammerman, 1995).

A study by Taylor & Ingram (1999) utilised a high-risk paradigm to investigate schematic processing in the children of depressed mothers. The children took part in a memory task and the dependent variable of interest was the differential recall of negative or positive information. Half of the participants were tested after a negative mood induction procedure, while the other half were tested in a normal mood. Participants, who were in the mood induction group, were more likely to recall negative information than participants in the neutral condition. This implies that cognitive reactivity may be a feature of “at risk” individuals, and not the result of cognitive “scar” as a result of a previous episode of depression. Again, this result is consistent with a schema activation hypothesis of depression (Beck, 1967, 1976). However, this result does not adequately establish that the recall of negative material was due to the participants holding negative self-schemata per se. For example, negativity in general (e.g., living in a depressed environment with a depressed parent) could be the reason why negative material is better recalled. Furthermore, Taylor & Ingram did not assess if the recalled material was strongly associated with the self. Thus, one cannot assume that the recalled material is representative of negative self-schemata. Lastly, Taylor & Ingram did not address the issue of the congruence between the stimuli and hypothetical negative self-schema. In other words, perhaps a negative bias would have been observed in the neutral condition to words that were more congruent with the hypothetical content within negative self-schemata.

This issue is indeed what Alloy et al., (1999) addressed. Alloy et al. found that high-risk individuals (as measured by cognitive style) showed preferential self-referent processing of negative depression-relevant material involving schematic themes of incompetence, worthlessness, and low motivation on implicit processing tasks compared to depression-relevant words related to themes of low mood. This was shown by faster reaction times to judge whether schematic themed words were an attribute of the self or not to, and superior recall of schematic themed words on a memory task. This last point is highly important as Beck (1967) suggests that depression prone people have specific negative self-schemata related to these domains. Thus information biases should be limited to stimuli that are congruent with the content hypothetically embodied in the self-schemata and not with stimuli that reflect other domains. This could be one reason why researchers have failed to find a consistent processing bias related to schematic activity in the absence of a depressed mood. Indeed the high-risk individuals in this longitudinal study were at a significantly higher risk, compared to individuals classified as low risk, of developing a depressive disorder.

Therefore this study provides good evidence that schematic activity is measurable in the absence of a depressed mood. However, the fact that participants had to rate the words as being representative of the self before the task raises serious questions as to whether this was really an implicit task. There is the possibility that self-presentational and controlled processing strategies could have confounded the results. Furthermore, asking individuals to concentrate and rate words as being self-descriptive may have induced some form of schema activation through unintentional priming similar to Hedlund & Rude's, (1995) self-focus manipulation technique.

3.6. Methodological problems in schema research

The research discussed in this chapter strongly supports a latent schema model of depression. There is very little strong evidence of schematic activity in the absence of a low or depressed mood or without the aid of mood or priming techniques. However, there are several methodological flaws. Firstly, the use of questionnaires such as the DAS (e.g., Blackburn, Jones et al., 1986; Dohr et al., 1989) to assess conditional attitudes ("I could be defective if I lose..."). However, negative self-schemata have been argued to be unconditional in nature ("I am defective") (Young, 1990). In other words, conditional dysfunctional attitudes may only be the function of a depressed mood, while core unconditional beliefs (e.g., of being defective) may be more accessible in the absence of a depressed mood. Indeed, many studies demonstrate that hypothetical schema activation produces higher scores on questionnaires such as the DAS (e.g., Segal et al., 1999). Therefore, DAS scores could be regarded as the products and processes of depressive cognition, but not cognitive structure or negative schemata per se (Young, 1990; Gemar et al, 2001). Indeed, many of the questionnaires and tasks used in the studies discussed above measure the products and processes of depressive cognition (e.g., ASQ; CST and task used in Hartledge et al, 1993) rather than cognitive structure or schemas as defined by Beck's cognitive theory of depression (1967, 1976).

The heavy dependence on questionnaires is a limitation that researchers should consider if they intend to measure schemata in depression. Demand and presentational strategies may affect responses on questionnaires or on tasks that demand controlled judgments. When people fill out questionnaires or are asked to make judgments they may lie to try to present themselves in a positive light, or may be so unreflective or unaware of their beliefs or feelings on a certain matter that they cannot express their true attitude or belief (Nisbett & Wilson, 1977). According to Beck (1967, 1976) schemata affect information processing in an implicit way. Therefore, if explicit and controlled processes influence questionnaires and related tasks, then surely these ways of measuring schemata are not tapping into more implicit processes. Consequently, these methodologies may only be tapping into the products or processes of depressive cognition. Indeed, the little evidence of schematic activity

in high risk individuals (to depression) may also have been confounded by explicit processing confounds (e.g., Alloy et al., 1999). Participants in Alloy et al.'s study had to rate whether words were representative of the self before the experimental task. This process could be considered a form of priming due to the self-focus nature of this task.

Furthermore, instruments such as the Stroop task (e.g., Gotlib & Cane, 1987), recall tasks (e.g., Hedlund & Rude, 1995), and a dichotic listening task (e.g., Ingram & Ritter, 2000), although convincing in their measurement of implicit processes, may not be assessing negative self-schemata per se. How does the naming of a negative colour word or the recall of words, or listening and repeating words, bear any relation to how an individual regards him/herself at an implicit level or measure an individual's self-representational system? Such tasks may be more of a reflection of an individual's greater experience of low mood (e.g., familiarity or memory; De Houwer, 2006) rather than schematic structures.

There also seems to be a lack of consideration of the nature of the stimuli used in the aforementioned studies (e.g., Taylor & Ingram, 1999; Ingram et al., 1994; Teasdale & Dent, 1987). Young (1990) argues that dysfunctional schemata (for different mental disorders) may be related to specific themes and these themes may be specific to different psychopathologies. Therefore, the global negative words used in many of the studies may not be congruent with the content of negative self-schemata in depression. Indeed as Alloy et al. (1999) demonstrated, individuals who were vulnerable to develop depression only had a bias to process words related to certain schematic themes (e.g., worthlessness and incompetence). If one is to come to any conclusions regarding the operations of negative schemata in depression, and their role in causality of depression, one has to address the implicit operation of schemata rather than the explicit affects of schemata, the stimuli used, and whether the stimuli are implicitly associated with the self.

3.7. Summary

The research investigating schemas using individuals who have recovered or are in remission from depression, mood induction or priming procedures, high-risk individuals, and samples receiving cognitive therapy versus pharmacotherapy, provides very strong evidence that the schema construct is latent and becomes activated in response to low mood or depression. This undoubtedly raises potential problems for the role that schemata have been given with regard to their hypothetical causative function in the onset of depression. To be considered causal, schematic activity must predate actual depression or depressive mood, the evidence for which is not strong as shown from the results of research (Gemar et al., 2001; Segal, 1988). However, there are problems associated with approaches to schema measurement in depression. Scores on self-report measures are influenced by several factors that may confound any inferences one can make, e.g., self-presentational bias (Nisbett & Wilson, 1977). Furthermore it is doubtful whether the explicit or controlled processes that are

measured by self-report measures, actually taps in to schematic functioning, as schematic functioning may be better measured with implicit methods (Eysenck, 1991). Automaticity or implicit functioning is more in line with Beck's notion of biases of information processing in depression as a result of schematic activity (Bargh & Tota, 1988; Beck, 1967, 1976). There are, however, several methodological flaws with existing implicit measures used in research. These include the inappropriate choice of stimuli and whether the stimuli is congruent to content within a negative self-schema structure, and whether the implicit task relates to automatic self-evaluation (e.g., a negative self-schema) or to the influences of memory or to the familiarity related to general life experience (e.g., having lived within a depressing environment). Further, there is the issue of whether the implicit tasks are really measuring implicit processes, or are being influenced by controlled and explicit processes (Segal, 1988, De Houwer, 2003). The next chapter will deal with these issues in detail, along with a discussion of new developments in depression research, and the consideration of other important variables that may affect schematic processing that need to be addressed.

Chapter 4

Methodological considerations and future directions of schema research in depression

4.1. Introduction

In the last chapter, a range of research was discussed that strongly supports a latent schema model of depression (e.g., Gemar et al., 2001). Schematic activity appears to be dependent on the presence of a low or depressed mood, or affected by priming in some way. The chapter also identified several problems associated with the use of both explicit and implicit methodologies that are used in schema research. These methodological problems may explain the reasons why the evidence for schematic activity in the absence of depressed mood is not strong. The aims of this chapter are: to expand upon these methodological problems associated with research into schemata in depression, to introduce other important methodological issues, and to attempt to outline a framework for future research, as carried out within the studies in this thesis, that may facilitate the measurement of schematic functioning in the absence of a low or depressed mood.

The first section deals with the importance of the use of implicit or automatic information processing methodologies in order to tap into schematic functioning in depression. This will focus on theoretical issues associated with implicit or automatic schematic functioning in depression and provide evidence as to why self-report measures may lack validity in measuring schemata. The following section will focus upon the important role of the implicit association of the self or self-evaluation in implicit tasks and why this methodological facet may be crucial to measure schemata (De Raedt et al., 2006). Two new implicit processing tasks that measure self-evaluation will be discussed in this section. These are the Implicit Association Test (IAT; Greenwald, Banaji, & Schwartz, 1998) and the Extrinsic Affective Simon Task (EAST; De Houwer, 2003). Recent research investigating depression and affective disorders using these tasks has shown promise. Taking into consideration the methodological considerations discussed in this chapter, they might be useful in attempting to measure schemata in the absence of a low or depressed mood. The next section will discuss the importance of the attributes of stimuli used in schema research. This will be concerned with the importance of selecting stimuli that is congruent with the theoretical schema structure that one is trying to measure (e.g., Alloy et al., 1999). Further, issues pertaining to the actual structure of the stimuli used in tasks (e.g., length and emotionality of words) in implicit processing tasks will also be discussed as an important consideration in implicit tasks. Following on from this will be the issue of what kind of data one should consider from the information obtained from implicit tasks. Traditionally, researchers have focused on reaction times as a valid marker of congruency with hypothetical cognitive constructs like schemas. However, implicit tasks also provide a measurement of

errors. It has been argued that reaction time, although a good measure of implicit processing, may be influenced by both explicit and controlled processing, whereas errors may be less affected by explicit processes. Therefore, errors may be a more accurate measure of schematic functioning (Bargh & Tota, 1988; Kirsch & Lynn, 1999). The next topic to be dealt with relates to the importance of careful consideration of samples used in depression research. The sampling of different individuals and their position on the depressive continuum (e.g., recovered depressed versus at risk to depression) may have a bearing on the inferences one can make pertaining to schematic functioning in depression (Hammen & Krantz, 1985). Lastly, a research framework will be outlined that will draw on the methodological considerations highlighted in this chapter, that will form the basis for the following empirical chapters.

4.2. Implicit/automatic and explicit processes and the role of schemata¹

As discussed in Chapter 3, the use of self-report measures (e.g., Segal et al., 1999) and some implicit tasks (e.g., Dobson & Shaw, 1987) may only measure the results of explicit processing. Therefore, it is problematic to ascertain from the results of some research studies whether the data obtained is a result of explicit or implicit/automatic processes, and therefore the result of schematic functioning and vulnerability to depression. One of the main objectives of the research into schemata and depression (some might say an elusive objective) is to differentiate between negative implicit or automatic processing that is involved in vulnerability to depression, and automatic processing that is the direct result of a low or depressed mood. This point is important as Beck (1976, 1976) argues that schemata, when activated, influence information processing unconsciously or automatically and there is subsequently a bias to preferentially process material of a negative nature. Beck argues that the reporting of negative thoughts is the result of more controlled processes, which are the result of a low mood facilitated by negative unconscious schematic functioning. Thus, the consciously reported negative thoughts associated with depression are hypothetically only the consequences of activated schemata, but not of schematic activity per se. Consequently, measuring consciously controlled negative self-reported thoughts associated with a depressed mood does not measure vulnerability to depression and schematic functioning. Therefore, one of the most important methodological considerations if one is to investigate schematic functioning in depression is to measure the results of implicit processes, rather than the results of explicit processes. Greenwald (1997) concurs with this assessment. He argues that implicit and explicit processes operate independently of each other but simultaneously. Greenwald posits that the two different processes are not congruent - so what people explicitly report may not be congruent with their implicit response (Nisbett & Wilson, 1977). Further, Eysenck

¹ For the purposes of this thesis the terms implicit and automatic are used interchangeably.

(1991) argues that automatic or implicit processing and associated biases are most likely a result of a vulnerability factor, while explicit or controlled processing (e.g., high scoring on depression questionnaire) is the result of a low or depressed mood.

In agreement with an explicit/implicit dichotomy of cognitive functioning and schematic activity, Gotlib & Krasnoperova (1998) argue that self-report measures are not effective in the assessment of the existence and operation of schemata or associative cognitive networks. Gotlib & Krasnoperova (1998) examined this proposal in a study investigating individuals' responses in a consumer survey. Participants were asked to indicate which pair of stockings (out of 4 pairs) was the best quality (in reality there were no differences in quality). The majority of the participants chose the pair of stockings on the far right from the row of stockings. When the experimenters asked the participants why they chose the right hand pair, nobody mentioned the position of the stockings on the row. When asked if position was a factor, all of the participants denied it. Gotlib & Krasnoperova suggest that the participants started evaluating on the left of the row and may have impulsively wanted to avoid choosing the first pair they saw. Another reason may be that people implicitly associate left with "bad" or negative connotations and right with "good" and positive connotations because of socio-cultural influences (J. De Houwer, personal communication, December 10, 2000). Whatever the actual reason, the participants made an implicit decision but were unaware that the position was a factor in the choice they made. Therefore, serious and fundamental concerns over the accuracy of what questionnaires actually measure should be considered. It seems that aspects of explicit decision-making when responding to questionnaires may be affected by factors that remain outside awareness.

There are other problems with the use of questionnaires to assess depression, schemas and vulnerability to depression. Touched upon in the last chapter, these involve issues of vulnerability towards social desirability, self-deception, subjectivity, and experimental demand. Individuals may be susceptible to experimental demand, especially if a measure is re-administered in the same study (Gemar et al., 2001). Individuals may present themselves in a more favourable light by underreporting depressive symptoms to avoid evaluation by others, either initially or during post-treatment assessment of symptoms (Eysenck, 1991; Rudman, Greenwald, Mellot, & Schwartz, 1999). Researchers have found that several depression and anxiety measures are moderately to highly associated with social desirability. Tanaka-Matsumi & Kameoka (1986) found correlations ranging from -.49 to -.85 and -.19 to .32 between nine anxiety and depression scales and the Edwards Social Desirability Scale (ESDS: Edwards, 1957), and the Marlowe-Crowne Social Desirability Scale (MCSDS: Crown & Marlowe, 1960).

Continuing with the issue of self-deception and self-report measures, Shedler, Mayman, & Manis (1993) posit that individuals may deceive themselves about their psychiatric symptoms, and may have an illusion of mental health. Shedler et al. describe such individuals as “defensive deniers”. These individuals use the denial of psychological distress as a defense mechanism and thus may ignore their feelings, desires, and needs. It is argued that self-report measures may accurately measure distress in those who are manifestly distressed and in those who are genuinely healthy, but may not capture underlying distress in those who have illusory mental health (Shedler et al., 1993). Surprisingly, Taylor & Brown (1988) argue that mentally healthy people tend to have illusions of exaggerated positive self-evaluations, perceptions of control, and are overly optimistic. Further, they suggest that mentally healthy people have positive cognitive biases during encoding, interpretation, and retrieval. This in turn affects responses on self-report measures. Taylor and Brown argue that individuals with low self-esteem and/or depression somehow lack these positive cognitive biases and consequently view reality in a more realistic way. In essence Taylor & Brown posit that harbouring an illusory positive cognitive style may be helpful in overcoming setbacks, maintaining high self-esteem, and holding a positive view of the future. Conversely, defensive deniers who have an illusion of mental health may not fall into a category of being mentally healthy. More specifically, individuals who are defensive deniers showed higher levels of physiological reactivity under stress, and more verbal manifestations of defense than genuinely healthy and manifestly distressed participants. Therefore, self-report data may not be sensitive enough to tap into the underlying vulnerability to mental illness.

In summary, according to hypothetical definitions of schemata and the workings of schemata in depression, schemata operate at an automatic level and the effects of schemata are non-volitional (Beck, 1967, 1976; Teasdale & Barnard, 1993). Essentially, individuals who are vulnerable to depression may not accurately report their underlying cognitive processes since they may not be aware of them, or may not be accurate in their perception of these processes involved in their judgments, behaviour, and choices (Higgins, & King, 1981; Nisbett & Wilson, 1977). Thus, if one is to assess vulnerability to depression and schematic functioning, one has to consider theoretical postulates of the cognitive model of depression (Beck, 1967, 1976) and minimise the influence of explicit processes, which may not be the result of automatic schematic functioning. However, the importance of assessing automatic schematic functioning is not the only facet one must consider. One must also consider if automatic schematic functioning is related or associated with the self in some way. This issue will now be discussed.

4.3. The role of implicit or automatic self-evaluation

As is hopefully now becoming apparent, automatic or implicit functioning may be important (and is often overlooked) in the assessment of schemata in depression. However, as also discussed in Chapter 3, automatic self-evaluation or information processing assessing associations with the self is often not considered as being important in research endeavors investigating depression (e.g., Gotlib & Cane, 1987; Hedlund & Rude, 1995; Ingram & Ritter, 2000). Therefore, negative processing biases displayed on certain tasks by individuals vulnerable to depression (e.g., Alloy et al, 1999) may be the result of other constructs (e.g., memory), which are not the result of a self-schema structure. Indeed, there is some evidence that supports the idea that information processing is facilitated if there is some kind of context that implicitly guides information processing (Pollatsek & Rayner, 1989). So, if one is to measure negative self-schemata and vulnerability to depression (in the absence of a low or depressed mood), one needs not only to measure the automatic nature of schemata, but also to incorporate the context of the self-system into automatic processing tasks. This may be one reason why schematic functioning in the absence of a low or depressed mood has proved elusive for researchers.

It is becoming clear from recent research findings that self-esteem or self-evaluation at an automatic level is perhaps crucial if one is to assess schematic functioning in depression (De Raedt et al., 2006). In recent years researchers investigating the self have come to question the role of consciousness in the self-evaluation process (J.D. Brown, 1993; Epstein & Morling, 1995; Greenwald & Banaji, 1995). In view of the evidence that many important social and cognitive processes function without the need for conscious awareness (Bargh & Chartrand, 1999, Greenwald & Banaji, 1995), these researchers have argued that self-evaluations might operate at non-conscious levels of awareness. Therefore, implicit self-evaluations may provide a more accurate measurement of potential vulnerability to mental disorders like depression, rather than self-report measures focusing on self-judgments. Experimental findings have supported this notion as an accumulation of evidence has shown that people display a pervasive implicit positive bias in their evaluations of self-associated stimuli (e.g., first letters of first name; Nuttin, 1985), even though they lack any awareness of this self-favouring bias. This suggests that self-evaluation may occur in the absence of conscious self-reflection (Greenwald & Banaji, 1995). Thus, a positive automatic self-evaluative bias, a lack of one, or enhanced negative self-evaluative bias in individuals vulnerable to depression, may be a key factor in the role of schemata and vulnerability to depression (De Raedt et al., 2006; de Jong, 2000).

In the last few years, new tasks have been developed that could provide a satisfactory way of assessing the underlying schema processes or ontological insecurity related to

vulnerability to depression. These new tasks measure implicit self-esteem or the automatic evaluation of the self (Greenwald & Banaji, 1995). The Implicit Association Test (Greenwald et al., 1998) is one such task that generated much interest in the measurement of implicit self-esteem. In the IAT participants are asked to categorise words that appear in the middle of a computer screen as belonging either to themselves (e.g., I), or someone else (e.g., they), or as negative (e.g., worthless), or as positive (e.g., successful) by pressing one of two keys. There are usually two test blocks on a standard IAT. In one test block, one key is pressed for words belonging to the participant (self) and negative words and the “other” person key is pressed for words that belong to other people and positive words. The response times and/or errors in this block are compared to the other test block in which self words and positive words are assigned the same key and words related to other people and negative words are assigned the other key. The research findings confirm that healthy individuals categorise words significantly quicker in the block when words related to self and positive words are assigned to the same key. These results are argued to be indicative of the presence of a positive self-esteem or positive automatic self-evaluation (De Raedt et al., 2006; Greenwald & Farnham, 2000).

Another task, the Extrinsic Affective Simon Task (De Houwer, 2003) is a modified version of the IAT whereby one is able to compare performance on subsets of trials within a single test block rather than a comparison of performance on two different test blocks. There are three phases in the EAST; 2 practice blocks and 1 test block. The first practice block asks participants to classify words associated with the self (e.g., first name) and words related to someone else (e.g., a name of another hypothetical person) in the center of a computer screen. Participants press one key for words related to self and another key for words related to “another” person. The second practice block involves categorising words by colour (blue or green) rather than by their meaning. Participants press one key for blue words and the other key for green words. Half of the blue words and half of the green words are positive and half are negative, ensuring an equal number of positive and negative words are assigned to both keys. The test phase involves both the coloured words, self-words, and words related to another hypothetical person being randomly presented. It has been found in healthy undergraduate samples that performance (faster response time and lower error rates) for categorising positive coloured words are superior when they are assigned the same key as the key used to categorise words related to the self. At an implicit or automatic level, positive self-evaluation is taking place because processing is superior when positive and self is associated, even though positive and negative words are categorised only on colour not on their meaning (De Raedt et al., 2006; De Houwer, 2003; Gamar et al., 2001).

Even though implicit or negative automatic self-evaluation is argued to be a crucial aspect in vulnerability to depression, research that has investigated self-evaluation in depression is very sparse especially with new tasks such as the IAT and EAST. However, these two new tasks may provide a way to measure latent self-schemata and hypothesised vulnerability to depression. It is a consistent finding that implicit self-esteem measures, such as the EAST and IAT alongside other similar implicit measures, do not correlate very strongly with explicit self-report measures (Greenwald & Banaji, 1995; Bosson, Swann, Pennebaker, 2000; De Houwer, 2002). This is in line with the idea that automatic or implicit processes and controlled explicit processes stem from different sources and should be regarded as different cognitive constructs (Rudman, 2004). More importantly, from the perspective of Beck's (1967, 1976) theory of the automatic nature of schematic functioning, the fact that self-report measures do not correlate strongly with the IAT and EAST may indicate that IAT and EAST may be valuable in measuring schematic functioning.

Of the limited research that has used these two new tasks (IAT & EAST) in assessing negative self-schemata in depression, some curious findings have resulted. Gemar et al. (2001) used the IAT to examine mood related changes in explicit and implicit self-esteem in those who recovered from depression and never depressed individuals. Following a negative mood induction, the recovered depressed group showed a greater shift towards a negative implicit and explicit self-esteem compared to the never depressed group. However, both the recovered depressed and never depressed controls displayed a preferential implicit positive self-esteem before, as well as after, the negative mood induction compared to a negative implicit self-esteem. Further, the difference between the pre and post mood induction performance in the recovered depressed group was in fact due to a more positive implicit self-esteem before the negative mood induction. In other words, the recovered depressed were greatly affected by the mood induction procedure (labile positive self-esteem) and displayed a greater negative implicit self-evaluative shift as a result of the negative mood induction. However, after the mood induction, when comparing the recovered depressed individuals with the never depressed individuals, no actual significant difference was evident on implicit self-evaluation between the two groups. This means that although the recovered depressed group did seem to react in a typical way to a negative mood induction (Miranda & Pearsons, 1988), essentially this group had overall an implicit positive self-evaluative bias. Nevertheless, the recovered depressed group's self-evaluation was more labile. This is partly in accord with schema activation theory of a fluctuating self-schematic bias in the face of a mood challenge (Beck, 1967, 1976).

These findings suggest that implicit self-evaluation remains positive in depressed individuals despite a mood challenge. This is not in line with Beck's cognitive theory of

depression. Beck argues that self-schematic processing should become more negative as a result of a mood challenge (Beck, 1967, 1976). There were, however some methodological irregularities with the scoring procedure of the IAT conducted by Gamar et al. that are not in line with scoring procedures recommended by Greenwald et al (1998) and thus could have produced unreliable results.

The role of positive implicit self-esteem was also investigated by De Raedt et al. (2006) using both the IAT and EAST tasks. De Raedt et al. found in a group of currently depressed individuals that they did indeed show evidence of positive implicit self-esteem by faster reaction times when positive adjectives were associated with the self, and this was not significantly different from a group of healthy controls. Surprisingly, the healthy controls displayed a weaker implicit positive self-esteem on the EAST task compared to the depressed individuals.

The findings of Gamar et al. (2001) and De Raedt et al. (2006) are not completely in line with cognitive theories of depression, but are partly in accord with some research findings and arguments that support the role of positive self-esteem or positive schemata being implicated in vulnerability to depression and related affective disorders (de Jong, 2000; Tanner et al., in press; Taylor & Brown, 1988). It has been found that the self-schemata of depressed people do not lack positive content. Individuals vulnerable to depression are argued to have mixed negative and positive self-schemata, but the fashion by which the positive content is activated, processed and organised might differ in depressed individuals compared to non-depressed individuals (Dozois & Dobson, 2001; Greenberg & Alloy, 1989). Perhaps implicit positive self-evaluation is more labile in individuals vulnerable to depression (as shown by Gamar et al, 2001) and perhaps in more ecologically valid conditions the labile positive self-esteem may falter and create a vulnerability to spiral into depression. Indeed it has been argued that individuals who are not vulnerable to depression differ from individuals who are vulnerable to depression as they possess a more stable and even perhaps unrealistic positive self-esteem, which protects them from developing depression (Taylor & Brown, 1988; Roberts & Munroe, 1992; 1994).

Other research using the IAT with individuals suffering from social anxiety supports the growing amount of research that implicates a dysfunctional automatic self-evaluation as a crucial aspect related to affective disorders (Tanner et al., in press; de Jong, 2000). In de Jong's (2000) study with socially anxious individuals using the IAT, the crucial difference between socially anxious and non-socially anxious individuals was that the socially anxious had a weaker positive automatic self-evaluative bias. Indeed, Tanner et al. (in press) found that after a social-threat activation task (similar to a negative mood induction) that socially anxious individuals did show a positive automatic self-evaluative bias, but this was weaker in

the socially anxious group compared to non-socially anxious individuals. Therefore, it seems that further investigations into the role of a positive automatic self-evaluation are warranted. De Jong (2000) argues that a weak positive self-evaluative bias may be what differentiates those who are vulnerable to affective disorders, compared to those who are not vulnerable. The use of the IAT and EAST tasks are relatively recent and more research needs to be done to ascertain the role of automatic or implicit self-evaluation, vulnerability to depression, and associated schematic functioning.

4.4. Other important methodological considerations: The role of stimuli, errors versus reaction time, and sampling

Much of the research into schemata and depression does not consider fully the choice of stimuli used in implicit tasks and whether it is congruent with the content within the theoretical schema structure. Further, the attributes of stimuli (e.g., word length and inherent emotionality of words) are frequently not considered when designing implicit processing tasks. Other neglected aspects within research are the role of errors produced on implicit tasks, and the consideration of the samples used. These criticisms can also be attributed to the recent depression research that has used the IAT and EAST (e.g., De Raedt et al., 2006; Gerner et al., 2001).

4.4.1. The role of stimuli

In most studies, there is very little in-depth discussion of the reasons why researchers choose their stimuli and its specifications (e.g., De Raedt et al., 2006; Gerner et al., 2001). There is rarely any mention with regards to the word frequency of stimuli, syllable count in words, letter length, and measures of emotionality and pleasantness of words. These are very important issues that affect performance on information processing tasks. Significant discrepancies between stimuli and their specifications in groups of words may significantly alter response time to them on a given task. For example, Baron & Strawson (1976) showed that high frequency words (i.e., more commonly used words) are processed quicker than low frequency words, and words that differ on certain attributes (e.g., number of syllables and letters) are processed quicker if they have fewer letters and fewer syllables compared to words with more letters and syllables (Taft, 1985). Differences in the inherent emotionality and pleasantness of stimuli also affect processing performance. It has been shown in some studies that groups of words rated higher in emotionality and pleasantness are processed differently (slower or faster depending on the task) compared to words with low ratings of emotionality and pleasantness (Eysenck, 1991). Therefore, in the context of measuring automatic schematic functioning in depression, if significant discrepancies between groups of words used in implicit tasks are present, then this may significantly affect reaction speed and errors obtained, and may also introduce more controlled and explicit processing strategies

(Eysenck, 1991). Such discrepancies would make any inferences regarding the nature of “true” automatic processing difficult to interpret especially in the context of measuring the automatic nature of schemata.

Another important issue is the congruence between the stimuli and the hypothetical construct to be measured. If one is to assess negative self-schemata one presumably has to choose stimuli that reflect the schema structure and the etiological development of the structure. For example, Beck argues that negative self-schemas develop due to the result of negative early experiences revolving around themes of being incompetent or worthless (Alloy et al., 1999; Beck, 1967, 1976). Using generic negative and positive stimuli may not be congruent with a negative self-schema and automatic self-evaluations inherent in a self-schema’s etiological development (e.g., Gamar et al., 2001). Indeed, Parker et al. (2000) found that certain schematic themes (vulnerability to harm and lack of worth) are stable themes that may be implicated in individuals who are vulnerable to develop depression. Also, schematic themes of defectiveness and abandonment were significantly correlated with depression in a study that examined relationships between schemas and psychopathology (Stopa, Thorne, Waters, & Preston, 2001). Plus, schematic themes of abandonment related to early mother-child attachment and risk for later adult depressive symptomatology have been frequently observed (Bowlby, 1969, 1973, 1980; Claesson & Sohlberg, 2002; Pielage, Gerlsma, & Schaap, 2000). Therefore, using stimuli that are descriptive of depressive moods, rather than themes associated with schema development in depression, may only assess the automatic self-evaluation of how an individual associates him/herself with low mood states or be reflective of a present mood state, but may not be assessing negative self-schemata related to vulnerability to depression. As mentioned in Chapter 3, Alloy et al. (1999) found that individuals who were cognitively vulnerable to develop depression demonstrated greater processing of negative self-referent information and inferior processing of positive self-referent information. The information used in this study was depression related adjectives (e.g., incompetence, worthlessness) and positive adjectives (e.g., successful, loveable). There are doubts however if the tasks used in this study were purely assessing implicit processing. The processing tasks involved measuring the speed of participants’ judgments about whether certain words were self-descriptive or not, and the recall of the words, that were rated as either self-descriptive or not, in a recall task. Participants therefore had to make an effortful judgment to decide if an adjective was descriptive of them or not, and make an effortful judgment to try to remember the words in the recall task. Thus, effortful and controlled processes could have confounded the results.

However, one should be careful of assuming that schema function is directly related with a belief system, sets of attributions, or even themes related to etiological development of

schemas (e.g., Sheppard & Teasdale, 2004; Alloy et al., 1999; Rude et al., 2001). De Houwer (2002) argues that implicit tasks measuring automatic self-evaluation, for example, do not specifically measure beliefs or cognitive content of a structure per se (e.g., the belief of being defective), but rather measure the relative strength of associations (e.g., the concept of the self being negative generally). In other words, automatic self-evaluation tasks may only provide indirect evidence so one can make inferences of what these associations might mean with regard to probable self-reported beliefs. In essence, automatic tasks may only measure an individual's orientation to the world and to the self, but not a belief structure per se. This idea highlights the difference between the epistemological framework and an ontological framework as discussed in Chapter 2.

This fits in well with Rudman's argument that automatic and controlled self-evaluations stem from different sources and should essentially be seen as different constructs (Rudman, 2004). From an etiological standpoint, and theories regarding the development of schemas from a neuroscientific (Le Doux, 1998), psychoanalytical (Bucci, 2000), and cognitive standpoints (Beck, 1967, 1976), schemas are hypothesised to develop at an early age (e.g., infancy, childhood) where complex language and language based attributional styles have not yet developed. Indeed, language may not have developed at all. Thus negative self-schemata may initially develop at a sub-symbolic level before formal language development. Therefore, schemata may not be a set of attributions of beliefs. Rather, negative schemata associated with vulnerability to depression may be better conceptualised as a theoretical and embodied plan of action or unconscious habitual ways of being (Wheeler, 2006; Heidegger, 2001; Merleau-Ponty, 1962). From this perspective, it is only through controlled and conscious reflection that themes and beliefs then come into actuality.

4.4.2. Error rates versus reaction time

Very few researchers investigating schemata and vulnerability to depression, including Gemar et al. (2001) and De Raedt et al. (2006), address the issue of error rates as opposed to reaction times on implicit processing tasks. Indeed Greenwald et al., (1998) posit that error rates may be very good indicators of automatic self-evaluation and may be more sensitive to vulnerability to depression as response rates may be confounded by controlled or explicit processes (Eysenck, 1991). Bargh & Tota (1988) argue that response latencies may be inappropriate indices of efficient or automatic processing because multiple factors besides the activation of stored constructs may influence response speeds. They identified several factors that may be responsible for effects on response times. These were self-presentational strategies within the experimental situation (Ferguson, Rule, & Carlson, 1983), and the subject's degree of confidence in his or her judgment which affect reaction time. Latencies therefore may (in some contexts) reflect the contribution of both automatic and attentional

forces that operate simultaneously but independently (Logan, 1979; Posner & Snyder, 1975; Shiffrin & Schneider, 1977).

The problems associated with response times provide difficulties in the interpretation as to what degree are response times due to automatic processing. One is not able to ascertain to what extent response speed was due to the relatively automatic processing, and how much of it was due to the effects of more controlled and explicit processing strategies (Bargh & Tota, 1988). Therefore response times may be confounded with aspects of explicit or more controlled explicit processes and may not be a true reflection of automaticity. Kirsch & Lynn (1999) argue that non-volitional responses can be generated and altered by the expectancy of their occurrence (e.g., the response expectancy; Kirsch, 1985). Response expectancies are the anticipations of automatic subjective and behavioural responses to particular situations or stimuli. Their effects are regarded as a form of self-fulfilling prophecy. In effect they are predictions of an individual's experience and behaviour. Research suggests that response expectancies are important factors in the etiology and vulnerability to depressive disorders (Kirsch & Lynn, 1999).

To specifically relate this issue to performance on implicit processing tasks, an individual who is vulnerable to depression may anticipate (unconsciously) that his or her self will be associated with negative stimuli or attributes (Kirsch & Lynn, 1999). Therefore on a task measuring automatic self-evaluation, the individual who is vulnerable to depression may implicitly expect negative stimuli to be associated with the self and not expect positive stimuli to be associated with the self. He/she may therefore make more errors when positive stimuli are associated with the self (indicative of a negative automatic self-evaluation) and fewer errors when negative stimuli are associated with self. This therefore may be indicative that the person has a less positive implicit self-evaluation or more negative self-schemata. It may therefore be fruitful to investigate the role of errors and implicit self-evaluation in vulnerability to depression.

4.4.3. Problems of sampling

Many studies investigating schemata and depression can be questioned in relation to the nature of the sampling of participants and theoretical notion of depressive scarring (Sheppard & Teasdale, 2004; Lewinsohn et al., 1981). If one is to try to investigate negative self-schemas and vulnerability to depression, participants who have already experienced episodes of depression may produce some serious confounds with regard to claims of measuring vulnerability to depression. The scar theory suggests that the experience of an initial depressive episode is required before negative personal constructs are organised schematically. If this is the case it provides problems for studies investigating vulnerability to depression. If one is to truly investigate vulnerability to depression, one would preferably (on

theoretical grounds) need to use samples of individuals who had never suffered a depressive episode (Hammen et al., 1985), individuals who are regarded as being at high-risk to develop depression (e.g., trait depression; Zemore et al., 1990), currently depressed individuals and individuals successfully treated for clinical depression. Using this kind of systematic methodology may shed light on the nature and influence of negative self-schemata from the “scar” perspective and “vulnerability perspective. One would be able to ascertain if negative schemata develop or are present before an episode of depression or are present in those hypothetically at risk to develop depression, or develop as a result of a depressive episode. Further, one would be able to ascertain how negative schemata change as a result of successful treatment, if at all, from pharmacological and psychological based therapies.

4.5. Summary and future directions

There is strong support from research that schemata in depression are latent and only effect information processing in the presence of a low or depressed mood. However, problems with the methodology traditionally used in depression research to assess schemata may have prevented the measurement of schemata in the absence of a depressed or low mood. Several methodological issues were discussed, that if considered and adopted, may facilitate the measurement of schemata in the absence of depression or low mood. It was argued that the importance of measuring schemata at an implicit or automatic level may be crucial from a theoretical perspective of how schemata function and affect information processing. Further, the role of an implicit association of “the self” or self-evaluation might also be a fundamental consideration when attempting to measure schemata. Indeed, newly developed tasks like the IAT (Greenwald et al., 1998) and EAST (De Houwer, 2003) show promise in their potential to measure the role of self-evaluation and vulnerability to affective disorders (de Jong, 2000; De Raedt et al., 2006). A pragmatic approach to the choice of stimuli used in implicit tasks is also an important consideration. If one is to measure schemata in depression, perhaps the stimuli should be congruent to the hypothetical content within the schematic structure (Parker et al., 2000; Alloy et al., 1999). Furthermore, stimuli should not differ on crucial variables (e.g., word-length, emotionality, frequency). If stimuli differ on these variables it may affect the speed of processing and facilitate more controlled and explicit processing strategies (Baron & Strawson, 1976; Taft, 1985). It was also highlighted that few researchers use error rate data to assess schematic functioning in depression (e.g., De Raedt et al., 2006; Gemar et al., 2001). Bargh & Tota (1988) argue that error rate data may be a true reflection of automatic processes as controlled and explicit processes may confound reaction time data. Lastly, the issue of relying only on certain samples of individuals for research into schemata in depression (e.g., recovered depressed individuals) limits the inferences one can make pertaining to schemata and vulnerability to depression (Lewinsohn et al., 1981). Using a wide

of range of samples may provide a clearer picture of the nature of schematic functioning at different stages on the depressive continuum. The schema is a very useful heuristic concept. Considering these methodological issues in future research, not only might it facilitate and provide useful information as to the nature of schematic functioning in the absence of a depressed or low mood, but it may provide improved insight into the role of automatic cognition, and its interface with explicit and controlled cognition associated with depression, and how this relates to vulnerability to depression. In turn this would provide researchers with useful information to develop improved or new treatment approaches. It may also provide insight into the mechanisms of cognitive change in individuals who have been successfully treated for depression and provide clues as to why psychopharmacological treatments result in higher relapse rates compared to cognitive orientated therapies (Paykel et al., 1999). The following empirical chapters will now attempt to address these issues.

Chapter 5

General introduction to empirical chapters and Study 1: Automatic self-evaluation in an analogue sample of depressed individuals

5.1. General introduction

In response to the methodological problems of schema research addressed in Chapter 4, this chapter starts by giving a brief outline of the empirical chapters of this thesis. This empirical work is an effort to provide a more comprehensive analysis of negative self-schemata and their functioning in depression. This will be followed by a more detailed description of Study 1 and its aims and hypotheses.

The main facet of the cognitive model of depression is that depression is triggered by latent cognitive structures called negative self-schemata (Beck, 1967, 1976). Negative self-schemata are hypothesised to develop as a result of negative early childhood experiences. Schemata are hypothesised to be activated by a low or depressed mood. This activation subsequently biases information processing so that negative material is preferentially processed, which in turn maintains the depressed mood state (Beck, 1967, 1976; Teasdale & Barnard, 1993). However, evidence for negative-self schematic information processing in the absence of a depressed mood or without the aid of priming or negative mood inductions is not strong (e.g., Rude et al., 2001; Hedlund & Rude, 1995; Gemar et al., 2001). Beck (1967, 1976) argues that schemata can be active or latent and this may in part explain the failure to find information processing biases in the absence of a depressed or low mood. Nevertheless, the concept of a latent schema is inherently problematic because it makes the theory potentially falsifiable, as schemata can never be measured in the absence of a low or depressed mood. However, as pointed out in the previous chapter, there are several problems with conventional methods of measuring schemata in depression, which may account for the poor evidence of schematic activity in the absence of a depressed mood or as a result of priming/negative mood inductions. These problems relate to the use of self-report measures (Nisbett & Wilson, 1977), lack of a self-referent or self-evaluative aspect inherent in implicit information processing tasks (e.g., Hedlund & Rude, 1995), the incongruence between stimuli and the hypothetical content within the negative self-schema (Alloy et al., 1999), unbalanced stimuli that could produce response confounds (e.g., word-length or frequency; Eysenck, 1991), and a heavy reliance on response times, while ignored error rate data might provide important information with regard to vulnerability to depression and evidence of schematic activity (Bargh & Tota, 1988; Kirsch & Lynn, 1999). Also many researchers rely on certain samples of individuals for research into schemata in depression (e.g., individuals who have recovered from depression). This limits the inferences one can make pertaining to schemata and vulnerability to depression (Lewinsohn et al., 1981). Therefore, using a broad range of

samples may provide a clearer picture of the nature of schematic functioning at different stages on the depressive continuum. New implicit tasks like the IAT (Greenwald et al., 1998) and the EAST (De Houwer, 2003) as highlighted in Chapter 4, have shown promise in psychopathology research and may provide a way of effectively measuring schematic activity in the absence of a depressed mood. The research that has used these tasks has indicated that automatic self-evaluation may play an important role in depression and related affective disorders (Gemer et al., 2001; de Jong, 2000; Tanner et al., in press; De Raedt et al., 2006).

In an effort to redress these issues, the studies that follow in this and subsequent chapters attend to these methodological problems that may be responsible for the lack of evidence of schematic processing in the absence of a low or depressed mood. Study 1 in this present chapter and Study 2 in the following chapter have two main areas of focus. These are 1) firstly to develop the IAT and EAST tasks (for the subsequent studies); and 2) to investigate how analogue depression affects automatic self-evaluation, and if the IAT and EAST are effective in measuring automatic self-evaluation. Studies 3 & 4 focus on the effects of hypothetical vulnerability and non-vulnerability to depression (e.g., individuals who have high and low psychological and behavioural traits indicative of increased risk of developing clinical depression; Zemore et al., 1999) and the effects of mood and depressive symptomatology on automatic self-evaluation. Study 3 is particularly concerned with the effects of automatic self-evaluation in individuals classified as high-trait depressed and low-trait depressed, and if differences in automatic self-evaluation exist between these two different groups of individuals at the same time as controlling for depressive symptomatology. The aim of Study 4 is to investigate the effects of a negative mood induction on automatic self-evaluation in high-trait and low-trait depressed individuals. This is to ascertain whether automatic self-evaluation in high-trait depressed individuals becomes more negative as a result of an induced low mood, and if automatic self-evaluation is a more stable construct in low-trait depressed individuals and thus not affected by low mood. The aim of Study 5 is to investigate the effects of antidepressant treatment in clinically depressed individuals. This study was planned as research and has shown that antidepressant treatment may not be effective in reducing enduring vulnerability to depression. This is because antidepressant treatment is argued not to be effective in changing the negative self-schemata theoretically held responsible for triggering depression (Paykel et al., 1999; Beck, 1967, 1976). Thus, the aim of this study is to determine if enduring vulnerability to depression in clinically depressed individuals, who have been successfully treated with antidepressants, is measurable at a level of automatic self-evaluation.

5.1.1. Introduction to Study 1: Background, aims, and hypothesis

There is an emerging consensus that the experience of depression lies on a continuum, and that subthreshold or subsyndromal depression differs quantitatively rather

than qualitatively from major depression. In other words, there is a phenomenological continuity of the experience of depression (Cox, Enns, & Larson, 2001; Cox, Enns, Borger, & Parker, 1999; Akiskal, Judd, Gillin, & Lemmi, 1997). Therefore, the use of an analogue depressed sample in this study may provide useful information as to the nature of implicit and explicit cognition in depression, especially if the same methodology is used in conjunction with other samples of depressed individuals at other points on the depression continuum; i.e., individuals classified as trait depressed and recovered depressed in studies 3, 4, & 5 (Vredenburg, Flett, & Krames, 1993). Research has shown that the self-schemata of depressed people do not lack positive content. It has been argued that depressed individuals and individuals vulnerable to depression have mixed negative and positive self-schemata. However, the way in which the positive content is activated, processed, and organised might differ in depressed individuals compared to non-depressed individuals, or in individuals who are vulnerable to depression compared to non-vulnerable individuals (Dozois & Dobson, 2001; Greenberg & Alloy, 1989). Therefore, the use of the EAST and IAT tasks in this study may be able to shed light on the role of automatic self-evaluation in depression and role of positive and negative self-schemata in analogue depression, and how this compares to recovered or clinically depressed individuals (i.e., Study 5).

However, the research findings using the IAT and EAST have so far been mixed. As discussed in Chapter 4, the research findings obtained by Gemar et al. (2001) and De Raedt et al. (2006) are to a certain extent not in line with cognitive theories of depression. Gemar et al. used the IAT to examine mood related changes in implicit and explicit self-esteem in formerly and never depressed controls before and after a negative mood induction. After the mood induction, the formerly depressed individuals showed a more negative implicit self-esteem compared to the control group. However, on closer examination both the formerly and depressed and control group showed a stronger positive implicit self-evaluative bias before as well as after a negative mood induction. Moreover, a group of currently depressed individuals, who did not undergo the negative mood induction as part of their study, showed a similar positive self-evaluative bias compared to a group of recovered depressed and non-depressed control group. In De Raedt et al.'s study using the EAST, a group of currently depressed individuals showed a stronger positive self-evaluative bias compared to a group of non-depressed controls. On the IAT, the depressed individuals displayed a similar positive automatic self-evaluative bias compared to a group of non-depressed controls. The results from these two studies are not what one would expect in currently depressed individuals. One would expect, from the perspective of the cognitive model of depression, such individuals would exhibit a negative implicit self-evaluation or negative implicit information processing bias (Beck, 1967, 1976). However, as highlighted in Chapter 4, the research that has used the IAT (Gemar et al., 2001) and the EAST (De Raedt et al., 2006) failed to address the issue of

error rates as a valuable measure of automatic self-evaluation or schematic functioning. Both studies also failed to address the problems associated with stimuli selection and if the stimuli was congruent with the content within depressive schemata (Alloy et al., 1999), the effects of unbalanced stimuli (e.g., word frequency, emotionality, & pleasantness), and how these may affect the efficiency of information processing or biases that may occur. More specifically, De Raedt et al. used negative stimuli in their EAST task that seemed to be unrelated to depression (i.e., false, mean, hostile, boring, hateful).

Nevertheless, Gemar et al. and De Raedt et al.'s studies do support, to some degree, research findings that support the role of positive self-evaluation or positive schemata being somehow implicated in vulnerability to depression and the related affective disorders (de Jong, 2000; Lewinsohn, Mischel, Chaplin, & Barnton, 1980; Taylor & Brown, 1988). Indeed, it is argued that a more positive implicit self-evaluation compared to explicit self-evaluation is associated with an unstable explicit self-esteem (e.g., individuals vulnerable to depression), whereas a more positive explicit self-evaluation compared to a more negative implicit self-evaluation is related with a stable explicit self-evaluation; e.g., individuals not vulnerable to depression (Bosson et al., 2000). Alternatively, other recent research concerned with individuals suffering from related affective disorders using tasks like the IAT & EAST have implicated a weak positive self-evaluative bias in social anxiety, and it is argued that such a weak bias may also play a role in the vulnerability to develop depressive disorders (de Jong, 2000; Tanner et al., in press). Therefore, the role of implicit or automatic self-evaluation in depression, vulnerability to depression, and related affective disorders is unclear. Consequently, more research needs to be done to see what role automatic self-evaluation plays in depression and how the EAST and IAT can be developed to measure automatic self-evaluation. At the same time, the role of errors and the careful selection of stimuli used in these implicit tasks must be considered if one is to attempt to measure schemata in depression.

The aims of this study were to 1) investigate automatic self-evaluative information processing in an analogue sample of depressed individuals who have significantly higher symptoms of depressive symptomatology (as measured by the Beck Depression Inventory-II; BDI-II; Beck, Steer, & Brown, 1996) compared to a group of low BDI scorers; 2) to ascertain whether the IAT and EAST are useful instruments to advance our understanding of the role of schemas in depression; and 3) to combine these two automatic self-evaluative processing tasks with traditional self-report measures of depression and compare the two approaches. In other words, this aim was to investigate the similarities and differences between implicit and explicit cognition in depression to see how self-report instruments that measure the products, processes, and structure of depressive cognition compare to more indirect implicit measures.

The hypothesis for the EAST¹ task was, following the cognitive model of depression (Beck, 1967, 1976), that a high scoring BDI group would be quicker and make fewer errors (compared to a low scoring BDI group) when presented with negative words that had to be classified with the same key that had to be used to classify self-words. This would indicate that the high BDI group had a stronger negative automatic self-evaluative bias. Conversely, it was hypothesised that the low BDI group would be quicker and make fewer errors (compared to the high BDI group) when presented with positive words that had to be classified with the same key that had to be used to classify self-words. This would indicate that the low BDI group had a stronger positive automatic self-evaluative bias.

With regard to the IAT², it was hypothesised that the low BDI group would be quicker and make fewer errors (compared to the high BDI group) on the compatible block of the IAT when self-words and positive words had to be classified with the same key, compared to the incompatible test block when self-words and negative words had to be classified with the same key. This would indicate that the low BDI group had a stronger positive automatic self-evaluative bias.

On the self-report measures, it was predicted that individuals with higher self-rated symptoms of depression (the high BDI group) would score higher on self-report questionnaires measuring the cognitive contents, processes, and structure associated with depression compared to individuals with lower self-rated symptoms of depression (the low BDI group).

A subsidiary aim was to investigate how different types of negative stimuli affect information processing. As discussed in Chapter 4, if one is to assess negative self-schemata one presumably has to choose stimuli that reflect the schema structure and etiological development of the structure (Alloy et al., 1999). Therefore, two types of negative stimuli were chosen to be used in this study that were descriptive of negative or low mood, and negative schematic themes associated with themes of abandonment and defectiveness. Parker, Gladstone et al. (2000) found that certain schematic themes (vulnerable to harm, and lack of worth) are stable themes that may be implicated in individuals who are vulnerable to depression. Alloy et al. (1999) also found that individuals at risk of developing depression showed preferential self-referent processing of negative depression-relevant material involving themes of incompetence, worthlessness, and low motivation on implicit processing tasks compared to depression-relevant words related to themes of low mood. Similarly, schematic themes of defectiveness and abandonment were significantly correlated with depression in a study that examined relationships between schemata and psychopathology

¹ See Chapter 4 and method section of this chapter for a full explanation of the EAST task.

² See Chapter 4 and method section of this chapter for a full explanation of the IAT task.

(Stopa et al., 2001). Lastly, it has been frequently observed that schematic themes of abandonment related to early mother-child attachment is linked to a greater risk for the development of later adult depression (Bowlby, 1969, 1973, 1980; Claesson & Sohlberg, 2002; Pielage, Gerlsma, & Schaap, 2000). No specific hypothesis was made as it was expected that the high BDI group would have a self-evaluative bias to both types of negative material as they would have significantly elevated symptoms of depression and, in accordance with other research, would have a bias to all material related to depression (Alloy et al., 1999.). However, the two types of stimuli used in this study were for development purposes. This was to test for a specific schema content hypothesis in later studies contained in this thesis investigating trait depression, individuals successfully treated with antidepressants, and associated hypothetical vulnerability to depression or relapse.

5.2. Method

Participants

Three-hundred students (undergraduates and post-graduates) were screened using the Beck Depression Inventory II (Beck et al., 1996). Sixty participants took part in the experiment. Thirty participants with scores between 2-13 (minimal depression on the BDI-II) on the BDI comprised the low BDI group (mean=5.95, SD=1.98). Individuals who scored below 2 were excluded as scores of 0 or 1 may represent other forms of psychopathology being present (e.g., psychopathy, hypomania) rather than, or in addition to, the absence of depression (Hammen, 1980). Thirty participants with scores between 14 and 28 (mild and moderate depression on the BDI-II) formed the high BDI group (mean=19.83, SD=3.9). The two groups differed significantly on BDI scores, $t(58) = 17.76, p < .001$. The mean age of the low BDI group was 24.16 years (S.D. = 2.92) and consisted of 12 males, and 18 females. The mean age of the high BDI group was 22.93 years (S.D. = 3.6) and consisted of 14 males and 16 females. The two groups did not differ on age, $t(58) = 1.46, p > .05$, or gender, $\chi^2(1) = .271, p > .05$. Exclusion criteria included the use of any medication that would interfere with motor reactions, previous history of depression, colour blindness or eyesight problems, and English as a second language.

Materials

Self-report measures (See Appendix III for questionnaires). A range of questionnaires was used to encompass the cognitive content, processes, and structure associated with depression to investigate how they compared to the implicit measures. The BDI-II (Beck, et al., 1996) was used to select the high and low depressive symptom group. The Beck Anxiety Inventory (Beck, 1980) was used to evaluate differences in anxiety between the groups, as anxiety and depression are commonly co-morbid. The Young Schema Questionnaire (Young & Brown, 1994) and the DAS (Weismann & Beck, 1978) were used to measure early maladaptive schemas, and dysfunctional assumptions respectively. The Evaluative Beliefs

Scale was included as Chadwick, Tower, & Dagnan (1999) argue that it measures a class of pure beliefs specifically related to depression. The Automatic Thoughts Questionnaire (Hollon & Kendall, 1980) was used to measure a class of negative beliefs (negative automatic thoughts) as these are known to be elevated in low mood states and highly associated with the cognitive content characteristic of depression (Beck, 1967, 1976).

The Beck Depression Inventory-II (BDI-II; Beck et al., 1996). The BDI-II is a 21 item self-report inventory designed to measure the presence and severity of depressive symptomatology in accordance with the DSM-IV. This instrument has been accepted as one of the better self-report measures of depression and has been used extensively in both research and practice (Beck, Steer, & Garbin, 1988; Dozois, Dobson & Ahnberg, 1998). Each item is rated on a scale ranging from 0-3; total scores thus range from 0-63. Scores from 0-13 indicate minimal depression, 14-19 indicate mild depression, 20-28 indicate moderate depression, and 29-63 indicate severe depression. The BDI-II is a revision of earlier versions of the BDI (Beck & Steer, 1987). The internal consistency and factor structure of the BDI-II has received ample support among outpatient samples of adults and adolescents (coefficient alphas typically at or above .90; e.g., Beck et al., 1996, Steer, Ball, Ranieri, & Beck, 1997, 1999; Steer, Kumar, Ranieri, & Beck, 1998; Steer, Rissmiller, & Beck, 2000) and college student samples (Beck et al., 1996), indicating that the BDI-II is a very reliable and well validated as an index of depressive symptom severity.

The Beck Anxiety Inventory (BAI; Beck, 1980). The BAI is a 21 item self-report scale that measures symptoms of anxiety. Each item is rated on a 4-point scale from 0 (not at all) to 3 (severely). The BAI has good internal consistency reliability of (.92), a correlation of .51 with the Hamilton Anxiety Rating Scale and of .48 with the BDI, and good test retest reliability (Beck, Epstein, Brown & Steer, 1988). In a large sample investigation of college students, Reynolds (1991b) reported an internal consistency reliability coefficient of .89 for the BAI and a correlation of .53 with the BDI.

The Dysfunctional Attitudes Scale (DAS; Weismann & Beck, 1978). This study used the 40-item DAS A form. The DAS was developed to assess dysfunctional assumptions such as "If I fail at my work than I am a failure as a person" that are thought to be characteristic of depressed individuals. Items are rated on a 7-point Likert scale where 1 represents "totally agree" and 7 represents "totally disagree". Internal consistency of the DAS is high (DAS-A 0.898; Power, Katz, McGuffin, Duggan, Lam, & Beck, 1994). Correlations between the DAS parallel forms and total test score range from .84 to .97. The DAS has good test-retest reliability of .73 over a six-week period (Oliver and Baumgart, 1985). The DAS has good discriminant validity as 73% of subjects scoring high on the DAS met an Independent Research Diagnostic Criteria for a diagnosis of clinical depression, compared to 36% of the

low scorers on the DAS (Nelson, Stern, Cicchetti, 1992). The DAS is also sensitive to the effects of cognitive therapy (Beck, Brown, Steer & Weissman, 1991).

Young Schema Questionnaire (YSQ short version; Young & Brown, 1994). The YSQ comprises 75-items and measures 15 maladaptive schemas. Each item is rated on a 6-point scale ranging from 1 (completely untrue of me) to 6 (describes me perfectly). The fifteen schemas measured by the YSQ (short version) include: emotional deprivation, abandonment, mistrust/abuse, social isolation, defectiveness, failure, dependence/incompetence, vulnerability to harm, enmeshment, subjugation, self-sacrifice, emotional inhibition, unrelenting standards, entitlement, and insufficient self-control/self-discipline. Preliminary validation studies show the YSQ has good convergent validity (Schmidt, Joiner, Young & Telch, 1995). The factor structure is in accordance with Young's description (Schmidt et al., 1995) and the YSQ also discriminates well between patients with Axis I and Axis II disorders (Calvete, Estevez, de Arroyabe, & Ruiz, 2005; Stopa et al., 2001).

The Evaluative Beliefs Questionnaire (EBS; Chadwick, Trower, & Dagnan, 1999). The EBS is an 18-item questionnaire with three sub-scales designed to measure evaluative beliefs. The three subscales comprise judgements about the self (i.e., I am a failure), and judgements about how one thinks other people judge one (i.e., other people think I am a failure), and judgements about how one thinks about others (i.e., other people are total failures). Items are rated on a 5-point scale ranging from 3 (agree strongly) to 0 (disagree slightly) and 0 (disagree strongly). The sub-scales have good internal reliability (self-self 0.90, other-self 0.92, and self-other 0.86). Good concurrent validity was also found when compared to the Hamilton Anxiety and Depression Scale, and it discriminated well between different forms of psychopathology such as anxiety and depression (Chadwick et al., 1999).

Automatic Thoughts Questionnaire (ATQ; Hollon & Kendall, 1980). The ATQ measures the frequency with which each of 30 negative automatic thoughts has been experienced during the past week on a 1 (not at all) to 5 (all the time) scale. The ATQ also measures the extent to which an individual believes these thoughts on the same five-point scale. Examples include "I don't think I can go on"..... "No one understands me"..... "It's just not worth it". The ATQ also discriminates extremely well between depressed people and non-depressed people (Blackburn, Jones et al., 1986), non-psychiatric medical patients, and remitted depressed patients (Harrell & Ryon, 1983).

The ATQ's psychometric properties have been evaluated in a number of studies. Internal consistency estimates (i.e. split-half and coefficient alpha) have been shown to be high (in the .96-.97 range) across a range of subjects (Dobson & Breiter, 1983; Harrell & Ryon, 1983; Hollon & Kendall, 1980), and correlates strongly with (i.e. around .63) with severity of depressive symptoms (Dobson & Breiter, 1983). The ATQ has a good reliability (.98; Harrell & Ryon, 1983).

Implicit Processing Tasks.

Extrinsic Affective Simon Task (EAST; De Houwer, 2003). The EAST is a computer task in which participants have to respond to words presented on a computer screen on the basis of a categorical attribute. In this study the categories were related to PERSON (SELF and OTHER) and COLOUR (BLUE & GREEN). The PERSON words were related to attributes of the participant (e.g., first name) or to another person (e.g., another person's first name). The category (COLOUR) comprised a set of words that included negative and positive words. The set of negative words and the set of the positive words were each presented once in the colour green and once in the colour blue (see Appendix IV and below for details of words used in the EAST). Participants were instructed to press left or right keys (on a response box) in response to self or other words that were presented in white, and to press the same keys in response to words that were either blue or green. The task allows the experimenter to measure participants' reaction times and errors to four combinations of words: positive words with the self-key, positive words with the other-key, negative words with self-key, and negative words with other-key.

The EAST comprises three phases. In the first phase (practice) 16 self-words and 16 other- words are presented randomly in 32 trials. Participants are instructed to press either the left or right key (depending on counterbalancing) when SELF words (e.g., participants surname) appear and the opposite handed key for OTHER words (e.g., another person's surname). In the second phase (practice) participants respond to words according to colour and press the right key for GREEN words and the left key for BLUE words. This phase comprised 32 trials; 8 negative words presented in blue and green (16 trials) and 8 positive words also presented in blue and green (16 trials). Key allocation for coloured words was counterbalanced across participants. The third phase is the test phase. Both types of words (SELF-OTHER and BLUE-GREEN) are presented in this phase, which comprises 64 trials (32 SELF-OTHER and 32 GREEN-BLUE). Phase three is essentially a combination of phases one and two.

All stimulus words were presented on a black background. SELF and OTHER words appeared in white. GREEN words used the values (red, 0) (blue, 46) and (green, 38). The BLUE words used the values (red, 0) (green, 38) and (blue, 46). All words were presented in uppercase lettering, in font size 60, and presented in the centre of the computer screen. There was a 2000 milliseconds stimulus interval between a participant's response to a stimulus and the presentation of the next stimulus. Participants were informed that a large red X would appear in the centre of the screen for 500 milliseconds if they made an incorrect response to a stimulus (pressed the wrong key), after which the EAST would continue as normal. Participants were told to proceed as quickly and accurately as possible when presented with a stimulus. All responses below 300ms and above 3000ms were recoded as 300ms and 3000ms

respectively in accordance with previous research (Greenwald et al., 1998), and responses that were inaccurate (e.g., pressing the wrong key in response to a stimulus) were classified as errors.

The EAST task was run on a computer using Superlab experimental laboratory software with a RB-400 response box (Cedrus corporation), which is compatible with a standard PC with Microsoft windows.

Implicit Association Test (IAT; Greenwald et al., 1998). The IAT is another computer task in which participants have to respond to two different categories of words presented on a computer screen on the basis of a categorical attribute and in this study the two categories of words used were person (SELF & OTHER) and valence (NEGATIVE & POSITIVE). (See Appendix IV for details of words used in the IAT in this study.) This task allows the experimenter to measure participants' self-evaluative bias on one set of test blocks when words related to the self are associated with negative words (by being allocated the same key), and when words related to another person are associated with positive words, compared to another set of test blocks when words related to the self are associated with positive words, and words related to another person are associated with negative words. Self-evaluation is measured by comparing mean reaction times and error rates on the compatible block (self with positive words and other with negative words) to the incompatible block (self with negative words and other with positive words).

The IAT consisted of seven blocks. It commenced with a practice block (block 1) of 32 trials where target concept words (words related to self or to other people) had to be correctly categorised by pressing one of two keys on a response box (e.g., right key for self words, left key for words related to other). Four words related to self were presented randomly four times each, as were four words related to other. The second practice block (block 2) involved 32 trials where attribute concept words (negative or positive words) had to be correctly categorised by pressing one of two keys (left or right). There were 8 negative words, and 8 positive words each of which were randomly presented twice. Following the two practice blocks, two test blocks (blocks 3 & 4) followed. These two blocks each had 32 trials where target concepts (4 self words x 2, and 4 words related to other x 2; 16 trials total) and attribute concepts (8 negative words x 1, and 8 positive words x 1; 16 trials total) were presented in a random order. Following the two test blocks a practice block (block 5) of 32 trials with target concepts was presented using a new key assignment - the right and left response keys assigned for targets concepts in block 2 (e.g., right key-self words, & left key-words related to other) was reversed. This practice block was followed by two further test blocks (block 6 & 7) of 32 trials each, comprising randomly presented 16 target concept trials, and 16 attribute concept trials, as specified in blocks 3 and 4.

The key assignments (left or right) for categorising target concepts and attribute concepts were counterbalanced across participants. Compatibility was also counterbalanced across participants i.e. half the participants were given the compatible blocks 3 & 4 first and the incompatible blocks 6 & 7 second. The other half of the participants were given the incompatible blocks first followed by the compatible blocks.

All stimulus words were presented in white on a black background in the centre of the computer screen. During each block of trials, the categories assigned to the left and right key were designated by labels in the top left or right hand of the computer screen (e.g., SELF, OTHER, POSITIVE, NEGATIVE). These labels would appear 500ms after the presentation of a word in the center of the computer screen and disappear after the participant had made a correct response. All words were presented in uppercase lettering, and in font size 60. There was a 2000 milliseconds stimulus interval between a participant's response to a stimulus and the presentation of the next stimulus. Participants were informed that a large red X would appear in the centre of the screen for 500 milliseconds if they made an incorrect response to a stimulus (pressed the wrong key), after which the IAT would continue as normal. Participants were told to proceed as quickly and accurately as possible when presented with a stimulus. Again, all responses below 300ms and above 3000ms were recoded as 300ms and 3000ms respectively in accordance with previous research (Greenwald et al., 1998), and responses which were inaccurate (pressing the wrong key) were classified as errors.

The IAT task was run on a computer using Superlab experimental laboratory software with a RB-400 response box (Cedrus corporation), which is compatible with a standard PC with Microsoft windows.

Stimulus words used in the EAST and selection procedure. There were 8 self-words and 8 words related to another person used in the EAST. Self-words comprised first name, second name, hometown, place of birth, month of birth, subject studied, nationality, and region of birth of each participant. This information was taken from each participant at the time of the experiment and programmed into the EAST computer program. Words related to another person described a hypothetical person and included the same 8 items of information as self-words. The words related to another person were changed if there were any matches with self-words of any participant.

The negative and positive words that comprised the COLOUR category used in the EAST comprised three different types of words. The negative words were made up of words descriptive of a negative mood and of negative schematic themes. The schema words were intended to reflect the abandonment and defective schematic themes in Young's (1990) typology. Two types of negative words were chosen, to investigate as one aim of this thesis, a specific schema content hypothesis (e.g., Alloy et al., 1999), and to see how analogue depressed individuals responded to words reflective of a depressed mood, and words

hypothetically related to the etiological development of schemata in depression. Themes of defectiveness (Alloy et al., 1999; Parker, Gladstone, Mitchell, Wilhelm, & Roy, 2000) and abandonment (Bowlby, 1969, 1973, 1980; Claesson & Sohlberg, 2002; Pielage, Gerlsma, & Schaap, 2000) were chosen as they have been implicated as schematic themes associated with vulnerability to depression. The positive words were made up of words intended to be descriptive of positive concepts. Initially, 30 positive words, 30 low mood words, and 40 schema words (20 abandonment, 20 defectiveness) were collated by the experimenter to be subjected to independent rating to ascertain how well the words fitted their respective category (i.e., positive, low mood etc). Ten experienced clinical psychologists rated the negative mood and schema words on a 1 (not applicable) to 10 (very applicable) scale to assess how representative each word was of its respective category. All groups of words were rated on a 10-point scale for emotionality and pleasantness (1=not very pleasant or not very emotional, 10=very emotional or very pleasant), by 20 post-graduate students. Low mood words and schema words were selected if they had a mean applicability rating (to the schema or low mood categories) of at least 7 (by the clinical psychologists), a mean of 3 or lower for pleasantness, and 7 or more for emotionality (from the post-graduates' ratings). Positive words were selected if they had a mean score of 7 or more for emotionality and pleasantness (from the post-graduates' ratings). After rating, 4 schema words (defective, unworthy, insecure, abandonment), 4 low mood words (misery, desolate, despair, pessimistic), and 8 positive words (enchancing, exotic, humorous, miracle, victory, adorable, courageous, ecstatic), were selected for use in the implicit tasks.

The three categories of words (positive words, negative schema words, low mood words) did not differ on number of letters, number of syllables, word frequency, or emotionality, but did significantly differ on pleasantness. The positive words were rated significantly more pleasant than the negative schema and low mood words. However, the ratings of the negative schema and low mood words did not significantly differ on pleasantness. The type of word (e.g., noun, adjective verb) did not differ between the three groups of words. See Appendix IV for word lists and statistical details of the attributes of words used in Study 1.

Stimulus words used in conjunction with the IAT. The same groups of negative and positive words used in the EAST were also used in the IAT-see Appendix IV. However, the negative and positive words were presented in white and had to be classified on valence unlike the EAST, where negative and positive words had to be classified on the basis of colour. The words that comprised the SELF category of words in the IAT were: ME, MINE, I, and the first name of the participant. The words that comprised the OTHER category were: THEY, THEM, HIS, and BRUCE (this was amended if the participant shared this name).

Procedure

Participants were screened with the BDI-II. Participants who met the criteria for either the high or low BDI groups were invited to participate in the next stage of the experiment and given an information sheet and a consent form to sign. Participants completed the IAT followed by the EAST³. The instructions told participants to classify words in the PERSON category (related to self or other) and in the COLOUR category (blue or green) by pressing the right or left hand keys on a response box (in the case of the EAST), and by PERSON and VALENCE (negative or positive in the case of the IAT). Testing took place in a small well lit and noise proofed cubicle with no outside light. Participants sat approximately 12-18 inches from the computer screen and held the response box on their laps. Participants were left on their own to complete the EAST and IAT. Next, participants completed the self-report questionnaires. At the end of the experiment, participants were debriefed and paid £3 or given course credits. The whole procedure took approximately one hour for each participant to complete.

5.3. Results

Self-report measures

Table 1 shows the scores of the two groups on the self-report measures. The high BDI group scored significantly higher than the low BDI group on the ATQ, the DAS, and on the BAI. Using a Bonferroni adjusted p value of .017 (.05/3) to correct for multiple comparisons, the high BDI group scored significantly higher on the self-self and other-self subscales of the EBS. Scores on the EBS self-other subscale did not significantly differ between the groups. This result is consistent Chadwick et al.'s (1999) finding that depressed and non-depressed individuals do not differ in their judgments about other people although they do differ in their views of self and their beliefs about judgments made about them by other people. The high BDI group scored significantly higher than the low BDI group on the emotional deprivation, abandonment, mistrust/abuse, social isolation, defectiveness, failure, dependency, vulnerability to harm, subjugation and self-sacrifice subscales of the YSQ using a Bonferroni adjusted p value of .003 (.05/15) to correct for multiple comparisons. The enmeshment ($p = .004$), emotional inhibition ($p = .007$), and entitlement subscales ($p = .007$) just failed to reach significance using this stringent criterion. The unrelenting standards and insufficient self-control subscales did not differ between the two groups.

³ Before the EAST and IAT tasks all participants completed a dichotic listening task as part of a separate research project conducted by the author.

Table 1

Means and Standard Deviations on Self-Report Measures by the Low and High BDI Group

Questionnaire	low BDI group		high BDI group		t result	p value
	mean	SD	mean	SD		
DAS	124.5	25.79	160.33	19.25	6.09	<.001
ATQ	37.26	12.49	62.2	15.58	6.83	<.001
BAI	27.6	05.5	36.13	6.76	5.35	<.001
EBS: self-self	0.02	0.10	0.61	0.56	5.51	<.001*
EBS: self-other	0.08	0.17	0.14	0.15	1.43	.16
EBS other-self	0.08	0.17	0.46	0.43	4.49	<.001*
YSQ subscales:						
Abandonment	1.84	0.76	3.2	1.28	5.24	<.001**
Emotional deprivation	1.72	0.90	3.58	0.95	7.72	<.001**
Mistrust/abuse	1.94	0.77	3.5	0.82	7.53	<.001**
Social isolation	1.72	0.93	3.4	0.94	7.27	<.001**
Defectiveness/shame	1.33	0.42	2.98	1.10	7.68	<.001**
Failure	1.47	0.55	3.04	1.10	6.91	<.001**
Dependence	1.35	0.33	2.22	0.64	6.57	<.001**
Vulnerability to harm	1.5	0.60	2.69	1.09	5.17	<.001**
Enmeshment	1.42	0.62	1.99	0.85	2.97	.004
Subjugation	1.48	0.46	2.81	0.93	6.99	<.001**
Self-sacrifice	2.85	0.87	3.95	1.37	3.71	<.001**
Emotional inhibition	1.79	0.66	2.53	1.28	2.83	.007
Unrelenting standards	3.24	1.43	3.60	0.88	1.17	.247
Entitlement	1.79	0.66	2.53	1.28	2.83	.007
Insufficient self-control	2.33	0.80	2.94	1.36	2.10	.40

* Statistically significant using Bonferroni corrected *p* value of .017** Statistically significant using Bonferroni corrected *p* value of .003

Analysis of the EAST

The EAST produced two types of data: reaction times and error rates. Each of these is presented separately. Reaction times were log-transformed to normalise extreme scores in accordance with previous research (Greenwald et al., 1998). The following set of EAST analyses focuses on the differences of positive and negative words on self-evaluation first (schema and low mood combined), and then secondly an analysis investigating the differences between schema words and low mood words on self-evaluation. The EAST analyses used a 2 (word-type: negative and positive or schema and low mood words) x 2 (person: key assigned with self or other) x 2 (group) repeated measures ANOVA's, the first two factors being within subjects factors, the latter a between subjects factor. An alpha level of .05 was used for all initial ANOVA's. The hypothesis was that the high BDI group would have a stable and weaker positive automatic self-evaluative bias by virtue of having slower reaction times and more errors when positive words were associated with the self (key) compared to the low BDI group. Alternatively, this could be shown by faster reaction times and fewer error rates when negative words were associated with the self (key) by the high BDI compared to the low BDI group. Unless reported in the results, non-significant results and minor effects and interactions from the ANOVA's from the EAST and IAT analyses are presented in Appendix V. Mean reaction times in milliseconds (untransformed) and error rates from the EAST are presented below in Table 2.

Table 2

Mean Reaction Times (ms), Error Rates, and Standard Deviations on the EAST for the High and Low BDI Group*

	low BDI group		high BDI group	
	reaction time	errors	reaction time	errors
Positive				
words with self	681.8 (1.86.14)	0.63 (0.85)	697.04 (164.10)	1.23(1.38)
Positive				
words with other	678.32 (151.33)	0.67 (1.02)	657.93 (185.87)	0.73 (0.90)
Low mood				
words with self	675.20 (180.05)	0.63 (0.89)	605.99 (176.09)	0.20 (0.40)
Low mood				
words with other	681.55 (186.7)	0.40 (0.77)	679.87 (181.18)	0.63 (0.67)
Schema				
words with self	681.95 (163.6)	0.43 (0.72)	582.61(147.49)	0.43 (0.82)
Schema				
words with other	647.61 (110.3)	0.43 (0.67)	687.42 (221.88)	0.60 (0.77)
**Negative				
words with self	678.95 (148.38)	1.37 (1.32)	593.83 (155.14)	1.07 (1.14)
**Negative				
words with other	666.27 (129.82)	1.33 (1.37)	682.54 (181.30)	1.63 (1.34)

* Standard deviations in brackets, ** negative words combination of schema and low mood words

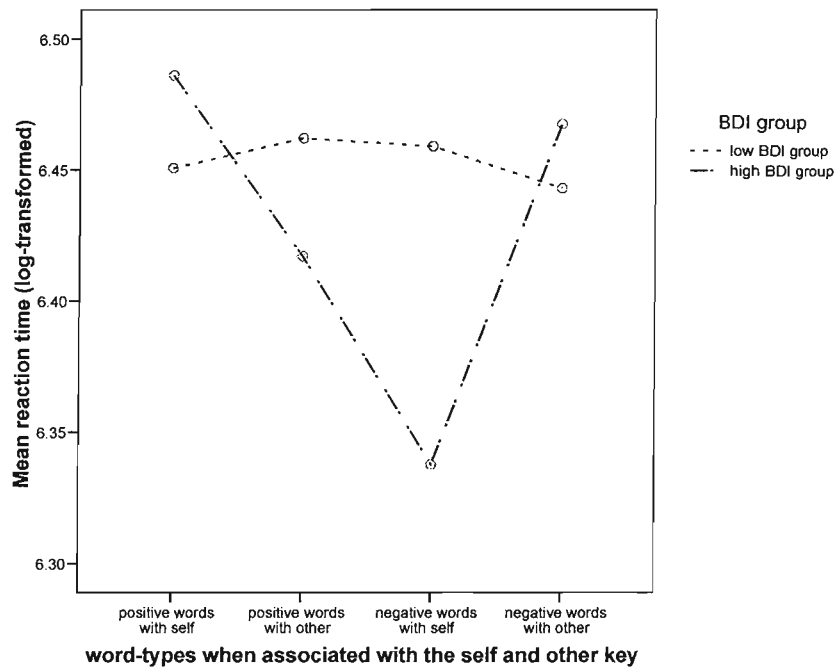
EAST reaction time analysis

There was a significant word-type by person by group interaction, $F(1, 58) = 13.46$, $p < .001$, which is illustrated in Figure 1 (overleaf), and there was also a significant word-type by person interaction, $F(1, 58) = 7.74$, $p < .01$. In order to explore this 3-way interaction,

independent t-tests and paired t-tests within groups were carried out using a corrected Bonferroni p value of .012 (.05/4). There was trend for the high BDI group to respond more quickly when negative words were associated with the self (key), $t(58)=2.43$, $p=.018$, although this just failed to reach significance using this stringent criteria. A paired t-test analysis with a corrected p value of .008 (.05/6) showed that the high BDI group responded more quickly when negative words were paired with self than when positive words were paired with self, $t(29)=4.49$, $p<.001$. The high BDI group was also faster when positive words were associated with other compared to when negative words were associated with self, $t(29)=3.37$, $p<.001$, but not when positive words were associated with other compared to when negative words were associated with other, $t(29)=2.49$, $p=.019$. Finally, the high BDI group was faster to respond when negative words were associated with self compared to when negative words were associated with other, $t(29)=4.5$, $p<.001$. There were no significant differences within the low BDI group (highest $p=.60$). This reaction time analysis confirms the hypothesis that the high BDI group would have a weaker positive automatic self-evaluative bias compared to the low BDI group.

In order to investigate whether the two groups responded preferentially to different types of negative stimuli (schema and mood related words), the latter analysis was repeated but in this case the within subject factor of word-type contained schema and low mood words only. There was a significant person by group interaction, $F(1, 58)=10.41$, $p<.01$, but no interaction of word-type by person by group, $F(1, 58)=1.03$, $p>.05$. Post-hoc paired t-test analyses using a corrected p value of .025 (.05/2) showed that the high BDI group was faster to respond to both types of negative words when they were associated with self than when they were associated with other, $t(29)=4.52$, $p<.001$. Thus, automatic self-evaluation in the high BDI group was not affected by different types of negative stimuli.

Figure 1. Reaction time word-type by person by group interaction on the EAST.

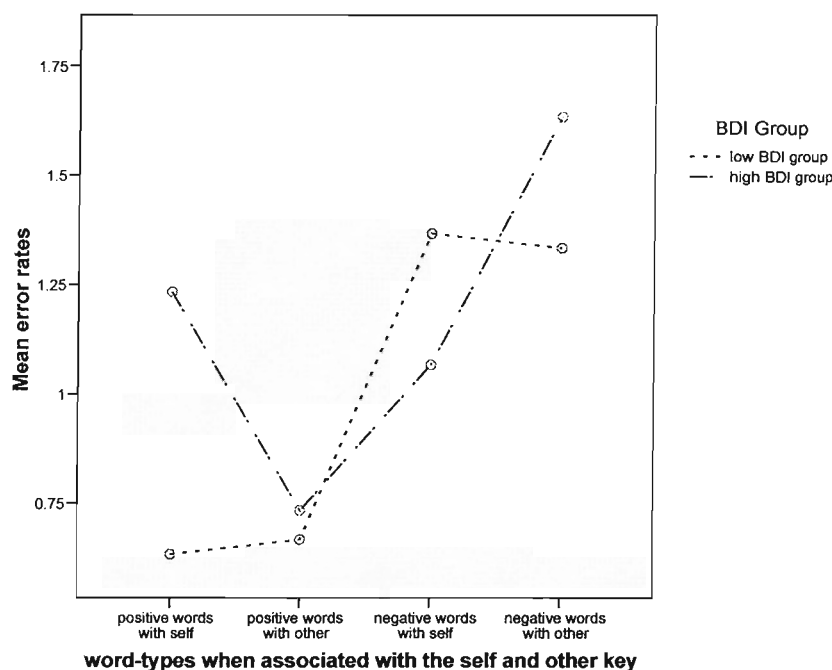


EAST error rate analysis

Errors are assumed to result from an incompatibility between stimulus and response and are recorded when a participant presses the incorrect key in response to a stimulus (Greenwald et al., 1998). The first ANOVA investigated the difference between positive words and negative words (schema and low mood words combined). There was a significant word-type by person by group interaction, $F(1, 58) = 5.65, p < .05$, which is illustrated in Figure 2 below. There was also a significant person by word-type interaction, $F(1, 58) = 4.40, p < .05$, and a main effect of word-type, $F(1, 58) = 28.89, p < .001$. Independent t-tests using a corrected p value of .012 (.05/4) revealed a non-significant result for the high BDI group to make more errors than the low BDI group when positive words were associated with self, $t(58) = 2.03, p = .047$. Within the groups, paired t-tests showed that the high BDI group made more errors when negative words were associated with other compared to when positive words were associated with other, $t(29) = 4.16, p < .001$. The low BDI group made more errors when negative words were associated with self than when positive words were associated with self, $t(29) = 3.43, p < .01$. The low BDI group also made more errors when negative words were associated with other compared to when positive words were associated with other, $t(29) = 3.44, p < .01$, and made more errors when negative words were associated with the self compared to when positive words were associated with other, $t(29) = 3.34, p < .01$. This error

rate analyses confirms the hypothesis that the high BDI group would have a weaker positive automatic self-evaluative bias compared to the low BDI group.

Figure 2. Error rate word-type by person by group interaction on the EAST.



In order to investigate whether there were any differences in the way the two groups responded to schema and mood related word, the ANOVA was repeated but in this case word-type contained schema and low mood words only. In accordance with the hypothesis there was no 3-way interaction of word-type by person by group, $F(1, 58) = 1.52, p > .05$, but there was a significant person by group interaction, $F(1, 58) = 6.13, p < .05$. Paired t-test analysis using a corrected p value of .025 (.05/2) showed a marginally non-significant result in that the high BDI group made more errors in response to both types of negative words when they were paired with other than when they were paired with self, $t(29) = 2.34, p = .026$, again showing automatic self-evaluation in the high BDI group was not affected by different types of negative stimuli. There were no significant differences within the low BDI group.

Analysis of the IAT

Like the EAST, the IAT produced two types of data: reaction time and error rates. Again, each of these were analysed separately. Reaction times were log-transformed to normalise extreme scores in accordance with previous research (Greenwald et al., 1998). The predictions for the IAT, on both the reaction time and error analyses, were that the high BDI group would show a weaker positive automatic self-evaluative bias compared to the low BDI group. This would be shown by overall slower reaction times and increased error rates on the compatible blocks (self associated with positive words and other associated with negative

words) relative to the incompatible blocks (self associated with negative words and other associated with positive words) of the IAT. Thus, the IAT is a measure of the relative positivity and negativity assigned to the self and to other people. Statistical analysis for each IAT involved a 2 (compatibility) x 2 (group) repeated measures ANOVA, compatibility being a within subjects factor and group a between subject factor. An alpha level of .05 was used for all ANOVA's. A separation of low mood and schema words was not carried out in these analyses, as the IAT does not lend itself to this type of analysis (e.g., Greenwald et al., 1998). See Table 3 for mean untransformed reaction times and error rates from the IAT.

Table 3

Mean Reaction Times (ms), Error Rates, Standard Deviations on the IAT for the High and Low BDI Group*

	low BDI group		high BDI group	
	reaction time	errors	reaction time	errors
Compatible				
phase: self				
with	758.67 (184.85)	3.23 (2.76)	770.29 (272.90)	4.03 (4.43)
positive/other				
with negative				
Incompatible				
phase: self				
with	793.77 (235.63)	3.33 (3.68)	728.44 (174.90)	3.93 (2.99)
negative/other				
with positive				

* Standard deviations in brackets.

IAT-Analysis of reaction times

There was no main effect of compatibility, $F(1, 58) = .008, p > .05$, and no interaction of compatibility by group, $F(1, 58) = .696, p > .05$. There were no other significant results (highest $p = .42$).

IAT-Analysis of error rates

Again there was no main effect of compatibility, $F(1, 58) = .000, p > .05$, and no interaction of compatibility by group, $F(1, 58) = .032, p > .05$. There were no other significant results (highest $p = .33$).

IAT analyses summary

These results are not in accord with the hypothesis. There was no difference in positive automatic self-evaluation between groups. Further, unlike previous research, no overall main effect of compatibility whereby participants generally are faster on the compatible blocks of the IAT relative to the incompatible blocks (Greenwald et al., 1998).

5.4. Discussion.

This study had two principal aims: 1) to investigate self-evaluative information processing in an analogue sample of depressed individuals, and to see whether the IAT and EAST are useful instruments in advancing our understanding of the role of schemas in depression; and 2) to combine these two automatic self-evaluative processing tasks with traditional self-report measures of depression to see how they compare in measuring depressive cognition. A subsidiary aim was to investigate how different types of negative stimuli affect information processing.

The results from the EAST suggested that, negative self-concept that is a characteristic of depression as reflected in self-report measures, is also reflected in the EAST. Both the reaction time and error data from the EAST supported the hypothesis that analogue depressed individuals (as shown by the BDI, ATQ, & BAI) have a weaker positive automatic self-evaluative bias compared to non-depressed individuals.

The low BDI group made more errors when negative words were associated with the self than when positive words were associated with the self. The low BDI group also made more errors when negative words were associated with other than when positive words were associated with other. This confirms previous research which indicates that non-depressed individuals show a self-serving bias and strong positive automatic self-evaluative bias on implicit tasks (e.g., Hedlund & Rude, 1995, Rude et al., 2001; Gerns et al., 2001). The high BDI group showed clear differences in their responses to negative and positive stimuli that are consistent with previous research (Hedlund & Rude, 1995). The high BDI group was faster to respond when negative words were associated with the self than when positive words were associated with the self, and faster when positive words were paired with other than when negative words were associated with self. The high BDI group was also faster when positive words were associated with other than when positive words were associated with self. Finally, the high BDI group was faster when negative words were associated with self than when negative words were associated with other, and made more errors when negative words were associated with other than when they were associated with self. This pattern of results suggests that negative words are associated with self-concept, whereas positive words are associated with other people. However, a more negative automatic self-evaluative bias (on errors or reaction time) was not observed in the high BDI group on the IAT. Conversely, the low BDI group surprisingly did not exhibit a strong positive automatic self-evaluative bias on

either the error or reaction time data. The IAT result is troubling in contrast to the results obtained with the EAST. A detailed analysis of the IAT task will be discussed in more detail later.

The results from the EAST provide evidence for the proposal that individuals high in self-rated depression have a weaker positive self-evaluative bias and a stronger positive other-evaluative bias compared to individuals with low self-rated depression. The self-report measures (DAS, YSQ, EBS) that were included in this study to provide information about explicit views of the self are consistent with this view. The high BDI group reported more dysfunctional assumptions than the low BDI group. They rated themselves higher on 10 out of the 15 schemas on the YSQ (these included abandonment and defectiveness) and they obtained higher scores on two of the EBS subscales that measure views of the self (self-self and other-self). In other words, the high BDI group saw themselves more negatively (and had more negative thoughts) compared to the low BDI group, and they also believed this is how other people see them. However, the high BDI group did not differ in their view of other people. The consistency of results across the two types of implicit and explicit measurement is in accord with the cognitive model of depression (Beck, 1967, 1976), whereby, high scores on self-report measures of depression are also linked to negatively biased information processing of the self (Alloy et al., 1999; Segal et al., 1999). This suggests that combining both implicit and explicit measures is a potentially useful way to approach schema research.

However, it has to be acknowledged that the similarity between the explicit and implicit measures found in this study is not in accord with several studies comparing implicit and explicit self-esteem or self-evaluation that have found dissociations between the two measures (Spalding & Hardin, 1999; Farnham, Greenwald & Banaji, 1999; de Jong, 2000). In other words, individuals may report a negative explicit self-esteem, but implicitly have a positive self-esteem. In one of the rare studies that have used the EAST with individuals suffering from clinical depression, De Raedt et al. (2006) found that currently depressed individuals had a stronger positive automatic self-evaluative bias even though they had elevated scores on self-report measures of depression. However, the negative stimuli used by De Raedt et al. in their EAST task was more descriptive of negative personality attributes (e.g., false, mean, hostile, boring, hateful), but not negative attributes related to depression per se, unlike the stimuli in this study. Indeed, there was no mention on what basis De Raedt et al. chose the stimuli for their study. Therefore, if one is to measure implicit self-esteem or self-evaluation, especially pertaining to psychopathology or vulnerability to depression, one has to seriously consider the construct one is attempting to measure. Theoretically, there may well be a discrepancy between an implicit and an explicit measure especially if there is no functional relation between the two measures. It is for this reason that stimuli selection, as discussed in detail in Chapter 4, must bear some relation to the phenomenon or schemata that

one is investigating. Overall, the research with the EAST and its relation to explicit attitudes and depression is in its infancy, but as the results in this study show, they are in accord with contemporary cognitive theories of depression (Segal, 1988; Segal et al., 1999; Teasdale & Barnard, 1993; Beck, 1967, 1976).

The subsidiary aim of this study was to investigate whether there was a difference in processing between schema related words and mood related words on the EAST. The results of the EAST, in accordance with the hypothesis, unsurprisingly showed no differences between the two types of words. It has been shown in other research that biases to material related to hypothetical schema content, but not all depression related material content, may only be observed in asymptomatic states (Alloy et al., 1999). On the other hand, biases to depression related material, irrespective of whether it is related to hypothetical schema content, are readily observable in individuals with high self-rated depression (Dobson & Shaw, 1987). However, in accordance with previous research, it seems that the schema content words (and low mood words) used in this study were suitable for use as stimuli assessing information processing biases in depression. To test for a specific schema content hypothesis, specific biases to certain kinds of stimuli, and vulnerability to depression (Alloy et al., 1999), it might be necessary to increase the number of words/trials for future studies to compare information processing biases to depressive mood related words and schema related words and the effects of mood. This issue was addressed in Study 3 and 5.

The results of the IAT in this study were disappointing. There was no evidence of a weaker positive or negative automatic self-evaluative bias in the high BDI group. Further, there was no evidence of a strong positive automatic self-evaluative bias in the low BDI group. Although the use of the IAT is relatively recent in depression research, the absence of a relatively positive automatic associative bias between self and positive concepts on the IAT, specifically with non-depressed individuals or individuals suffering from other affective disorders, is not consistent with previous research findings (Spalding & Hardin, 1998; Farnham et al., 1999; De Raedt et al., 2006; Gemar et al., 2001; de Jong, 2000). Individuals usually exhibit a compatibility effect (faster reaction time or fewer errors when self is associated with positive and other with negative), even though those vulnerable to affective disorders may show a weaker compatibility effect (de Jong, 2000; Tanner et al., in press). In particular, the failure to find a positive self-evaluative bias in the non-depressed group raises questions about the validity of the IAT used in this study.

One reason why an overall positive automatic self-evaluative bias was not observed may have been due to the design of the IAT. The labels for the key assignments of attribute and target concepts (e.g., SELF/POSITIVE in one corner of computer screen) were not present on the screen at all times. In other words, when the word, FAILURE, or ME was presented in the middle of the computer screen, participants had to wait 500 milliseconds for

the key assignment labels to appear in their respective corners of the computer screen. In previous research, the target concept and attribute concept labels are always present in their respective corners on the computer screen (i.e., De Raedt et al., 2006; Gamar et al., 2001; de Jong, 2000; Greenwald et al., 1998). The disruptive effects of the order of presentation of labels and stimuli might have contributed to the utilisation of more effortful and controlled processing skills employed by participants' response to the stimuli (Hsieh, 2002; Brebner & Welford, 1980). This may have cancelled out the effects of "true automaticity" related to self-evaluation and/or created confusion as to the demands of the task (Bargh, 1984; Eysenck, 1991). The next study in the following chapter was conducted to address these issues and the expected result in accordance with previous research using the IAT was obtained.

One potential criticism of this study is that the results obtained in this study do not genuinely reflect evidence of schematic processing, but instead reflect cognitive distortions that typically accompany dysphoric mood. This is because the participants in this study were selected on the basis of current elevated depressed mood and symptomatology. Therefore, the information processing differences on the EAST between the high and low BDI groups may only be reflective of the influence of mood on information processing, but not the existence of depressive self-schemata in the high BDI group *per se*. While the use of an undergraduate population who were selected on the basis of self-reported symptoms of depression does limit the generalisability of the study, there are a number of important counters to the above criticism. First, the importance of the EAST is that it fulfills the criteria put forward by a number of researchers for a tool that minimises voluntary responding (Rude et al., 2001) and that it is less transparent than self-report measures (Segal & Swallow, 1994). If this study had simply used questionnaire measures to compare the two groups, this would be a valid criticism. However, the use of the EAST and the results from this task do suggest that cognitive self-structures or automatic views of the self (at least from a latent schema model of depression; Beck, 1967, 1976) were being measured because of the concordance between the self-report measures and EAST. It is also possible that the high BDI group was more prone to depression (and thus have negative self-schemata) than the low BDI group as elevated levels of depressive symptomatology in student samples is an index of trait depression and hypothetical increased vulnerability to depression (Zemore et al., 1990; Beck, 1967, 1976). Further, the high BDI group in this study did not have a history of clinical depression and, therefore, it could be argued that the negative implicit views of the self were not due to cognitive scarring and associated negative self-schematic organisation as a result of a history of clinical depression (Lewinsohn et al., 1981), but indeed related to genuine vulnerability to depression.

Clearly, the best test of whether the high BDI group in this study were genuinely showing evidence of biased negative self-schematic activity or a negative self-evaluation

related to vulnerability to depression, would be to repeat the experiment with participants who are in a normal mood state but who have been selected because they are vulnerable to or prone to develop depression. This approach should allow one to evaluate whether the results obtained on the EAST require the activation of dysphoric mood or can be observed in a normal mood state with a vulnerable population. Studies 3 and 4 took this approach.

This study has shown that the EAST is a useful tool to be used in schema research alongside self-report measures of depression. The results of this study suggest that individuals with mild to moderate levels of depressive symptomatology (and associated depressive negative beliefs) showed evidence of biased information processing or a weaker positive automatic self-evaluative bias compared to a group of individuals with low levels of depressive symptomatology. Study 3 investigated whether this bias is evident in individuals who are hypothetically more prone to develop depression but who are in a normal mood state: individuals classified as trait depressed. Study 3 also addressed the issue of stimuli related to hypothetical schema content and how this affects information processing in trait depression compared to depressive non-schematic material. However, the design problems of the IAT as discussed in this chapter were addressed in the following study.

Chapter 6

Study 2 - The Implicit Association Test: In search of a positive automatic self-evaluative bias

6.1. Introduction

Positive automatic self-evaluation and potential differences in self-evaluation between individuals who are vulnerable to affective disorders and, those who are not, are under investigation with new tasks such as the IAT (Tanner et al., in press; De Raedt et al., 2006; de Jong, 2000; Gamar et al., 2001). In Study 1, the absence of an implicit or a positive automatic associative bias between self and positive concepts on the Implicit Association Test is not consistent with previous research findings (Greenwald et al., 1998; Greenwald & Banaji, 1995; De Raedt et al., 2006; Gamar et al., 2001; de Jong, 2000). In particular, the failure to find a positive bias in the non-depressed group raises questions about the validity of the IAT used in that study.

One possible reason for the failure to find a positive automatic self-evaluative bias may have been due to a design problem in the IAT that was used in Study 1. The normal procedure in the IAT is that the target concept and attribute concept labels (e.g., SELF/POSITIVE in one corner of computer screen) are always present in their respective corners on the computer screen (De Raedt et al., 2006; Gamar et al., 2001; de Jong, 2000; Greenwald et al., 1998). However, in Study 1 the target and attribute concept labels indicating key assignments were not present on the screen at all times. In other words, when the words FAILURE or ME were presented in the middle of the computer screen, participants had to wait 500 milliseconds for the key assignment labels to appear in their respective corners of the computer screen. The effect of stimuli appearing and disappearing (the labels) (Brebner & Welford, 1980), and the disruption of the order of presentation of labels and stimuli (Hsieh, 2002) might have contributed to the utilisation of more effortful and controlled processing skills employed by participants' responses to stimuli. This may have cancelled out the effects of "true automaticity" and the implicit nature of automatic self-evaluation (Bargh & Tota, 1988; Eysenck, 1991).

Aims and Hypothesis

The aim of this study was to re-design the IAT so that its presentation was consistent with previous research. In the re-designed IAT the target concepts (SELF-OTHER) and attribute concepts (POSITIVE-NEGATIVE) were visible on the computer screen at all times during the appropriate test and practice blocks of the IAT. The study tested the hypothesis that a group of non-depressed individuals would show an implicit or a positive automatic self-evaluative bias. That is, they would be faster and make fewer errors on the compatible block of the IAT when positive concepts were associated with the self and negative concepts were associated with other.

6.2. Method

Participants

A healthy, non-depressed sample of 20 psychology undergraduates from the University of Southampton was recruited through advertisement and opportunity sampling (7 males and 13 females). Participants were questioned to verify that they had never been depressed or had been treated for depression. They volunteered to participate in the experiment for course credits, which counted towards their undergraduate degree course. English was the first language of all participants. The mean age of the participants was 21.2 (SD=2.06) with a range of 18 to 26 years.

Materials

Implicit Association Test (IAT; Greenwald et al., 1998). The IAT consisted of seven blocks. It commenced with a practice block (block 1) of 32 trials where target concept words (related to self or to other people) had to be correctly categorised by pressing one of two keys on a computer keyboard (e.g., right key for self words, left key for words related to other). Four words related to self were presented randomly four times each, as were words related to “other”. The second practice block (block 2) comprised 32 trials where attribute concept words (negative or positive words-see Appendix IV) had to be correctly categorised also by pressing one of two keys (left or right). There were 8 negative words, and 8 positive words each of which was randomly presented twice. Following the two practice blocks, two test blocks (blocks 3 & 4) followed. These two blocks each had 32 trials where target concepts (4 “self” words and 4 “other” words were each presented twice producing a total of 16 trials) and attribute concepts (8 negative words and 8 positive words producing 16 trials in total) were presented in a random order. Following the two test blocks, participants practiced a new key assignment using the target concepts only (block 5). In this practice block participants were instructed to use the opposite keys for self and other to the keys used in blocks 1-4. This block comprised 32 trials where the 4 self words and the 4 words related to other were randomly presented 4 times each. The two test blocks (block 6 & 7) comprised 32 trials each, and included 16 randomly presented target concept trials, and 16 randomly presented attribute concept trials, as specified in blocks 3 and 4.

The key assignments (left or right) for categorising target concepts and attribute concepts were counterbalanced across participants. The compatible and incompatible test blocks were also counterbalanced so that half the participants completed the compatible test blocks first (blocks 3 & 4; self assigned same key as positive words) followed by blocks 6 & 7 (when self assigned the same key as negative words). The other half of the participants completed the incompatible test blocks first (blocks 6 & 7) then completed the compatible test blocks (blocks 3 & 4). During each block of trials, the concepts assigned to the left and right key would be designated by labels in the top left or right hand of the computer screen (e.g.,

SELF, OTHER, POSITIVE, & NEGATIVE). These labels would be present on the screen throughout each block unlike the IAT in Study 1. Each word was presented on the screen until a response was given. The response stimulus interval was 400ms. If a response was incorrect a red cross would appear in the center of the screen. The participant had to press the correct key to continue and the next trial was initiated 400ms after the red X disappeared as a result of the corrected response.

All responses below 300ms and above 3000ms were recoded as 300ms and 3000ms respectively in accordance with other implicit processing paradigms (Greenwald et al., 1998), and responses which were inaccurate (pressing the wrong key) were classified as errors.

The IAT was operated using a Turbo Pascal 5.0 Program that operated in graphics mode using an IBM compatible PC. The stimuli presented in the center of the computer screen was presented in white, in arial font uppercase, 7mm high and 5mm wide, as were the target and attribute concept labels in the top right and left corners of the computer screen. The background of the computer screen was black. Participants responded to stimuli using the Q and P keys on a standard QWERTY keyboard.

Stimuli used in Association with IAT. The IAT used a set of target concept words that represented the self (me, mine, I, first name of the participant), and represented other people (they, them, his, name of the author-this amended if it was the same as a participant). The attribute concept words comprised 8 negative words related to the maladaptive schemas of abandonment and defectiveness (Young & Brown, 1994) and the same 8 positive words that were used in the IAT in Study 1. These two sets of words did not significantly differ on the number of letters, frequency, number of syllables, and ratings of emotionality. However, the negative and positive words did, as planned, significantly differ on the ratings of pleasantness assigned to them. The word-type composition for sets of words (e.g., verb, adjective etc) did not differ significantly between the groups of words. The negative and positive stimuli used in the IAT were selected by the method outlined in Study 1. See Appendix IV for attributes of words used in Study 2.

Procedure

Participants were given an information sheet to read and then asked to sign a consent form if they agreed to take part. Participants were told that the experiment was about people's information-processing capabilities in relation to different kinds of words and that it would take around ten minutes to complete. The author took the first name of the participant and programmed it into the IAT. Each participant completed the experiment individually in a small, well lit, and noise proofed cubicle with no outside light. Participants sat approximately 12-18 inches from the computer screen with their fingers poised on the two response keys (P and Q). Participants were given instructions on the computer screen before each practice and test block of the IAT. The instructions told participants to categorise words by pressing one of

two keys (e.g., P for words related to self and negative words, Q for words related to other people and positive words). Participants were told whether the block was a test block or a practice block. Participants were asked to respond as quickly as possible but also as accurately as possible. Lastly, they were informed that if they pressed the wrong key in response to a word that a large red X would appear in the center of the screen, and they would have to press the correct response key to enable the IAT to continue. The whole procedure took approximately 30-45 minutes for each participant to complete.

6.3. Results

As in Study 1 two types of data were analysed: reaction times and error rates. Examination was conducted on both types of data using a repeated measures model of analysis on the one factor of compatibility, which had two levels. An alpha level of .05 was used for all ANOVA's. As in study 1, when referring to the IAT analysis, the compatible blocks refer to the two test blocks when the same key is used for self-words and positive words and the words related to other people and negative words share the same key. The incompatible blocks refer to the two test blocks when self-words and negative words share the same key, and words related to other people and positive words share the same key.

All reaction times were log-transformed to normalise extreme scores and the transformed data were used in all analyses. Table 4 overleaf shows mean untransformed reaction times and the error rates. Only reaction times on the test blocks with correct responses were analysed. Reaction times on the first trial of each block were discarded. Reaction times below 300ms and above 3000ms were recoded to 300ms and 3000ms respectively in accord with Greenwald et al.'s (1998) recommendations. Similarly, errors were not recorded if they occurred on the first trial of a test block.

Table 4

Mean Reaction Times (ms), Error Rates, and Standard Deviations on the IAT*

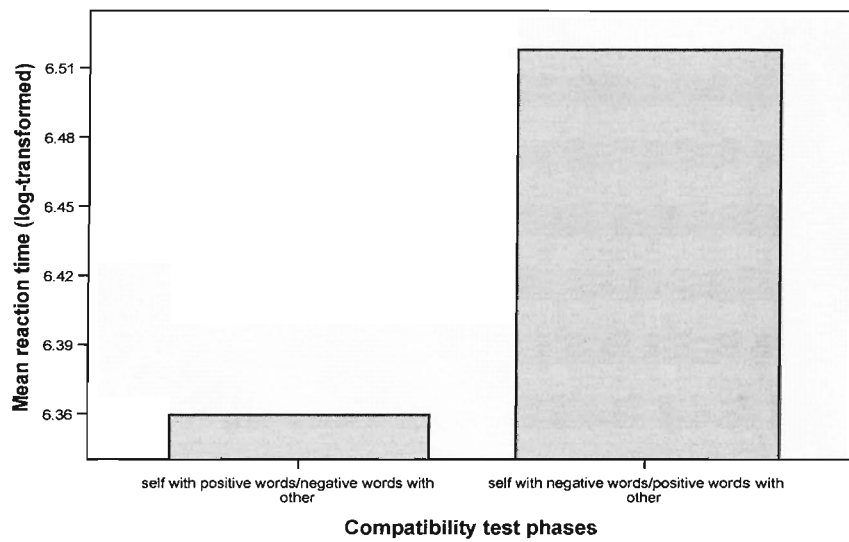
	<u>reaction time</u>	<u>errors</u>
Compatible phase	610.97 (98.42)	3.00 (2.57)
Incompatible phase	728.69 (122.61)	4.04 (3.02)

* Standard deviations in brackets.

IAT analysis of reaction time

In accord with the hypothesis, there was a significant effect of compatibility, $F(1, 19) = 62.09, p < .001$. This indicated that participants were significantly faster on the compatible blocks when self-words shared the same key as positive words (See Figure 3).

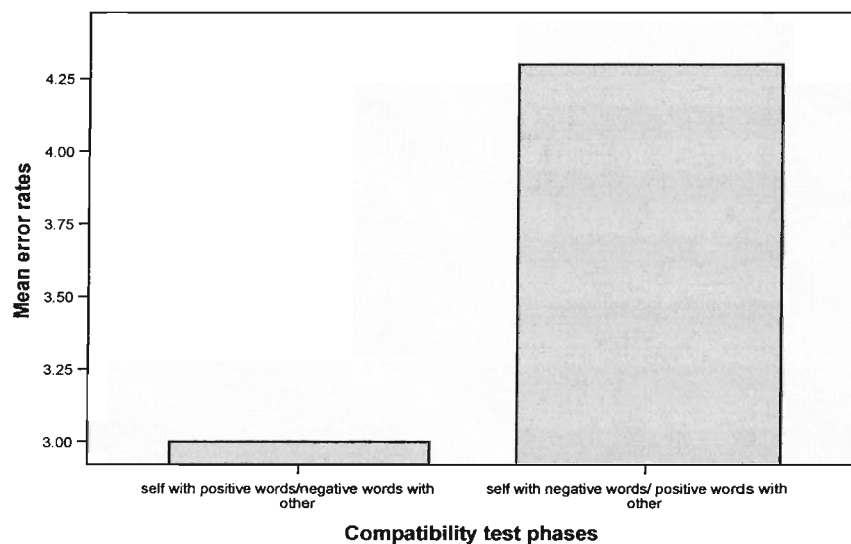
Figure 3. Graph showing main effect of compatibility on the IAT (reaction times).



IAT analysis of error rates

Again in agreement with the hypothesis, there was a main effect of compatibility, $F(1, 19) = 13.32, p < .01$. This indicated that participants made significantly fewer errors on the compatible blocks when self-words shared the same key as positive words (See Figure 4).

Figure 4. Graph showing main effect of compatibility on the IAT (error rates).



6.4. Discussion

The principal aim of this study was to re-design the IAT so that the task was consistent with previous studies that have used the IAT other research (e.g., Greenwald & Banaji, 1995; Greenwald et al., 1998; De Raedt et al., 2006; de Jong, 2000, Gamar et al., 2001) and thus to have a valid and reliable task to use in the following studies. These studies have shown that healthy non-depressed individuals possess a positive automatic or implicit self-evaluative bias which is in accordance with theories of automatic self-evaluation or self-esteem in healthy individuals (e.g., Swann, 1990). The revised task in this study that had the target and attribute concept labels (e.g., SELF-POSITIVE and OTHER-NEGATIVE) on the computer screen at all times during the test blocks demonstrated the hypothesised positive automatic self-evaluative bias on both error rates and reaction time analyses.

By having target and attribute labels constantly appearing and disappearing, as was the case in Study 1, it may have caused an excess in the shifting of attention, a slowing of automaticity, and a subsequent increase in reaction speed and deliberation in judgment (Brebner & Welford, 1980; Hsieh, 2002). The new design in this study seemed to minimise these influences and produce similar results consistent with other research (e.g., De Raedt et al., 2006).

As the revised IAT now appears to conform to previous research findings (e.g., Greenwald et al., 1998) this now leads us to be able to proceed further from the results and conclusions of Study 1. As discussed in Study 1, it was found on the EAST that individuals classified as analogue depressed had a weaker positive automatic self-evaluative bias compared to individuals classified as non-depressed. If such a positive automatic self-evaluative bias is present in healthy and non-depressed individuals as seen in this study, and in Study 1 on the EAST, then it would be interesting to investigate the nature of this bias in individuals who are classified as being high in traits indicative of and increased risk of developing depression (individuals hypothetically more vulnerable to develop depression as a result of the presence of negative self-schemata) and those regarded as being low in trait depression. The next study will address this issue using the improved IAT design from this study, and the EAST task used in study 1.

Chapter 7

Study 3 - Negative schematic processing in high and low trait depression

7.1. Introduction

In Study 1, which investigated implicit negative schematic processing in depression, individuals who scored highly on the Beck Depression Inventory-II (Beck et al., 1996) showed a weaker positive automatic self-evaluative bias on the EAST compared to individuals who scored low on the BDI. This result is consistent with the mood state hypothesis of depression (Gernar et al., 2001; Teasdale & Barnard, 1993; Beck, 1967, 1976), which states that when depressed people are in a depressed mood, or are experiencing significant depressive symptoms, information processing is more negatively biased. It is also consistent with Beck's proposal that a depressed mood activates latent schemata in depression-vulnerable individuals and that this activation results in negative information processing (Beck, 1967, 1976). However, the results do not concur with previous research that has used the EAST (De Raedt et al., 2006), and showed that depressed individuals have a stronger positive self-evaluative bias compared to non-depressed controls. Conversely, the results are consistent with other research that shows that depressed individuals have a more negative automatic self-evaluative bias compared to non-depressed individuals (Hedlund & Rude, 1995, Rude et al., 2001). With regard to the use of the IAT in study 1, the results were disappointing. The characteristic positive automatic self-evaluative bias found in healthy and non-depressed individuals in most research, and weakened positive automatic self-evaluative bias in depressed individuals, was absent (de Jong, 2000; Gernar et al., 2001; Greenwald et al., 1998). However, as already discussed there were some design problems with the IAT task that were identified and rectified in Study 2.

Nonetheless, these results from Study 1 cannot tell one whether the weak positive automatic self-evaluative bias observed in the high BDI group has a role to play in creating vulnerability to depression. This was because the two groups (high and low BDI groups) were selected on the basis of current levels of depressive symptoms. In other words, it is impossible to disentangle whether the obtained results were due to the effects of low mood associated with elevated levels of depressive symptomatology, or whether they were the result of dysfunctional cognitive schemas driving information processing. To answer this issue it would be useful to investigate schematic processing using the EAST and IAT in individuals who do not differ in mood or depressive symptomatology, but who do differ in the possession of stable traits indicative of depression proneness or who are at high-risk for developing depression; namely, individuals classified as having high traits of depression proneness compared to individuals classified as having low traits of depression proneness. This was the goal of Study 3 in response to the results of Study 1.

The proposal of stable traits being associated with vulnerability or proneness to depression has initiated much debate (Zemore et al., 1990). It has been argued that certain kinds of behaviour (intrapsychic and observable behaviour) elicited by individuals in response to a depressed mood are indicative of stable depressive traits and that these traits are associated with the subsequent development of clinical depression (Nolen-Hoeksema, Parker, Larson, 1994). Further, people prone to depression seem to experience an increased number of low moods and more severe low moods without necessarily developing clinical depression (Zemore et al., 1990). The next section will discuss the concept of trait depression and the applicability of this term for use in research investigating schemas and vulnerability to depression.

A biological theory of traits of depression

Recently researchers have investigated neurobiological abnormalities that may explain subsequent vulnerability to depression. Gold, Goodwin, & Chrousos (1988) and Bucci (2000) argue that stressful circumstances in early life (from infancy onwards) result in long-term changes in responsivity of the central nervous system, making such individuals hypersensitive to loss and external stressors. More importantly, early maladaptive experiences (infancy onwards) cause permanent changes to brain structure and function, and weaken resilience to stress. The process creates vulnerability in individuals, who then experience more frequent low moods or even develop depression in response to psychological challenges or environmental difficulties. Therefore individuals vulnerable to depression experience more frequent low moods and these low moods persist for longer, compared to non-vulnerable individuals. This neurobiological abnormality may contribute to the number of low moods experienced, how an individual experiences a low mood, and how the individual thinks, feels, and copes with psychologically challenging events. This process in turn, if the stressors and negative cognitive processes associated with low mood are sufficient, may contribute to clinical depression (McEwen & Magarinos, 2001; Nolen-Hoeksema & Morrow, 1991).

What the biological theory of trait depression suggests is that certain individuals, by virtue of negative early experience, acquire a hypersensitive nervous system. However, the question remains as to whether there are psychological or personality traits that can be measured in relation to vulnerability to depression? The next section will focus on this issue.

Personality traits of depression

The relationship between personality traits and depression proneness/vulnerability is complex and controversial. Several research endeavors have examined the relationship between personality and depression (Clark, Watson, & Mineka, 1994; Klein et al., 1988), and a number of different personality traits and characteristics have been examined (Barnett & Gotlib, 1988). This includes both broad traits, such as neuroticism and extraversion, as well as specific vulnerability factors, such as dependency and self-criticism (Blatt, 1974). Critics of

personality models of depression have argued that little evidence exists for a stable personality measure of vulnerability to depression (Coyne & Gotlib, 1983). Elevated levels on self-report measures of personality traits (e.g., neuroticism) may reflect the severity of depressive symptoms, and thus these measures may only be mood-state dependant (Coyne & Gotlib, 1983). Support for this view comes from a number of studies showing that scores on personality scales are either no different in remitted depressed individuals than in non-depressed controls, or are significantly lower in depressed patients tested in remission (Segal & Ingram, 1994). Because scores on personality measures generally decrease following treatment, critics have concluded that personality and vulnerability factors (e.g. scores of neuroticism and a negative implicit information processing bias) are not stable, but depend on the severity of depression or low mood. As such they cannot be used as markers of risk or vulnerability for depressive states (Brewin, 1988).

However, Spasojevic & Alloy (2001) have argued that there is some evidence in support of a biological and personality theory of traits, associated with vulnerability to depression. They found in a sample of initially non-depressed individuals followed over 2.5 years that negative cognitive styles, self-criticism, dependency, neediness, and a history of increased frequency of low moods (risk factors for subsequent vulnerability to depression) were all significantly associated with the amount of rumination. Rumination, measured by the Ruminative Responses subscale of the Responses to Depression Questionnaire (RDQ; Nolen-Hoeksema, & Morrow, 1991), mediated the predictive relationships of all risk factors. This subscale assesses responses to depressed mood that are self-focused (i.e. thinking about one's own short-comings). More importantly, it assesses the amount of time that one is self-focused. Depressive rumination is therefore conceptualised as an emotion-regulation strategy or a meta-emotional cognitive process (Gross, 1999). It is important to note that everyone experiences low moods and ruminates. However, individual differences exist in the way that people regulate their emotions. Trait depressed individuals seem to regularly engage in depressive rumination to a larger extent than non-trait depressed individuals. In an attempt to cope with and manage their depressive mood, trait depressed individuals seem to passively focus on their emotional state (Nolen-Hoeksema & Morrow, 1991).

In a very comprehensive evaluation of trait characteristics of depression closely connected to rumination research, Zemore et al. (1990) found that individuals classified as trait depressed seem to experience more low moods; the low moods last longer, and are more severe. Further, trait depressed individuals had more experience of ruminative thoughts relating to themes of feeling discouraged about the future, feeling lonely, perceptions of being a failure and being defective, difficulty concentrating, and lacking energy. Most people suffer at some time or another from all of the above, but with the trait depressed individuals the frequency is significantly higher. Indeed Zemore et al. (1990) have found that these traits, as

measured by the Depression Proneness Rating Scale (DPRS; Zemore et al., 1990), have considerable stability even while taking into account state depression. Therefore, high DPRS scores remain stable even when individuals are not suffering from high levels of state depression. Further, the DPRS has good predictive validity in individuals who go on to develop clinical depression.

Negative schemas and traits as a marker of vulnerability to depression

The ideas of biological developmental antecedents and psychological traits of depression relate to Young's (1990) concept of maladaptive schemas, which confer vulnerability to depression. They posit that early maladaptive experiences (antecedent risk factors) create templates (schemata) that guide an individual's processing of information. In other words, theoretically, early negative experience may form neurobiological abnormalities, which may in turn form stable negative cognitive schemas due to stress (e.g. as a result of poor parenting). Subsequently, this may affect how an individual evaluates and conceptualises a psychologically challenging event. It therefore seems viable to investigate the concept of vulnerability to depression, schemas and information processing from the trait perspective.

In the context of the present study, utilising the trait depressive concept and the ideas it yields, one is able to investigate some key ideas related to the schema concept in depression, and issues related to schema measurement. As discussed in detail in Chapter 3, negative schematic information processing biases related to depression are generally thought to be mood-state dependent (Gemar et al., 2001). However, as highlighted in Chapter 4, there may be methodological issues that prevent the measurement of schemas and vulnerability to depression in the absence of a depressed mood. The problems highlighted were whether implicit tasks measure the strength of association of stimuli to the self or self-evaluation/negative self-schemata, whether the appropriate stimuli being used is congruent to be able to measure the schema construct/content, and whether error rates may be a more useful way to measure schemata and vulnerability to depression. Thus, by using a sample of trait depressed individuals and comparing them with non-trait depressed individuals, along with two relatively new tasks (the EAST & IAT), one may be able to test the strength of association of different kinds of stimuli to the self; whether stimuli related to hypothetical content within the schema construct is a better measure of negative self-schemas rather than generic depression related material (e.g., Alloy et al., 1999). Further, one will be able to investigate the important issue of the role of errors as opposed to reaction time with regards automaticity and vulnerability to depression. Error rates to stimuli have been argued to be more representative of vulnerability to psychopathology and depression (see Chapter 4; Bargh & Tota, 1988; Kirsch & Lynn, 1999; Eysenck, 1991). By taking into account all of the above issues, one may be able to ascertain the role of schematic processing in individuals classified as being trait depressed and who are hypothetically at risk of developing depression.

Aims and hypotheses

The main aim of this study was to investigate automatic self-evaluation in individuals classified as being either high-trait depressed or low-trait depressed. This was to ascertain whether information-processing differences exist between the two groups, even though levels of depressive symptomatology do not. The second aim was to establish whether individuals who differ on measures of trait depression, differ in the ways they implicitly process different kinds of information. In other words, to ascertain if trait depressed individuals have a preferential bias to process negative information that is related to content theoretically within schemata and implicated in schematic development (e.g., Alloy et al., 1999; Beck, 1967, 1976) compared to stimuli that is generically related to depression. The third aim of this study was to ascertain if trait depressed individuals' error rates compared to non-trait depressed individuals' error rates to incongruent and congruent stimuli are a more accurate measure of schematic functioning compared to reaction speed.

It was hypothesised that 1) a group of low-trait depressed individuals would exhibit a stronger positive automatic self-evaluative bias compared to a group of high-trait depressed individuals. This would be shown by errors and/or reaction time differences when self is associated with positive stimuli versus negative stimuli on the implicit tasks (e.g., EAST & IAT); 2) a group of high-trait depressed individuals would have a significantly weaker positive automatic self-evaluative bias in response to negative stimuli related to themes implicated in the etiological development of schemas and schema content (themes of abandonment and defectiveness; Alloy et al., 1999) when associated with the self, compared to generic negative stimuli related to depression.

7.2. Method

Participants

Participants in this study were undergraduate students from the University of Southampton. Individuals were recruited from lectures, advertisement, and opportunity sampling methods. Exclusion criteria included the use of any medication that would interfere with motor skill, previous history of depression, colour blindness or eyesight problems, and English as a second language. Participants completed the informed consent, DPRS (Zemore et al., 1999), and BDI-II (Beck et al., 1996) before being included in the study. A median split was taken on the DPRS to classify individuals as either high-trait or low-trait depressed. A mean score between 1-5 on the DPRS put these participants into the low-trait depressed group. A mean score between 5-9 on the DPRS put these participants into the high trait depressed group. Individuals who scored below 2 on the BDI-II were excluded as scores of 0 or 1 may represent other forms of psychopathology being present (e.g., psychopathy, hypomania) rather than, or in addition to, the absence of depression (Hammen, 1980). Sixty-

six participants took part in the experiment: 35 participants formed the low-trait depressed group and had a mean DPRS score of 3.29 (SD=. 88). Thirty-one participants formed the high-trait depressed group and had a mean DPRS score of 6.02 (SD=. 92). The difference in DPRS scores between the groups was significant, $t(64) = 12.25, p < .001$. The mean age of the low-trait depressed group was 21.54 years (S.D. = 3.82) and the mean age of the high-trait depressed group was 20.96 years (S.D. = 2.77). The low-trait group comprised 21 males and 14 females, and the high-trait group comprised 17 males and 14 females. There was no difference between the groups on age, $t(64) = .691, p > .05$, or on gender composition, $\chi^2(1) = 1.67, p > .05$.

Materials

Self-Report measures. The rationale behind choosing the following questionnaires as dependent measures was to sample cognitive products, cognitive processes, and cognitive structures/schema as carried out in Study 1.

Depression Proneness Rating Scale (DPRS, Zemore et al., 1990). The DPRS is a 13 item self-report questionnaire that has two sections. The first section (comprising 3 questions) asks participants to compare their own experiences of depression to the experiences of most people whom they know according to the frequency, length and severity of depressive episodes. The second part (comprising 10 questions) asks participants to compare their experience of 10 commonly assessed depressive symptoms, to the symptoms experienced by other people whom they know. Scoring is on a nine-point scale, 1 being either much less often, much shorter, or much less deeply (experience of low moods in comparison to other people), 9 being either much more often, much longer, or much more deeply. All questions pertain to a participant's experience during the past two years. The test-retest reliability of the DPRS, measured in a sample of 100 undergraduates with a 9-week interval between administrations was .82. This correlation showed greater stability than current symptom severity, measured by the Beck Depression Inventory (Beck et al., 1979). Internal consistency was measured in a sample of 1,101 undergraduates. Cronbach's alpha was .90 and correlations between each item and total score ranged from .39 to .76. One test of validity of the DPRS involved comparing scores on the DPRS to a person's history of depression. Results showed a significant correlation, .41, in a group of 440 university students. This correlation was significantly greater than the relationship between current symptom severity measured by the BDI (Beck et al., 1979) and history of depression.

The Ruminative Responses subscale of the Response to Depression Questionnaire (RDQ; Nolen-Hoeksema & Morrow, 1991). Using a 4-point Likert scale on the ruminative responses subscale, participants are asked to indicate what they generally do when feeling low, sad, or depressed. A score of 1 indicates that someone almost never engages in rumination, while 4 indicates someone almost always engages in depressive rumination. The

Ruminative Responses subscale of the RDQ consists of 21 items assessing responses to depressed mood that are self-focused (e.g. Thinking about all your shortcomings, failings, faults, mistakes), symptom focused (e.g. Thinking about how hard it is to concentrate), or focused on possible causes and consequences of the depressive mood (e.g. Thinking “I won’t be able to do my job/work because I feel so badly”). Previous research has established that the Ruminative Responses subscale of the RDQ has good internal consistency ($\alpha = .89$; Nolen-Hoeksema & Morrow, 1991), a good 5-month test-retest reliability ($r = .80$; Nolen-Hoeksema et al., 1994), and good validity for predicting the onset of depression (e.g., Just & Alloy, 1997; Nolen-Hoeksema & Morrow, 1991).

For full details of the following questionnaires listed below please see the method section in Study 1, Chapter 5.

The Beck Depression Inventory-II (BDI II, Beck et al., 1996). Measures the severity of depressive symptomatology.

The Dysfunctional Attitudes Scale (DAS, Weismann & Beck, 1978). Measures conditional dysfunctional assumptions related to depression.

Young Schema Questionnaire (short version) (YSQ, Young & Brown, 1994). Measures stable cores beliefs, themes, or early maladaptive schemas related to different psychopathologies.

The Beck Anxiety Inventory (BAI, Beck, 1980). Measures the severity of anxious symptomatology.

The Evaluative Beliefs Questionnaire (EBS, Chadwick, Tower, & Dagnan, 1999). Measures a class of beliefs closely related to psychopathological disturbance and especially depression; self-depreciatory beliefs, one’s depreciatory beliefs regarding other people, and depreciatory beliefs regarding what one thinks others think of one.

Automatic Thoughts Questionnaire (ATQ, Hollon & Kendall, 1980). Measures the severity of negative automatic thoughts related to the presence and severity of depressive symptoms.

Rationale for the stimuli used to investigate schema content specificity. According to cognitive theories of depression, individuals who are vulnerable to depression have a bias towards processing negative information in relation to themselves (Beck, 1967, 1976). This process is hypothesised to be the result of the possession of negative cognitive self-schemas due to negative early life experiences (e.g., poor parenting, deprivation, stressful insecure environments etc; See Parker et al., 2000). Beck suggests (Beck, 1967, 1976) that depression-prone individuals have specific negative self-schemata related to incompetence, worthlessness and low motivation, but do not possess self-schemata related to other negative themes (Dykman et al., 1989; Alloy et al., 1999; Greenberg & Alloy, 1989; McLain & Abramson, 1985). Thus, Beck’s content specificity hypothesis suggests that information processing

biases should be limited to stimulus material that is congruent with the content of the negative self-schema. Indeed, in a study by Alloy et al (1999), they found that individuals who were at risk of developing depression showed preferential self-referent processing of negative depression relevant words involving themes of incompetence, worthlessness, and low motivation, but not to depression words unrelated to these themes.

The lack of a consideration for the possibility of specific schema content in depression research may be one reason for the elusive detection of schematic activity in the absence of depression or induced low mood. Stopa et al. (2001) found that themes of defectiveness and abandonment measured by the Young Schema Questionnaire were correlated with depression scores on the SCL-90 in a mixed psychiatric sample. Other research has confirmed that similar themes are implicated in vulnerability to depression. Parker et al. (2000) found significant associations between negative events in childhood and adult depression. Notably the depressed participants in Parker et al.'s study endorsed schema themes involving emotional deprivation, worthlessness, and/or being inadequate.

In this study, two types of negative stimuli were chosen to test the schema content specificity hypothesis. These two types comprised words related to depressive mood, and words related to the schematic themes of abandonment (encompassing emotional deprivation, loneliness and insecurity) and defectiveness/shame (encompassing ideas of defectiveness & unworthiness). It was felt that these schema themes were fitting for the study's purpose bearing in mind other research findings as discussed earlier (e.g., Alloy et al., 1999). The EAST task included both sets of negative words, while in the two IAT tasks, one IAT used negative schema words, whereas the other IAT used low mood descriptor words.

Stimulus words used in conjunction with Extrinsic Affective Simon Paradigm. The same words as used in Study 1 in the EAST task were used in the EAST task in Study 3. These are shown in Appendix IV.

Stimulus words used in conjunction with the Implicit Association Tasks. Participants completed two IAT tasks. In one version of the IAT (IAT Mood) a set of 8 words depicting a low mood and 8 positive words were used. The positive words and low mood words were balanced on the same variables as specified above in the EAST task (e.g. number of letters etc) and selected by the method outlined in Study 1. In the other version of the IAT (IAT Schema), 8 schema words, based on the early maladaptive schemas of abandonment and defectiveness, and the 8 positive words from Study 2 comprised the negative and positive stimuli. Again these sets of words were balanced for number of letters, syllables, frequency, and emotionality. The word-type composition for sets of words on both IAT's did not differ (e.g., verb, adjective, or noun) significantly between groups of words. See Appendix IV for attributes and details of words used in the IAT Schema and IAT Mood.

The Extrinsic Affective Simon Task (De Houwer, 2003). The specifications of the Extrinsic Affective Simon Task were similar to those described in Study 1 apart from some minor adjustments. There was an increase in the number of trials in the test block (Phase 3) in order to make a clearer distinction for the purposes of statistical analysis and improved power between the schema and low mood descriptor words in order to test the “schema content hypothesis”. Therefore, there were now 320 trials in phase 3, the test block: 20 schema word trials with the self-attribute key, and 20 schema word trials with the other person attribute key, 20 low mood word trials with self- attribute key, 20 low mood word trials with other person attribute key. There was also an increase in positive word trials: 40 trials with the self-attribute key and 40 trials with the other attribute key. There were also 40 presentations of self-related words (e.g., I) and 40 presentations of other attribute words (e.g., THEM). Phase 1 practice block (classifying words related to oneself or another person) remained identical to the previous experiment, while phase 2, the second practice block (classifying words as either blue or green) had 32 trials; 8 schema words trials, 8 low mood word trials, and 16 positive word trials. Each word-type (negative words and positive words) again was presented equally in blue and green. All stimuli were presented in a random fashion apart from the first 5 trials of the test block, which involved classification of self-attributes to minimise errors associated with the beginning of the test phase (e.g., surprise or startle). There were four different versions of the EAST task for counterbalancing purposes to ensure that participants had different key allocations for self/other attributes and coloured negative/positive words. Response keys were Q for left and P for right on a standard QWERTY keyboard.

The Implicit Association Task (IAT, Greenwald et al., 1998). The IAT design was identical to the task described in Study 2. There were two separate IAT’s in this study. One IAT (IAT Schema) used the list of words related to the early maladaptive schemas (defectiveness/shame & abandonment) as its negative stimuli, while the other IAT (IAT Mood) used negative words descriptive of low mood. Both IAT’s used the identical positive stimuli as used in Studies 1 & 2. Counterbalancing specifications for the IAT (response keys and compatibility) were identical as detailed in Study 1 and took into consideration that participants had completed two IAT tasks.

Procedure

After initial screening with the DPRS & BDI-II, participants who met the other study criteria described in the participant section were invited to participate in the next stage of the experiment. All participants were given an information sheet to read, and a consent form to sign. Testing took place in a small noise-proofed, well-lit cubicle with no outside light protruding. The participants were situated approximately 12-18 inches from the computer screen. All participants were told that they would have to complete three computer tasks. They were told that they would be presented with instructions telling them to classify words

by either colour or by person (i.e. their name or another person's name) by pressing one of two keys (P or Q) on the keyboard. In the case of the IAT tasks, participants were told that they would have to classify words related to them or words related to someone else, negative words, and positive words by pressing either P or Q on the keyboard. The experimenter then left the room after each experiment was set up and waited outside until participants finished each task. Participants then indicated to the experimenter that they had completed one task. Then the experimenter set up the next task. The order of computer tasks was counterbalanced across participants. After the computer tasks, participants completed the questionnaires in the following order: DAS, EBS, YSQ, ATQ, RDQ, BAI. The experimenter again left the room while the participants did this. After participants had completed the questionnaires they were debriefed. Participants were paid £5 for their participation or given credits in fulfilment of their respective degree course. The whole procedure took approximately one hour for each participant to complete.

7.3. Results

The questionnaire results are presented first, followed by the reaction time and error results of the EAST, and then the reaction time and error results of the two IAT's. This will be followed by the EAST and IAT analyses to test for the specific schema content hypothesis. This analysis was concerned with investigating schema words and low mood descriptor words and their effects on automatic self-evaluation. The reaction time data from the IAT's was treated in the same way as described in studies 1 & 2. Unless reported in the results, non-significant results and minor effects and interactions from the conducted ANOVA's on the IAT & EAST data are presented in Appendix V. An alpha level of .05 was used for all initial ANOVA's. All post hoc analyses (e.g., independent and paired t-tests) used Bonferroni correction techniques to reduce the likelihood of false positive results. This involved dividing the alpha level (.05) by the number of tests conducted. This will be referred to in the text as (.05/N).

The aim was to sample a high-trait group and low-trait group who did not differ on depressive symptoms and investigate information processing differences in high-trait and low-trait individuals who did not differ on depressive symptomatology. However, the high trait-group scored significantly higher on the BDI compared to the low-trait group (see overleaf). Therefore, to examine and control for the effects of differential levels of depressive symptoms between the high and low trait groups, BDI scores were added to the statistical analyses as a covariate. All analyses from the EAST and IAT's will take the form of reporting the results, firstly with no covariate added to the statistical analysis, secondly with BDI scores added to the analysis as a covariate. By carrying out the analysis in this way it gives an indication of the relative effects of depressive symptoms. As in Studies 1 and 2, log-

transformed reaction times in the EAST and IAT tasks were used for the purposes of the statistical analyses.

Self-report measures

Using independent t-tests, the high-trait group compared to the low-trait group scored significantly higher on the BDI, $t(64) = 7.35, p < .001$, ATQ, $t(64) = 6.10, p < .001$, RDQ, $t(64) = 3.03, p < .001$, BAI, $t(64) = 2.83, p < .05$, and DAS, $t(64) = 2.03, p < .05$. Using an adjusted Bonferroni corrected p value of .017, (.05/3) on the EBS, the high-trait group did not significantly differ from the low-trait group on the self-self judgments, $t(64) = 2.35, p = .022$, self-other judgments, $t(64) = 1.91, p = .060$, but did significantly differ on other-self judgments, $t(64) = 3.51, p = .001$. The high-trait group scored significantly higher than the low-trait group on the YSQ subscales of Defectiveness/Shame, $t(64) = 3.54, p = .001$, Failure, $t(64) = 3.71, p < .001$, and Insufficient Self-Control, $t(64) = 3.40, p = .001$, using a Bonferroni corrected p value of .003 (.05/15). See Table 5 below for mean scores on questionnaires.

Table 5

Means and Standard Deviations on the Self-Report Measures by the Low and High Trait Group

	<u>Low-trait group</u>		<u>High-trait group</u>	
	<u>mean</u>	<u>SD</u>	<u>mean</u>	<u>SD</u>
BDI	6.63	4.35	16.98	6.93
DAS	127.71	25.96	143.97	38.7
ATQ	46.14	14.98	73.39	21.07
BAI	8.05	5.81	11.41	6.15
RDQ	41.66	13.21	53.16	10.17
EBS: self-self	0.09	0.23	0.35	0.63
EBS: self-other	0.13	0.30	0.59	0.69
EBS other-self	0.12	0.31	0.33	0.55
<u>YSQ subscales:</u>				
Abandonment	2.05	0.94	2.72	1.17
Emotional deprivation	1.79	0.75	2.54	1.33
Mistrust/abuse	2.20	1.14	2.90	1.16
Social isolation	1.66	0.67	2.25	1.03
Defectiveness/shame	1.34	0.67	2.19	1.18
Failure	1.88	0.88	2.87	1.26
Dependence	1.81	0.65	1.95	0.76
Vulnerability to harm	1.60	0.59	1.99	0.76
Enmeshment	1.54	0.54	1.48	0.36
Subjugation	1.74	0.73	2.20	0.79
Self-sacrifice	2.87	1.06	3.05	1.26
Emotional inhibition	2.09	0.92	2.40	1.11
Unrelenting standards	3.18	1.13	3.36	1.47
Entitlement	2.29	0.90	2.50	1.16
Insufficient self-control	2.40	0.77	3.27	1.27

EAST analyses

The EAST produced two types of data: reaction times and error rates. These are presented separately. The following set of EAST analyses looked at positive and negative words, but did not sub-divide them into separate mood and schema word categories. The EAST analyses involved 2 (word-type) x 2 (person) x 2 (group) repeated measures ANOVA's, the first two factors being within subjects factors, the latter a between subjects factor. The hypothesis was that the high-trait group would have a weaker positive automatic self-evaluative bias by virtue of having slower reaction times and more errors when positive words were associated with the self, compared to the control group. Alternatively, this could be shown by faster reaction times and fewer error rates when negative words were associated with the self compared to the low-trait group. Mean reaction times (untransformed) and error rates from the EAST (with BDI scores added as a covariate and not added as a covariate) are presented in Table 6 below.

Table 6

Mean Reaction Times (ms), Error Rates, and Standard Deviations on the EAST for the High and Low Trait group with and without adding BDI Scores as a Covariate

	Group	RT	S.D	RT cov*	S.D.	Err **	S.D	Err ** cov*	S.D.
Positive words with self	Low-trait	624.09	103.79	616.17	112.71	2.51	2.87	2.35	3.61
	High-trait	582.82	84.02	591.76	114.74	3.42	3.21	3.62	3.68
Positive words with other	Low-trait	634.89	120.71	632.76	132.79	1.97	1.67	1.93	2.37
	High-trait	589.58	100.01	591.98	135.18	2.71	2.34	2.75	2.39
Low mood words with self	Low-trait	644.95	147.70	643.66	153.87	1.69	1.72	1.52	1.89
	High-trait	573.76	104.12	575.22	156.58	1.71	1.51	1.89	1.94
Low mood words with other	Low-trait	624.32	102.67	621.37	119.53	0.83	1.12	0.82	1.30
	High-trait	578.12	97.73	581.45	121.70	1.19	1.13	1.20	1.33
Schema words with self	Low-trait	666.98	177.13	665.38	176.12	1.40	1.35	1.05	1.92
	High-trait	589.98	105.07	591.78	179.24	1.90	1.95	2.29	1.94
Schema words with other	Low-trait	624.04	119.12	616.06	127.58	0.83	1.22	0.79	1.42
	High-trait	574.74	92.54	583.75	129.83	0.84	1.18	0.87	1.44
Negative words with self	Low-trait	655.69	154.95	654.78	157.23	3.09	2.54	2.57	3.25
	High-trait	581.99	99.58	583.02	160.02	3.61	3.07	4.18	3.28
Negative words with other	Low-trait	655.69	102.65	618.57	115.32	1.66	2.01	1.61	2.24
	High-trait	581.99	90.18	582.64	117.36	2.03	1.77	2.08	2.28

* Reaction time and error rate when adding BDI scores to the analysis as a covariate. **=Error rates

EAST reaction times with BDI scores not added as a covariate. The analysis carried out without adding BDI scores as a covariate did not yield a 3-way interaction of word type by person by group, $F(1, 64)=1.687, p>.05$, or any other significant results (highest $p=.069$).

EAST reaction times with BDI scores added as a covariate. A 3-way interaction (word-type x person x group) was just marginally non-significant, $F(1, 63)=3.92, p=.052$. As this result was very close to significance, a post-hoc analysis was carried out. Using

Bonferroni corrected p values for independent t -tests (.05/4) and paired t -tests exploring differences within groups (.05/5) the highest p value obtained using this stringent criteria showed that the low-trait group were slower when negative words were associated with the self compared to when positive words were associated with self, $t(34) = 2.40, p = .022$. Unfortunately, using Bonferroni correction this result was non-significant. There were no other significant results (highest $p = .159$).

EAST reaction time analyses summary. The EAST reaction time results were not in accord with the hypothesis. The low-trait group did not show a stronger positive automatic self-evaluative bias although they did show a non-significant trend (reaction time with BDI scores added as a covariate) to be quicker when positive words were associated with the self compared to when negative words were associated with the self.

EAST error analysis without BDI scores added as a covariate. This analysis without adding BDI scores as a covariate yielded a main effect of person, $F(1, 64) = 12.69, p < .01$, which showed that all participants made more errors when all types of words were associated with other. There was no hypothesised 3-way interaction of word-type by person by group, $F(1, 64) = .000, p > .05$. There were no other significant results (highest $p = .061$).

EAST error analysis with BDI scores added as a covariate. Again there was a main effect of person, $F(1, 64) = 6.77, p < .05$ indicating that participants made more errors when words were associated with other. There was no hypothesised 3-way interaction of word-type by person by group, $F(1, 63) = .301, p > .05$. There were no other significant results (highest $p = .094$).

EAST error analysis summary. Contrary to the hypothesis, the low-trait group did not exhibit a stronger positive automatic self-evaluative bias compared to the high-trait group.

IAT analyses

Like the EAST, the IAT produced two types of data: reaction time and error rates. Again, each of these was analysed separately. The hypotheses for each IAT (Mood & Schema) on both the reaction time and error analyses, was that the high-trait group would possess a weaker positive automatic self-evaluative bias compared to the low-trait group. This would be shown by overall slower reaction times and increased error rates on the compatible blocks relative to the incompatible blocks of the IAT's by the high-trait group, compared to the low trait-group. Statistical analysis for each IAT involved a 2 (compatibility) \times 2 (group) repeated measures ANOVA, compatibility being within subjects factors and group a between subjects factor. See Table 7 for mean untransformed reaction times and error rates from the IAT Mood and IAT Schema.

Table 7

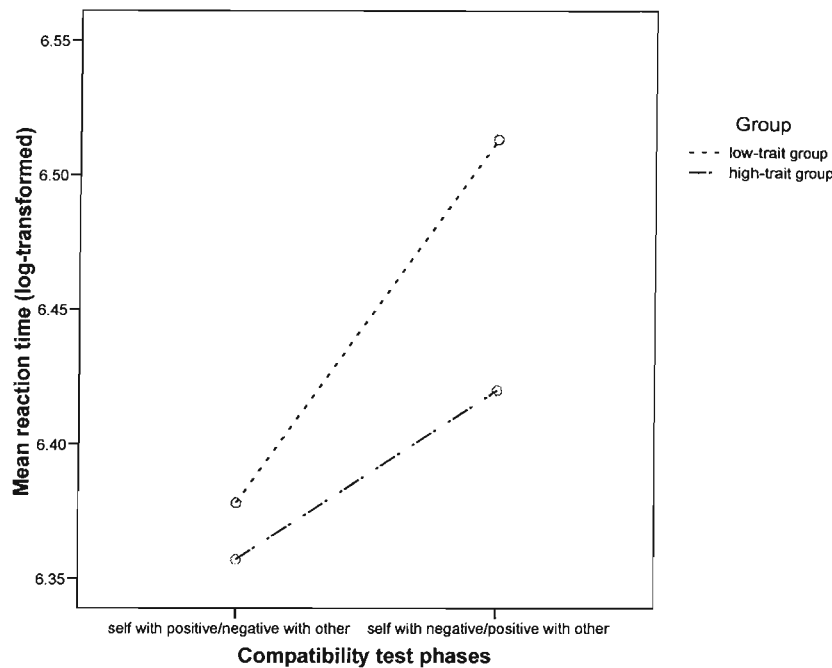
Mean Reaction Times (ms), Error Rates, and Standard Deviations on the IAT Mood & IAT Schema for the High and Low Trait Group*

IAT Phase	low trait group		high trait group	
	reaction time	errors	reaction time	errors
<u>IAT Mood</u>				
Compatible	630.81 (133.76)	3.92 (4.78)	612.76 (113.50)	5.76 (3.88)
phase:	630.46 (148.53)**	3.39 (5.38)**	613.15 (151.22)**	6.48 (6.01)**
Incompatible	737.52 (185.21)	5.82 (6.02)	653.21 (131.93)	5.66 (3.99)
phase:	726.26 (192.93)**	5.53 (6.52)**	665.92 (196.34)**	6.05 (5.73)**
<u>IAT Schema</u>				
Compatible	620.89 (138.01)	3.25 (3.26)	617.88 (117.60)	4.61 (4.30)
phase:	611.09 (172.95)**	2.73 (4.52)**	642.43 (192.33)**	5.30 (5.05)**
Incompatible	721.66 (170.26)	5.60 (5.37)	664.19 (146.56)	5.76 (3.76)
phase:	710.36 (217.02)**	5.70 (5.92)**	702.95 (242.51)**	5.62 (6.61)**

* Standard deviations in brackets, ** results with BDI scores added to the analysis as a covariate

IAT Mood- Analysis of reaction times with BDI scores not added as a covariate. The analysis with BDI scores not added as a covariate produced a main effect of compatibility, $F(1, 64) = 58.23, p < .001$, which showed that all participants were overwhelmingly faster when positive attributes were associated with self, and a two-way interaction of compatibility by group, $F(1, 64) = 7.75, p < .01$. As one can see from Figure 5 below, the low-trait group's difference in performance between the compatible block compared to the incompatible block in response time efficiency resulted in a very strong positive automatic self-evaluation. In comparison, the high-trait group did not show as strong a difference between the compatible versus the incompatible block and thus did not exhibit a strong positive automatic self-evaluative bias like the low-trait group.

Figure 5. Compatibility by group interaction (reaction time) without BDI scores added as a covariate (IAT Mood).



IAT Mood- Analysis of reaction times with BDI scores added as a covariate. There was a main effect of compatibility, $F(1, 63) = 21.54, p < .001$. This indicates that all participants were faster in the compatible block (e.g., faster when positive and self were assigned to the same keys), and therefore all had a positive automatic self-evaluative bias. There was no 2-way group by compatibility interaction, $F(1, 63) = 1.15, p > .05$. This indicates that neither group had a stronger or weaker positive automatic self-evaluation.

IAT Schema- Analysis of reaction times without BDI scores added as a covariate. The analysis with BDI scores not added as a covariate showed a main effect of compatibility, $F(1, 64) = 11.231, p < .01$, which indicates that all participants were overwhelmingly faster on the block when positive attributes were associated with self indicative of a stronger positive automatic self-evaluation. There was however a non-significant 2-way interaction of compatibility by group, $F(1, 64) = .248, p > .05$. This result again shows that neither group had a stronger or weaker positive automatic self-evaluation.

IAT Schema- Analysis of reaction times with BDI scores added as a covariate. There was a non-significant main effect of compatibility, $F(1, 63) = .836, p > .05$, indicating that overall participants performed similarly on both the compatible and incompatible blocks. There was also a non-significant 2-way group by compatibility interaction, $F(1, 63) = .571, p > .05$. Again this shows that neither group had a stronger or weaker positive automatic self-evaluation.

IAT reaction time analyses summary. On only one analysis of the IAT reaction time data (IAT Mood with BDI scores added as a covariate) did the low-trait group show a stronger positive automatic self-evaluative bias compared to the high-trait group.

IAT Mood-Analysis of error rates without BDI scores added as a covariate. The error analysis with BDI scores taken out as a covariate yielded a marginally non-significant main effect of compatibility, $F(1, 64) = 3.30, p = .074$. There was a compatibility by group interaction, $F(1, 64) = 6.18, p < .001$. This indicated that the low trait group had a stronger positive automatic self-evaluation (See Figure 6 overleaf).

IAT Mood-Analysis error rates with BDI scores added as a covariate. There was no main effect of compatibility, $F(1, 63) = .143, p > .05$. Again there was a compatibility by group interaction, $F(1, 63) = 4.46, p < .05$. As is evident from Figure 7 overleaf, the low-trait group had a stronger positive automatic self-evaluation compared to the high-trait group.

Figure 6. Compatibility by group interaction (error rates) without adding BDI scores as a covariate (IAT Mood).

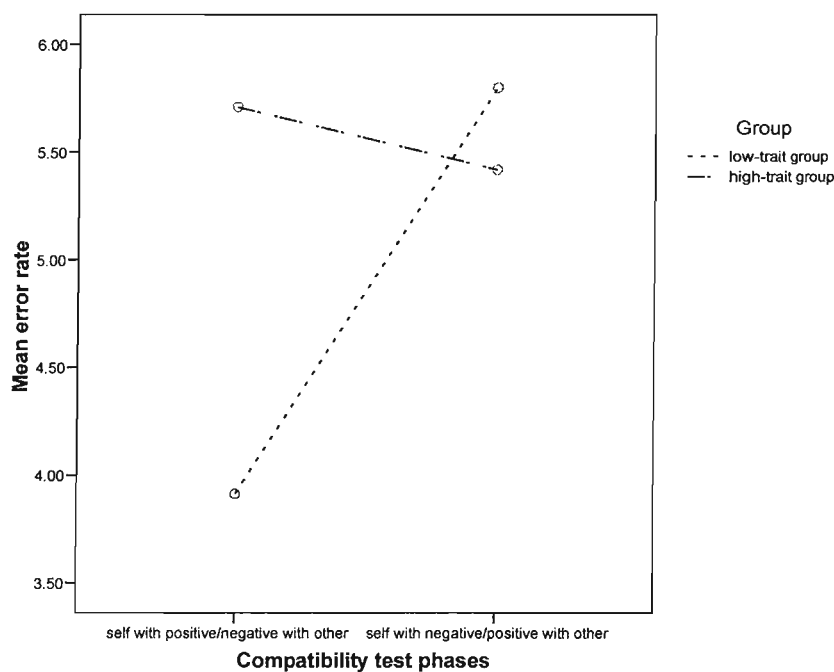
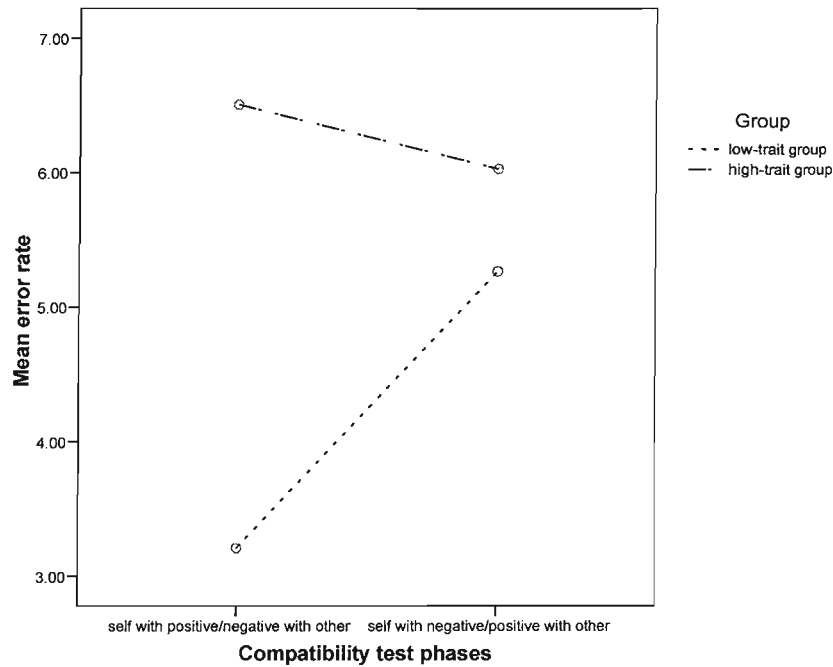
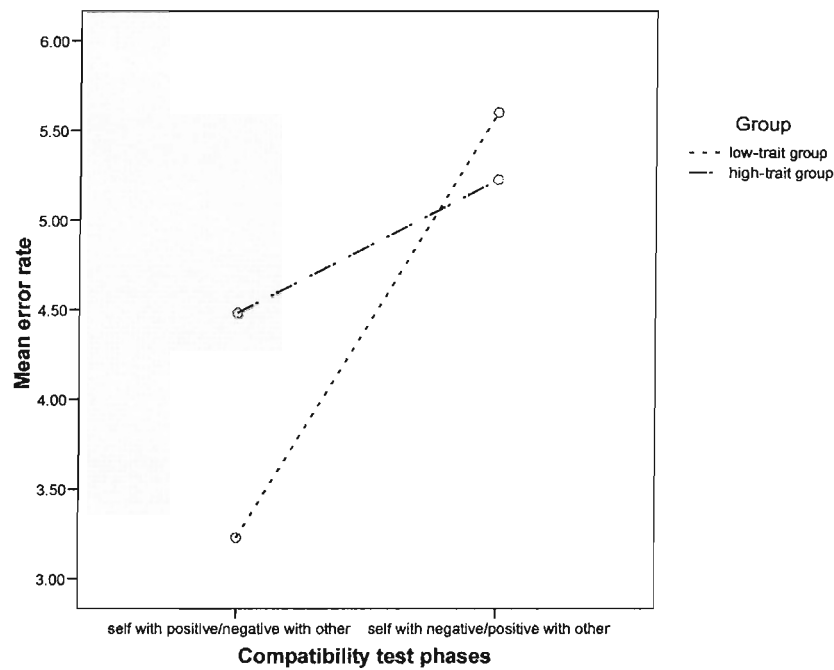


Figure 7. Compatibility by group interaction (error rates) with adding BDI scores as a covariate (IAT Mood).



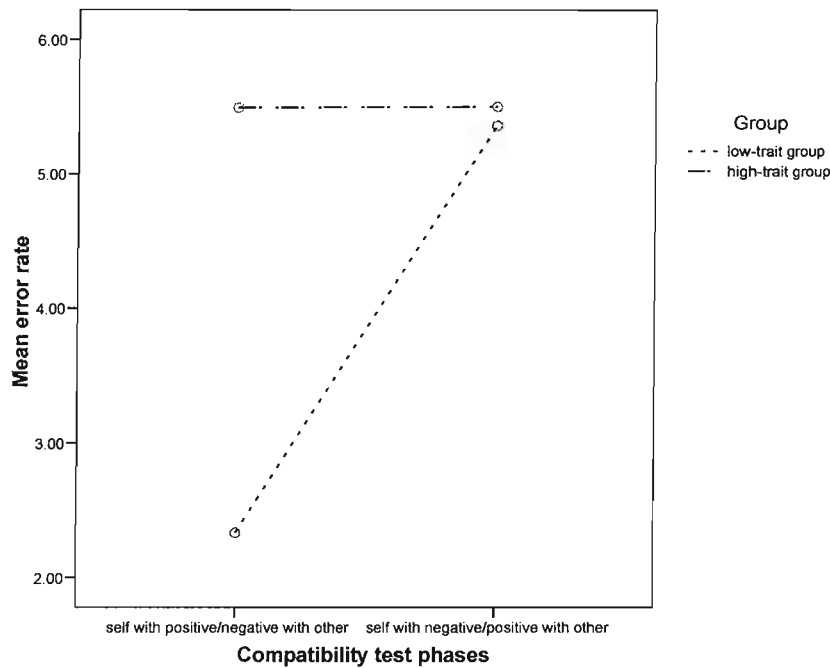
LAT Schema- Analysis of error rates without adding BDI scores as a covariate. This analysis with BDI scores not added as a covariate yielded a significant main effect of compatibility, $F(1, 64) = 16.39, p < .001$, indicating that, overall, all participants had a positive automatic self-evaluative bias. There was also a compatibility by group interaction, $F(1, 64) = 4.49, p < .05$, indicating that the low-trait group had a stronger automatic self-evaluative bias compared to the high-trait group (See Figure 8 below).

Figure 8. Compatibility by group interaction (error rates) without adding BDI scores as a covariate (IAT Schema).



IAT Schema- Analysis of error rates with adding BDI scores as a covariate. In this analysis, there was no main effect of compatibility, $F(1, 63) = .001, p > .05$, indicating that overall the participants did not have a bias to have either a positive or negative automatic self-evaluation. There was however a compatibility by group interaction, $F(1, 63) = 8.747, p < .01$, which indicated that the low trait group had a stronger positive automatic self-evaluation compared to the high-trait group (see Figure 9).

Figure 9. Compatibility by group interaction (error rates) with adding BDI scores as a covariate (IAT Schema).



IAT error rates analyses summary. The results of all the error analyses show that in accord with the hypothesis, the low- trait group showed a stronger positive automatic self-evaluative bias compared to the high-trait group as the low-trait group performed more efficiently on the between the compatible phase of the IAT. This was the case even when controlling for depressive symptomatology (BDI scores) and not controlling for depressive symptomatology.

Specific schema content analysis

The following investigations involved testing the specific schema content hypothesis that a stable and weaker positive automatic self-evaluative bias would be more pronounced in the high-trait group on the IAT Schema and the EAST towards material congruent with themes hypothetically associated with schema development (e.g., themes of abandonment and defectiveness (see Alloy et al., 1999; Parker et al., 2000). In other words, if self-schema structures (in people vulnerable to depression) contain beliefs of themes associated with abandonment and defectiveness, and little positive schematic content, then hypothetically a weak positive self-evaluative bias should be more evident on a schema content IAT compared to a mood descriptor IAT. This is because, individuals who are vulnerable to depression should, theoretically, have a stronger bias to associate the self with negative schema words compared to low mood words if schema material is congruent with the self-schema structure. This should detract from the strength of the positivity of the automatic self-evaluation.

EAST-Specific schema content analyses. The specific schema content EAST analysis involved subdividing the negative stimuli for analysis into one group of negative schema words and one group of low mood words. The hypothesis was that the high-trait group would be quicker and commit fewer errors when negative schema words were associated with the self. A 2 (word-type) x 2 (person) x 2 (group) repeated measures ANOVA was performed for both reaction time and error analyses (with and without adding BDI scores as a covariate). Word-type and person were within subjects factors, and group the between subjects factor.

EAST-Specific schema content reaction time analysis without BDI scores added as a covariate. This analysis did not yield a 3-way interaction of word-type x person x group, $F(1, 64) = .102, p > .05$. This indicates that neither the high-trait or low-trait group had a bias to associate schema nor low mood words with the self. There was, however, a main effect of group, $F(1, 64) = 4.05, p < .05$, which showed that the high-trait group was faster overall to respond to negative words of both kinds when associated with the self. There were no other significant results (highest $p = .088$).

EAST-Specific schema content reaction time analysis with BDI scores added as a covariate. The 3-way interaction was not significant ($F(1, 63) = .030, p > .05$), indicating that the high-trait group did not have a bias to schema words when associated with the self. There were no other significant results (highest $p = .155$).

EAST-Specific schema content error rate analysis without adding BDI scores as a covariate. The error analysis taking BDI scores out as a covariate did not yield a 3-way interaction of word-type x person x group, $F(1, 64) = 2.186, p > .05$. Therefore this analysis shows that there was no effect on either the high-trait group on the kind of negative stimuli to be classified or how efficiently they were processed when associated with the self or other. There was however a main effect of person, $F(1, 64) = 18.68, p < .001$, indicating that overall more errors were made with all word-types when they were associated with other. There were no other significant results (highest $p = .349$).

EAST-Specific schema content error rate analysis with adding BDI scores as a covariate. The hypothesised word-type x person x group interaction was not significant, $F(1, 63) = .229, p > .05$. There was again a main effect of person, $F(1, 63) = 21.28, p < .01$, indicating that overall more errors were made with all word-types when they were associated with other.

EAST-Specific schema content analyses summary. In disagreement with the hypothesis, the high-trait group did not show a weaker positive self-evaluative bias in response to stimuli hypothetically related to schema content when associated with the self.

Specific schema content analysis- IAT reaction time and error analysis. This analysis involved calculating IAT effects for each IAT for both reaction time and error rate data. An IAT effect is calculated by subtracting the average reaction time or error rate of the

compatible block from the incompatible block¹. The larger the score after this calculation is indicative of a stronger positive self-evaluative bias. Research has shown that participants reliably have faster reaction times or fewer error rates on the compatible block of the IAT compared to the incompatible block (de Jong, 2000). In other words, if Person A is very fast or very accurate on the compatible block of the IAT, but very slow or inaccurate on the incompatible block of the IAT, while Person B performs in a relatively similar way on both blocks, then Person A will receive a larger IAT effect score as a result of his/her superior performance on the compatible block. Person A's score will reflect a larger discrepancy between the compatible and incompatible block, which is indicative of a stronger self-evaluative bias. Therefore, by calculating different IAT effects for both IAT's (schema and mood) one can ascertain the relative strength of the positive automatic self-evaluation in each IAT.

The IAT analysis for both error rates and reaction times involved 2 (IAT-Type; Mood or Schema) x 2 (group) repeated measures ANOVA's (with and without adding BDI scores as a covariate), with IAT-type as a within subjects factor, and group as a between subjects factor. IAT effect reaction time scores used for the statistical analysis were calculated from log-transformed reaction times.

IAT Effect-Reaction time analysis without adding BDI scores as a covariate. There was a main effect of IAT type, $F(1, 64) = 4.81, p < .05$, which showed that all participants had a stronger positive automatic self-evaluative bias on the IAT low mood. There was no 2-way interaction of IAT-type by group, $F(1, 64) = 2.08, p > .05$, which shows that the two groups performed similarly on the two IAT's. There were no other significant results (highest $p = .061$).

IAT Effect-Reaction time analysis with adding BDI scores as a covariate. Again, there was a main effect of IAT type, $F(1, 63) = 5.39, p < .05$, which showed that all participants had a stronger positive self-evaluative bias on the IAT low mood. There was no 2-way interaction of IAT-type by group, $F(1, 63) = .002, p > .05$, which shows again that the two groups performed similarly on the two IAT's. There were no other significant results (highest $p = .261$).

IAT Effect-Error rate analysis without adding BDI scores as a covariate. There was no main effect of IAT type, $F(1, 64) = 1.93, p > .05$, which showed that levels of positive automatic self-evaluation in all participants were similar on the two IAT's. There was also no 2-way interaction of IAT-type by group, $F(1, 64) = .250, p > .05$, which shows that the two

¹ One can carry out this calculation the opposite way by subtracting the incompatible block from the compatible block. Either way of doing this calculation gives a relative score. The larger the score after this calculation, compared to another person's score (whether negative or positive) is an indication of a relatively stronger positive self-evaluative bias.

groups did not differ in performance on the two IAT's. There was however a main effect of group, $F(1, 64) = 9.46, p < .01$, which indicated that the low-trait group made fewer errors overall on the compatible blocks of the IAT's. This indicates, as one can see from Figure 10 overleaf, that the low-trait group had overall a stronger positive automatic self-evaluative bias by virtue of having a larger IAT effect score.

IAT Effect-Error rate analysis with adding BDI scores as a covariate. There was no main effect of IAT type, $F(1, 63) = 107, p > .05$, which showed that all participants did not show differences of positive self-evaluation on the two IAT's. There was also no 2-way interaction of IAT-type by group, $F(1, 63) = .106, p > .05$, which shows that the two groups did not differ on performance on the two IAT's. There was again a main effect of group, $F(1, 63) = 11.12, p < .01$, which indicated that the low-trait group made fewer errors overall on the compatible blocks of the IAT's. This indicates, as shown in Figure 11 overleaf, that the low trait group had overall a stronger positive automatic self-evaluative bias by virtue of having a larger IAT effect score.

Figure 10. Main effect of group-IAT effect (error rate without adding BDI scores as a covariate).

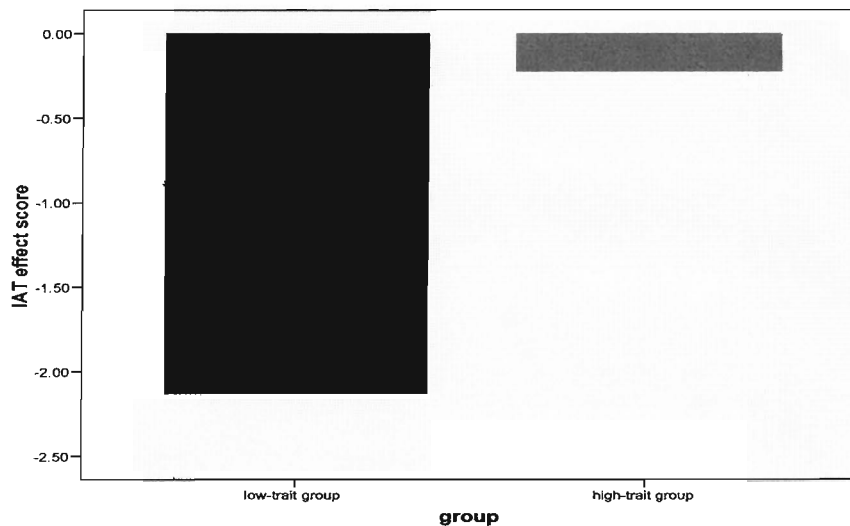
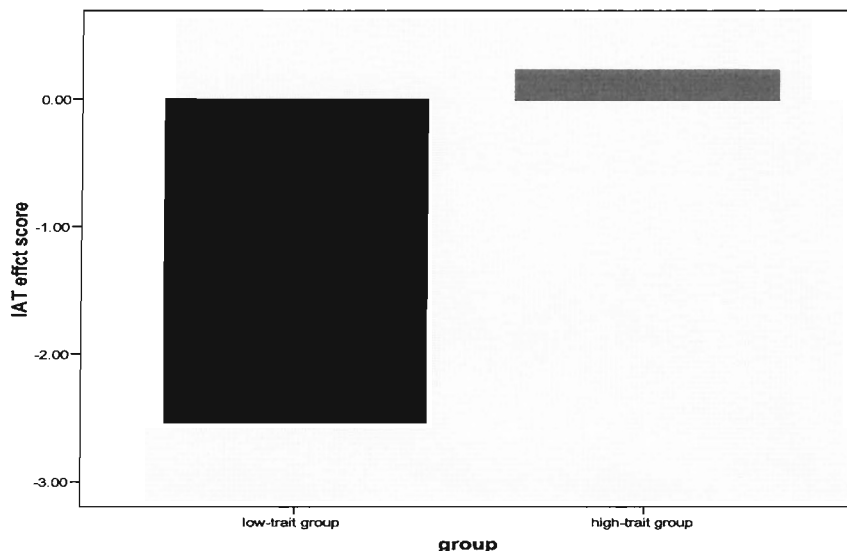


Figure 11. Main effect of group -IAT effect (error rate with adding BDI scores as a covariate).



IAT effect specific schema content analyses summary. In disagreement with the hypothesis, the high trait group did not show a weaker positive self-evaluative bias in response to stimuli related to theoretical schema content. However, on errors, the low trait group showed a stronger positive automatic self-evaluative bias demonstrated by fewer errors on the compatible blocks of the IAT's (larger IAT effect score) compared to the incompatible blocks both when controlling for depressive symptoms and when not controlling for them.

7.4. Discussion

The results in this study partially fit with the hypothesis that a group of low-trait depressed individuals would exhibit a stronger automatic self-evaluative bias compared to a group of high-trait depressed individuals. This was shown on all IAT error analyses (IAT Schema, IAT Mood, & IAT effect analyses) with and without adding BDI scores as a covariate, and on reaction time analyses on the IAT Mood (without BDI scores added as a covariate). Although the high-trait depressed group exhibited depressive characteristics as shown by higher self-report measure scores (e.g., higher DAS, ATQ, RDQ, BDI, BAI, YSQ-Defectiveness/Shame, Failure, Insufficient Self-Control, and EBS Self-Other and Self judgments), they still did not demonstrate, in comparison to the low-trait group, a strong positive automatic self-evaluative bias (on both IAT error analyses) even when controlling for depressive symptomatology. On the other hand, in disagreement with the hypothesis, disappointingly, there were no significant results with the EAST task, apart from a non-significant trend showing that the low-trait group has a stronger positive automatic self-evaluation measuring reaction time with BDI score added as a covariate. This issue will be

discussed later. Further, in disagreement with the specific schema content hypothesis (Alloy et al., 1999), there was no evidence that a group of high-trait depressed individuals had a weaker positive automatic self-evaluative bias to stimuli related to hypothetical schema content on both the EAST and IAT analyses.

Specific schema content biases versus positive automatic self-evaluation in vulnerability to depression

The fact that the low-trait group exhibited a stronger positive automatic self-evaluative bias compared to the high-trait group is in line with recent ideas on theoretical vulnerability to affective disorders using tasks like the IAT (Tanner et al., in press; de Jong 2000). It is argued that a weak positive automatic self-evaluation is a feature of individuals who are vulnerable to depression, and is measurable in non-depressed or asymptomatic states (Alloy et al., 1999). This study's findings fit well with arguments that the presence of a positive self-evaluative bias may be essential for good mental health and adaptation to stressful environmental situations that may give rise to a depressive episode (De Raedt et al., 2006; de Jong, 2000; Taylor & Brown, 1988).

However, with regard to the theoretical postulates of an epistemological (cognitive structural) model of schemas (Gamar et al., 2001; Segal, 1988) and vulnerability to depression (Beck, 1967, 1976), this study shows little evidence of specific schema content being implicated in vulnerability to depression, even taking into consideration mood and associated depressive symptoms. If content specificity is an important factor implicated in vulnerability to depression, then one would have expected the high-trait group to have a weaker positive automatic self-evaluative bias when hypothetical schema content stimuli is paired with the self on the implicit processing tasks. This certainly was not the case. The results of the IAT effect analyses (error and RT with and without BDI scores added as a covariate) and the EAST analyses comparing both types of negative words (with and without BDI scores added as a covariate), did not show a difference in automatic self-evaluation in the high-trait group when schema words were associated with the self. Further, the typical negative information processing biases seen in depression were also not evident in this study. If anything, it was the low-trait depressed who showed a greatest bias: a strong positive automatic self-evaluative bias.

This result is problematic as it is not in accordance with the cognitive model of depression and the notion of vulnerability being linked to self-schema constructs containing certain content, (Segal et al., 1999; Segal, 1988; Beck, 1967, 1976). However, it seems feasible to argue that the overwhelming evidence that depressed individuals or those vulnerable to depression (when induced into a low mood) and associated negative processing biases (see Ingram et al., 1998), may well be a function of mood (and possibility cognitive scarring from an episode of depression; Segal et al., 1999), but that vulnerability to depression

may be displayed more subtly. In other words, perhaps a lack of a strong positive self-evaluation bias is what is relevant to enduring vulnerability to depression but not a bias to negative words per se. From the results of research using the IAT, it has been argued that a strong positive automatic self-evaluative bias in individuals not vulnerable to affective disorders is what distinguishes them from those individuals who are vulnerable to affective disorders (de Jong, 2000). Agreeing with the idea of a weaker positive self-evaluation being linked to vulnerability to depression was research conducted investigating adolescent depression. Southall & Roberts (2002) observed that the existence of low self-evaluation coupled with high life-stress resulted in adolescents developing depression. Individuals with a positive self-evaluation reacted to life stress in a more adaptive way and did not subsequently develop depression. However, in their study self-evaluation was measured with explicit self-report measures. Therefore, perhaps the high scores on this measure may have been the result of a transient low mood or mild to moderate depressive symptomatology pre-existing before the development of depression (Miranda & Pearsons, 1988). However, other studies have typically failed to find a difference in explicit self-evaluation ratings of individuals who are at risk of developing depression and those who are not (Luxton & Wenzlaff, 2005). This conflicting evidence may highlight the lack of reliability of using self-report measures (Nisbett & Wilson, 1977). Nevertheless, Southall & Robert's (2002) study does provide some support that a stable and strong positive self-evaluation might be a protective factor from the development of depression.

Error rates versus response speed

The results of this study showed that error rates may be a useful tool to measure automatic self-evaluation and hypothetical vulnerability to depression. Overall, a stronger positive self-evaluative bias was found in the low-trait group when analysing reaction times on only one occasion with the IAT Mood analysis with BDI scores not added as a covariate. However, on all the error analyses of the IAT's (with and without adding BDI scores as a covariate), it was found that the stronger positive automatic self-evaluative bias in the low-trait group was evident. These results provide support for the usefulness of analysing error rates as opposed to response latencies from the data of implicit processing tasks, which is rarely addressed (e.g., Gamar et al., 2001; De Raedt et al., 2006). It has been posited that error rates may be a more accurate indicator of automatic self-evaluation (Greenwald et al., 1998) and may be more sensitive to tapping into vulnerability to depression (Eysenck, 1991). This is because response speed may be affected by controlled or explicit processes rather than implicit processes commonly attributed to schematic functioning (Beck, 1967, 1976; Bargh & Tota, 1988). The fact that De Raedt et al. (2006), who also used the IAT, did not find a weaker positive automatic self-evaluation in depressed individuals compared to non-

depressed individuals using only reaction time data in their analyses, perhaps points to the usefulness of using error rates as a measure of vulnerability to depression.

Problems of EAST and IAT compatibility results

The EAST results in this study were disappointing, as no significant results were obtained as opposed to the pattern of results obtained in the IAT's, especially considering the encouraging results obtained by the EAST in Study 1. However, the results obtained by the EAST in Study 1 may have been an artifact of the procedure used: the EAST was the final task for participants, but in this study the order of tasks was counterbalanced. Therefore the confounding effects of priming in Study 1 may have influenced the result (J. De Houwer, personal communication, November, 12, 2001).

However, in this study there may have been problems with stimulus selection that were overlooked, which may have involved important imbalances between the positive and negative words that were used in the EAST and IAT's. These imbalances may have contributed to the poor results of the EAST and also the lack of a consistent overall compatibility bias on the IAT's; participants were significantly quicker or more efficient on the compatible blocks of the IAT (e.g., Greenwald et al., 1998). With the IAT tasks, compatibility effects were only observed on four out of the eight IAT analyses, despite the revisions made to this task in Study 2. The problems of stimuli in both the EAST and IAT tasks may be to do with the fact that positive words were used which were not reflective of the opposites of the schema words or low mood words. Essentially, it would be preferable to use positive schema words in a task alongside negative schema words, instead of generic positive words, and when using low mood words, one uses in conjunction positive mood words. Furthermore, there was a grammatical mixture in the type of words used: some words were nouns and some were adjectives (although there was no statistically significant difference in word types between negative and positive words). Such slight alterations in the salience of words may dramatically alter response speed and error rate (De Houwer, 2001). It has been shown that non-words, or words that differ at some explicit or implicit level of physical or conceptual structure, can be evaluated more negatively than "true" negative words on tasks like the IAT due to the salience of a non-word (De Houwer, 2001). This argument is also applicable to this study's use of non-matching general positive words alongside negative schema or low mood words. It could be argued that the positive self-evaluative bias found in the non-trait depressed group could be an artifact of the positive words used. Overall, either group of positive words was not descriptive of positive mood or positive schemata. Therefore, if such words were used, the strong positive self-evaluative bias effects observed by the low-trait depressed group may not have occurred. The low-trait group may not have such a strong "association" with positive mood or positive schematic concepts with the self. The very "neutral" positive words may be more salient to the low-trait group as cultural or socially

ingrained concepts due to their life experiences, but may not be related to self-evaluation or self-esteem per se. These issues may be pertinent to the poor results of the EAST and irregular compatibility biases on the IAT's where poor matching of stimuli may have had deleterious effects (De Houwer, 2001, 2003; Brendl, Messner, & Markman, 2000).

Further methodological considerations

It must be noted that in this study there was not a "pure" sample of trait depressed individuals. The high-trait group differed significantly on explicit measures of anxiety, depressive rumination, dysfunctional attitudes, automatic thoughts, evaluative beliefs (Self-Self and Self-Other), maladaptive schemas (Defectiveness/Shame, Failure, & Insufficient Self-Control), as well as symptoms of depression. Ideally, it would be preferable to obtain a group of high-trait depressed individuals who do not differ on depressive symptomatology and associated depressive cognition and beliefs. However, by virtue of being high-trait depressed, statistically, it is probably extremely difficult to sample such a group who do not differ on some measures of depressive cognition and depressive symptoms. Depressive symptoms and associated depressive cognition are interlinked (Beck, 1967). Therefore, in retrospect perhaps using depressive symptoms as a covariate is problematic. One could argue that by using BDI scores as a covariate, one is taking out aspects of depressive traits as the BDI, although measuring depressive symptoms on the day, also takes into account symptoms over a two-week period. Thus, it would have been perhaps wiser to include a "present mood measure" like the Visual Analogue Scale (VAS; Grossberg & Grant, 1978) to use as a covariate instead of BDI scores. By being more precise and only factoring out present mood, any analyses may have provided more intriguing and accurate results. Using this method, one may not cancel out as many important variables associated with trait depression, but instead factor out solely the effects of mood on information processing (Zemore et al., 1990). However in the case of all the IAT error analyses, this did not seriously impede the results as the findings in both cases (BDI scores as covariate and BDI scores not added) were the same - the low-trait group showing a positive automatic self-evaluation.

A final criticism of the results of this study could be attributed to a central executive account of depression. Depression has been found to be associated with a dysfunction in executive processes. It has been argued that depressed or dysphoric individuals have impairment in allocating processing resources on tasks measuring information processing leading to poorer performance, compared to those who do not have depression (Channon & Green, 2006; Elliot, Baker, & Rogers, 1997; Watts, Macleod, & Morris, 1998; Hertel & Hardin, 1990; Elliot, Sahakian, McKay, 1996). As was observed in this study, the high-trait group in comparison to the low-trait group performed more similarly on the compatible and incompatible phases of the IAT's, suggesting that performance may have been impaired. In other words, the differences observed in performance by the high-trait group may not be due

to a lack of a positive automatic self-evaluative bias, but instead to dysfunctional executive processes. However, it has been argued that performance deficits associated with central executive dysfunction by depressed people only occur on tasks that involve controlled processes but not on tasks measuring automatic processes (Ellis & Ashbrook, 1998; Hasher & Zacks, 1979; Hertel, 1994; Teasdale & Barnard, 1993). Therefore, on such tasks like the IAT, a criticism of executive functioning affecting performance may not be applicable, as the IAT is supposed to measure automatic processes (Greenwald et al., 1998). However, De Houwer (2006) argues that controlled processes may at times affect performance on tasks like the IAT. Thus the IAT could measure controlled processes, automatic processes, or a combination of both. This issue of how executive function and automatic self-evaluation are related and how this affects interpretation of data from the IAT needs to be addressed by further research.

Conclusion

This study has shown that individuals classified as low-trait depressed, and who are hypothetically not vulnerable to depression, possess a positive automatic self-evaluative bias. The high-trait depressed, on the other hand, did not exhibit a strong positive automatic self-evaluation. This indicates that automatic self-evaluation may be implicated in vulnerability to depression. Further, the use of errors as a measure of automatic self-evaluation and hypothetical vulnerability to depression appears to be a valid way of measuring information processing biases. Levels of automatic self-evaluation were stable when using error rates as a measure, even when taking into consideration depressive symptoms and associated low mood. There was also no evidence that schema content specificity is implicated in theoretical vulnerability to depression. However, there were problems with the stimuli as some words were adjectives and some were nouns, which may affect information processing performance. It could also be argued that the positive stimuli used in this study may have to be revised for future studies to provide a clearer picture of automatic self-evaluation in its role in vulnerability and non-vulnerability to depression. Further, the specific measurement of present mood was not assessed in the participants. The role of executive function in the trait depressed affecting the interpretation of the results is also an issue that needs to be addressed by more research to make more firm conclusions of the role of automatic self-evaluation and executive function with tasks like the IAT. These issues have to be rectified to enable one to make more firm conclusions and inferences about the roles of automatic self-evaluation, schema content specificity, and error rates as a reliable measure with regards vulnerability to depression. It would be of interest to explore how a low mood or episode of depression affects error rates and automatic self-evaluation. If a strong and stable positive automatic self-evaluation is implicated in non-vulnerability to depression, one might expect in response to a low mood, a positive self-evaluation would remain stable. Study 4 in the next chapter addressed these issues.

Chapter 8

Study 4 - Negative schematic processing in high and low trait depression: The effects of a negative mood induction

8.1. Introduction

Following on from the previous study, the next logical step was to investigate the effects of an induced negative mood on automatic self-evaluation in high and low-trait depression. In Study 3 it was found that individuals who were classified as low in trait depression had a stronger positive automatic self-evaluation on the Implicit Association Task, compared to a group who were high in trait depression, even after controlling for depressive symptomatology. These results may indicate that a positive automatic self-evaluative bias may be a protective factor from the development of depression, whereas a weak or reduced positive automatic self-evaluative bias might represent vulnerability in depression. Therefore, an investigation into the effects of a negative mood induction may help to ascertain if a strong positive automatic self-evaluation is resilient to the effects of a low mood, and thus may hypothetically prevent a spiral into depression.

In Study 3, there was also no evidence of a negative self-evaluative bias in the high-trait depressed group, even though they were experiencing significant depressive symptomatology in comparison to the low-trait group (when BDI scores were not added as a covariate). This result is inconsistent with contemporary cognitive theories of depression and with ideas about the role of negative schemas and schema activation producing subsequent negative information processing biases (Beck, 1967, 1976; Segal et al., 1999; Gemar et al., 2001). However, it may well be that a negative information processing bias characteristic of depression is only exhibited under certain circumstances - for example, during periods of transient low moods (Gemar et al., 2001). Therefore, the high-trait group, even though they reported more depressive symptoms than the low-trait group, may not have been in a low enough mood to produce the characteristic negative information processing biases seen in schema research (e.g., Gemar et al., 2001; Segal et al., 1999; Hedlund & Rude, 1995; Rude et al., 2001). The next reasonable step, therefore, was to investigate the effects of an induced negative mood on automatic self-evaluation in individuals classified as having either high or low traits of depression. This may help to ascertain if an induced low mood is necessary to activate the characteristic negative information processing bias usually seen in depression, or if hypothetical vulnerability to depression is indeed characterised only by a lack of a positive automatic self-evaluative bias.

Mood induction research in depression

As discussed in detail in Chapter 3 (see 3.2.), much of the research has found that formerly depressed individuals who are hypothetically vulnerable to develop depression, after being induced into a low mood, preferentially process negative information. It has also been

found that individuals who are vulnerable to depression score higher on self-report measures of depressive cognition after a negative mood induction compared to non-vulnerable individuals (Gemar et al., 2001; Williams, 1988; Rude et al., 2001; Hedlund & Rude, 1995). However, when formerly depressed and non-depressed individuals are in a comparable mood state (normal mood), no differences have been found between groups on measures of information processing or on self-report measures of depression (Gemar et al., 2001; Gotlib & Cane, 1987). However, as discussed in detail in Chapter 4, there exist many methodological problems that potentially contribute to the reasons as to why there is little evidence of schematic activity in the absence of a depressed mood or without the effects of priming or a negative mood challenge.

New information processing paradigms and their application in mood induction research

The tasks used in this thesis (the IAT and EAST) could provide a satisfactory way of evaluating schema processes related to self-evaluation whilst filtering out the effects of mood. Greenwald et al. (1998) argue that the IAT may be sensitive enough to measure implicit constructs like self-esteem and not be affected by the potentially confounding effects of mood or controlled processing strategies that may affect other tasks (e.g., Stroop Task, Recall tasks; Hedlund & Rude, 1995; Rude et al., 2001). Indeed, as Stapel & Blanton (2004) demonstrated, automatic self-evaluation appears to be a stable construct and is not affected by changes in mood. This is also supported from other research that suggests that implicit self-evaluation is more resilient to the effects of mood compared to explicit self-evaluation (Pelham & Hetts, 1999). As observed in Study 3, the low-trait group had a stronger positive automatic self-evaluative bias (on the IAT) compared to the high-trait group when controlling for significant differences in symptoms of depression between the two trait groups. However, when depressive symptoms were not controlled for, there was still no evidence of a negative bias in the high-trait depressed group, and the strong positive self-evaluative bias was still evident in the low-trait depressed group. The results of previous research and the results from Study 3 may indicate automatic self-evaluation is a stable construct and is not influenced by mood.

The results of other research initiatives using the IAT in depression research, however, are mixed. De Raedt et al. (2006) found that currently depressed individuals had a relatively positive self-evaluative bias, even though they scored significantly higher (more negative) on the Beck Depression Inventory (Beck et al., 1979). However, De Raedt et al. did not look at error rates as a measure of self-evaluation. This is important, as Bargh (1988) argues that error rates may be a more accurate reflection of vulnerability to depression. Gemar et al. (2001) found that those individuals who had recovered from depression showed a negative self-evaluative bias on the IAT after, but not before, a negative mood induction. However, if Gemar et al. had analysed their data differently they would have observed that, overall, the self-evaluative bias in the recovered depressed group was only more labile as a

result of the mood induction and that actually the positive self-evaluative bias was less pronounced in the recovered depressed group (De Raedt et al., 2006). Further, like De Raedt et al.'s study, Gemar et al. did not use error rates as a measure of self-evaluation and, thus, this crucial data may have been more informative as to the nature of self-evaluation in individuals who had recovered from depression.

However, de Jong (2000), in accordance with the results found in Study 3, is in agreement with the hypothesis that a weak positive automatic self-evaluative bias is implicated in those vulnerable to affective disorders. In de Jong's (2000) study with socially anxious individuals using the IAT, the crucial difference between socially anxious and non-socially anxious individuals was that the socially anxious individuals had a weaker positive automatic self-evaluative bias. Indeed, Tanner et al. (in press) found that after a social-threat activation task (similar to a negative mood induction), the socially anxious individuals did show a positive automatic self-evaluative bias, but this was weaker in the socially anxious group compared to non-socially anxious individuals. Therefore, there it seems that further investigation into the role of positive automatic self-evaluation is warranted. Indeed, Taylor & Brown (1988) argue that an overly positive self-evaluation may be crucial as a protective factor for good mental health. However, there is very little research using the IAT or EAST with depression that investigates the effects of negative mood. Therefore, this study may be able to advance knowledge in this area and the role of automatic self-evaluation in relation to vulnerability to depression and the effects of mood.

Aims and Hypothesis

The aim of this study was to investigate the effect of a negative mood induction on individuals with high and low trait depression, and to see whether inducing a negative mood produces a negative self-evaluative bias at an implicit and explicit level of cognition in individuals classified as being high-trait depressed. Another aim of this study was to ascertain if a positive automatic self-evaluation is a stable construct in individuals classified as being low-trait depressed and thus a hypothetical protective factor for individuals not to develop depression. A subsidiary aim, following from the results in Study 3, was to investigate the role of errors compared to reaction times in assessing automatic self-evaluation.

It was hypothesised that 1) a group of individuals classified as being high-trait depressed would not exhibit a strong positive automatic self-evaluative bias (as shown by errors and/or reaction time on the IAT) as compared to the low-trait depressed group, and that the high-trait group's automatic self-evaluative bias would become more negative as a result of the negative mood induction; 2) a group of individuals classified as low-trait depressed would exhibit a stronger positive automatic self-evaluative bias, and that this bias would be relatively stable and unchanged compared to the high-trait depressed group, despite the effects of a negative mood induction.

82. Method

Participants

Participants in this study were undergraduate students from the University of Southampton. Individuals were recruited from lectures, advertisement, and opportunity sampling methods. Exclusion criteria included the use of any medication that would interfere with motor skill, previous history of depression, colour blindness or eyesight problems, and English a second language. Fifty-four participants took part in the experiment. Twenty-nine participants were assigned to a low-trait depressed group and had a mean DPRS (measure of trait depression-see below) score of 3.68 (SD=. 92), and 25 participants were assigned to a high-trait depressed group and had a mean DPRS score of 5.78 (SD=. 70). The difference in DPRS scores between the groups was significant, $t(52) = 9.20, p < .001$. The mean age of the low-trait depressed group was 21.37 years (S.D. =2.32) and the mean age of the high-trait depressed group was 22.12 years (S.D. = 4.54). The low trait group comprised 14 males and 15 females, and the high trait group comprised 11 males and 14 females. There was no statistical difference between the groups on age, $t(52) = .769, p > .05$, or on gender composition, $\chi^2(1) = .099, p > .05$.

Materials

Self-Report measures. For full details of the DPRS and BDI please see method section of Study 3.

Depression Proneness Rating Scale (DPRS, Zemore et al., 1990). The DPRS measures thoughts and behaviours indicative of being depressive traits associated with a risk of developing depression.

The Beck Depression Inventory-II (BDI II; Beck et al., 1996). Measures the severity of depressive symptomatology.

Visual Analogue Scale (VAS; Grossberg & Grant, 1978). Participants rated current mood on a visual analogue scale (see Appendix III). The VAS is a horizontal line measuring 140mm. The descriptor SAD is located at the left end point, while the descriptor HAPPY is located at the right end point. Participants were instructed to place a mark on the line to indicate how happy or sad they presently felt. The position of the mark on the line indicated how strongly an individual felt happy or sad. Scoring consisted of measuring mood in millimeters and (0-140): 0 being extreme sadness, 140 being extreme happiness.

Dysfunctional Attitudes Scale-A & B Forms (DAS, Weissman & Beck, 1978). This questionnaire was used to assess the endorsement of conditional dysfunctional beliefs that theoretically guide a person's self-evaluation before and after a negative mood induction. These beliefs are implicated in accounts of cognitive vulnerability to depression because they are presumed to be more enduring than the negative automatic thoughts that are characteristic

of depression and associated depressed mood (Kovacs & Beck, 1978). However, Gemar et al. (2001) found that DAS scores were affected by negative mood and thus may not be as stable as previously supposed. The DAS has two forms (A & B), which show good equivalency (Weissman, 1979; Weissman & Beck, 1978; see Hammen & Krantz, 1985 for a review), good internal consistency (alphas ranging from .89 to .93) and good test re-test reliabilities ranging from .71 to .84 (Hamilton & Abramson, 1983; O'Hara, Rehm, & Campbell, 1982; Weissman, 1979).

Negative Mood Induction. Participants were asked to listen to a piece of music presented on a Walkman and to try and recall a time in their lives when they felt sad. The piece of music was the orchestral introduction by Prokofiev entitled "Russia under the Mongolian Yoke" from the film *Alexander Nevsky*. The piece of music was re-mastered at half speed and presented through headphones for 5 minutes. This type of mood induction, combining elements of music associated with sad mood and autobiographical recall, is established as an effective procedure for bringing on a transient dysphonic mood state that lasts for several minutes (Clark & Teasdale, 1985; Martin, 1990).

Positive Mood Induction. At the end of the experimental session, participants were asked to listen to a piece of music presented on a Walkman and to try and recall a time in their lives when they felt happy. This procedure was employed to counter any lingering effects of the negative mood induction. The piece of music used was Schubert's *Scherzo: Presto*. This is a very upbeat piece of music and, because of its underlying musical grammatical structure and the associated autobiographical recall, is effective in producing a transient positive mood state (Govern, & Marsch, 1997). This piece of music lasted approximately 5 minutes.

The Implicit Association Task (IAT, Greenwald et al., 1998). The IAT design was similar to the IAT in Studies 2 and 3 apart from one exception; the lists of positive and negative words were altered to take into consideration factors that may have affected the results obtained in Study 3 regarding response time and error rates. This involved the salience of words (discrepancies in thematic categories between words) and word structure (grammatical differences in words; e.g., adjectives or nouns) that could affect processing efficiency (De Houwer, 2001). The IAT task used in this study was the IAT Schema as used in the previous study. However, unlike Study 3, all the words were now adjectives. The negative words used were related to the early maladaptive schemas of defectiveness/shame & abandonment. The positive words comprised a list of positive schema words that were considered the categorical opposites of the negative schema words. The positive schema words were selected with a similar procedure to that described in the method section in Study 1; 20 post-graduate students had to rate if a positive schema word was a categorical opposite of a negative schema word. A positive schema word was selected if it had a mean

applicability rating (being the opposite of a negative schema word) of at least 7. The negative schema words were also selected using the same procedure as Study 1. The two groups of words were balanced for frequency, number of letters, syllables, emotionality, and pleasantness. See Appendix IV for details of words used in Study 3.

Procedure

Participants first completed a VAS (to measure initial mood), the DPRS trait depression measure, and the BDI as a measure of depressive symptomatology. A median split was taken on the DPRS to classify individuals as being either high-trait or low-trait depressed. Mean scores of 1 to or equal to 5 on the DPRS assigned participants to the low-trait depressed group and mean scores of above 5 to 9 assigned participants to the high-trait group. Individuals who scored below 2 on the BDI were excluded as scores of 0 or 1 may suggest that other forms of psychopathology are present (e.g., psychopathy, hypomania) (Hammen, 1980). After initial screening with the DPRS & BDI, participants who met the other study criteria (see participant section) were invited to participate in the next stage of the experiment. Participants were given an information sheet to read, and a consent form to sign that ensured confidentiality. Testing took place in a small cubicle with optimum lighting with no outside light or noise protruding. Participants were situated approximately 12-18 inches from the computer screen. Participants were told that they would complete two computer tasks, a variety of questionnaires, and listen to two pieces of music on a Walkman. With regard to the IAT, participants were told that they would be presented with instructions telling them to classify words related to themselves or words related to someone else, negative words, and positive words by pressing the either the P or Q key on the computer keyboard. During the entire experimental procedure the experimenter left the room after each experimental task was set up, and waited outside until the participant finished each task. The order of tasks for each participant after the initial screening (i.e., DPRS, VAS & BDI) was as follows: VAS 2, 1st IAT, DAS A or B (counterbalanced across participants), VAS 3, Negative Mood Induction, VAS 4, 2nd IAT, VAS 5, DAS A or B (again counterbalanced), Positive Mood Induction, VAS 6¹. Like Studies 1, 2, & 3, key assignments on the IAT were counterbalanced and to take into consideration practice effects as each participant completed two IAT tasks. After they had completed all the stages of the experiment, participants were debriefed. Participants were paid £5 for their participation or given credits in fulfilment of their respective degree course. The whole procedure took approximately 1-1/2 hours for each participant to complete.

¹ The VAS was administered 6 times during the course of the experiment. The VAS analysis therefore looked at the change of mood over time uses 6 time points. All participants received the negative mood induction first. The positive mood induction was used to restore mood state after the negative mood induction for ethical purposes.

8.3. Results

The questionnaire results are presented first followed by the reaction time and error rate results of the two IAT's. The reaction time data from the IAT's was treated in the same way as described in Study 3. Unless reported in the results, non-significant results and minor effects and interactions from the conducted ANOVA's on the IAT and self-report data are presented in Appendix V. An alpha level of .05 was used for all initial ANOVA's. All post hoc analyses (e.g., independent and paired t-test's) used Bonferroni correction techniques to reduce the likelihood of false positive results. This involved dividing the alpha level (.05) by the number of tests conducted. This will be referred to in the text as (.05/N).

To examine and control for the effects of differential levels of mood and depressive symptomatology between the high and low trait groups, all analyses from the IAT's will take the form of reporting the results of the IAT's, firstly with no covariate added to the statistical analysis, secondly with an mean VAS mood measure (average VAS scores over the six VAS administrations) added as a covariate, and lastly with BDI scores added to the analysis as a covariate. Carrying out the analysis in this way gives an indication of the relative effects of depressive symptoms (BDI) and present mood (VAS) and how this affected implicit processing performance on the two IAT's. The reason that an average VAS measure was used instead of the degree of change on the VAS as a result of the negative mood induction was that 1) despite all participants' mood being significantly lowered by the negative mood induction, the negative mood induction did not in any way affect participants' performance on the second IAT; and 2) at no point was mood between the high and low trait groups significantly different as measured by the VAS, 3) but overall over the six time points (as shown below) the high-trait group had a significantly lower mood rating.

VAS Mood Measure

To investigate changes in mood levels throughout the experiment and the effects of the negative and positive mood inductions, a 6 (time) x 2 (group) repeated measures ANOVA was performed (See Table 8 for mean VAS scores). There was a main effect of time, $F(5, 260) = 65.57, p < .001$, a main effect of group, $F(1, 52) = 4.23, p < .05$, but a non-significant interaction of time by group, $F(5, 260) = 1.15, p > .05$. These results indicated that participants' moods changed over time and that, overall, the high-trait group had a sadder mood ($M = 82.9, SD = 20.5$) compared to the low-trait group ($M = 94.4, SD = 20.5$), but that no significant changes of mood as a function of time was evident when comparing groups at individual time points.

Pairwise, t-tests using a Bonferroni corrected p value of .003 (.05/15) indicated that the negative mood induction (time 3 compared to time 4) had the effect of significantly lowering mood, $t(53) = 10.08, p < .001$. Further the positive mood induction at time 6 had the effect of making participants significantly happier, $t(53) = 8.50, p < .001$. Therefore, the

negative and positive mood inductions were successful in altering mood in the desired directions.

Time 1 mood ratings were significantly happier than time 3, $t(53) = 3.76, p < .001$, and time 5, $t(53) = 7.19, p < .001$, but not time 6, $t(53) = 1.78, p > .05$. These results indicate that after the first administration of the IAT and subsequent administration of the DAS and third VAS, mood was becoming significantly sadder even before a negative mood induction, and that mood levels at the end of the experiment had returned to the similar levels before the experiment had began.

There was a significant difference in mood between time 4 and time 5, $t(53) = 5.98, p < .001$, which indicated that mood had become significantly happier by the end of the second IAT (post negative mood induction), before the positive mood induction. This indicated that the increase of negative mood was transient, and that the effects of the negative mood induction were already wearing off. However, mood was still significantly sadder at time 5 compared to time 1, indicating that the negative mood persisted to some extent. The lowest mood ratings were also observed at time 4 after the negative mood induction.

Table 8

Mean Visual Analogue Scale Mood Ratings for the High and Low Trait Group

	Group	Mean VAS score (mm)	S.D
VAS Time 1	High-trait	94.4	27.5
	Low-trait	104.7	23.0
VAS Time 2	High-trait	87.8	25.4
	Low-trait	103.2	21.8
VAS Time 3	High-trait	86.2	22.4
	Low-trait	97.7	21.7
VAS Time 4*	High-trait	54.8	26.3
	Low-trait	71.7	28.5
VAS Time 5	High-trait	73.8	20.4
	Low-trait	82.3	26.7
VAS Time 6**	High-trait	100.6	20.2
	Low-trait	106.9	21.4
VAS Average	High-trait	82.9	20.5
	Low-trait	94.4	20.3

* ratings after negative mood induction, ** ratings after positive mood induction.

Beck Depression Inventory

The two groups significantly differed from each other on BDI scores, $t(52) = 4.47, p < .001$. The mean BDI score for the high trait group was 14.04 ($SD = 8.83$), and the low trait group's mean BDI score was 6.06 ($SD = 3.38$). This showed that the high trait group had higher levels of depressive symptomatology and this was just within levels characterised by Beck et al., (1996) as mild levels of depressive symptomatology.

Dysfunctional Attitudes Scale

To investigate whether DAS scores were affected by the negative mood induction, a 2 (time) x 2 (group) repeated measures ANOVA was performed. There was a main effect of group, $F(1, 52) = 4.89, p < .05$. This indicated that the high-trait group overall scored significantly higher on the DAS ($M = 146.08, SD = 26.80$), compared to the low trait group ($M = 131.46, SD = 21.46$) (See Table 9 for DAS scores).

There was no main effect of time, $F(1, 52) = 1.40, p > .05$, and a non-significant time by group interaction, $F(1, 52) = 1.68, p > .05$. These results indicate that overall DAS scores did not significantly change with the effects of the negative mood induction, and that DAS scores in either group were not significantly affected by the negative mood induction. Lastly, it also indicates that there was no difference in DAS scoring between groups before or after the negative mood induction.

To check the equivalence of the two DAS forms, a 2 (form; A or B) x 2 (Group; AB, or BA) repeated measures ANOVA was performed to see if overall the order of presentation of the DAS forms had an effect on DAS scoring; group AB receiving form A before the negative mood induction, and group BA receiving form B before the negative mood induction. There was a main effect of form, $F(1, 52) = 10.06, p < .01$, but a non-significant form by group interaction, $F(1, 52) = .201, p > .05$. This indicated that participants scored significantly higher on the DAS B form (B form- $M = 141.31, SD = 26.52$; A form- $M = 131.14, SD = 26.09$), but that the order of presentation did not affect DAS scores. Therefore, the negative mood induction did not affect scoring on the DAS.

Table 9

Mean DAS Scores of the Low and High Trait Group

	Group	Mean DAS Score	S.D.
DAS pre-negative mood	High-trait	145.96	28.28
induction	Low-trait	134.06	22.26
DAS post-negative	High-trait	146.20	27.44
mood induction	Low-trait	128.86	23.80
DAS Average	High-trait	146.36	26.83
	Low-trait	131.41	21.46

IAT Analysis

The IAT reaction time and error analysis involved testing the hypothesis that the low-trait group would be quicker to respond and/or make fewer errors to stimuli on the compatible block (self associated with positive schema words) compared to the high-trait group. It was also hypothesised that the low-trait group would not be affected by the negative mood induction, unlike the high-trait group whose automatic self-evaluation would become more negative as a result of the negative mood induction. This would be evidence that the low-trait group had a more stable and stronger positive automatic self-evaluative bias compared to the high-trait group. A 2 (compatibility) x 2 (time) x 2 (group) repeated measures ANOVA was performed on the reaction time and error data, firstly with no covariate added, then with an average VAS score, followed by BDI score, added as covariates to examine and control for the effects of mood and depressive symptomatology. The reaction time results are presented first, followed by error results. See Table 10 and 11 below for mean reaction times and error rates from the IAT's. The reaction time data from the IAT's was treated in the same way as described in the previous three studies.

Table 10

Mean Reaction Times (ms) and Errors on the IAT Pre and Post Negative Mood Induction for the High and Low Trait Groups

Group	Compatibility test phase	Pre/Post NMI*	Mean reaction time	S.D.	Mean error rate	S.D.
High- trait	Compatible	Pre NMI	693.13**	120.3	3.52**	2.38
			691.15 (VAS)	123.9	3.38 (VAS)	2.41
			683.48 (BDI)	132.45	3.54 (BDI)	2.64
		Post NMI	642.69**	104.2	5.280 **	4.39
			635.85 (VAS)	104.95	3.60 (VAS)	2.51
			632.08 (BDI)	114.2	3.80 (BDI)	2.76
	Incompatible	Pre NMI	813.01**	242.1	3.76**	2.48
			802.27 (VAS)	247	5.27 (VAS)	4.53
			817.96 (BDI)	268.4	5.43 (BDI)	4.87
		Post NMI	724.42**	178.3	5.0 **	3.56
			712.41 (VAS)	179.3	4.73 (VAS)	3.60
			723.93 (BDI)	197.8	5.01 (BDI)	3.95
Low- trait	Compatible	Pre NMI	665.06**	120.18	2.31**	2.37
			666.77 (VAS)	123.41	2.42 (VAS)	2.39
			673.38 (BDI)	120.73	2.29 (BDI)	2.60
		Post NMI	597.67**	104.10	4.13**	4.38
			603.57 (VAS)	104.53	3.16 (VAS)	2.50
			606.85 (BDI)	112.76	2.99 (BDI)	2.72
	Incompatible	Pre NMI	832.90**	241.83	3.03**	2.49
			842.15 (VAS)	246.08	4.14 (VAS)	4.51
			828.63 (BDI)	264.96	4.00 (BDI)	4.79
		Post NMI	756.02**	178.13	5.58 **	3.55
			766.38 (VAS)	178.61	5.76 (VAS)	3.59
			756.45 (BDI)	195.29	5.57 (BDI)	3.90

* Pre or post negative mood induction, ** No covariate added, (VAS) =VAS added as a covariate, (BDI) = BDI added as a covariate.

Table 11

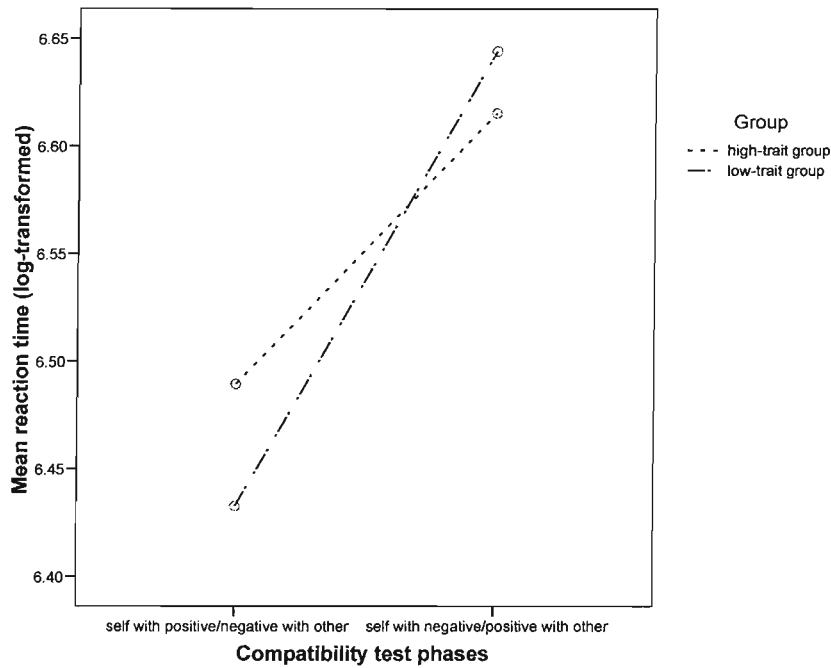
Mean Reaction Times (ms) and Errors on the IAT Overall

Group	Compatibility test phase	<u>Average of Pre and post NMI IAT's</u>			
		Mean reaction time	S.D.	Mean error rate	S.D.
High-trait	Compatible	667.91*	104.80	3.64*	2.18
		663.50 (VAS)	107.05	3.45 (VAS)	2.20
		657.76 (BDI)	115	3.67 (BDI)	2.42
	Incompatible	768.71*	199.40	5.14*	3.54
		757.34 (VAS)	202	5.03 (VAS)	3.64
		770.95 (BDI)	221.20	5.22 (BDI)	3.93
Low-trait	Compatible	631.37*	104.69	2.67*	2.18
		635.17 (VAS)	106.63	2.79 (VAS)	2.19
		640.12 (BDI)	113.51	2.64 (BDI)	2.39
	Incompatible	794.46*	199.22	4.86*	3.54
		804.27 (VAS)	201.21	4.95 (VAS)	3.62
		792.54 (BDI)	218.37	4.79 (BDI)	3.88

* No covariate added, (VAS) = VAS added as a covariate, (BDI) = BDI added as a covariate.

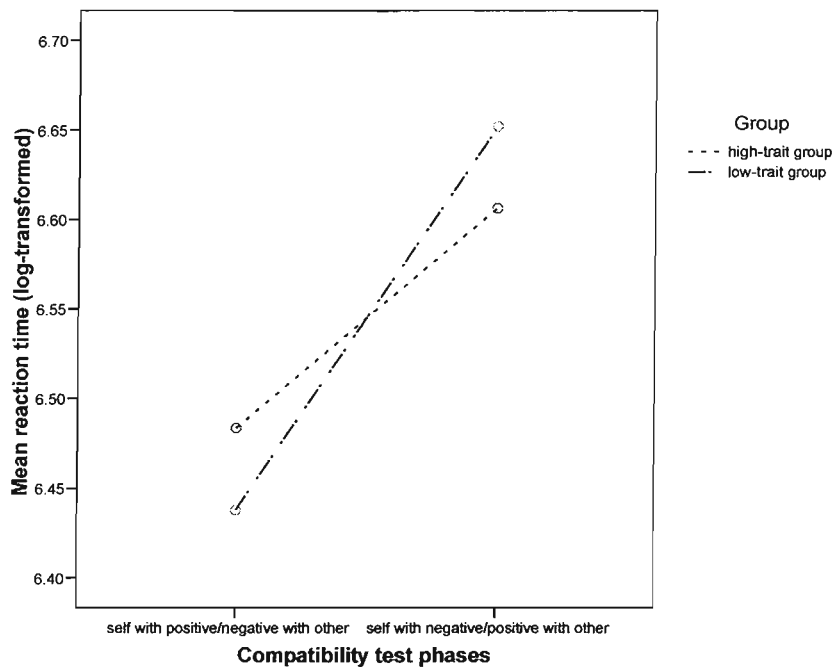
IAT- RT analysis with no covariate. There was a main effect of compatibility, $F(1, 52) = 65.00, p < .001$, and a main effect of time, $F(1, 52) = 47.18, p < .001$. This indicated that participants were faster overall on the compatible blocks of the IAT, and became faster on the post mood induction IAT compared to the pre mood induction, probably reflecting the effects of practice. There was a significant compatibility by group interaction, $F(1, 52) = 4.24, p < .05$. This indicated that the low-trait group overall showed a stronger positive automatic self-evaluative bias (See Figure 12) compared to the high trait group. Further, the mood induction had no effect on automatic self-evaluation in either group, as there was a non-significant compatibility by time by group interaction, $F(1, 52) = .700, p > .05$. There were no other significant results (highest $p = .782$).

Figure 12. Compatibility by group interaction-IAT reaction time with no covariate added to the analysis.



IAT- RT with VAS scores added as a covariate. There was again a main effect of compatibility, $F(1, 51) = 4.975, p < .05$, and a significant compatibility by group interaction, $F(1, 51) = 4.34, p < .05$, and non-significant compatibility by time by group interaction, $F(1, 51) = .656, p > .05$. These results again show that all participants were overall quicker on the compatible blocks of the IAT, and that the low-trait group overall had a stronger positive automatic self-evaluative bias compared to the high-trait group, and this was unaffected by the mood induction (see Figure 13). There were no other significant results (highest $p = .234$).

Figure 13. Compatibility by group interaction-IAT reaction time with mean VAS scores added as a covariate.



IAT- RT analysis with BDI scores added as a covariate. There was a main effect of compatibility, $F(1, 51)=27.49, p<.001$, and a main effect of Time, $F(1, 51)=16.531, p<.001$, which indicated that overall participants were faster on the compatible blocks of the IAT and became faster over time, therefore revealing practice effects. Contrary to the hypothesis, the compatibility by group interaction was not significant, $F(1, 51) = 1.387, p>.05$, and there was no 3-way interaction of compatibility by time by group, $F(1, 51) = .732, p>.05$. There were no other results of significance (highest $p=.729$). This analysis, contrary to the previous two, shows that there was no difference in positive automatic self-evaluation between groups.

IAT RT analyses summary. In accordance with the hypothesis, the low trait group exhibited an overall stronger positive automatic self-evaluative bias compared to the high trait group on two of the IAT analyses (without adding a covariate and when controlling for mood; e.g., VAS scores added as a covariate). This was not the case when controlling for depressive symptomatology (e.g., BDI scores added as a covariate). However, contrary to the hypothesis, the high-trait group's automatic self-evaluation did not become more negative as a result of the negative mood induction.

IAT- Error analysis with no covariate. There was a main effect of compatibility, $F(1, 52)=18.85, p<.001$, which indicated that all participants made fewer errors on the compatible blocks of the IAT. There was also a non-significant trend for all participants to make fewer

errors with time, $F(1, 51) = 3.27, p = .076$, and an almost significant time by group interaction, $F(1, 52) = 3.53, p = .066$, indicating that the low-trait group had a tendency to make more errors over time (increased errors on the 2nd IAT). Contrary to the hypothesis that the low-trait group would exhibit an overall stronger automatic self-evaluative bias, the compatibility by group interaction was not significant, $F(1, 52) = .659, p > .05$. There was also no 3-way interaction of compatibility by time by group, $F(1, 52) = 1.16, p > .05$, indicating that the negative mood induction did not affect self-evaluation in either group. There were no other significant results (highest $p = .365$).

IAT- Error analysis with VAS scores added as a covariate. This analysis was conducted by adding an averaged VAS score (over 6 time points) to the ANOVA as covariate. Participants did not perform with the usual bias on the compatible blocks of the IAT, as shown by a non-significant main effect of compatibility, $F(1, 51) = .457, p > .05$. Again the compatibility by group interaction was, contrary to the hypothesis, not significant, $F(1, 51) = .484, p > .05$, as was the 3-way interaction of compatibility by time by group, indicating the negative mood induction did not affect automatic self-evaluation in either group, $F(1, 51) = 1.748, p > .05$. There was, however, a time by group interaction, $F(1, 51) = 4.601, p < .05$. This revealed that the low-trait group made more errors overall on the post negative mood induction IAT. There were no other significant results (highest $p = .120$).

IAT-Error analysis with BDI scores added as a covariate. There was a main effect of compatibility, $F(1, 51) = .606, p < .05$, which indicated that participants made fewer errors on the compatible blocks of the IAT, and a non-significant trend for the low-trait group to make more errors over time, $F(1, 51) = 3.047, p = .087$. Again, the compatibility by group interaction, $F(1, 51) = .349, p > .05$, was contrary to the hypothesis, not significant, as was the compatibility by time by group interaction, $F(1, 51) = 1.28, p > .05$, which showed that the negative mood induction did not affect automatic self-evaluation in either group. There were no other results of significance (highest $p = .373$).

IAT Error summary. In disagreement with the hypothesis, the low-trait group did not exhibit overall a stronger positive automatic self-evaluative bias compared to the high-trait group, as the low-trait group did not make fewer errors overall on the compatible blocks of the IAT compared to the high-trait group. This was the case on the three error analyses when controlling for mood and depressive symptomatology, and when not adding any covariates to the analysis. Further, contrary to the hypothesis, the negative mood induction did not affect the automatic self-evaluative bias in the high-trait group to become more negative.

IAT Overall summary. To summarise the results of the IAT analyses, the low-trait group overall, compared to the high-trait group, had a stronger positive automatic self-evaluative bias on two of the reaction time analyses: one without adding a covariate, and on the analysis adding VAS scores to the analysis as a covariate. These results were not

replicated on the error analyses. The negative mood induction had no effect on automatic self-evaluation in either group as measured by reaction time or error rates with or without any of the covariates being added to any analyses.

8.4. Discussion

The aim of this study was to investigate the effect of a negative mood induction on individuals with high and low trait depression to see whether inducing a negative mood produces a negative bias at an implicit and explicit level of cognition. A second aim was to ascertain if a positive automatic self-evaluation is a stable construct in low-trait depression and thus a hypothetical protective factor for individuals not to develop depression. A subsidiary aim was to investigate the role of errors compared to reaction times in assessing automatic self-evaluation. It was hypothesised that the high-trait group, compared to the low-trait group, would show a more negative automatic self-evaluative bias after the negative mood induction, and that the low-trait group would overall show a stronger positive automatic self-evaluative bias, but that this would be relatively stable despite the effects of the negative mood induction. In accordance with the hypothesis, a stronger positive automatic self-evaluative bias, compared to a group of high-trait depressed, was found in a group of low-trait depressed. This was shown by reaction times on the IAT without any covariate added and with VAS scores added as a covariate (e.g., reaction times were significantly shorter for the low-trait depressed on the compatible IAT test blocks-self with positive key, other with negative key). Further, the observed overall pattern of a strong automatic self-evaluation in the low-trait group was unaffected by the effects of the negative mood induction, even though they reported a significantly lower mood as shown by the VAS score. Contrary to the hypothesis, the negative mood induction did not affect automatic self-evaluation in the high-trait group, although the VAS scores of the high-trait group after the negative mood induction did show a significantly lower mood had been obtained. The overall positive self-evaluative bias found in the low-trait group in this study confirms the findings in Study 3 of this thesis, where a stronger positive self-evaluative bias was observed in low-trait depressed compared to high-trait depressed, even when controlling for depressive symptomatology.

Yet, unlike the previous study (Study 3) a significantly stronger positive automatic self-evaluative bias was not evident in the low-trait group on all the error analyses (no covariate added, and with VAS scores & BDI scores added as covariates). There were no mood-linked changes in either group on the self-reporting of dysfunctional attitudes scores (DAS; Weissman & Beck, 1978) as a result of the negative mood induction. Overall however, the high-trait group did score significantly higher on the DAS compared to the low-trait group. This implies that dysfunctional attitudes were a stable feature of the high-trait depressed group. The high-trait depressed were also suffering from significantly elevated levels of depressive symptoms as measured by the BDI-II (Beck et al, 1996), compared to the

non-trait group although this was just within levels associated with mild depression. However, adding BDI scores to both the error and reaction time analyses as a covariate did not reveal a significant pattern of results from the IAT. This may indicate that using BDI scores as a covariate may take out of the analysis certain key attributes that are inherent within trait depression. It may also indicate the lack of reliability of self-report measures as in Study 3 on both IAT error analyses (IAT Schema & Mood), adding BDI scores as a covariate did not affect levels of automatic self-evaluation. Although the negative and positive mood induction worked equally well for both groups, neither group was significantly more affected by either mood induction. Both groups responded in a similar direction (sadder or happier) and in similar ways to the transient mood states. However, as already mentioned, mood linked changes did not affect automatic self-evaluation (errors or reaction time) on the IAT in any of the groups. The lack of a change in automatic self-evaluation may be linked to the temporal quality of the negative mood induction, as by time 5 there was a significant shift in all participants to report feeling happier compared to time 4. However, participants at time 5 were still significantly sadder compared to time 1, 2, 3, and time 6 indicating that relatively, low mood was still enduring.

Lack of an automatic self-positivity and vulnerability to depression

Similar self-positivity biases in healthy non-depressed individuals, individuals who are not vulnerable to depression, or other affective disorders, as found in this study in the low-trait group, have been found in other research initiatives (Taylor & Brown, 1988; de Jong, 2000). The absence of a positive bias or a weaker positive bias in comparison to healthy individuals (Alloy & Abramson, 1979), as well as a more pessimistic bias has been shown to be associated with psychological distress such as depressive symptoms (Vazquez, 1987; Scheier & Carver, 1992). This certainly was the case in this study where the high-trait group were faster on the compatible phases of the IAT despite having a significantly more negative mood overall (VAS), more dysfunctional assumptions (DAS), and more symptoms of depression (BDI), although they did not show a similarly strong positive automatic self-evaluative bias compared to the low-trait group. Furthermore, the weaker positive automatic self-evaluation in the high-trait group was still evident even when controlling for the effects of low mood (VAS score). Thus it could be argued that the results from this study support the growing notion that a lack of a significantly strong positive automatic self-evaluative bias is implicated in vulnerability to depression and perhaps other affective disorders (e.g., de Jong, 2000).

The findings from this study, and from study 3, fit well with notions that positive illusions (such as a strong positive self-evaluation) are important for good mental health (Taylor & Brown, 1988). Strong positive self-evaluations are thought to be highly adaptive when individuals receive negative feedback or are threatened in a negative way (Taylor &

Brown, 1988). One can speculate from the results of this study that the positive self-evaluative bias observed in the low-trait group (in comparison to the high-trait group) may be a potential non-vulnerability factor that protects individuals from developing depression as a result of negative life events (de Jong, van den Hout, & Merckelbach, 1995).

Error rates versus reaction time

However, a positive automatic self-evaluative bias in the low-trait group was predicted to occur as a function of error rates (as found in Study 3) as well as reaction times to stimuli. One reason why the error analyses did not reach significance may have in part something to do with practice effects as participants had to complete two IAT's. On both IAT's (error and reaction time analyses without covariates added) there were significant main effects of time (error analysis was marginally non-significant). This showed that the effects of practice may have contributed to reduced error rates and improved response efficiency of performance on the IAT. However, Greenwald et al (1998) and De Houwer (2006) argue that response latency and error rates may both be good indicators of measuring vulnerability to affective disorders. Performance on implicit tasks like the IAT may depend on a complex interaction between the familiarity of the stimuli, practice, confidence, personality, and the complex elements that represent implicit or automatic associations of attributes with the self (De Houwer, 2002, 2003; Zurroff, Mongrain, & Santor, 2004). Indeed, Townsend & Ashby (1983) argue that efficiency (speed and accuracy) of responding to stimuli on implicit tasks is a complex interaction of the aforementioned factors. However, they also argue that one must also consider the speed accuracy trade off on implicit tasks if the data are difficult to interpret². One important reason why reaction times proved a more reliable indicator of automatic self-evaluation in this study may have something to do with the choice of stimuli used. The stimuli in this study were more balanced than the stimuli used in Study 3. All words were adjectives and more importantly, the positive words and negative words were balanced conceptually. The words were designed to be reflective of positive and negative schematic themes. This may have facilitated more fluid and efficient processing of the stimuli (at the level of reaction speed) unlike conceptually different stimuli (De Houwer, 2001).

The absence of mood-linked changes on the DAS and hypothetical vulnerability to depression

In this study there were not any mood linked changes in information processing or on DAS scoring after the negative mood induction. This is not in accord with much of the previous research (Ingram et al, 1994, Ingram & Ritter, 2000; Miranda & Pearsons, 1988) and

² In Study 3 unreported calculations based on Townsend Ashby's (1983) "efficiency index" still showed the low-trait group to have positive self-evaluative bias on both IAT's with, and without adding BDI score to the analysis while the high-trait group still did not show a positive self-evaluative bias. In this study unreported efficiency index calculations yielded no significant effects. Implicit bias is a very complex area, but the efficiency index tool taking into account speed accuracy trade off can be used in contexts where the data is difficult to interpret (Townsend & Ashby, 1983).

the cognitive model of depression implicating a latent schema model of depression (Beck, 1967, 1976). Studies using individuals who have recovered from depression in mood induction studies usually find some kind of negative information processing bias or increased scores on self-report measures of depression after participants are induced into a transient negative mood state (Dykman, 1997). However, in this study, the high-trait group was not composed of individuals who had recovered from depression, but a sample of individuals who had been classified as possessing elevated levels of traits of depression proneness. Thus, this group is only hypothetically at more risk to develop depression.

It has been argued that there is insufficient evidence to justify the use of analogue depressed and analogue trait depressed samples in depression research and the findings being used for inferences into “true” depression. Depue & Monroe (1978) argue that the depressions experienced by analogue samples (especially university students) are markedly different from those of clinically depressed samples or from those who are vulnerable to depression. These differentiating characteristics include age, level of education, socioeconomic status, IQ, and marital status. Further, Coyne & Gotlib (1983) and Gotlib (1984) argue that the characteristics of analogue depressed samples are markedly different from the depression manifested by depressed psychiatric patients. This is because measures of depression in college students are correlated with measures of a variety of other psychiatric disorders. Coyne and Gotlib (1983) conclude that student depression may be nothing more than general psychological distress. Therefore, the lack of an induced negative automatic self-evaluative bias and also lack of mood-linked DAS changes in the high-trait group in this study may be indicative that this group of individuals may only be representative of individuals showing general psychological distress, or are vulnerable to develop other kinds of psychological disorder. Thus, in the context of this study, the individuals that comprised the high-trait group may not be representative of true vulnerability to depression. Another possibility that the high-trait group was not representative of true vulnerability to depression is implicated in what is known as the “scar” theory of depression. This idea suggests that the experience of an initial episode of depression is required before schematic organization occurs leading to a hypovalent schema in Beck’s (1967, 1976) theory (Hammen et al., 1985). Thus, the high-trait individuals used in this study, because they had no history of clinical depression, theoretically may not have the required schematic organization that leads to negatively biased information processing as a result of the effects of a negative mood.

Another reason why DAS scores may not have risen in the high-trait group after the negative mood induction may have to do with the arbitrary cut-off scores for the selection of the high and low trait depressed groups. A median split was used to select the two groups and thus “false” differences in trait depression between the two groups may have been sampled. The differences in depressive traits between the two groups may not have been wide enough

for a “true” difference in trait depression to emerge. Thus sampling more participants on the upper and lower quartiles on the DPRS may have been more reflective of individuals who were more representative of individuals who were potentially more vulnerable to developing depression. If more extreme sampling on the DPRS was carried out (e.g., upper and lower quartiles) then two distinct trait groups may have emerged. Thus theoretically, in the high-trait group an elevated DAS score may have been observed after the negative mood induction as in accordance with previous research (e.g., Gamar et al., 2001; Miranda & Pearsons, 1988)³.

Lastly, the lack of a typical DAS change in response to low mood in the high-trait group (Miranda & Persons, 1988) may indicate that self-report measures are at times unreliable. This fits with Nisbett & Wilson’s (1977) and Farnham et al.’s (1999) arguments that there are many variables that may affect self-report measures, but desire to please, lying, illusion of health or ill-health, and other self-presentational effects may influence responding (e.g., social desirability or social pressure). Further, it is argued that explicit self-report measures may reflect different constructs compared to implicit measures (Wilson, Lindsey, & Schooler, 2000; Eysenck, 1991).

Further methodological considerations

The final two methodological issues that have to be considered pertain to the complexity of the experimental procedure and the role of executive function and these two elements may have some bearing on the interpretation of the results. There were nine stages to complete, of which two were the IAT tasks and two were the DAS questionnaires (A & B) and five VAS mood measures. Therefore, the experimental procedure demanded a great deal of attention, concentration, and commitment. This has implications with regards the nature of depression and the issue executive function in depression. For example depressed people of those vulnerable to depression have a tendency to tire during complex tasks and find it hard at times to concentrate and pay attention to the details or instructions of tasks set before them (Channon & Green, 2006). This aspect of fatigue associated with depression may have had an effect on the outcome of results in this study due to its complexity (e.g., DAS scores or IAT performance in the high-trait group). It may have been a better to have four groups take part in this study. This could involve a high and low trait depressed group completing the IAT

³ The criticism of arbitrary DPRS cut-off scores for the selection of the high and low trait depressed groups could also be levelled at Study 3. A median split was also used to select the two groups, and thus “false” differences in trait depression between the two groups may have been sampled. However, it must be noted that significant differences between the two trait groups on depressive symptomatology was observed and the information processing differences between the two groups was evident with and without adding the BDI score to the analyses in this study. Nevertheless, sampling more extreme high trait depressed and low-trait depressed individuals may be an issue to consider for further research investigating automatic self-evaluation and schemata.

and DAS and associated mood measure each once only with no mood induction, and a low and high trait depressed group completing the negative mood induction and then completing the IAT, DAS, and VAS. This would minimise the effects of fatigue and perhaps yield a clearer picture of the nature of automatic self-evaluation, mood, and trait depression.

Related to the issue of fatigue is the problem associated with executive function as highlighted as a methodological problem in Study 3. As discussed in Study 3, depressive type disorders have been associated with central executive dysfunction which can affect information processing capabilities (Channon & Green, 2006; Elliot et al., 1997; Watts et al., 1998; Hertel & Hardin, 1990; Elliot et al., 1996). As was observed in this study, the high-trait group in comparison to the low-trait group performed more similarly overall pre & post negative mood induction) on the compatible and incompatible phases of the IAT's perhaps suggesting some kind of executive dysfunction. In other words the differences in performance by the high-trait group may not be due to a lack of a positive automatic self-evaluative bias, but instead to dysfunctional executive processes affecting performance on the IAT's. Further, any executive dysfunction in the high-trait group may have been compounded by the complexity and potential fatigue inducing elements of the experimental procedure of this study and this may have had a bearing on the results. As discussed in Study 3, it has been argued that performance deficits associated with central executive dysfunction by depressed individuals only take place on tasks that involve controlled processes but not on tasks measuring automatic processes (Ellis & Ashbrook, 1998; Hasher & Zacks, 1979; Hertel, 1994; Teasdale & Barnard, 1993). Consequently, on such tasks like the IAT, a criticism of executive functioning affecting performance may not be applicable as the IAT is argued to measure automatic processes and not controlled processes (Greenwald et al., 1998). However, De Houwer (2006) argues that controlled processes might at times affect performance on the IAT. Therefore, the IAT may measure the effects of controlled processes, automatic processes, or a mixture of both. This issue needs addressed by other researchers to ascertain the role of executive function and fatigue on automatic self-evaluation using a task like the IAT.

The cognitive model of depression re-visited

This study found no evidence for a negative schematic bias in a group of high-trait depressed individuals as a result of a negative mood induction. The main finding was that overall, the high-trait depressed showed a weaker positive automatic self-evaluative bias compared to the low-trait depressed. In essence, there was no evidence for schemata being activated in response to a low mood. This is not in accordance with the cognitive model of depression and previous research (Beck, 1967, 1976; Gemar et al., 2001; Teasdale & Barnard, 1993; Hedlund & Rude, 1995; Rude et al., 2001). This model, and the results of previous research, postulates the existence of negative schemas in individuals who are depressed or

vulnerable to depression, which become activated in response to a low mood (e.g., Beck, 1967, 1976). However, de Jong (2000) argues that discrepancies in automatic self-evaluation may be the key factor between individuals who are vulnerable to develop psychopathologies rather than an activated cognitive system (e.g., Beck, 1967, 1976). This fits nicely with other ideas on self-evaluation. Southall & Roberts (2002) observed that pre-existing weak positive self-evaluation coupled with high environmental stress resulted in adolescents being vulnerable to develop depression. In addition, other findings demonstrate that automatic self-evaluation appears to be a stable construct and is not affected by changes in mood (Stapel & Blanton, 2004; Pelham & Hetts, 1999). These findings support the results of this study and of the idea that a stable weaker positive automatic self-evaluation may be implicated in vulnerability to depression (de Jong, 2000). Certainly the IAT used in the context of vulnerability to psychopathology and depression is in its infancy, but the results of this study (and the previous study) show that information-processing differences in the guise of automatic self-evaluation is measurable in individuals who are theoretically vulnerable to depression. More importantly, it seems that such differences in automatic self-evaluation are not affected by low mood.

Conclusion

This study observed a stronger positive automatic self-evaluative bias in a group of low-trait depressed compared to a group of high-trait depressed using the relatively new IAT task. The superiority of automatic self-evaluation in the low-trait group over the high-trait group was unaffected even when controlling for current mood state. More surprisingly, this study showed that a negative mood induction did not produce the characteristic negative information biases in the high-trait group, which are usually seen in individuals who are theoretically vulnerable to develop depression (e.g., Gemar et al., 2001). Therefore, the results of this study alongside those from Study 3 have shown promise in measuring schematic functioning and hypothetical vulnerability to depression in the absence of a depressed or low mood. These results challenge the cognitive model of depression and the hypothetical latent schema structure inherent in this (Beck, 1967, 1976). If automatic self-evaluation is implicated in vulnerability to depression, it may be fruitful to investigate automatic self-evaluation and vulnerability to depression in clinical samples. It has been argued that antidepressants are not as effective in preventing depressive relapse and that cognitive therapy or variants of cognitive therapy may be superior in preventing relapse in individuals who are vulnerable to a depressive relapse (Teasdale, Scott, Moore, Hayhurst, Pope, & Paykel, 2001). If a positive automatic self-evaluative bias is not engendered with the use of antidepressants, then perhaps this is why relapse rates with drug treatments compared to psychologically orientated treatments is superior. Therefore, it would be extremely informative to measure automatic self-evaluation alongside explicit measures of cognition in a sample of clinical

depressed individuals at different points during antidepressant treatment. If a weak positive automatic self-evaluation is not affected by a low mood and is measurable in the absence of a low mood, one may be able to ascertain whether successful antidepressant treatment (reduced self-reported symptoms of depression) results in generating a strong positive automatic self-evaluative bias during treatment and by the end of treatment. Therefore, the role of antidepressants and their effects on automatic self-evaluation was investigated in the following study.

Chapter 9

Study 5: The psychological effects of selective serotonin reuptake inhibitors in depression measured with implicit and explicit methods

9.1. Introduction

In the previous study, participants with low levels of trait depression had a stronger positive automatic self-evaluative bias (as shown by reaction time data) compared to participants with high levels of trait depression.¹ This bias was evident even after a negative mood induction and controlling for mood. The results of Study 4 together with those of Study 3 indicate that a positive automatic self-evaluation might be a protective factor by creating low levels of vulnerability to depression. Conversely, a weak positive automatic self-evaluation could confer vulnerability to depression. If a weak positive self-evaluative bias is a vulnerability factor for the risk of developing depression (or having a depressive relapse), then this aspect of implicit functioning could be a useful avenue to research in regard to different treatments for depression and their relative efficacy. In other words, one could investigate the claims of some researchers that antidepressant treatment is not as effective as cognitive therapy in the prevention of relapse (e.g., Hensley et al., 2004). If antidepressant treatment for depression leaves a latent vulnerability for a depressive relapse, it may be feasible to ascertain if a weak positive automatic self-evaluative bias is still present after successful antidepressant treatment. It may be that the reason antidepressant treatment does not protect from a depressive relapse (as well as cognitive therapy) is because it does not restore a strong positive automatic self-evaluation. Therefore, the next logical step is to investigate the effects of treatment for depression and automatic self-evaluation. This was the goal of this present study.

Current status of evidence comparing the efficacy of psychotherapy versus antidepressants

There is some good evidence to suggest that antidepressant treatment is not as effective in the prevention of depressive relapse compared to cognitive therapy (Hensley et al., 2004; Paykel, 2001; Scott, Palmer, Paykel, & Teasdale, & Hayhurst 2003; Evans et al., 1992), psychological therapy based on mindfulness techniques (Teasdale, Segal, Williams, Rideway, Soulsby, Lau, 2000), and a variety of other psychological therapies; e.g., dynamic

¹ In Study 4 reaction time from the IAT data was the measure that showed differences between low and high trait depressed groups, while in Study 3 it was errors rates that were the significant measure. De Houwer (J. De Houwer, personal communication, 14th September 2005) argues that either reaction time or errors rates on implicit tasks like the IAT or EAST are valid measures of self-esteem or self-evaluation. Kirsch & Lynn (1999) and Bargh & Tota (1988) argue that error rates may be a more valid measure of automaticity especially related to vulnerability to affective disorders as error rates are less affected by explicit and controlled processes compared to reaction time data. On the other hand, Townsend and Ashby (1983) argue that reaction time and error rate data from implicit tasks have to be evaluated carefully and one has to consider the speed accuracy trade-off if the results are difficult to interpret. In other words one may have to, if the data permits, measure the relative efficiency of a performance on an implicit task to measure “true automaticity”.

psychotherapy (see Antonuccio, Danton, & DeNelskey, 1995 for a review). Beck (1967, 1976) argues that individuals who are vulnerable to depression have acquired negative self-schemas or templates of the self that guide information processing. The evidence from research from the perspective of Beck's theories of depression suggest that cognitive and other psychological therapies are thought to effect change in the self-schema or self-evaluation of the vulnerable individual, whereas pharmacotherapy does not (Jacobson & Gortner, 2000; Segal, 1988). Therefore, hypothetically antidepressants may merely alleviate symptoms but not change the negative self-system or self-evaluative system of vulnerable individuals, which is needed to prevent future depressive relapse.

However, although it has been shown that antidepressants reduce negative automatic thoughts associated with depression, the evidence on their effects on deeper level cognition such as schemata is more equivocal as there is little evidence of the impact of antidepressants on implicit cognition (see Gemar et al., 2001; Ingram et al., 1998). Therefore, by investigating the role of self-evaluation, self-schemas, or self-esteem via implicit methods in people successfully treated with a certain type of antidepressants (i.e., SSRI's), one may be able to ascertain whether antidepressant treatment changes negative self-evaluation at an implicit as well as an explicit level. Such information may provide an explanation for the difference in relapse rates for pharmacotherapy compared to psychological type therapies. In the next section, an overview of the theoretical background of antidepressant treatment and how this relates to depression will be discussed. Following this, the mechanisms of change inherent in psychological treatments, and the unresolved issues in psychotherapy and pharmacotherapy research, will be presented.

Antidepressants and mechanisms of action

One of the mechanisms hypothetically involved in the genesis of depression and feelings of low self-worth is attributed to a serotonin dysfunction in the brain (Boyer et al., 1996). In many studies it has been inferred that low levels of serotonin and/or dysfunctional serotonin transmission is associated with depression and low self-esteem. Studies of mammals, such as monkeys and rats, that display withdrawn, fearful, and depression-like symptoms after negative experiences, have lower levels of serotonin or dysfunctional serotonin transmission, compared to normal rats or monkeys (see James, 1998 for a review). In humans, using various methods² of measuring serotonin levels, it has been found that depressed individuals have abnormal serotonin levels and/or dysfunctional serotonin

² Brain imaging techniques and measuring levels of by-products of serotonin or chemicals essential in its manufacture in spinal fluid, blood or urine. There are arguments that these methods of measuring serotonin metabolism lack validity. Serotonin receptors and serotonin metabolism are found elsewhere in the body as well as the brain (McNeal & Cimbalic, 1986).

production compared to non-depressed individuals (S.L. Brown, 1991; Roy, Virkkunen, & Linnoila, 1990).

Persuasive evidence for a relationship between serotonin and depression comes from experiments where depressed individuals have been given drugs that increase serotonin levels/transmission (e.g., Paroxetine or Fluoxetine). Whilst taking the drugs the depression tends to diminish, and individuals start to feel an increased self-esteem, although this does not take place for several weeks. As these two events correlate, it is inferred by biologically orientated scientists that a rise in serotonin levels or in serotonin transmission causes the depression to remit (S.L. Brown, 1991; Roy et al., 1990). This theory is further advanced by evidence that indicates that 80-90% of people who suffer a bout of depression eventually suffer a subsequent one if they stop taking the drugs (Boyer et al., 1996).

However, the evidence supporting a serotonin theory of depression is not so straightforward. Serotonin has also been implicated in a myriad of other human behaviours such as aggression (O'Keane, 1992; Von Knorring, 1987; Schukit, 1987), substance misuse (Boyer et al., 1996), compulsive disorders e.g., gambling (Coccaro et al., 1990; Zuckerman, 1984), anorexia (Brewerton et al., 1990), and social phobia (Bower & Stein, 1998). McNeal & Cimbalic (1986) argue that there are multiple functions of serotonin in the brain other than regulation of mood, and that a complex interaction of other neurotransmitters (e.g., noradrenaline and acetylcholine) is also implicated in depression.

Further doubt regarding the serotonin hypothesis of depression is presented by Thase (2002) who notes that 35-50% of patients respond to antidepressant medication compared to 25-30% who respond to placebo. This is interpreted as indicating that 10-20% of depressed clinical trial patients show a true drug effect. This means that 80-90% of patients do not show a specific drug effect. Even though the effect between an actual drug effect and placebo effect is small, on average there is still a significantly greater response to antidepressants, compared to an inert placebo. What is uncertain is the reason for this difference in response. It may be a drug effect (true antidepressant effect), but it also may well be an enhanced placebo effect associated with the perception of negative side effects. It is well known that antidepressants produce more side effects than inert placebos (Mulrow et al, 1999) and that an antidepressant effect is not convincingly demonstrated in studies that use placebos that produce side effects akin to antidepressants (Moncrieff, 2003). In other words, a superior antidepressant effect is not demonstrated by "real antidepressants" compared to a placebo that also produces side-effects akin to those found with the use of antidepressants. The evidence from brain imaging studies is mixed for showing differential brain changes in placebo responders to antidepressant responders. Leuchter, Cook, Witte, & Abrams (2002) found differential brain changes in individuals treated with antidepressants compared to placebo, while Mayberg et al. (1999) found similar brain changes in both the placebo and antidepressant groups. Kirsch &

Lynn (1999) argue that the positive drug responses in depression may be a combination of the effects of an active placebo, expectation of treatment and other non-specific effects such as the therapeutic relationship between the doctor and patient.

Although the serotonin hypothesis of depression is inconclusive, cognitive therapies and other psychotherapies that seem to fare better in preventing relapse are still being overlooked in favour of antidepressant treatment, which is favoured as a first line treatment for depression (Hensley et al., 2004). Differential brain changes have been observed in patients who received psychotherapy versus the antidepressant treatment. For example Martin, Martin, Rai, Richardson, & Royall (2001) found increased limbic blood flow in patients who received interpersonal therapy versus antidepressant therapy. This may, at a neurochemical level, explain why psychotherapies are better at preventing relapse. Le Doux (1998) argues that neuronal pathways in the amygdala situated in the limbic system are implicated in affective disorders. Thus, a treatment that affects this area may in some way correct dysfunctional neuronal organisation of the amygdala. However, if relapse rates are poorer with antidepressants than with psychological therapies, the question remains what do cognitive and other psychotherapies accomplish that pharmacotherapy does not?

Psychotherapy and mechanisms of action

Beck (1967, 1976) argues that negative schematic change occurs as a result of cognitive therapy due to an improved meta-cognitive relationship between the individual and his/her negative thoughts of the self, the world, and the future. Further, an individual's cognitive reactivity (e.g., schematic activation and subsequent negative thoughts and symptoms) to negative events/negative thoughts becomes less negatively biased. Concurring with this idea, Beevers & Millar (2005) found that cognitive therapy compared to pharmacotherapy significantly reduced the rate at which negative cognition increases as a function of the increasing severity of depression. In other words, the rate at which negative cognition increased as the symptoms of depression increased was lower for individuals treated with cognitive therapy. This finding suggests that in some way cognitive therapy decouples negative cognition from symptoms of depression. This is in accord with Teasdale, Moore, Hayhurst, Pope, Williams, & Segal's (2002) suggestions. They argue that cognitive therapy may help individuals relate more functionally to their negative thoughts. More specifically, cognitive therapy may help individuals treat negative thoughts as mental events that may or may not have a basis of truth. Therefore, when the symptoms of depression increase, an individual is less likely to concur with his/her negative thoughts.

The uncoupling of negative cognition from negative affect was also confirmed in findings from a study by Teasdale et al. (2001), which compared cognitive therapy and pharmacotherapy. They concluded that cognitive therapy reduces relapse through a reduction in absolutist, dichotomous thinking style. Therefore, cognitive therapy seems to prevent

relapse by training individuals to change the way that they process depression-related material rather than changing depressive thought content. In other words, the thought content in those who had recovered from depression remained essentially similar, but the way that individuals dealt with it became more functional with cognitive therapy. From the perspective of a schema model of depression, this process supposedly weakens the negative self-schematic system responsible for automatic information processing and puts into place a more functional or positive self-schema system (Beck, 1967, 1976).

However, in a critique of a purely cognitive explanation of depression, Jacobson & Gortner (2000) argue that it is the behavioural activation component of cognitive (behavioural) therapy that is responsible for the protective gains made from the therapy. Their argument is based on a clinical trial run four years earlier (Jacobson et al., 1996). In this study, they found that the punitive cognitive change mechanisms involved in cognitive behavioural therapy were not necessary for the success of the treatment. The behavioural activation component, which made no attempt to change thinking, worked as well as both a complete cognitive therapy treatment, and an additional treatment that targeted just negative automatic thoughts, both in maximising acute treatment response and in relapse prevention over a two-year period. Jacobson & Gortner (2000) argue that a behavioural activation hypothesis of relapse prevention in depression provides an alternative to the defect/disease models of depression (e.g., cognitive dysfunction and faulty brain chemistry). A behavioural activation hypothesis is based on the assumption that negative thinking is a realistic by-product of stressful negative events and negative thinking will change automatically as behavioural reinforcers return to a normal level in an individual's life.

There are also other arguments as to why psychotherapy is effective in treating depression. An interpersonal theory has been put forward as being responsible for positive psychotherapeutic change (Drew, Dobson, & Stam, 1999). They argue that certain aspects of the psychotherapeutic relationship contribute to the process of socially re-establishing a patient's positive identity by enabling the patient to test out hypotheses with the therapist about the patient's social identity. Rehm (1995) also asserts that cognitive therapies are effective because of the therapeutic relationship. Rehm (1995) and Illardi & Craighead (1994) argue that the greatest improvements in symptoms during the course of cognitive-behavioural therapy occur early, before many of the interventions have been introduced. Therefore it may be possible to argue that the positive positioning between therapist and patient may help patients retrieve positive self-identities or hold themselves in positive self-evaluation. In other words, the actual context of a "meeting" in psychotherapy with another human being to help and support an individual is what contributes to a change in the negative self-schema or self-esteem of the individual who is vulnerable to depression (see Frank, & Frank, 1991).

If the psychotherapeutic relationship is important in cognitive orientated therapies, how do other psychotherapies compare, and what is the evidence for the therapist-patient relationship being of prime importance in resolving depression? In response to the latter point, it has been found that non-specific treatment and therapist effects (e.g., therapeutic relationship and empathy) are likely to make a significant contribution to recovery from depression (Burns & Nolen-Hoeksema, 1992; Kendall & Lipman, 1991; Simons, Lustman, Wetzel, & Murphy 1985). With regards to the former point, reviews of comparative outcome studies for depression, in common with other psychological disorders, have generally concluded that different psychotherapeutic treatments are broadly equivalent in effectiveness (Shapiro, Barkham, Rees, Hardy, Reynolds, & Startup, 1994; Jones & Pulos, 1993; Robinson, Berman, & Neimeyer, 1990; Shapiro & Shapiro, 1982; Smith, Glass, & Miller, 1980; Stiles, Shapiro, & Elliot, 1986).

In consideration of the evidence when comparing pharmacotherapy and psychotherapy for resolving depression, it appears that both the specific effects and non-specific effects of psychological treatments may play a part in the process of therapeutic change and in preventing relapse. However, there are problems in the methodologies used in psychotherapeutic depression treatment research.

Unresolved Problems in psychotherapy and pharmacotherapy research

A major problem with studies investigating the effectiveness of treatments for depression and cognitive effects of treatments is the overuse of measures that tap into explicit or more controlled processing. Nisbett & Wilson (1977) point to the dangers of relying on self-report measures, as the social pressure to conform to external expectations may influence the results. Very few studies have investigated implicit and explicit processes concurrently within a treatment context and looked at the similarities and differences between these two different types of measure (Hedlund & Rude, 1995). This oversight is troublesome considering the fact that it has been argued succinctly that implicit measurement of information processing biases are more accurate in measuring vulnerability to affective disorders (Gemar et al., 2001). It has been found in other studies that a weak or dysfunctional positive automatic self-evaluation as measured by implicit processing tasks, like the IAT (Greenwald et al., 1998) or the EAST (De Houwer, 2003), may be implicated in vulnerability to depression or other affective disorders (Gemar et al., 2001; de Jong, 2000). The usefulness of implicit tasks is given further credence when one considers that individuals who are vulnerable to depression and who score low on self-report measures of depression still exhibit a weak positive automatic self-evaluative bias (Alloy et al., 1999). Indeed, as shown in Study 3 (with error data only) and Study 4 (with reaction time data only) in this thesis, individuals classified as being high-trait depressed had a weaker positive automatic self-evaluation on the

IAT compared to low-trait depressed individuals, even when controlling for current depression.

If a positive automatic (implicit) self-evaluation is an important aspect of non-vulnerability to depression, then perhaps antidepressant treatments do not protect from an increased risk of relapse. This may be because patients who take antidepressants are not exposed to the specific effects of the treatment (e.g., Teasdale et al., 2002) and to the non-specific effects of a psychotherapeutic relationship (e.g., Rehm, 1995) that instil a more positive self-evaluation. Enduring vulnerability to depression may be masked by the use of self-report measures of depression (Nisbett & Wilson, 1977) but may be tapped into using implicit measures of cognition (Gemar et al., 2001). Therefore, one would expect that if a drug treatment for depression is unsuccessful in resolving latent vulnerability to depression, then antidepressants may only lower scores on self-report measures of depression (e.g., symptoms or automatic thoughts), but a weak positive automatic self-evaluation would still be evident.

Aims and hypotheses

The main aim of Study 5 was to investigate how SSRI's affect positive automatic self-evaluation and scoring on explicit self-report measures of depression over a period of six months. It was hypothesised that 1) at baseline (within two weeks of beginning antidepressant treatment) a depressed group (which will be called the SSRI group for the purposes of this study) would score significantly higher on explicit self-report measures of depression, and show a weaker positive automatic self-evaluation compared to a non-depressed control group (as shown by errors and reaction time on the EAST and IAT tasks); 2) after six months of treatment, the SSRI group's self-report measure scores would have reduced to similar levels compared to the control group, but the SSRI group would continue to show a weaker positive automatic self-evaluative bias compared to the control group (as shown on the EAST and IAT); and 3) the weak positive automatic self-evaluative bias of the SSRI group would be stable over time, whereas a strong positive automatic self-evaluative bias would be stable in the control group over time as shown by the IAT and EAST tasks.

A subsidiary aim, continuing on from the investigations in Study 3, was to investigate how different types of negative and positive stimuli affect information processing. It was hypothesised that a weak positive automatic self-evaluative bias would be more evident in the SSRI group in response to specific stimuli congruent to hypothetical content within negative self-schemata (e.g., reflective of themes related to abandonment and defectiveness).

9.2. Method

Participants

Two groups of participants were selected for this study: a group of individuals who had been diagnosed by their GP as being depressed and prescribed an SSRI antidepressant

within approximately two weeks of taking part in the study,³ and a control group of non-depressed individuals who were not currently depressed and who did not have a history of depression. Individuals were recruited via advertisement (e.g., Southampton University campus notice boards, local library notice boards, local internet notice boards, and through a local G.P.). All participants were native English speakers and the two groups were matched as far as possible on socioeconomic status. Potential participants were excluded if they had colour blindness or eyesight problems. The depressed participants, who made up the SSRI group, were screened using the SCID (see details overleaf) diagnostic interview to confirm that they met DSM-IV criteria for major depression. The control group participants were questioned to confirm that they did not currently suffer from depression, were being treated for depression, or had a history of depression or treatment for depression. The SSRI group comprised 7 males and 8 females and had a mean age of 27.93 (SD=8.85). The control group comprised 5 males, and 11 females with a mean age of 28.56 (SD=9.01). There was no difference between the groups on age, $t(29) = .196, p > .05$, or on gender composition, $\chi^2(1) = .776, p > .05$.

Materials

Self-Report measures

The rationale behind choosing the following questionnaires as dependent measures was to have a sample of questionnaires that took into account cognitive products, cognitive processes, and cognitive structures/schema associated with depression, and to ascertain how these variables changed over the course of antidepressant treatment. A questionnaire was also used to evaluate the change in anxious symptomatology over time, as anxiety is highly associated with depressive symptomatology. For full details of the questionnaires below, see the method section in Study 1 and Appendix III.

The Beck Depression Inventory-II (BDI-II, Beck et al., 1996). Measures the severity of depressive symptomatology.

The Dysfunctional Attitudes Scale (DAS, Weismann & Beck, 1978). Measures conditional dysfunctional assumptions related to depression.

³ Selective serotonin reuptake inhibitors are a certain class of drugs (e.g., paroxetine, fluoxetine) that selectively prevent the reuptake of serotonin at pre-synaptic neurons. Participants were selected if they had only recently started taking antidepressants (e.g., within 2 weeks approximately). Research has shown that the effects of SSRI's can take approximately 2 weeks to begin to exert an effect on depressive symptoms (Boyer et al., 1996). Therefore, selecting participants within two weeks of starting their medication should have ensured that the therapeutic effects of the SSRI's would have been minimal, and the levels of depressive symptomatology of participants should have been as close to baseline levels before treatment began. All potential depressed participants were questioned to confirm their Doctor's diagnosis of depression and medication status. Participants were not excluded if they had experienced a previous episode of depression.

Young Schema Questionnaire (short version) (YSQ, Young & Brown, 1994).

Measures stable cores beliefs, themes, or early maladaptive schemas related to different psychopathology.

The Beck Anxiety Inventory (BAI, Beck, 1980). Measures the severity of anxious symptomatology.

The Evaluative Beliefs Questionnaire (EBS, Chadwick, Tower, & Dagnan, 1999).

Measures a class of beliefs closely related to psychopathological disturbance, especially depression; self-deprecatory beliefs, one's negative beliefs regarding other people, and negative beliefs regarding what other people think of one.

Automatic Thoughts Questionnaire (ATQ, Hollon & Kendall, 1980). Measures the severity of negative automatic thoughts related to the presence and severity of depressive symptoms.

Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1996). All depressed participants that made up the SSRI group were assessed with the SCID to determine if they met diagnostic criteria for major depressive disorder. The author (Bruce Scott) administered the SCID after receiving specialist training from Dr. Lusia Stopa, a qualified clinical psychologist.

Stimuli used in the IAT & EAST tasks and for investigation into specific schema content hypothesis⁴

EAST stimuli. Following on from Study 3 and investigations into the specific schema content theory and vulnerability to depression (e.g., Alloy et al., 1999), the EAST used four sets of words: negative schema words, positive schema words, low mood words, and positive mood words. With these groups of words, it was possible to investigate the effects of automatic processing to the different types of negative and positive words when they were associated with the self or other people. To investigate automatic self-evaluation in general (not considering different kinds of negative and positive words and effects on automatic processing), the negative schema and negative mood words were combined to make a set of negative words, while the positive schema and positive mood words were combined to make a set of positive words.

IAT Stimuli. There were two IAT's used in this study. One IAT (IAT Mood) used negative mood words as its negative stimuli and positive mood words as its positive stimuli. The other IAT (IAT Schema) was the same as used in Study 4. The IAT Schema used negative schema words and positive schema words as its stimuli. To investigate the specific

⁴ See Appendix IV & for attributes and details of words used in Study 5.

schema content hypothesis with the IAT, comparisons of performance on each IAT was used to examine the effects of the mood related words versus schema related words on automatic self-evaluation.

Stimulus selection. The positive mood words were selected by a similar method outlined in Study 3. Twenty post-graduate students had to rate if a list of positive mood words (collated by the experimenter) was reflective of a positive mood. A positive mood word was selected if had a mean applicability rating of at least 7. The negative schema words, negative mood words, and positive schema words were selected as described in Studies 1 and 4 respectively. All groups of words used in each task were balanced for frequency, syllable count, number of letters, and emotionality. However, as highlighted in previous studies in this thesis, the positive words were rated significantly more pleasant than the negative words, but the different groups of negative words did not significantly differ on the ratings of pleasantness. All words were also adjectives to control for saliency confounds (see method discussion section in Study 3 regarding stimuli saliency).

Implicit Processing Tasks

Extrinsic Affective Simon Task (EAST; De Houwer, 2003). The specifications of the EAST were almost identical to those described in Study 3. The only difference was that 50% of the negative words were schema words and 50 % were mood descriptors. Likewise, 50% of the positive words were schema words and 50% were mood descriptors. This composition of words in each category allowed a comparison between schema related and mood related words. As in Study 3, there were four different versions of the EAST task for counterbalancing purposes regarding key allocations.

The Implicit Association Task (IAT, Greenwald et al., 1998). The specifications of the two IAT's in this Study (IAT Mood & IAT Schema) were the same as those in the IAT in Study 3. As in all previous studies, counterbalancing procedures ensured that participant key allocations were controlled for over the course of the experiment.

Procedure

Participants who volunteered for the experiment were first screened to make sure that they met the criteria for the study as outlined in the participant section. In addition, the SSRI group had to undergo a SCID interview to confirm that they met a diagnosis of Major Depressive Disorder. Participants were then asked to complete three computer tasks and fill out the questionnaires. Testing took place in either a small cubicle or a room with optimum lighting (i.e., no or little outside light) and low levels of external noise. The participants completed the three computer tasks first (2 IAT's & EAST) then filled out the questionnaires (DAS, EBS, ATQ, YSQ, BAI, BDI). Each participant repeated this procedure on another two occasions; at approximately three months (Time 2), and six months (Time 3) after initial

testing (Time 1)⁵. As there were three computer tasks, the order of tasks was counterbalanced at each time point and across the three time points. Participants were paid £5 on each testing occasion. After the third testing point, each participant was then debriefed. Each individual testing session took approximately one hour for each participant.

9.3. Results

The questionnaire results are presented first followed by the results from the individual EAST and the two IAT's. This will be followed by the EAST and IAT analyses to test for the specific schema content hypothesis. This analysis was concerned with investigating schema words and mood descriptor words, and the effects on performance to these different categories of words. Log-transformed reaction time scores were used in the statistical analysis of the EAST and IAT's. An alpha level of .05 was used for all initial ANOVA's. Unless reported in the results, non-significant results and minor effects and interactions from the ANOVA's from the self-report measures and EAST and IAT's analyses are presented in Appendix V. All post hoc analyses after initial ANOVA's (e.g., independent and paired t-tests) used Bonferroni correction techniques to reduce the likelihood of false positive results. This involved dividing the alpha level (.05) by the number of tests conducted. This will be referred to in the text as (.05/N).

Self-Report Measures

The hypothesis was that all scores by the SSRI group on self-report measures (and associated subscales) would significantly reduce over time to levels comparable with those levels of the control group. The statistical analyses for each self-report measure without individual subscales (e.g., BDI, ATQ, BAI, DAS) involved a 3 (time) x 2 (group) repeated measures ANOVA, with time as a within subjects factor and group as a between subjects factor. For self-report measures with individual subscales (e.g., YSQ=15 subscales, EBS=3 subscales) a 3 (time) x 15 or 3 (subscales) x 2 (group) repeated measures ANOVA was used. Again time and subscale were within subjects factors and group was a between subjects factor. See Tables 12 & 13 for mean scores of all self-report measures.

⁵ Individuals from the SSRI group took part in initial testing (Time 1) approximately within 2 weeks of starting SSRI treatment.

Table 12

Mean Scores on BDI, BAI, DAS, ATQ, & EBS Subscales by the SSRI and Control Group

Questionnaire	Group	Time 1		Time 2		Time 3		Mean overall	SD
		Mean	SD	Mean	SD	Mean	SD		
BDI	SSRI	30.26	11.55	16.66	9.47	14.93	11.04	20.62	9.62
	Control	7.31	7.89	8.25	7.75	9.12	7.87	8.22	6.97
ATQ	SSRI	89.20	28.45	67.86	28.45	63.73	27.77	73.60	24.95
	Control	48.56	15.52	56.50	31.93	56.31	29.87	53.79	23.27
DAS	SSRI	156.80	29.98	145.20	27.89	141.86	26.20	147.95	25.46
	Control	125.31	30.46	122.56	31.55	125.81	41.78	124.56	29.81
BAI	SSRI	16.80	10.86	11.46	7.54	10.73	8.47	13.0	8.07
	Control	4.87	5.00	7.0	5.31	6.18	4.81	6.02	4.62
EBS: self-self	SSRI	0.24	0.26	0.49	0.86	0.58	0.81	0.43	0.63
	Control	0.10	0.11	0.57	0.78	0.79	0.84	0.49	0.54
EBS: self-other	SSRI	0.59	0.50	0.51	0.49	0.41	0.38	0.50	0.40
	Control	0.24	0.32	0.16	0.24	0.24	0.37	0.21	0.26
EBS: other-self	SSRI	1.04	0.89	0.82	0.77	0.99	0.88	0.95	0.80
	Control	0.28	0.39	0.36	0.55	0.45	0.65	0.36	0.43

Beck Depression Inventory. There was a main effect of group, $F(1, 29) = 16.99, p < .001$, and a main effect of time, $F(2, 58) = 16.98, p < .001$, indicating that the SSRI group scored higher overall on the BDI, and that BDI score reduced over time. There was an expected significant 2-way interaction of time by group, $F(2, 58) = 25.28, p < .001$, and the results of further post hoc tests using a Bonferroni corrected p value of .017 (.05/3), showed that the SSRI group had a significantly higher BDI score compared to the control group at Time 1, $t(29) = 6.49, p < .001$, at Time 2, $t(14) = 1.05, p = .308$, but not at Time 3, $t(29) = 1.69, p = .101$.

Further paired sample t -tests also using a corrected p value of .017 investigating within group changes revealed that the BDI score for the SSRI group significantly reduced from Time 1 to Time 2, $t(14) = 6.41, p < .001$, did not significantly differ from Time 2 to Time 3, $t(14) = 1.05, p = .308$, but BDI scores at Time 1 were significantly higher than at Time 3, $t(14) = 6.13, p < .001$. This indicated that depressive symptomatology had significantly reduced in the SSRI group after six months of antidepressant treatment. Control group comparisons revealed no differences in BDI scores comparing Time 1 to Time 2, $t(15) = .501, p = .623$, Time 2 to Time 3, $t(15) = 1.24, p = .231$, and Time 1 to Time 3, $t(15) = 1.01, p = .328$.

As hypothesised, depressive symptomatology significantly reduced over the course of treatment for the SSRI group to levels that were not significantly different to that of the control group.

Automatic Thoughts Questionnaire. There was main effect of group, $F(1, 29) = 5.23$, $p < .05$, indicating that the SSRI group had a higher ATQ score overall than the non-depressed group. More importantly, an expected significant 2-way interaction of time by group, $F(2, 58) = 9.69$, $p < .001$, followed by post-hoc independent t-tests using a corrected Bonferroni p value of .017 (.05/3) indicated that the SSRI group scored higher on the ATQ at Time 1, $t(29) = 4.98$, $p < .001$, but not at Time 2, $t(29) = 1.04$, $p > .104$, or Time 3, $t(29) = .715$, $p = .480$, compared to the control group.

Paired sample t-tests (.05/3) investigating differences within the SSRI group showed a significantly lower ATQ score at Time 2 compared to Time 1, $t(14) = 3.36$, $p = .005$, a non-significant difference at Time 2 compared to Time 3, $t(14) = .840$, $p = .415$, but a significant difference at Time 1 compared to Time 3, $t(14) = 4.01$, $p = .001$. Paired sample t-tests within the control group revealed no differences in ATQ scores over time; Time 1 compared to Time 2, $t(15) = 1.58$, $p = .133$, Time 2 compared to Time 3, $t(15) = .031$, $p = .976$, and Time 1 compared to Time 3, $t(15) = 1.27$, $p = .223$.

In accord with the hypotheses, the SSRI group's ATQ score significantly reduced over time to similar levels to that of the control group.

Beck Anxiety Inventory. Again there was main effect of group, $F(1, 29) = 8.86$, $p < .01$, indicating overall that the SSRI group had a higher BAI score, and a time by group interaction, $F(2, 58) = 9.11$, $p < .001$. Further analysis to explore this interaction using independent t-tests (.05/3) revealed that the SSRI group scored significantly higher on the BAI at Time 1, $t(29) = 3.96$, $p < .001$, but not at Time 2, $t(29) = 1.91$, $p = .065$, or Time 3, $t(29) = 1.85$, $p = .074$, compared to the control group.

Paired t-tests (.05/3) showed that the SSRI group's BAI score did not significantly reduce from Time 1 to Time 2, $t(14) = 2.41$, $p = .030$, or from Time 2 to Time 3, $t(14) = .59$, $p = .565$, but did from Time 1 to Time 3, $t(14) = 3.09$, $p = .008$. Within the control group no significant differences in BAI scores were observed when comparing Time 1 to Time 2, $t(15) = .228$, $p = .037$, Time 2 to Time 3, $t(15) = 1.37$, $p = .191$, and Time 1 to Time 3, $t(15) = 1.25$, $p = .229$.

The BAI analysis confirms the hypothesis that levels of anxious symptomatology would reduce to similar levels compared to the control group as depression was treated with SSRI's.

Dysfunctional Attitudes Scale. There was a main effect of group, $F(1, 29) = 5.483$, $p < .05$, which indicated that overall the SSRI group scored higher on the DAS. There was no main effect of time, $F(2, 58) = 1.48$, $p > .05$, and the time by group interaction was not

significant, $F(2, 58) = 1.28, p > .05$. This indicates that, contrary to the hypothesis, the SSRI group's DAS score was not higher at the beginning of treatment compared to the control group and did not significantly change during treatment.

Evaluative Beliefs Scale. There was a significant main effect of group, $F(1, 29) = 4.14, p = .05$, and a main effect of EBS subscale, $F(2, 58) = 3.32, p < .05$, which was qualified by a significant EBS subscale by group interaction, $F(2, 58) = 3.66, p < .05$. There was also a time by subscale interaction, $F(4, 116) = 9.520, p < .001$, which indicated that participants scored significantly lower on the self-self subscale at time 1. However, there was no effect of time $F(2, 28) = 2.81, p > .05$, no interaction of time by group, $F(2, 58) = 1.80, p > .05$, and no significant interaction of time by EBS subscale by group, $F(4, 116) = .762, p > .05$. Further t-test's (.05/3) to explore the EBS subscale by group interaction revealed that the SSRI group did not significantly differ compared to the control group on self-self beliefs, $t(29) = .250, p = .804$, or self-other beliefs, $t(29) = 2.42, p = .022$, but did report more negative beliefs regarding what they thought other people thought of them (other-self beliefs), $t(29) = 2.57, p = .015$. There were no significant results from an analysis using paired t-test's using Bonferroni corrected p value of .017 (highest $p = .033$) to investigate within group differences.

In summary, the SSRI group held stable beliefs over six months, which indicated that they believed that other people think more negatively of them than the control group. Surprisingly, no difference was exhibited on the self-self beliefs subscale (how negatively one thinks about oneself) between the SSRI group and control group at Time 1 when symptoms were significantly higher in the SSRI group. These results are not in accord with the hypothesis that EBS scores on associated subscales would reduce in the SSRI group to comparable levels of the control group.

Young Schema Questionnaire. There was a main effect of group, $F(1, 29) = 8.78, p < .01$, which showed that the SSRI group scored higher overall on average on the YSQ individual subscales. There was also a main effect of YSQ subscale, $F(14, 406) = 11.95, p < .001$, and a YSQ subscale by group interaction, $F(14, 406) = 2.35, p < .01$, but no time by subscale by group interaction, $F(28, 812) = 1.683, p > .05$, time by group interaction, $F(2, 58) = 1.34, p > .05$. Further investigation to ascertain the nature of the YSQ subscale by group interaction, using independent t-tests and a Bonferroni corrected p value of .003 (.05/15), indicated that the SSRI group scored significantly higher overall on the Social Isolation subscale, $t(29) = 4.55, p < .001$. The Failure subscale was just marginally non-significant, $t(29) = 3.16, p = .004$.

The 2-way interaction of time by group was not significant, $F(2, 28) = 1.33, p > .05$, nor was the 3-way interaction of time by YSQ subscale by group, $F(28, 812) = 1.68, p > .05$,

indicating that change over time on YSQ (average scores of all subscales) or individual YSQ subscale scores was not evident in either group.

These results are not in accord with the hypothesis. YSQ scores in the SSRI group (and on any subscale) did not reduce as a result of successful resolution of depressive symptomatology (e.g., BDI & ATQ). Interestingly, only the early maladaptive schema of Social Isolation was significantly higher and stable over time in the SSRI group compared to the control group.

Table 13

Mean Scores on the YSQ Subscales by the SSRI and Control Group

YSQ subscales:	Group	Time 1		Time 2		Time 3		Mean overall	SD
		Mean	SD	Mean	SD	Mean	SD		
Abandonment	SSRI	3.26	1.57	2.29	.922	1.74	1.18	2.43	0.93
	Control	2.40	1.13	2.50	1.46	2.26	1.34	2.38	1.19
Emotional deprivation	SSRI	3.18	1.12	2.78	1.58	2.70	1.23	2.89	1.13
	Control	1.98	1.02	2.17	1.18	2.16	1.48	2.10	1.14
Mistrust/abuse	SSRI	3.01	1.10	2.82	1.13	2.44	1.36	2.76	0.90
	Control	2.53	1.29	2.18	.868	2.33	1.20	2.35	0.99
Social isolation	SSRI	3.82	1.60	3.69	1.41	3.48	1.21	3.66	1.04
	Control	2.06	0.95	2.12	1.16	1.95	1.09	2.04	0.94
Defectiveness/shame	SSRI	2.85	1.36	2.26	1.13	2.09	0.89	2.40	0.90
	Control	1.56	0.71	1.77	1.06	1.85	1.26	1.72	0.94
Failure	SSRI	3.17	1.76	2.82	1.60	14.33	5.76	6.77	2.45
	Control	1.80	0.88	1.75	0.96	9.00	5.44	4.18	2.19
Dependence	SSRI	2.52	1.15	2.37	0.95	12.73	7.98	5.87	2.97
	Control	1.63	0.62	1.66	0.87	9.18	4.91	4.16	1.85
Vulnerability to harm	SSRI	2.41	0.95	2.13	0.83	1.60	0.83	2.04	0.68
	Control	1.75	0.59	1.88	0.80	1.86	0.82	1.83	0.67
Enmeshment	SSRI	1.48	0.60	1.74	0.83	2.04	1.42	1.75	0.75
	Control	1.61	0.73	1.45	0.65	1.36	0.54	1.47	0.58
Subjugation	SSRI	2.50	1.10	2.36	0.83	2.24	1.08	2.36	0.60
	Control	2.30	1.15	2.02	1.10	2.13	1.35	2.15	1.11
Self-sacrifice	SSRI	3.00	1.15	3.06	0.83	3.30	0.97	3.12	0.76
	Control	3.00	1.00	2.81	0.88	2.63	0.86	2.81	0.85
Emotional inhibition	SSRI	2.92	0.97	2.92	1.23	2.77	1.36	2.87	1.03
	Control	2.20	1.01	2.20	0.98	2.05	1.13	2.15	0.88
Unrelenting standards	SSRI	3.85	1.30	3.88	1.28	4.09	0.91	3.94	0.94
	Control	3.20	1.32	3.42	1.24	3.15	0.90	3.25	0.91
Entitlement	SSRI	2.93	1.11	2.78	0.97	2.76	0.92	2.82	0.83
	Control	2.51	1.21	2.75	1.25	2.67	1.34	2.64	1.17
Insufficient self-control	SSRI	3.49	1.35	3.04	1.21	3.20	1.05	3.24	0.91
	Control	2.16	0.96	2.52	1.29	2.38	1.18	2.35	1.03

EAST Analyses

The EAST produced two types of data: reaction times and error rates. Each of these is presented separately. The following set of EAST analyses looked at positive and negative words, but did not sub-divide them into separate mood and schema word categories. The EAST analyses involved two, 2 (word-type) x 2 (person) x 3 (time) x 2 (group) repeated measures ANOVA's, the first three factors being within subjects factors, the latter a between subjects factor. The hypothesis was that the SSRI group would have a stable and weaker positive automatic self-evaluative bias by virtue of having slower reaction times and more errors when positive words were associated with the self compared to the control group. Alternatively, this could be shown by faster reaction times and fewer error rates when negative words were associated with the self compared to the control group. Mean reaction times (untransformed) and error rates from the EAST are presented in Tables 14 & 15.

EAST-Reaction time. There was a main effect of time, $F(2, 58) = 4.765, p < .05$, which showed that all participants became faster over time. This might represent the effects of practice as participants may have become familiar with the task over subsequent testing sessions. There was also a significant 3-way interaction of word-type by person by time, $F(2, 58) = 4.84, p < .05$. Paired t-tests using a Bonferroni corrected p value of .004 (.05/12) indicated that participants were quicker when self was associated with negative words at time 3 compared to time 1, $t(30) = 3.13, p = .004$, and were quicker when positive words were associated with other at time 3 compared to time 1, $t(30) = 4.11, p < .001$. There were no other significant main effects or interactions (highest $p = .09$), although a 3-way (word-type x person x group) indicated a trend towards significance, $F(2, 58) = 3.87, p = .059$.

These results are not in accord with the hypothesis that the SSRI group, despite resolution of depressive symptoms, would still have a stable and weaker positive automatic self-evaluative bias compared to the control group.

Table 14

Mean Reaction Time on the EAST by the SSRI and Control Group

Word-type								Mean	
when		Time 1		Time 2		Time 3		overall	
associated with	Group	Mean	SD	Mean	SD	Mean	SD	***	SD
Low	SSRI	694.39	127.45	646.82	115.66	654.53	119.08	665.25	96.62
mood/other	Control	713.41	129.68	664.11	95.75	670.94	129.51	682.82	99.58
Low mood/self	SSRI	724.18	175.14	647.06	110.27	628.75	139.56	666.66	115.38
	Control	714.43	146.15	680.94	102.11	650.92	113.85	682.10	108.06
Positive	SSRI	712.50	149.05	655.10	104.01	640.54	119.76	669.38	110.89
mood/other	Control	718.09	133.34	636.41	93.11	656.38	118.60	670.29	102.80
Positive	SSRI	670.36	124.82	672.21	109.04	633.95	112.52	658.84	91.83
mood/self	Control	697.23	140.59	624.42	95.30	646.51	106.06	656.06	91.22
Positive	SSRI	668.32	113.26	658.43	86.13	624.70	87.65	650.48	68.02
schema/other	Control	737.01	149.78	682.14	132.68	684.85	137.67	701.33	116.88
Positive	SSRI	696.60	139.25	685.58	123.12	662.11	134.45	681.43	112.96
schema/self	Control	691.43	141.84	655.96	104.23	634.21	101.47	660.53	100.70
Negative	SSRI	652.28	125.51	647.45	127.98	684.10	202.63	661.28	124.06
schema/other	Control	697.97	121.81	635.25	85.62	664.00	115.85	665.74	94.59
Negative	SSRI	666.42	170.92	653.16	179.93	597.56	80.35	639.05	95.20
schema/self	Control	705.78	124.49	672.47	102.98	663.36	102.78	680.54	94.00
Negative*	SSRI	673.34	117.49	647.13	106.32	669.32	137.83	663.26	100.30
words /other	Control	705.69	118.70	649.68	83.37	667.47	118.33	674.28	93.94
Negative*	SSRI	695.30	158.77	650.11	116.33	613.15	89.36	652.85	86.66
words/self	Control	710.10	128.19	676.71	94.18	657.14	100.28	681.32	94.49
Positive**	SSRI	690.41	94.77	656.77	67.41	632.62	79.72	650.48	68.02
words/other	Control	727.55	124.54	659.28	107.18	670.61	115.06	701.33	116.88
Positive	SSRI	683.48	103.40	678.90	89.72	648.03	92.42	670.14	70.00
**words/self	Control	694.33	133.02	640.19	86.23	640.36	93.96	658.29	86.59

*=negative schema and low mood words combined, **=positive mood and positive schema words combined, ***=mean over 3 time points.

EAST- Error rates. This analysis produced a significant word-type by group interaction, $F(1, 29) = 6.01, p < .05$, and a significant person by group interaction, $F(1, 29) = 4.90, p < .05$. These results were qualified by a hypothesised significant 3-way word-type by

person by group interaction, $F(1, 29) = 4.38, p < .05$. However, the 4-way interaction of word-type by person by time by group, $F(2, 58) = .08, p > .05$, was not significant. Further analysis exploring the significant 3-way interaction of word by person by group using independent t-tests and a corrected p value of .012 (.05/4) revealed a non-significant trend for the SSRI group to make fewer errors, compared to the control group when negative words were associated with the self, $t(29) = 2.57, p = .016$. Paired t-tests (.05/4) within the SSRI group did not provide any significant results. However, paired t-tests within the control group showed that they made more errors when negative words were associated with the self, compared to when negative words were associated with other, $t(15) = 4.33, p = .001$ (see Figure 14). This showed that the control group overall did not have a strong negative automatic self-evaluation, but did have a strong negative “other” evaluation. The control group therefore thought more negatively of others than they did of themselves.

Figure 14. Control group error rates when negative words are associated with self and other (EAST).

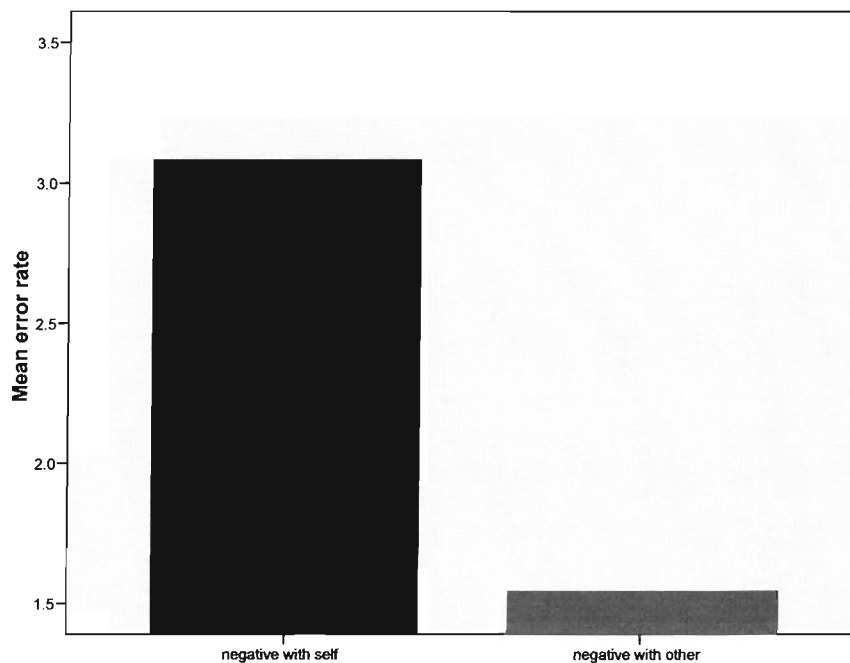


Table 15

Mean Error Rates on the EAST by the SSRI and Control Group

Word-type when associated with self and other		Time 1		Time 2		Time 3		Mean overall	
	Group	Mean	SD	Mean	SD	Mean	SD	***	SD
Low mood/other	SSRI	1.13	2.32	0.93	1.86	1.13	1.55	1.06	1.80
	Control	1.18	1.16	0.75	0.93	1.12	1.50	1.08	1.03
Low mood/self	SSRI	0.73	.961	0.87	0.91	0.60	0.50	0.73	0.42
	Control	1.56	1.45	1.75	1.73	1.25	1.39	1.62	1.30
Positive mood/other	SSRI	1.0	1.13	0.80	0.77	1.13	1.06	0.98	0.78
	Control	0.56	0.89	0.62	1.14	1.12	1.14	0.73	0.93
Positive mood/self	SSRI	0.93	1.03	1.73	1.43	1.0	1.00	1.22	0.10
	Control	0.94	1.52	0.87	1.02	1.06	1.12	1.00	1.14
Positive schema/other	SSRI	1.93	3.32	1.26	2.54	1.26	1.27	1.48	2.28
	Control	1.37	2.12	0.62	0.80	1.25	1.65	1.06	1.10
Positive schema/self	SSRI	1.0	1.55	1.13	1.18	0.80	1.20	0.98	1.21
	Control	0.69	1.13	0.87	1.25	0.87	1.50	0.79	0.95
Negative schema/other	SSRI	1.20	2.56	1.06	1.38	1.0	1.46	1.08	1.63
	Control	0.31	0.60	0.31	0.48	0.75	0.93	0.46	0.62
Negative schema/self	SSRI	0.80	1.014	1.06	1.03	0.40	0.73	0.75	0.74
	Control	1.43	1.36	1.18	1.04	1.68	1.70	1.45	1.15
Negative* words /other	SSRI	2.33	4.76	2.00	3.13	2.13	2.55	2.15	3.37
	Control	1.50	1.50	1.25	1.39	1.87	1.74	1.54	1.38
Negative* words/self	SSRI	1.53	1.76	1.93	1.62	1.00	1.00	1.49	1.03
	Control	3.00	2.47	3.12	2.57	3.12	2.89	3.08	2.18
Positive** words/other	SSRI	2.93	3.36	2.06	2.71	2.40	1.84	2.46	2.37
	Control	1.93	2.56	1.25	1.52	2.18	2.34	1.78	1.62
Positive** words/self	SSRI	1.93	2.40	2.86	2.09	1.80	1.89	2.20	1.97
	Control	1.62	2.47	1.75	2.11	2.0	2.25	1.79	2.00

*=negative schema and low mood words combined, **=positive mood and positive schema words combined, ***=mean over 3 time points.

IAT Analyses

Like the EAST, the IAT produced two types of data: reaction time and error rates. Again, each of these were analysed separately. The hypothesis for each IAT (Mood & Schema) on both the reaction time and error analyses was that the SSRI group would possess

a stable and weaker positive automatic self-evaluative bias despite having been treated with SSRI's. In other words, the control group would perform more efficiently (fast RT and low error rate) on the compatible block of the IAT (when the self was associated with positive words) compared to the incompatible block. Therefore the discrepancy in performance between the two test blocks would be greater in the control group.⁶ This would reflect a stronger positive automatic self-evaluative bias. Statistical analysis for each IAT involved a 2 (compatibility) x 2 (time) x 2 (group) repeated measures ANOVA, time and compatibility being within subjects factors and group a between subject factor. See Table 16 for mean reaction times and error rates from the IAT Mood and IAT Schema.

IAT Mood-Reaction time. There was a main effect of compatibility, $F(1, 29) = 24.584, p < .001$, a main effect of time, $F(2, 58) = 6.44, p < .01$, and a marginally non-significant interaction of compatibility by time, $F(2, 58) = 3.02, p = .056$. These results indicate that all participants were quicker on the compatible block of the IAT (irrespective of time), that all participants became quicker over time, and that there was a non-significant trend for all participants to become quicker on the compatible block of the IAT over time. The hypothesised compatibility by group interaction was non-significant, $F(1, 29) = 1.26, p > .05$. This analysis disconfirms the hypothesis that the SSRI group would have a stable and weaker positive automatic self-evaluation in comparison to the control group. There were no other significant results (highest $p = .242$).

IAT Schema-Reaction time. As with the IAT Mood, there was a main effect of compatibility, $F(1, 29) = 14.68, p < .01$, and a main effect of time, $F(2, 58) = 4.09, p < .05$, but a non-significant compatibility by time interaction, $F(1, 58) = .843, p > .05$. This showed that overall, participants were quicker on the compatible blocks of the IAT, and that they developed quicker reactions times over time, but that this was not related to compatibility. More importantly, there was, contrary to the hypothesis, no compatibility by group interaction, $F(1, 29) = 1.96, p > .05$. This shows that the SSRI group, like the control group, had a positive automatic self-evaluative bias and that this was maintained over time. There were no other significant results (highest $p = .32$).

⁶ Research has shown that participants reliably have faster reaction times or fewer error rates on the compatible block of the IAT compared to the incompatible block (de Jong, 2000). In other words, if Person A is very fast or very accurate on the compatible block of the IAT, but very slow or inaccurate on the incompatible block of the IAT, while Person B performs in a relatively similar way on both blocks, then Person A will receive a smaller IAT effect score as a result of his superior performance on the compatible block. Person A's score will reflect a larger discrepancy between the compatible and incompatible block, which is indicative of a stronger self-evaluative bias.

Table 16

Mean Reaction Times and Error Rates on the IAT Mood and IAT Schema by the SSRI and Control Group

		<u>IAT Mood</u>						Mean overall ***	SD
Group		Mean	SD	Mean	SD	Mean	SD		
		<u>Time 1</u>		<u>Time 2</u>		<u>Time 3</u>			
Compatible* phase RT	SSRI	727.11	142.91	660.50	108.56	679.87	126.51	689.16	114.28
	Control	726.16	126.04	725.64	163.25	698.59	148.47	716.80	125.45
Incompatible** phase RT	SSRI	834.06	234.08	766.06	117.89	725.36	160.62	775.16	139.72
	Control	941.21	229.70	846.86	209.64	793.48	168.55	860.52	170.97
Compatible* phase errors	SSRI	3.13	1.92	2.80	2.75	3.80	2.70	3.24	2.14
	Control	2.62	2.15	2.37	2.15	3.06	1.87	2.68	1.76
Incompatible** phase errors	SSRI	2.53	2.44	4.73	2.65	3.93	1.79	3.73	1.97
	Control	4.12	2.82	4.25	4.23	5.12	3.50	4.50	3.05

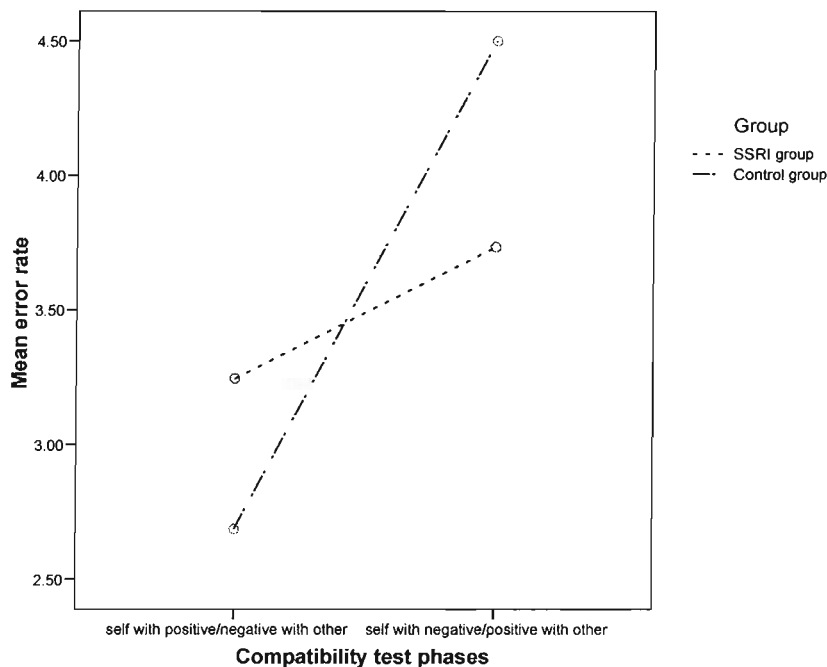
		<u>IAT Schema</u>						Mean overall ***	SD
Group		Mean	SD	Mean	SD	Mean	SD		
		<u>Time 1</u>		<u>Time 2</u>		<u>Time 3</u>			
Compatible* phase RT	SSRI	755.82	175.04	678.19	125.93	661.22	96.53	698.41	111.84
	Control	683.29	198.09	694.64	136.79	659.56	107.83	679.16	122.00
Incompatible** phase RT	SSRI	826.18	202.25	769.27	133.40	704.84	110.34	766.77	125.21
	Control	886.03	265.61	820.40	172.04	761.29	124.23	822.58	155.75
Compatible* phase errors	SSRI	3.13	2.13	3.13	2.13	2.06	1.48	2.77	1.36
	Control	2.87	2.70	2.75	2.67	2.62	2.028	2.75	2.17
Incompatible** phase errors	SSRI	3.33	2.58	3.20	2.30	3.66	2.19	3.40	1.76
	Control	3.81	3.85	4.25	2.62	3.56	2.36	3.87	2.54

*= Self with positive words/negative words with other, **=self with negative words/positive words with other, ***=mean reaction time and error rate over 3 time points.

IAT Mood-Error rates. There was a main effect of compatibility, $F(1, 29) = 14.08$, $p < .01$, and a main effect of time, $F(2, 58) = 4.08$, $p < .05$. However, there was a non-significant (although on a trend to becoming significant) two-way interaction of compatibility by time, $F(2, 58) = 2.82$, $p = .068$. These results indicate that overall all participants made fewer errors on the compatible block of the IAT, made fewer errors over time, and there was a trend to make fewer errors on the compatible block over time.

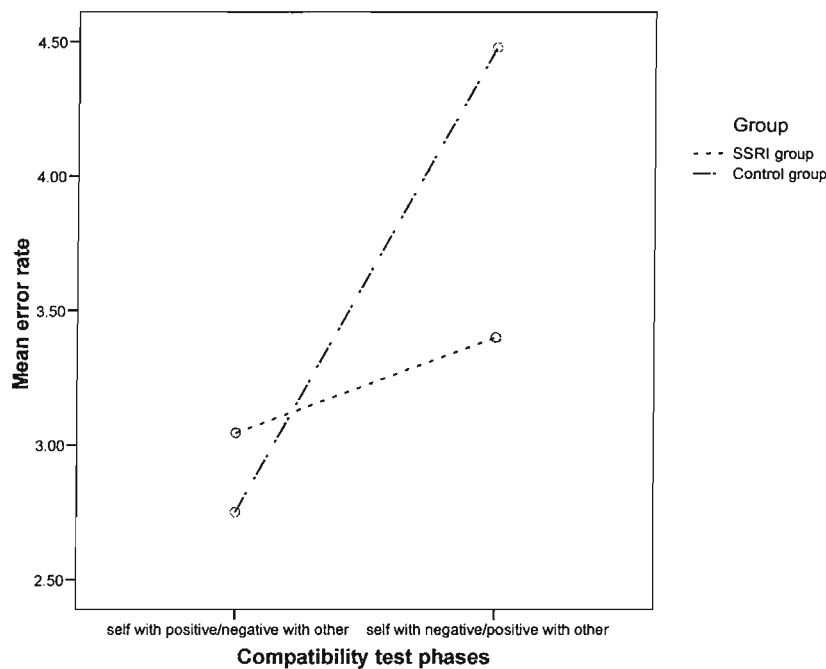
More importantly, there was a significant two-way interaction of compatibility by group, $F(1, 29) = 4.66, p < .05$, and a non-significant three-way interaction of compatibility by time by group, $F(2, 58) = 1.91, p > .05$. The control group had more errors on the incompatible block (i.e. self with negative and other with positive). This indicates, as one can see from Figure 15, that the control group had stable and a stronger positive self-evaluative bias compared to the SSRI group. This IAT error analysis indicates that the SSRI group had a stable and weaker positive automatic self-evaluative bias compared to the control group. There were no other significant results (highest $p = .26$).

Figure 15. Compatibility by group interaction on the IAT mood with error rates.



IAT Schema- Error rates. Again, there was a main effect of compatibility, $F(1, 29) = 12.31, p < .01$, indicating participants made fewer errors on the compatible block of the IAT. More importantly, however, there was a two-way compatibility by group interaction as hypothesised, $F(1, 29) = 5.34, p < .05$ (see Figure 16), and a non-significant three-way interaction when adding time, $F(2, 58) = 1.44, p > .05$. This again indicates that the SSRI group had a stable and weaker positive automatic self-evaluative bias as compared to the control group. There were no other significant results (highest $p = .35$).

Figure 16. Compatibility by group interaction on the IAT Schema with error rates.



Specific schema content analyses

The following investigations involved testing the specific schema content hypothesis that a stable and weaker positive automatic self-evaluative bias would be more pronounced in the SSRI group on the IAT and on the EAST that uses material congruent with themes hypothetically associated with schema development (e.g., themes of abandonment and defectiveness (see Alloy et al., 1999; Parker et al., 2000). In other words, if self-schema structures (in depressed people or those vulnerable to depression) contain beliefs of themes associated with abandonment and defectiveness, and little positive schematic content, then hypothetically a weak positive self-evaluative bias should be more evident on a schema content IAT compared to a mood descriptor IAT and on the EAST towards negative schema related stimuli.

Specific schema content IAT analyses. This analysis involved calculating IAT effects for each IAT for both reaction time and error rate data. As described in Study 3 (see IAT effect description in the results section of Study 3 for full details of this calculation), an IAT effect is calculated by subtracting the average reaction time or error rate of the compatible block from the incompatible block. The larger the relative score after this calculation is indicative of a stronger positive self-evaluative bias.

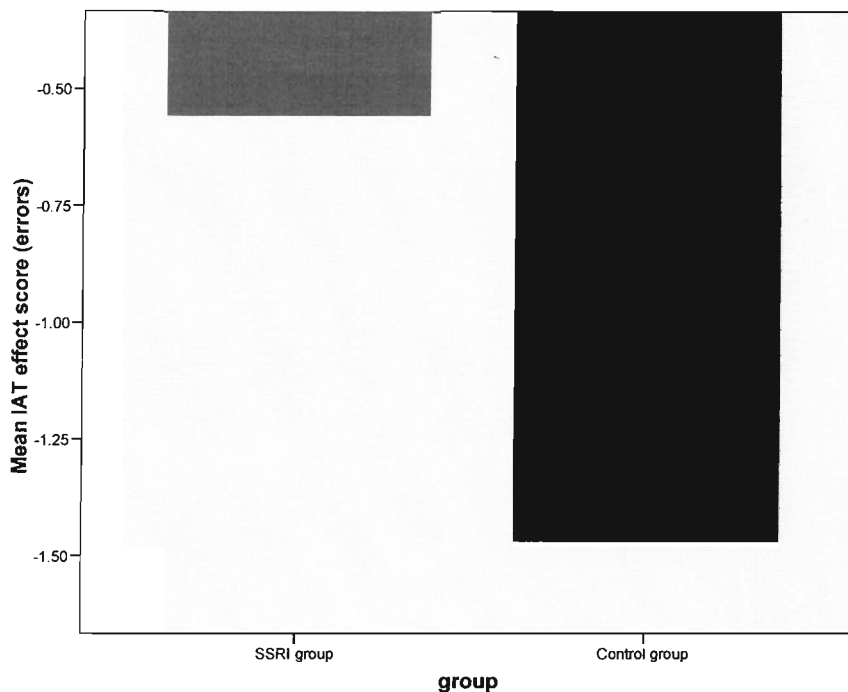
The IAT analysis for both error rates and reaction times involved a 3 (time) x 2 (IAT-Type; Mood & Schema) x 2 (group) repeated measures ANOVA, with time and IAT-type as

within subjects factors, and group as a between subjects factor. IAT effect reaction time scores used for the statistical analysis were calculated from log-transformed reaction times.

Specific schema content IAT Effect-Reaction time. The main effect of time was not significant (although this was on a trend to becoming significant), $F(1, 29) = 3.00, p = .077$, which indicated that there was trend for participants to become faster over time. There was a non-significant effect of IAT-Type, $F(1, 29) = .30, p > .05$, which indicated that overall participants performed similarly on both IAT's. There was also a non-significant IAT-Type by Group interaction, $F(2, 29) = .408, p > .05$, and a non-significant time by IAT effect by Group interaction, $F(2, 58) = .108, p > .05$. This indicated that the SSRI group did not show a weaker positive self-evaluative bias on the IAT using schema material, and that the bias did not change over time. All other results were non-significant (highest $p = .13$).

Specific schema content IAT Effect-Error rates. The same analysis conducted on error rates yielded a main effect of group, $F(1, 29) = 4.17, p = .05$, which indicated overall (over both IAT's) that the control group had a stronger positive automatic self-evaluative bias compared to the SSRI group (see Figure 17). The main effect of IAT-Type was not significant, $F(1, 29) = .738, p > .05$, which indicated that overall, participants performed similarly on both IAT's. The effect of time was not significant, $F(2, 58) = 1.872, p > .05$, indicating participants' error rates did not change over time. Finally, the IAT-Type by Group interaction, $F(1, 29) = 1.71, p > .05$, and time by IAT-Type by group, $F(2, 58) = 2.49, p > .05$, were non-significant. This showed that the two groups performed similarly on the two IAT's and this was not affected by time. There were no other significant interactions (highest $p = .30$).

Figure 17. Specific schema content IAT effect-Main effect of group (error rates).



One can conclude from both of the IAT effect analyses that the type of negative material did not affect automatic self-evaluation in the SSRI group. These results disconfirm a specific schema content theory of depression. However, the results partly confirm the hypothesis (only with error rate data, not with reaction times) that the control group would exhibit a stable and more positive automatic self-evaluative bias compared to the SSRI group.

EAST: Specific schema content analyses. The specific schema content EAST analysis involved subdividing the negative and positive stimuli for analysis (positive and negative schema words, happy and low mood words). The hypothesis was that the SSRI group would be quicker and commit fewer errors when negative schema words were associated with the self. See Table 19 for means and standard deviations of reaction times and error rates from the EAST. A 4 (word-type) \times 2 (person) \times 3 (time) \times 2 (group) repeated measures ANOVA was performed for both reaction time and error analyses. Word-type, person, and time were again within subjects factors, and group the between subjects factor.

EAST: Specific schema content-Reaction Time. There was a main effect of time, $F(2, 58) = 4.82, p < .05$, which showed that all participants became faster over time when classifying words during the task. There was also a non-significant word-type by person by group interaction, $F(3, 87) = 1.30, p > .05$. There were no other results of significance (highest $p = .11$).

EAST: Specific schema content-Error rates. There was a significant person by group interaction, $F(1, 29) = 4.90, p < .05$, and a significant key by time interaction, $F(2, 58) = 4.69, p < .05$. These results indicated that the control group made more errors when pressing the key associated with self and that all participants made more errors at time 2 when pressing the key associated with self. Again a non-significant 3-way interaction of word-type by person by group, $F(3, 87) = 2.14, p > .05$, disconfirmed the hypothesis that the SSRI group would show a stronger negative automatic self-evaluation in response to specific schema related material. There were no other significant main effects or interactions (highest $p = .08$).

This EAST reaction time and error analyses disconfirmed the specific schema content hypothesis. A significantly stronger and stable negative self-evaluative bias was not evident in response to material related to hypothetical schema content in the SSRI group.

9.4. Discussion

The main aim of Study 5 was to investigate the effects of SSRI antidepressant treatment on depressed individuals using both explicit and implicit measures, compared to a non-depressed control group. In accord with the hypotheses, depressive symptoms measured by the BDI, significantly reduced over six months to levels similar to those in the control group. Furthermore, negative automatic thoughts (ATQ) and anxious symptomatology (BAI) both associated with the syndrome of major depressive disorder, also reduced significantly to levels comparable with those of the control group. Therefore, one can conclude that SSRI's are successful in reducing depressive symptomatology and associated depressive cognition. However, in disagreement with the hypotheses, self-reported dysfunctional attitudes (DAS), themes of social isolation (YSQ), and negative thoughts about other people's opinions of the self (EBS), remained stable and were higher in the SSRI group. With regard to performance on the implicit processing tasks, on the IAT error analyses (IAT Schema, IAT Mood, & IAT Effect) and the EAST error analysis (negative versus positive words), the control group displayed a stable and stronger positive automatic self-evaluation compared to the SSRI group. This difference in automatic self-evaluation between the two groups was not affected by the treatment gains made by the SSRI group (i.e., reduced BDI, ATQ, BAI). These results are only partly in accord with the hypotheses set out for this study. This is because, unexpectedly, no significant results from the implicit tasks were obtained from the reaction time data. This issue will now be discussed below.

One subsidiary aim of this study was to investigate whether there was any evidence that material related to hypothetical etiological antecedents of depression and schema formation affected information processing. On the EAST and both IAT effect analyses (errors and reactions times), there was no evidence that the schema material engendered an information processing bias linked to themes associated with etiological antecedents of

schema formation. This finding is not in accordance with the hypothesis for this part of the study.

Error rates versus reaction time

With regard the main aim of investigating automatic self-evaluation and the effects of SSRI treatment, a stable weak negative automatic self-evaluation was found in the SSRI group, despite a reduction in depressive symptoms to levels comparable to that of the control group. Yet, this was only observed with error rates on the EAST and IAT's. This is problematic, as reaction times have been traditionally used as a measure of performance on implicit processing tasks (e.g., Segal, 1988; De Raedt et al., 2006; Gamar et al., 2001).

However, very few researchers have addressed the possibility or usefulness of measuring error rates as opposed to response latencies in implicit processing in depression research (e.g., Gamar et al., 2001; De Raedt et al., 2006). It has been posited that error rates may be good indicators of automatic self-evaluation (Kirsch & Lynn, 1999; Bargh & Tota, 1988; Greenwald et al., 1998) and may be more sensitive to tapping into vulnerability to depression (Eysenck, 1991). The issue of the utility of errors rates versus reaction times may have specific relevance to the research involved with schematic activity and vulnerability to depression. As discussed in detail in Chapter 4, there has been little evidence of schematic activity in the absence of a low or depressed mood obtained from research into depression (Gamar et al., 2001; Allot et al., 1999). This paucity of evidence for schematic activity in the absence of a low or depressed mood may well therefore be reflective of the dismissal of important data (error rates) and the over-reliance on the measurement of reaction time which, as discussed, may be more reflective of explicit or controlled processes, but not true automaticity (Kirsch & Lynn, 1999; Bargh & Tota, 1988). Therefore, the results obtained in this study and in Study 3, suggest that error rates may be a fruitful alternative avenue to measure automatic self-evaluation, if explicit processes or other confounding variables affect response speed. Indeed, Townsend & Ashby (1983) suggest that researchers should consider all the data from implicit tasks and investigate speed-accuracy trade-offs if the data is difficult to interpret and, if necessary, use a relative measure of efficiency as an index of implicit processing. This facet of performance on implicit tasks obviously needs further investigation in order to be able to make firmer inferences regarding the role of errors versus reaction time, the role they play in schemata, and vulnerability to depression.

Explicit versus implicit measures

The idea of a dysfunctional automatic self-evaluative system, as found in this study in the SSRI group, is starting to be considered in vulnerability to depression and other affective disorders (De Raedt et al., 2006; de Jong, 2000). Traditionally, researchers have focused on ideas about negative self-schemata which, when activated, produce distortions in information processing (Segal, 1988). However, such biases in information processing or scoring on

questionnaires have normally been found to be mood-state dependent (Gemar et al., 2001; Zuroff et al., 1999; Segal et al., 1999). Evidence of enduring vulnerability to depression in asymptomatic states may have been thwarted by the methods of schema measurement used in other studies. For example, traditional self-report measures are influenced by self-presentational demands (Nisbett & Wilson, 1977) and implicit or automatic processing tasks may be measuring more explicit rather than implicit processes (Bargh & Tota 1988). This study, with the use of two relatively new automatic-processing tasks, demonstrates that enduring vulnerability to depression in successfully treated depressed patients by SSRI's, may be measurable and may not be mood-state dependent.

However, although a weaker positive automatic self-evaluation was observed in the SSRI group, contrary to the hypothesis, the SSRI group also showed higher scores on certain questionnaires. The SSRI group showed stable and overall higher levels of dysfunctional attitudes (DAS), social isolation (YSQ), and negative thoughts regarding how they thought other people view them (EBS-self-other). This occurred even though depressive symptoms (BDI) and associated negative cognition (ATQ) in the SSRI group had reduced with treatment and returned to levels similar to those of the control group. These findings are difficult to reconcile considering the study's hypothesis, that negative cognition would only be shown by the SSRI group at an implicit level (i.e., on the EAST and IAT's). It could be argued that the depressive symptoms in the SSRI group had not been adequately resolved as they still did show moderate levels of depressive symptomatology. This may have contributed to the high scores on the other self-report measures (i.e., DAS etc) and the lack of a positive automatic self-evaluative bias. It could also mean that self-report measures of dysfunctional attitudes (DAS), the schema of social isolation (YSQ) and negative self-other thoughts (EBS-self-other) are also markers of enduring vulnerability to depression (Parker et al., 2000; Alloy et al., 1999; Segal et al., 1999). Contrary to this idea, Miranda and Pearsons (1988) argue that scores on the DAS are mood-state dependent. This would then indicate that the SSRI group had indeed significant residual depressive symptomatology that contributed to the high scores on the aforementioned self-report measures. However, it has been argued that individuals who have recovered from clinical depression are different from individuals who are vulnerable to depression but have not yet experienced a depressive episode. In other words, Lewinsohn et al. (1981) and Segal (1988) postulate that elevated scores on certain questionnaires (e.g., DAS) may be a feature of individuals who have acquired negative self-schematic organisation due to an episode of depression. This kind of schematic organisation may not be evident in vulnerable individuals who have not had an episode of depression (Lewinsohn et al., 1981). Thus, the high scores on some self-report measures may be a result of the sample characteristics (i.e., individuals who have recovered from depression and thus who have acquired negative self-schematic organisation).

Nevertheless, the results found in this study may suggest that one needs to consider both implicit and explicit views of the self and to ascertain how they interact and influence vulnerability to depression. It is postulated that explicit controlled processing and implicit processing measure different constructs and thus should be treated differently, but that implicit processing biases are the direct result of vulnerability to psychopathology (Eysenck, 1991). Thus, it could be argued that a weak positive automatic self-evaluation is intrinsically linked with the surfacing of beliefs of being socially isolated, negative dysfunctional attitudes, and fear of being judged by others. Holding such negative explicit beliefs may in turn perpetrate a weak positive automatic self-evaluation, or a weak positive automatic self-evaluation may contributed to the formation of negative explicit beliefs. These complex facets of the phenomenology of depression may not be mutually exclusive in relation to vulnerability to depression, or depressive relapse. They may influence each other, be related to an individual's overall self-evaluation or self-schema, and affect how one reacts to environmental stressors (Beck, 1967, 1976). Certainly, self-report measures may at times be unreliable and individuals may not be fully aware of the extent of their negative comportment in the world. However, the SSRI group were aware that they were suffering from depression and thus may to some extent have started constructing a "depressive narrative" to explain their depression (or may have been aided by their doctor in constructing one). These processes may have helped bias in some way the SSRI group's responses on the questionnaires via self-presentational strategies (Nisbett & Wilson, 1977).

In summary, more research needs to be done to investigate the role of implicit and explicit beliefs/self-evaluation on current depression and vulnerability to depression. Bearing this in mind, the issue of implicit schematic content or the content of schematic beliefs has also to be addressed and how this relates to explicit belief or processes and vulnerability to depression. This issue will now be discussed.

Specific schema content

A specific schema content theory was not supported in this study. It has been argued that depressed individuals may possess negative self-schemas that contain negative self-representations or content as a result of the developmental antecedents of schema formation; themes of being defective or abandoned as a result of negative early childhood experiences (Alloy et al., 1999; Segal, 1988; Beck, 1967, 1976). As found in this study, the weak positive automatic self-evaluative bias of the SSRI group was not affected by schema or mood related material.

However, De Houwer (2002) argues that tasks measuring automatic self-evaluation, do not specifically measure beliefs or cognitive content or structure per se, but rather the relative strength of associations (e.g., the concept of the self being negative generally). In other words, automatic self-evaluation tasks provide indirect evidence so one can make

inferences as to what these associations might mean with regard to probable beliefs or themes. The awareness or knowledge of connectedness of personal constructs is a conscious, controlled, and explicit activity that develops through reflection, and considered thought (e.g., psychotherapy). Whereas a negative or positive automatic self-evaluation or comportment towards the world may be a more embodied, intuitive, and atheoretical ontological way of being that comes before language (Wheeler, 2006; Heidegger, 2001; Greenfield, 1998; Merleau-Ponty, 1962; Freeman, 2001; Dreyfus, 1989).

This conceptualisation of self-evaluation concurs with Rudman's (2004) argument that automatic (before reflection and thus language) and controlled self-evaluations (based in language) stem from different sources and should essentially be seen as different constructs. From neuroscientific (Le Doux, 1998), psychoanalytical (Bucci, 2000), and cognitive theories of schema development (Beck, 1967, 1976), schemas are hypothesised to develop at an early age where complex language and attributional styles have not yet been developed. Thus the negative self-schema develops initially at a level that may be sub-symbolic and is reflected through an individual's overall ontological orientation. Therefore, a schema may not be a set of attributions, self-representational system, or language based belief system. Rather a schema may be better described as a negativity of self or ontological orientation, the essence of which guides a person in a certain way (e.g., towards depression or other psychopathology). An individual may well formulate from this orientation, through later reflection or psychotherapy, certain complex negative beliefs, self-representations, or attributions (Heidegger, 2001; Merleau-Ponty, 1962). Therefore, the depressed individuals in this study, although showing a weak positive automatic self-evaluative bias, also had high scores on certain self-report measures. This might reflect some kind of understanding on their part of the reasons why, or how, they feel depressed. It may be that their weak positive automatic self-evaluation or ontological insecurity plays a part in creating the consciously reflected thoughts and beliefs represented by self-report measures. Thus, schemata related to vulnerability to depression may be better thought of as a negative automatic ontological view of the self, while the beliefs and thoughts associated with depression and vulnerability to depression may be better considered as afterthoughts to explain the negative self-ontology.

Cognitive models of depression and treatment for depression

In agreement with similar arguments put forward (Hensley et al., 2004; Paykel et al., 1999; Teasdale et al., 2002; Teasdale et al., 2000), this study perhaps provides important information as to why antidepressant medication may leave a residual vulnerability for further episodes of depression. It seems that a weak positive automatic self-evaluation may not be augmented by antidepressant medication. Further, contemporary conceptualisations of a schema being a latent structure that only becomes activated in response to low mood are not supported (e.g., Gemar et al., 2001; Segal et al., 1999; Beck, 1967, 1976). In other words, the

SSRI group showed a stable and weaker positive automatic self-evaluation compared to the control group, despite significant reductions in depressive symptomatology and mood. Therefore, the results of this study are not in accordance with cognitive theories of depression, the latent schema model, and enduring vulnerability to depression following the use of antidepressant medication (e.g., Beck, 1967, 1976; Gemar et al., 2001; Segal et al., 1999; Ingram et al., 1998).

Instead of vulnerability to depression being linked with a latent and mood reactive negative self-schema, vulnerability may be better regarded as a dysfunction in self-evaluation at an automatic level that exerts a subtle effect in the absence of a low mood. This is in agreement with (Alloy et al., 1999; de Jong, 2000) who argue that vulnerability to affective disorders may be implicated in a disordered automatic self-evaluative system. Indeed, Dozois & Dobson, 2001; Greenberg & Alloy, 1989) confirm this view with research findings that suggest that the way in which positive self-evaluation is organised might be the key differentiating factor that separates those who are not vulnerable to depression and those who are. The results from this study and studies 3 and 4 support the idea of a dysfunctional automatic self-evaluative system and also challenge the latent schema model of depression.

These findings fit well with the idea that the presence of a positive self-evaluative bias is essential for mental health and adaptation to stressful environmental situations (de Jong, 2000; Taylor & Brown, 1988). A strong positive automatic self-evaluation thus may be advantageous in situations where individuals are faced with negative environmental events. In the case of those people who are vulnerable to depression, weak positive self-evaluation may theoretically be insufficiently strong enough to protect them from negative environmental events. This idea is in line with Beck's (1967, 1976) and Teasdale & Barnards's (1993) ideas that individuals who are vulnerable to depression react to the environment in a negative and dysfunctional way. A weak self-evaluative system may make an individual prone to deal with environmental stressors in a dysfunctional way. Indeed, elevated scores in the SSRI group on the DAS, Social Isolation (YSQ), and negative thoughts regarding how they thought others judged them may be reflective of potential dysfunctional attributes. These attributes may be caused by having a weak positive self-evaluation and these attributes may in turn affect how one deals with environmental stressors. However, this argument is very much conjecture. It assumes a causal direction from having a weak positive automatic self-evaluation as the cause for dealing with the environment in a dysfunctional way. There is no evidence from this study that this is the case. It could well be that dealing with the environment in a dysfunctional fashion contributes to a weak, self-evaluative system. Thus, more research needs to be done in this area.

Nevertheless, it has been argued that antidepressant medication like SSRI's may produce effects that make social interaction and accurate elaborated cognitive formulation of

experience and the environment difficult to accomplish. SSRI's have been argued to produce emotional blunting, which can affect motivation to engage in, and formulation of social interaction (Tracy, 1994). SSRI's are also reported to inhibit social interaction, as individuals can feel like social misfits for having to rely on a drug to be well. Individuals can also feel defective in comparison to others for being depressed, which in itself can carry a stigma, and in turn induce apathy for social interaction (Vernarde, 1999). Thus, it could be argued, as the findings in this study suggest, that SSRI's do not target issues related to dysfunctional attitudes, social isolation, and thoughts of being negatively judged, which are all related to self-evaluation, but only address issues of depressive symptomatology.

Methodological issues

There are a number of methodological issues that warrant consideration in the interpretation of the findings in this study. These are the effects regarding residual depressive symptomatology, the length of treatment of the SSRI group, differing treatment effects of the different SSRI's (e.g., paroxetine versus fluoxetine), the effects of differing dosages on individuals, and the issue of executive dysfunction associated with depression.

Although the SSRI group did not significantly differ on depressive symptomatology compared to the control group at six months, their BDI score still indicated mild depressive symptomatology ($M=14.93$). Therefore, it could be argued that levels of depression in the SSRI group had not entirely resolved. Thus the lack of a strong positive self-evaluative bias may have been attributable to residual symptoms of depression. Likewise, high scores on the DAS, Social Isolation (YSQ), and self-other beliefs (EBS) may have contributed to implicit processing differences.

Six months is regarded as an optimum period of treatment for SSRI's (Boyer et al., 1996). However, some individuals need longer periods of maintenance medication to become well and recover from depression. Therefore, a follow-up of the SSRI group further into treatment might have produced more informative results. It should also be noted that all the participants were continuing treatment at the close of the study. Therefore it would be preferable to investigate symptoms of depression and automatic self-evaluation at a point when participants were drug free and in remission.

Also, different types of SSRI's can produce different effects (e.g., side-effects and treatment efficacy). Paroxetine is reported to be better in treating depressions with high levels of associated anxiety and social phobia (Boyer et al., 1996) and has more side-effects like irritation or sedation (Healy, 2003). Fluoxetine, on the other hand, is better suited for depressions with little accompanying symptoms of anxiety, as one of the possible side-effects of this drug can be increased anxiety (Boyer et al., 1996). Therefore, to enable one to make firmer inferences of the effects of SSRI's on explicit and automatic self-evaluation, it would have been informative to consider the different types of SSRI's that participants were

prescribed. To carry out this kind of investigation one would preferably need more participants that were engaged in this study.

The differing dosages of each individual's SSRI prescription was not taken into consideration in this study, and this aspect may have some bearing on the resolution (or not) of participants' depressive symptoms (Dorne, Walton, Slobb, & Renwick, 2004; Boyer et al., 1996) and changes in automatic self-evaluation. It would have been informative to take into account the dosage of SSRI prescribed to each participant to see if different dosages had different effects on explicit and implicit cognition. Again, however, one would need more participants to carry out this kind of investigation.

Although research has indicated that cognitive therapy may be superior in resolving abnormal cognition (Teadsale et al., 2002), problematic behaviour patterns (Jacobson & Gortner, 2000), and relapse in depression, one can only speculate whether CBT would engender a positive automatic self-evaluative bias like the control group in this study. Therefore it would be informative to have a CBT group to use as a comparison.

As discussed in Study 4, depression has been linked with central executive dysfunction which can influence performances on information processing tasks (Channon & Green, 2006; Elliot et al., 1997; Watts et al., 1998; Hertel & Hardin, 1990; Elliot et al., 1996). This fact has to be considered in light of the findings in this study. The SSRI group in comparison to the control group did not show as great an information processing bias on the EAST or IAT. In other words, it did not have a strong bias to either negative or positive stimuli when associated with the self or other. This observation suggests that executive dysfunction may have been present in the SSRI group. This possibility is given further strength considering that the SSRI still had mild symptoms of depression after six months of antidepressant treatment. Therefore, the differences in performance by the SSRI group may not be due to a lack of a positive automatic self-evaluative bias, but instead to dysfunctional executive processes affecting performance. Obviously, this issue needs addressing in future research.

Conclusion

Whilst taking into consideration the methodological problems, this study has shown that in a group of individuals being treated with SSRI's, although depressive symptomatology was significantly reduced, treatment did not restore a strong positive automatic self-evaluation to the same levels observed in a control group of non-depressed individuals. Further, levels of negative dysfunctional attitudes, beliefs of social isolation, and thoughts of being negatively judged by others were not resolved with the use of SSRI's. These findings may explain why antidepressant medication may not be as effective in reducing depressive relapse compared to cognitive therapy. Therefore, automatic self-evaluation as measured by tasks like the EAST and IAT may prove useful tools in further research investigating

vulnerability to depression. Further research is also needed to explore the relation between dysfunctional attitudes, beliefs of social isolation, beliefs of being negatively judged by others, and automatic self-evaluation. This study also found little evidence for a specific schema content hypothesis being associated with vulnerability to depression. Rather, hypothetical vulnerability was expressed through a weak positive automatic association of positive concepts and the self relative to negative depression related concepts. Lastly, this study provides some tentative evidence for the potential value of error rates as well as response latencies for tapping into implicit or automatic-processing biases associated with depression.

Chapter 10

General discussion

For in every action what is primarily intended by the doer, whether he acts from natural necessity or out of free will, it is the disclosure of his own image...nothing acts unless by acting it makes patent its latent self. — Dante

10.1. Introduction

Much of the research into schemata in depression has found little evidence for schematic activity in the absence of a low or depressed mood. This has led to a widespread view that schemata in depression are latent and only influence information processing in certain conditions, such as in the presence of negative affect (e.g., Gemar et al., 2001). The aim of this thesis was to investigate schematic activity using two relatively new information-processing paradigms, whilst at the same time taking into consideration certain methodological problems related to existing schema research discussed at length in Chapter 4. These methodological issues included the potential confounds of self-report measures (Nisbett & Wilson, 1977); failure to recognise error rates as a useful measure of information processing biases (e.g., Bargh & Tota, 1988; Kirsch & Lynn, 1999); an inadequate consideration of hypothetical specific schema content in depression (e.g., defectiveness; Alloy et al., 1999); the lack of an association between stimuli and the self in implicit tasks (e.g., Hedlund & Rude, 1995) and/or a disregard for the potentially important role of automatic self-evaluation in vulnerability to affective disorders, which some researchers are only now starting to realise (e.g., De Raedt et al., 2006; Tanner et al., in press; de Jong, 2000). Failure to fully recognise these issues in schema research may have hampered the detection of schematic activity in the absence of a depressed mood and mood priming initiatives.

This general discussion chapter starts with a summary of the main findings of the studies reported within this thesis. This focuses on the role of automatic self-evaluation, the role of errors versus reaction time, the specific schema content hypothesis, and the latent schema activation hypothesis. This is followed by a discussion of how these findings relate to contemporary cognitive models of depression and the schema concept (i.e., Beck, 1967, 1976) along with some explorative suggestions for a possible re-conceptualisation of schemata in depression. This chapter ends with a discussion of the limitations of the research reported within this thesis, suggestions for future research, and an overall conclusion.

10.2. Automatic self-evaluation

It was observed that automatic self-evaluation was a principal finding in the results from the series of studies within this thesis. The importance of implicit or automatic self-evaluation was proposed in Chapter 4 to be an important aspect of information processing in depression. It was argued, as shown from recent research findings, that self-esteem or self-evaluation at an automatic level might be an important factor if one is to evaluate schematic

functioning in depression (e.g., Alloy et al., 1999; Gemar et al., 2001; De Raedt et al., 2006). In recent years researchers investigating the self have come to query the role of consciousness in the self-evaluation process (J.D. Brown, 1993; Epstein & Morling, 1995; Greenwald & Banaji, 1995). Evidence suggests that many important social and cognitive processes operate without the need for conscious awareness and thus researchers have argued that self-evaluations may operate at non-conscious levels of awareness (Bargh & Chartrand, 1999, Greenwald & Banaji, 1995). Therefore, implicit self-evaluations may provide a more precise measurement of vulnerability to depression compared to self-report measures that focus on explicit self-judgments. This is because self-report measures may only be representative of the effects of a low or depressed mood (Miranda & Pearsons, 1988), and are susceptible to the effects of potentially confounding explicit processes (Nisbett & Wilson). Indeed it is argued that if one is to assess schematic functioning in depression and vulnerability to depression, one has to tap into implicit rather than explicit processes as the former are argued to be a central aspect of schematic functioning from the perspective of the cognitive model of depression (Beck, 1967, 1976).

In Study 1 on the EAST, as predicted, the high BDI group showed a weak positive automatic self-evaluative bias by a propensity to process negative words more efficiently (on reaction time only) when associated with the self, while the low BDI group was more efficient when positive words were paired with the self (errors only). The results from Study 1 also showed that the EAST task is sensitive in measuring information-processing biases, characteristic of individuals who suffer from significantly different levels of depression. The high BDI group in this study also scored significantly higher compared to the low BDI group on the majority of the self-report measures of depression. This shows that the use of implicit tasks measuring automatic self-evaluation may be informative in assessing schematic functioning in depression alongside explicit measures.

Although Study 2 was conducted to construct a better IAT design because of the poor results of the IAT in Study 1, this study showed that a strong positive automatic self-evaluative bias was evident in individuals who did not have a history of depression. This was shown by reaction time data and error rates from the IAT. However, as current depression or mood were not assessed and there was no “currently” depressed group to compare the supposedly non-depressed group with, it is difficult to make any firm inferences from the results of this study

In Study 3 on the two IAT tasks, individuals classified as low-trait depressed had a stronger positive self-evaluative bias (on errors only) compared to the high-trait depressed. The fact that the high-trait depressed group showed a weaker positive automatic self-evaluative bias on the IATs with and without adding depressive symptoms (BDI scores) to the

analysis as a covariate, showed that automatic self-evaluation may be measurable in the absence of a low mood and associated depressive symptomatology.

Study 4 also showed that the low-trait depressed had a stronger positive automatic self-evaluative bias compared to the high-trait depressed, and that these levels of automatic self-evaluation in both groups were unaffected by a negative mood induction. This was shown by reaction time data on the IAT tasks. More importantly, the weaker positive automatic self-evaluative bias in the high-trait depressed was evident even when controlling for self-reported low mood (VAS). One could infer from this study that automatic self-evaluation and hypothetical vulnerability to depression may be measurable in the absence of a depressed mood, and without the necessity of schematic activation argued to be crucial by Beck (1967, 1976) for information processing biases in depression to occur. Therefore, relying solely on explicit self-report measures, which are extremely sensitive to the effects of mood (Miranda & Pearsons, 1988), may be unreliable in measuring schematic functioning in the absence of a depressed mood. Interestingly, when BDI scores were controlled for, no differences in automatic self-evaluation were observed between the high and low trait depressed groups. This may indicate the lack of reliability of self-report measures (e.g., Nisbett & Wilson, 1977), as levels of automatic self-evaluation were not affected in Study 3 when controlling for BDI scores.

Study 5 showed that a control group of non-depressed individuals had an overall stronger positive automatic self-evaluative bias compared to a group of depressed individuals treated with SSRI antidepressants. This was shown by error rates on the EAST and IAT tasks. Study 5 also provided important supporting evidence to explain why antidepressant treatments may not be effective in preventing depressive relapse compared to cognitive therapy, as found by other researchers (e.g., Hensley et al., 2004; Paykel et al., 1999; Teasdale et al., 2002; Teasdale et al., 2000). This was indicated by the finding that a strong positive automatic self-evaluation was not engendered in successfully treated depressed individuals. Yet on the majority of self-report measures of depression at six months, the SSRI group scored no differently compared to the control group. This study again provides evidence that relying solely on self-report measures (i.e., ATQ, BDI) of depression to measure schematic functioning and enduring vulnerability to depression may not be a valid method. This study also shows that a weak positive automatic self-evaluation might be a more valid measure of enduring vulnerability to depression.

The results presented in this thesis confirm a growing amount of research that implicates automatic self-evaluation as an important construct involved in depressive and affective disorders (Tanner et al., *in press*; Alloy et al., 1999; de Jong, 2000). The results highlight the informative value of using implicit or automatic processing tasks as a promising way of measuring schematic functioning in depression without the problems inherent in the

use of self-report measures (Nisbett & Wilson, 1977). More importantly, these results show how schematic functioning and enduring vulnerability to depression may be measurable in the absence of a low or depressed mood.

Although differences in automatic self-evaluation were an important and consistent finding in the series of studies, there were nevertheless discrepancies in the findings that perhaps make the results less clear-cut. In some studies error rates were indicative of significant differences in automatic self-evaluation, whereas in other studies reaction times were indicative of significant differences in automatic self-evaluation. This issue will now be discussed in more detail.

10.3. Automatic-processing and issues of measurement: response latencies versus error rates

In Chapter 4, it was proposed that error rates on implicit tasks might be an important measure to consider when studying schemata in depression, and that this issue has to date been neglected in schema research. This oversight may have contributed to the lack of evidence of schematic activity in the absence of a low or depressed mood (e.g., De Raedt et al., 2006). Error rates proved to be a good indicator of differences in automatic self-evaluation in Studies 1, 2 (as well as reaction time), 3 and 5. However, in Study 4 a difference in self-evaluation between groups (high and low-trait depressed) was obtained only with reaction time. These inconsistent findings may challenge any firm inferences one can make regarding the validity of the results discussed in this thesis, and claims by some that error rates may be a better measure of automatic self-evaluation (i.e., Bargh & Tota, 1988). Bargh & Tota (1988) argue that response latencies may be inappropriate indices of efficient or automatic processing because multiple factors, besides the activation of stored constructs, may influence response speeds. These factors are self-presentational strategies within the experimental context (Ferguson et al., 1983), and the subject's degree of self-confidence in his or her decision-making, which affect reaction time. Latencies therefore may (in some contexts) reflect the contribution of both automatic/implicit and explicit forces that operate simultaneously but independently (Logan, 1979; Posner & Snyder, 1975; Shiffrin & Schneider, 1977). Therefore, reaction time results may not be the result of "truly" implicit processes. This is problematic as Beck (1967, 1976) argues that schematic functioning is an implicit affair, and thus explicit measurement may be tapping into other constructs not related to implicit schematic functioning (Bargh & Tota, 1988).

Contrary to Bargh & Tota (1988), Greenwald et al., (1998) and De Houwer (J. De Houwer, personal communication 14 of February 2005) argue that both reaction time data and error rates are both valid measures of implicit processes and self-evaluation. Townsend & Ashby (1983) take a stricter approach. They postulate that one must consider the speed/accuracy trade-off during implicit processing tasks. In other words, if the data from implicit tasks show a bias for favoring error or reaction time data as the significant measure,

this may be a valid indicator of implicit processing. However, if the data is very difficult to interpret, one should consider the speed/accuracy trade-off (the relative efficiency or performance on a task) to measure “true automaticity” and conduct efficiency index calculations.

The debate regarding what kind of data one should collect from implicit tasks is encumbered with difficulties and requires further research. However, it seems from the results of the series of studies in this thesis, that both reaction time and error rates were good indicators of automatic self-evaluation. It appears that automatic self-evaluation and the way one measures it (error rates and/or reaction time data) may be important to consider when attempting to measure schematic functioning in depression and hypothetical vulnerability to depression. Indeed, in past research investigating depression, error rates and consideration of a speed accuracy trade-off has been ignored in favour of reaction time (e.g., De Raedt et al., 2006; Gamar et al., 2001). This may have prevented important information from coming to light with regards schematic to functioning and vulnerability to depression. This brings us to another important and frequently neglected part of schema research. This involves the failure by many researchers (e.g., De Raedt et al., 2006; Gamar et al., 2001) to consider schema content when examining vulnerability to depression, and associated information processing biases. This complex issue will now be discussed in the next section below.

10.4. The specific schema content hypothesis

It has been argued that negative self-schemata in depression contain specific content, and that this content is related to specific themes such as abandonment and defectiveness (Alloy et al., 1999; Beck, 1987; Blatt, 1974; Bowlby, 1969, 1973, 1980; Claesson & Sohlberg, 2002; Pielage et al., 2000; Stopa et al., 2001). In other words, negative self-schemata contain negative themes and consequently any information processing bias (or self-evaluative bias) should be congruent with these themes. A failure to address this aspect of schemata may be another reason why there is little evidence of schematic functioning in the absence of a depressed or low mood.

To address this problem, the specific schema content hypothesis was tested in Studies 3 & 5. In Study 3, two groups of negative words were used. One group of negative words was related to schematic themes of abandonment and defectiveness whilst the other group of negative words was related to themes of low mood. There were no observed differences in automatic self-evaluation between the high and low trait depressed groups using these two types of negative words as stimuli. In Study 5, there were no differences observed in automatic self-evaluation between the two groups when comparing schema words (negative and positive schema words), and mood words (negative and positive mood words). Therefore, the results from the studies reported here do not support the theoretical suggestion or the results of other studies, which propose that individuals hypothetically vulnerable to

depression have negative self-schemas containing specific content relating to the etiology of schemata (e.g., Beck, 1967, 1976, 1987; Alloy et al., 1999).

However, it may well have been that the core maladaptive schema theme appropriate to test the specific schema content hypothesis was not correctly identified. In Study 5, the maladaptive schema of social isolation was a stable theme that emerged in the recovered the SSRI group as measured by the Young Schema Questionnaire (Young & Brown, 1994). The theme of social isolation was not a theme that was used as part of word lists used alongside the implicit tasks (although abandonment, which was included, is thematically related to social isolation in some ways). Furthermore, it is highly speculative that the schemata of all individuals who suffer from depression or who are vulnerable to depression may contain the same specific content. It could be that on an individual-to-individual basis, individuals may possess unique personalised schematic themes. If this were the case, it would make sense to assess what these individualised schematic themes were and integrate them into the design of the implicit task being used. Again, more research needs to be done in this area to ascertain the validity of a specific schema content hypothesis.

10.5. The latent schema model of depression

As well as the specific schema content hypothesis being addressed, another important aspect of the cognitive model of depression was also explored. This involved investigating automatic self-evaluation and the idea of the latent schema model, which has a hypothetical role in vulnerability to depression (Beck, 1967, 1976). It is a widely held assumption that negative self-schemata are activated by the effects of negative mood, and subsequently negatively bias information processing (e.g., Beck, 1967, 1976; Segal et. al, 1999; Hedlund & Rude, 1995; Gemar et al., 2001). In other words negative self-schemata lie dormant when an individual is in a normal mood and do not affect information processing.

In studies 3, 4, & 5, the latent negative self-schemata model of depression was tested. In Study 3, the high-trait depressed group showed evidence of a weaker positive automatic self-evaluative bias on all the IAT error analyses compared to the low-trait depressed. This was evident, even when controlling for depressive symptomatology (BDI). In Study 4, a stable and weaker positive automatic self-evaluative bias was observed in the high-trait depressed group on the IAT (reaction times only) compared to the low-trait depressed, despite undergoing a negative mood induction and when controlling for mood (VAS). In Study 5, a stable and weaker positive self-evaluative bias was observed (error analyses from IATs and EAST) in a group of depressed individuals compared to a non-depressed control group. This was evident despite the SSRI group being relatively symptom free after six months of SSRI antidepressant treatment. These results suggest that a low or depressed mood was not necessary for differences in automatic self-evaluation or information processing biases to occur between the different groups of individuals used in the respective studies. As a result,

these studies are not consistent with a latent schema model of depression (Beck, 1967, 1976; Gemar et al., 2001; Segal et al., 1999). However, they do confirm other findings where individuals vulnerable to developing depression and free from depressive symptomatology (and therefore schematic activation) still showed a negative implicit self-evaluative bias (Alloy et al., 1999).

10.6. The cognitive model of depression re-visited

The results from the studies in this thesis indicate that automatic or implicit self-evaluation may be an important aspect in differentiating individuals hypothetically vulnerable to depression (e.g., high-trait depressed and individuals successfully treated for clinical depression), compared to individuals who are not vulnerable to depression (e.g., low-trait depressed and non-depressed individuals). However, there was no evidence to support a latent schema model of depression and no evidence that schemata contain specific negative themes (i.e., abandonment and defectiveness). As such, the results are not in agreement with the cognitive model of depression and associated latent schema theory of depression (e.g., Beck, 1967, 1976; Gemar et al., 2001; Segal, 1988).

One reason why research may have found very little evidence of schematic activity may lie partly in the fact that researchers have typically conceptualised schemata from a classical cognitive framework. From this perspective (e.g., Rector et al., 1998; Segal 1988, Beck, 1967, 1976), a self-schema or self-structure is seen as a relatively stable, stored body of negative knowledge, negative self-information, or self-representation that interacts with incoming information by shaping selective attention, and expectancies. However, this structure only affects information processing when activated by a negative mood (Teasdale & Barnard, 1993). On the other hand, the results of some of the studies in this thesis suggest that negative mood may not be a necessary condition for schema activation, particularly when implicit measures are used. They also suggest that it might be useful to re-visit the way in which schemata have traditionally been conceptualised, and consider whether conceptualisation based on different philosophical premises may provide both a richer theoretical account and also a better fit with the available evidence reported here. To this end it might be useful to borrow Segal's (1988) cognitive self-representationalist framework for understanding schemata. Segal (1988) outlined a useful format for different ways to think about schemata or cognitive self-representation in depression via theories of 1) schema availability, 2) schema accessibility, and 3) the negative self-schema model. Each of Segal's theoretical schema models will be discussed in turn in the following section focusing specifically on how they relate to the findings in this thesis.

The Availability model supposes that depressed people have an increased number of stored negative personal constructs (e.g., due to negative childhood experiences). In an episode of depression the individual will have increased cognitive content that is negative

concerning the self and associated negative implicit information processing biases. When the depression has lifted the cognitive content has been altered, is no longer as negative, is replaced with more positive content and negative implicit information processing is no longer present. However, this does not yield much insight into vulnerability to depression in the absence of a low mood. Firstly, if in a non-depressed state, the self, cognitive content, or self-schemata are constructed more or less in positive terms, measurement of vulnerability will only be visible in a depressed or low mood. This implies that negative environmental events (those that induce a low mood) “activate” a predisposition for the person to reconstruct “the self” in more negative terms.

The key problem for the availability model is how it fits with Beck’s cognitive model of depression (Beck, 1967, 1976). From the availability perspective, two questions are raised. Firstly, does the depressed mood reconstruct a dormant self-structure, which has negative content, which then biases information processing in a negative way, producing the cognitive aspects characteristic of a depressed episode? Or, secondly, do negative schemata (or negative personal constructs) bias information processing, which then produce the cognitive content of a depressed episode? It is not clear from the majority of research (Ingram et al., 1998) if the schema is first and foremost responsible for biasing information processing to produce low mood and negative personal constructs, or if the low mood and conscious cognition containing negative self-constructs comes first to activate latent schemata. This issue is not clear in Beck’s cognitive model of depression (Beck, 1967, 1976). Furthermore, the availability model cannot tackle notions of vulnerability to depression in the absence of a depressed mood. From the availability perspective, vulnerability to depression can only be conceptualised as having characteristics that are state dependent, which to some extent is a circular argument in etiological terms. It is difficult to ascertain whether the content of cognition about the self has been altered (to be more positive) in the absence of a depressed mood or if the content/structure has been de-activated.

So how does the availability model relate to the results from the five studies within this thesis? It could be argued that in Study 1 the depressed group did have available more stored personal negative constructs. The high BDI group made fewer errors when self was associated with negative words on the EAST compared to the non-depressed group (low BDI group). The high BDI group also responded in a more negative way on the self-report measures of depression and cognition. However, the availability model posits that in the absence of a low or depressed mood the content of cognitions about the self are altered, becoming more positive. Therefore, the results from Study 1 cannot provide any evidence to test this aspect of the model, as the participants were only tested once during the presence of increased depressive symptomatology. However, from the results of Studies 3 (low & high trait depressed), 4 (high & low trait depressed with a negative mood induction), and 5

(depressed & non-depressed control group), there was evidence of discrepancies in the availability of positive self-constructs in the absence of a depressed mood between the respective groups. In Study 3, the high-trait depressed had a weaker positive automatic self-evaluative bias (compared to the low-trait depressed) even when controlling for depressive symptoms. In Study 4, this again was the case even when controlling for concurrent low mood. In Study 5, the depressed group (SSRI group), at all 3 time points (baseline, 3 months, and at 6 months where depressive symptoms had been significantly resolved) showed a weaker positive automatic self-evaluative bias compared to a non-depressed control group. These results, partly in agreement with the availability model, showed that availability to negative self-constructs in the absence of a depressed or low mood was not evident. However, in disagreement with the availability model, the results showed that the high-trait depressed (Studies 3 & 4) and recovered depressed (Study 5) still had a reduced availability to positive self-constructs as shown by a weak positive automatic self-evaluative bias. Therefore, these findings are for the most part contrary, to the availability model's feature of the self becoming positive in the absence of a low or depressed mood, at least in relation to automatic self-evaluation. It must be noted however, that the SSRI group at six months did still show some evidence of residual depressive symptomatology (although the levels of depressive symptomatology in the SSRI group was not significantly different from the control group's levels of symptoms). They also reported significantly more negative feelings of being socially isolated and negative thoughts regarding how they thought other people judged them.

The Accessibility model of schematic functioning was also not fully supported by the findings in this thesis either. The accessibility model proposes that depressed people and non-depressed people have differences in the accessibility of personal constructs. In other words, both non-depressed and depressed individuals have equal numbers of negative and positive personal constructs. However, for those who are vulnerable to depression, a low or depressed mood increases and maintains the accessibility of negative personal constructs. This in turn affects information processing, which becomes more negatively biased and subsequently produces the negative cognitive triad that is characteristic of depression. According to the accessibility model, the depressed person in remission no longer has increased access to negative self-constructs. Further, in remission, negatively biased information processing ceases to occur, and thus there is no evidence of the negative cognitive triad. From the perspective of the accessibility model, like the availability model, any evidence of vulnerability is elusive when there is no depressed mood present.

The results of these studies did not show any evidence of increased accessibility of negative self-constructs, at least at the implicit level, although the theory did have support at the explicit level. Study 1 partly supported the accessibility model. The high BDI group, compared to the low BDI group, had higher levels of depressive symptomatology, more

negative scores on other self-report measures of depression, and a weaker positive self-evaluative bias on the EAST. Therefore, the high BDI group appeared to have accessibility to self-constructs that were more negative in nature. However, like the Availability model and its relation to Study 1, there are limitations to what inferences one can make from this study. It was not possible to prove that the low mood of the high BDI group was maintaining or increasing accessibility to negative self-constructs at an explicit or implicit level, and that in the absence of a dysphoric mood, accessibility to negative self-constructs would no longer be dominant. These aspects of the accessibility theory were addressed by Studies 3, 4, and 5. The high-trait depressed group in Study 3 still had a weaker positive automatic self-evaluative bias compared to the low-trait depressed group, even when controlling for depressive symptomatology. In Study 4, despite undergoing a negative mood induction, the high-trait depressed group's automatic level of self-evaluation was not affected. Even when controlling for elevated levels of low mood, the high-trait depressed group still exhibited a weaker positive automatic self-evaluation compared to the low-trait group. In Study 5, a weaker positive automatic self-evaluation was observed in the depressed group (SSRI group) at the beginning of treatment when depressive symptoms and mood were significantly higher compared to the non-depressed control group. However, this weak positive automatic self-evaluation in the SSRI group remained stable, despite significant reductions in depressive symptomatology over six months of receiving SSRI treatment, to levels not significantly different to the control group. Again it must be noted, the SSRI group in Study 5 still did have elevated scores on some self-report measures of depression. Therefore, it may be possible that residual levels of depression had not been resolved and were responsible for the weaker positive self-evaluative bias in the SSRI group.

Overall however, at an implicit level, the findings in this thesis, contrary to the accessibility model, showed that a depressed mood did not increase and maintain the accessibility of negative self-constructs and in the absence of a depressed mood, accessibility to negative self-constructs remained in an inverse way. This was shown by an automatic self-evaluation lacking in positivity.

From the perspective of both the availability and accessibility models, Segal (1988) states that there appears to be a mood congruity relationship between affect and schema accessibility/availability. In other words, the information that matches the individual's mood state is more accessible or available (at an implicit level), and more easily recalled or reported during the depressed mood state. Segal argues that the availability and accessibility models of schematic functioning describe correlations between mood and cognitive constructs. However, he posits that these correlations do not fully explain whether these cognitive constructs develop from an organised self-schema or are merely representations from other cognitive systems (e.g., memory). Segal also argues that to demonstrate the existence of a

negative self-schema one must go beyond merely looking at the content of information stored in a such a structure to determine to what degree the content being attended to, or reported, is related to a self-structure. Put simply, if one is to measure negative personal constructs or the availability and accessibility of such constructs, one must specifically measure the relation of the self to these negative constructs. Simply measuring a person's reaction to a negative word or recording an explicit self-description from an individual, might not be representative of, or strongly associated with, the negative self-schema or structure per se. Such a measure might only be representative of a person's negative memory or the person's familiarity with a concept facilitated by the congruency of a dysphoric mood (De Houwer, 2002). In other words, the EAST and IAT as used in this study, by evaluating the implicit or automatic association of the self with concepts, may hypothetically measure a specific "self-structure", rather than a memory construct associated with the self. This meets Segal's requirements for an adequate test of a schema hypothesis as described in his negative self-schema model.

The importance of the self is incorporated into Segal's negative self-schema model. In this model he argues that there are differences in the interconnectedness of personal constructs between depressed and non-depressed people. In the depressive episode a specific "self structure" is activated, which negatively biases information processing, and produces the output that is characteristic of the negative cognitive triad. In the absence of a negative mood, information processing is no longer negatively biased, but the interrelations between the negative self-schema structure remains. If content alone was important, as is the case in the availability and accessibility models of schemata, a model relying on a cognitive structure approach would not necessarily be superior to a mood congruent availability/accessibility account. This is because, once primed, the same pattern of construct activation would be expected to occur. However, if content plus structure are crucial, then a different pattern of findings would be predicted. Due to the interconnection between individual elements in the negative cognitive schematic structure, schemata could hypothetically exert an influence and be active in the absence of a depressed mood. This means that an individual's negative self-schema could persist beyond the depressive episode and well into recovery. Furthermore, a person vulnerable to develop depression may possess negative self-schemata that exist in some mode, but due to the lack of pragmatic research methodology (see 5.4), evidence of schematic activity is not convincing in the absence of a low or depressed mood (Gemar et al., 2001; Rude et al, 2001; Hedlund & Rude, 1995).

The results from the studies in this thesis are partly in line with Segal's negative self-schema model. Differences in the interconnectedness of personal constructs was shown in Studies 1, 3, 4 and 5 where the high BDI, high-trait depressed and SSRI groups scored higher not only on self-report measures of depression, but also showed a weaker positive automatic self-evaluative bias. However, Studies 3, 4, and 5 also showed that a low or depressed mood

was not necessary for the activation of a more negative self-structure contrary to the theory. In Study 3, the high-trait group still displayed a weaker positive self-evaluative bias compared to the low-trait group when controlling for depressive symptomatology. In Study 4, a negative mood induction was not necessary to activate a latent negative self-structure, as the high-trait group's level of automatic self-evaluation was not affected by it. In Study 5, even though the SSRI group's depressive symptoms had resolved in comparison to the non-depressed control group, they still exhibited a weaker positive automatic self-evaluative bias. Thus in accordance with Segal's (1988) claim, the results in this thesis seem to show that negative self-schemata may still be intact in the absence of a low or depressed mood and "reactive" as measured by the IAT & EAST. However, this issue needs to be clarified. For the most part, the results showed that self-schemata that were intact were not so much "negative", but rather self-schemata lacking in positivity.

In light of the obtained results reported in this thesis, and taking into account the methodological considerations highlighted as problems preventing the measurement of schematic functioning in the absence of a low or depressed mood (e.g., the implicit or automatic association of stimuli with the self), it seems logical to tentatively explore a re-conceptualisation of depressive schemata. The next section will be a discussion of some new suggestions to re-conceptualise and think about schemata and depression in light of the results presented in this thesis. However, section 10.8 will address the limitations of these suggestions as further research is needed to come to firmer conclusions.

10.7. Preliminary suggestions for the re-conceptualisation of schemata in depression in light of the findings in this thesis

As discussed in Chapter 4, one important aspect often overlooked in depression research is the consideration of the implicit measurement of the self with the stimuli. With the use of two new tasks, the IAT and EAST, which implicitly measure the association of the self with negative concepts, it has been shown from the results in this thesis, that differences in self-structure or self-evaluation are evident in the absence of a low or depressed mood in individuals hypothetically vulnerable to depression (i.e., in the high-trait depressed and the recovered depressed). This is troubling, considering Segal's (1988) and Beck' (1967, 1976) assertions that negative self-schemata structures are latent and remain potentially reactive. However, it may well be that inherent differences in implicit judgments regarding the self and vulnerability to depression are measurable without the presence of a low mood to activate other kinds of information processing biases (e.g., memory bias). Perhaps the only reactivity needed when one is measuring self-structure is to present stimuli that are related in some way to the self. This alone may be enough to "activate" some kind of bias or existing self-structure.

Indeed, Stapel & Blanton (2004) and Gilbert, Giesler, & Morris (1995) concur with this argument. These authors have found in their research that mere subliminal exposure to certain information that is related to a person's self-evaluation is enough to activate a negative or positive self-evaluative bias. More importantly, Stapel & Blanton (2004) found that levels of mood did not affect levels of implicit self-evaluation, and also that being exposed to an implicit self-evaluative task (the subliminal information related to the self) did not result in mood being affected in any way. In effect, their study shows that implicit self-evaluation is a stable construct despite fluctuations in mood. Therefore, implicit self-evaluation may be an extremely sensitive measure that can be "activated" without the effects of mood being required. Therefore, the tasks used in this thesis (IAT & EAST) may be more sensitive than other tasks as used in depression research (e.g., memory/recall tasks; Hedlund & Rude, 1995). Such tasks that measure recall of depression words may only be a mood congruent phenomenon, tapping into memories not related to a self-structure per se, and thus not representative of a specific self-structure implicated to self-evaluation and vulnerability to depression. If other non-self-evaluative tasks had been used alongside the IAT and EAST as part of this thesis, information-processing differences may not have been observed in the conditions where a low or depressed mood was absent (i.e., Studies, 3, 4, & 5). These ideas are obviously speculative, but future research could compare implicit self-evaluative tasks like the EAST and IAT to implicit tasks that measure information processing simply towards negative and positive stimuli that are not linked to the self.

However, there are other potential problems with a schema activation hypothesis. If one assumes, as Segal (1988) argues, that individuals who are not vulnerable to depression possess a greater interconnectedness of positive personal constructs within their self-schema structure, does this mean that it takes a positive reaction (reactivity of a self-structure to a positive mood) to activate their positive self-structure? This does not seem to be the case. Individuals who are not vulnerable to develop depression or who are regarded as mentally healthy do not seem to develop a positive information bias as a result of an induced positive mood (See Ingram et al., 1998 for a review). Indeed as Stapel & Blanton (2004) demonstrated, automatic self-evaluation in such individuals seems to be a stable construct and is not affected by changes in mood. This is also supported from the results of other research that suggest that implicit self-esteem is very more resilient compared to explicit self-esteem, which is more sensitive to the effects of mood (Pelham & Hetts, 1999). Therefore, it is reasonable to assume that if implicit self-evaluation is in general a stable construct and resilient to "reactivity", it might be the lack of a strong positive automatic self-evaluative bias, rather than an "activated negative-self-structure" that is important in understanding vulnerability to depression.

There is some support for this argument in Studies 3, 4, and 5. The low-trait depressed (Studies 3 & 4) and the non-depressed control group (Study 5) showed greater relative differences in automatic self-evaluation compared to the low-trait depressed and non-depressed controls respectively. These groups of individuals had a very strong positive automatic self-evaluative bias. In other words, the high-trait depressed in Studies 3 and 4 and the depressed SSRI group in Study 5 did not show a strong bias towards either a positive or negative automatic self-evaluative bias (relative to the low trait and non-depressed control groups). Therefore, the lack of a positive self-evaluative bias may be what is relevant to measuring enduring vulnerability to depression. This inference however has to be treated with caution. In Study 1 the high BDI group did on one occasion (on the EAST reaction time analysis) show a preferential negative bias when self was associated with negative words, compared to the low BDI group. Nevertheless, the results of research studies that have used the IAT support the argument that a stronger positive automatic self-evaluative bias in individuals who are not vulnerable to affective disorders is what distinguishes them from individuals who are vulnerable to affective disorders (de Jong, 2000). The results of a study by Southall & Roberts (2002) partly concur with this idea. They found that the existence of low self-esteem coupled with high life-stress resulted in adolescents developing depression. Those with a more positive self-esteem reacted to life stress in a more adaptive way and did not spiral into depression. This study provides some evidence that a stable and strong positive self-evaluation might be implicated as a protective factor in the development of depression. In other words, a strong positive self-esteem may help individuals deal with environmental challenges in a more adaptive way, which consequently protects from low and depressed moods developing. However, in this study self-esteem was measured explicitly via self-report and thus high scores on this measure may have been the result of mild to moderate depressive symptomatology pre-existing before the development of a full blown episode of depression. Further, other research has typically failed to find a difference between the explicit self-esteem ratings of persons who are at risk to develop depression (see Luxton & Wenzlaff, 2005 for a review). However, these two studies again may highlight the lack of reliability of using self-report measures (Nisbett & Wilson, 1977).

Overall, the results of this thesis are not in accordance with a major idea inherent in theories of schematic functioning and depression, that is the idea of latent self-schema structure as described by Segal (1988) and Beck (1967, 1976). The results are also not in accordance with the notion of thinking about schemata in terms of containing specific content (e.g., Alloy et al., 1999; Beck, 1967, 1976, 1987). As shown in this thesis, there was no evidence for specific schema content being implicated in automatic self-evaluation. Instead, the results of the studies conducted in this thesis suggest that what may differentiate individuals who are vulnerable to depression from individuals who are not is a reduced

positive automatic self-evaluative bias. However, there is a greater prevalence of research that has investigated the role of negative events or themes hypothetically related to early childhood, and themes that are implicated as being specific content within schemata (e.g., Alloy et al., 1999; Bowlby, 1969, 1973, 1980; Claesson & Sohlberg, 2002; Pielage et al., 2000). This preponderance of investigation of the negative content/themes in schemata in depressed individuals, may have neglected the investigation of the positive content/themes in schemata in individuals who are not vulnerable to develop depression. Therefore, looking for specific negative content within schemata in vulnerable individuals may be an overly constrained methodology. Perhaps it may prove fruitful to investigate the ratio of hypothetical positive and negative content of schemata in individuals who are and are not hypothetically vulnerable to develop depression. There may be very specific events and themes that typify the schemata of individuals who are not vulnerable to develop depression and the ration of such themes may be important. One such idea is that a certainty of one's beliefs or the security of one's ontology is what differentiates those who are vulnerable to develop depression and those who are not (Luxton & Wenzlaff (2005).

This last point brings us to an alternative way of thinking about schemata and vulnerability to depression, which is from the ontological and neuroscientific perspectives rather than from more traditional epistemological perspectives. Obviously an in-depth discussion of these perspectives is beyond the remit of this thesis, but a re-conceptualisation of schemata from different perspectives might provide a way to develop new ways of thinking about schemata and depression. Essentially, an ontological approach to depression does not deny that human beings have mental states in which their minds are directed towards objects or stimuli. However, the world for the ontologically insecure individual is not so much a belief system (unconscious self-representation) or a range of stimuli impinging upon the schema system and consequently activating it, but is instead an atheoretical, intuitive, and embodied way of perception and of dealing with the world. Therefore, the way one deals with the world (implicitly) is nonlinguistic, non-conceptual, and does not involve structures or specific cognitive self-representations that have to be activated (Wheeler, 2006; Heidegger, 2001; Merleau-Ponty, 1962; Dreyfus, 1989). This idea that a negative/insecure being, essence, or ontology exists in individuals vulnerable to depression fits well with the suggestion, as discussed above, that a lack of a strong positive automatic self-evaluation may be implicated in vulnerability to affective disorders (de Jong, 2000). By placing importance on the word "automatic", the process of human "being" (Heidegger, 2001) occurs without an individual considering the underlying reasons or explicit ideas he/she holds, which results in his/her orientation in the world.

Neuroscientific research, like the ontological perspective, counters the notion of a "structure" containing cognitive self-representations that biases incoming information

processing. Walter Freeman (2001), a founding figure in neuroscience, was one of the first scientists to take seriously the idea of the brain as a nonlinear dynamic system that could operate (towards a goal) without the brain in any way representing that goal in advance of achieving it. This idea is very much like Greenfield's (1998) theory of emergent consciousness; stimuli impinge upon the individual from an epicenter, spread out over non-specialised neurons having effects over the whole brain, and the strength of neuronal activity determines the degree of consciousness of impinging stimuli. In effect there is no center of consciousness (or center of unconsciousness) that holds categorisations or representations of the world or self. From the ontological and neuroscientific approaches, the construction of the "self" occurs at a conscious level of language, but the self is not situated in a neural network at an unconscious level. When conscious self-construction occurs, then one begins to ascertain the nature of one's ontology that has not up to that point ever been brought into awareness or been attended to in any great detail. Indeed, De Houwer (2002) argues that implicit tasks like the IAT do not measure unconscious beliefs or self-representation per se, but one can infer from implicit self-evaluative judgments how a person might view him/herself at a conscious level. Thus, the implicit judgment of the self is only a rough measure of possible conscious beliefs, but does reveal the relative positive or negativity that person holds at an ontological level.

These suggestions for a re-conceptualisation of schemata are only intended to stimulate different ways of thinking of schemata and vulnerability to depression. These results concur with previous research (Alloy et al., 1999) but are discordant with more traditional schema models of depression (Beck, 1967, 1976; Segal, 1988). However, several methodological issues must be considered with regard the findings reported in this thesis which may have a bearing on any inferences that can be firmly stated. These will now be discussed along with some suggestions for further research which can hopefully generate in the future a clearer picture of schemata in depression and the role of automatic self-evaluation.

10.8. Methodological considerations and future directions

By using two relatively new implicit tasks (the IAT & EAST), it was found that automatic self-evaluation was significantly weaker in individuals who were hypothetically vulnerable to develop depression compared to non-vulnerable individuals. This difference between vulnerable and non-vulnerable individuals seemed to be evidently measurable in the absence of a low or depressed mood. However, it would be informative and would further clarify the role of automatic self-evaluation in vulnerability to depression by carrying out similar research using these two tasks (IAT & EAST) alongside other traditional implicit tasks used in depression (e.g., memory recall of depressed and positive words; Hedlund & Rude, 1995). One could investigate whether automatic self-evaluation is a stable feature of

vulnerability to depression using the IAT and EAST and compare this with a range of other traditional measures. One would then be able to look at the effects of negative mood on performance on the IAT and EAST to see how they compare to these other non-self-evaluative implicit tasks. One would be able then to discern directly if automatic self-evaluation as measured by the IAT and EAST was a more reliable and subtle marker of vulnerability to depression compared to these other non-self-evaluative implicit tasks.

It could be argued that the way in which the specific schema content hypothesis (with the use of themes related to abandonment & defectiveness) was investigated, as part of this thesis was inaccurate. For example, it is highly speculative that the schemata of all individuals who suffer from or who are vulnerable to develop depression may contain the same content. A more reliable methodology might be to assess each individual's core maladaptive schema themes (e.g., with the YSQ; Young & Brown, 1994), and incorporate these individual themes into information processing tasks. One could also go a little further. In-depth qualitative interviews could be used to draw out individual key themes or schemata unique to each individual, which subsequently could be incorporated into implicit processing tasks. Adopting these methodological procedures may shed light on the specific schema content that theoretically may influence information processing in depression.

It has been argued in this thesis that a weak positive automatic self-evaluation may be a vulnerability factor for depression. However, it could also be argued that a weak positive automatic self-evaluation may only be a vulnerability factor related to affective disorders in general, or even a vulnerability factor related to the development of other psychiatric disorders, but not specifically to depression (de Jong, 2000). Thus, in order to disentangle the role of automatic self-evaluation and vulnerability to depression, a more ambitious aim would be to use longitudinal research. For example, following different groups of "at risk" individuals (e.g., anxiety, depression, schizophrenia) over many months or even years, and assessing automatic self-evaluation. One might then be able to ascertain if a weak positive automatic self-evaluation was a stable and unique factor for vulnerability to develop depression, or was only an index of vulnerability to mental disorder in general.

Study 5 provided some interesting evidence as to why antidepressant treatment (SSRI's) may not provide sufficient protection from a depressive relapse: a stable weak positive automatic self-evaluative bias was not restored as a result of treatment in the SSRI group. However, as discussed, all the participants in Study 5 were still actively engaged in SSRI treatment at the close of the study. It would have been preferable to follow-up depressed individuals before, during and after treatment. Further, one could compare currently depressed individuals (pre-treatment), depressed individuals in treatment (e.g., SSRI's), and those who have recovered from depression, with other groups of individuals hypothetically vulnerable to depression (e.g., high-trait depressed). Using this kind of design, one may be

able to draw out stronger assumptions on the role of automatic self-evaluation and the role it plays in clinically depressed individuals, and individuals hypothetically at risk to develop depression.

It is the fashion of experimental psychology in depression to use laboratory tasks to measure cognition, attention and perception. However, it may be appropriate to use other more naturalistic or observationalist methodologies alongside traditional laboratory methodologies. If automatic self-evaluation is a factor in vulnerability to depression, then it may be helpful to assess how this emerges in an individual's natural setting. Although the series of studies within this thesis indicate that automatic self-evaluation could be an important factor in depression and hypothetical vulnerability to depression, it is possible that such tasks induce individuals to "perform" in a certain way (e.g., like a depressed person). Therefore, such conditions may not be an ecologically sound method of assessing self-evaluation as such artificial conditions may induce what Merleau-Ponty (1962) calls the *experimenter's error*. This concept essentially describes the process where the artificial situation of the laboratory makes people act in the way that they think is expected of them. Thus a depressed person may act in a depressed way, in much the same way as explicit controlled processes affect self-report measures (Nisbett & Wilson, 1977; Merleau-Ponty, 1962). Therefore, it may be more informative to investigate the way individuals act and behave in naturalistic situations using tasks that measure automatic self-evaluation. Through this kind of methodology one may be able to ascertain behavioural correlates of vulnerability to depression and clinical depression related to self-evaluation, while at the same time taking into consideration the potential confounding influence of laboratory experimentation.

A further issue pertaining to the results of this thesis that limit any firm inferences could be attributed to a central executive account of depression. Depression has been argued to be associated with a central executive dysfunction (Channon & Green, 2006). It has been found that depressed individuals have difficulty in allocating processing resources leading to poorer performance on information processing tasks compared to non-depressed individuals (Channon & Green, 2006; Elliot et al., 1997; Watts et al., 1998; Hertel & Hardin, 1990; Elliot et al., 1996). As observed in Studies 3, 4, and 5, the high-trait groups and SSRI group in comparison to the low-trait groups and control group respectively showed no bias in either direction to negative or positive words when associated with the self or other on either the EAST or IAT respectively. Only in Study 1 did the high BDI group show the characteristic bias to negative words on the EAST as seen in previous research (e.g., Hedlund & Rude, 1995). This overwhelming lack of an information processing bias observed in these groups (high-trait and SSRI groups) in the respective studies therefore may not be attributable due to a lack of a positive automatic self-evaluative bias, but instead to dysfunctional executive processes. However, it has been argued that performance deficits associated with central

executive dysfunction by depressed people only occur on tasks that involve controlled processes but not on tasks measuring automatic processes (Ellis & Ashbrook, 1998; Hasher & Zacks, 1979; Hertel, 1994; Teasdale & Barnard, 1993). Therefore, on such tasks like the IAT or EAST, a criticism of executive functioning affecting performance may not be applicable as these tasks are supposed to measure automatic processes (Greenwald et al., 1998). However, De Houwer (2006) argues that controlled processes may at times affect performance on tasks like the IAT and EAST tasks. Thus the IAT or EAST could measure controlled processes, automatic processes, or a combination of both. This issue of how executive function and automatic self-evaluation are related and how executive function in depression affects performance on tasks like the IAT and EAST needs to be addressed by further research before firmer conclusions can be made. Perhaps investigating how generalised executive function affects performance on the IAT and EAST would be a productive avenue for future research.

Another potential criticism of Studies 3 and 4 could be attributed to the arbitrary cut-off scores for the selection of the high and low trait depressed groups. A median split on the trait depression measure (DPRS; Zemore et al., 1990) was used to select the high and low trait depressed groups. Therefore, “false” differences in trait depression between the two kinds of trait groups may have been sampled. The differences in depressive traits between the high and low trait depression groups may not have been large enough for a “true” difference in trait depression to emerge. By sampling more individuals on the upper and lower quartiles on the DPRS may have been a more accurate representation of low and high trait depression. The arbitrary DPRS cut-off may have been the reason for the lack of a distinct information processing bias in the high-trait groups compared to the low-trait groups. Thus, if high and low trait depression groups had been chosen on the basis of scores from the upper and lower quartiles of the DPRS it may have been possible to observe more differences in automatic self-evaluation (e.g., a stronger negative automatic self-evaluative bias in the high-trait groups). However, it must be noted that statistically significant differences between the two trait groups on depressive symptomatology was observed and the information processing differences between the two groups was evident with and without adding BDI score to the analyses. Nevertheless, sampling more extreme high trait depressed and low-trait depressed individuals may be an issue to consider for further research investigating automatic self-evaluation and schemata.

10.9. Conclusion

The schema concept in depression is fraught with difficulties, as there is little evidence for schematic activity in the absence of a depressed mood (Segal, 1988). This may in part be due to traditional cognitive conceptualisations of schemata in depression (i.e., as a potentially activated structure containing specific content) and the methodological difficulties inherent in implicit tasks traditionally used to measure them.

In this thesis, the use of two relatively new tasks (the IAT and EAST) showed potentially important differences in automatic self-evaluation between analogue depressed individuals, high-trait depressed, and those who had recovered from clinical depression (a weaker positive self-evaluative bias) compared to non-depressed controls and low-trait depressed individuals. More importantly, these observed differences in automatic self-evaluation were not affected by differences in mood or levels of depression. Thus, these results provide important and original evidence that schematic activity and associated hypothetical vulnerability to depression can be measured in the absence of a low or depressed mood. These results also support the growing appreciation for the role of automatic self-evaluation being implicated in the vulnerability to develop affective disorders and in resilient mental health (Greenwald et al., 1998; de Jong, 2000, Tanner et al., in press). Importantly, this thesis provides further evidence as to why SSRI antidepressant treatment alone may not be effective in preventing relapse in depression (Hensley et al., 2004), in that SSRI antidepressants do not engender a strong positive automatic self-evaluative bias.

These results are not in accordance with the cognitive model of depression (Beck, 1967, 1976). There was no evidence to support a specific schema content hypothesis (Alloy et al., 1999) or the latent schema model intrinsic to the cognitive model of depression (Beck, 1967, 1976). Tentative suggestions have been made to review contemporary cognitive conceptualisations of schemata and the term ‘schematic activation’ as coined by Segal (1988) in his theoretical self-schema model and Beck’s (1967, 1976) cognitive model of depression. It may be that the association between certain concepts and the self as measured by the IAT or EAST is sufficient to activate or tap into certain implicit self-structures. It may also prove useful to focus research upon the nature of positive automatic self-evaluation in healthy individuals who are hypothetically not vulnerable to develop depression. Research has shown that healthy individuals have a stable and strong positive self-evaluative bias that does not have to be activated by a positive mood for positively biased self-evaluation to occur (Stapel & Blanton, 2004; Greenwald et al., 1998). It may also prove productive to investigate the positive content of the schematic structures in healthy individuals who are not deemed vulnerable to depression, as opposed to concentrating upon hypothetical negative content in vulnerable individuals. It may also be fruitful to investigate the ratio of different kinds of positive and negative schematic content in individuals who are, and those who are not, vulnerable to depression. Theoretical perspectives from ontological philosophy and its ideas on psychopathology and perspectives from the field of neuroscience are just two other alternative suggestions that may provide a new framework and way forward to re-conceptualise schemata and vulnerability to depression (Wheeler, 2006; Heidegger, 2001; Merleau-Ponty, 1962; Dreyfus, 1989; Greenfield, 1998; Freeman, 2001).

However, there are still outstanding methodological issues to consider if one is to make firmer conclusions that automatic self-evaluation is a valid heuristic associated with vulnerability to depression. More research needs to be done to investigate the nature of automatic self-evaluation using the IAT and EAST alongside traditional or more established measures of implicit cognition. The specific schema content hypothesis may need to be more thoroughly investigated by assessing the core schematic themes of individuals (on an individual basis) and the obtained themes subsequently being used in experimental research. More longitudinal research is needed with samples of different classes of depressed and “at risk” individuals to ascertain the nature and role of automatic self-evaluation and how it is related to vulnerability to depression and other psychiatric disturbances. More ecologically valid or naturalistic studies of automatic self-evaluation and associated vulnerability to depression are needed in order to extricate the possible confounding variables of laboratory experimentation. There needs to be further research in order to ascertain the role of executive function and how this affects automatic self-evaluation on tasks like the IAT and EAST. Lastly, there must be more rigorous sampling methods of individuals who are presumed to be vulnerable to develop depression to establish a clearer role of automatic self-evaluation in vulnerable individuals.

References

- Abramson, L.Y., Garber, J., Edwards, N.B., & Seligman, M.E.P. (1978). Expectancy changes in depression and schizophrenia. *Journal of Abnormal Psychology*, 87, 102-109.
- Abramson, L.Y., Metalsky, G.I., & Alloy, L.B. (1989). Hopelessness depression: a theory-based subtype of depression. *Psychological Review*, 96, 358-372.
- Abramson, L.Y., Seligman, M.E.P., Teasdale, J.D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology*, 87, 49-74.
- Akiskal, H.S., Judd, L.J., Gillin, C., & Lemmi, H. (1997). Subthreshold depressions: clinical and polysomnographic validation of dysthymic, residual and masked forms. *Journal of Affective Disorders*, 45, 53-63.
- Alba, J.W., & Hasher, L. (1983). Is memory schematic? *Psychological Bulletin*, 93, 207-231.
- Alloy, L.B., & Abramson, L.Y. (1979). Judgment of contingency in depressed and non-depressed students: Sadder but wiser? *Journal of Experimental Psychology: General*, 108, 441-485.
- Alloy, L.B., Abramson, L.Y., Whitehouse, W.G., Hogan, M.E., Tashman, N.A., Steinberg, D.L., Rose, D.T., & Donovan, P. (1999). Depressogenic cognitive styles: Predictive validity, information processing and personality characteristics, and developmental origins. *Behaviour Research and Therapy*, 37, 503-531.
- Andrews, G., Neilson, M., Hunt, C., & Stewart, G. (1990). Diagnosis, personality and the long-term outcome of depression. *British Journal of Psychiatry*, 157, 13-18.
- Angst, J. (1986). The course of affective disorders. *Psychopathology*, 19 (suppl), 46-47.
- Antonuccio, D. O., Danton, W. G., & DeNelsky, G. (1995). Psychotherapy vs. medication for depression: Challenging the conventional wisdom with data. *Professional Psychology: Research & Practice*, 26, 574-585.
- Arib, M.A., Conklin, E.J., & Hill, J.C. (1987). *From schema theory to language*. Oxford: Oxford University press.
- Barber, J.P., & DeRubis, R.J. (1989). On second thought: where the action is in cognitive therapy. *Cognitive Therapy and Research*, 13, 441-457.
- Bargh, J.A. (1984). Automatic and cognitive processing of social information. In R.S. Weyer & T.K. Srull (Eds.), *Handbook of social cognition (Vol.3)* (pp.1-43). Hillsdale, NJ: Erlbaum.
- Bargh, J. A., & Chartrand, T. L. (1999). The unbearable automaticity of being. *American Psychologist*, 54(7), 462-479.

- Bargh, J.A., & Tota, M.E., (1988). Context-dependent automatic processing in depression: Accessibility of negative constructs with regard to self but not others. *Journal of Personality and Social Psychology*, 54, 925-939.
- Barnett, P.A., & Gotlib, I.H. (1988). Psychosocial functioning in depression: Distinguishing among antecedents, concomitants, and consequences. *Psychological Bulletin*, 104, 97-126.
- Baron, J., & Strawson, C. (1976). Use of orthographic and word-specific knowledge in reading words aloud. *Journal of Experimental Psychology: Human Perception and Performance* (2), 386-393.
- Beck, A.T. (1967). *Depression: Clinical, experimental, and theoretical aspects*. Hoeber, New York.
- Beck, A.T. (1976). *Cognitive therapy and the emotional disorders*. New York: International Universities Press.
- Beck, A.T. (1980). *Beck Anxiety Inventory*, New York: Psychological Corporation.
- Beck, A.T. (1987). Cognitive model of depression. *Journal of Cognitive Psychotherapy*, 1, 2-27.
- Beck, A.T., Brown, G.K., Steer, R.A., & Weissman, A.N. (1991). Factor analysis of the Dysfunctional Attitude Scale in a clinical population. *Psychological Assessment*, 3, 478-483.
- Beck, A.T., Epstein, N., Brown, G.K., & Steer, R.A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology*, 56, 893-897.
- Beck, A.T., Rush, A.J., & Emery, G. (1979). *Cognitive Therapy of depression*. New York: Guilford press.
- Beck, A.T., & Steer, R.A. (1987). *Manual for the revised Beck Depression Inventory*. San Antonio, TX: Psychological Corporation.
- Beck, A.T., & Steer, R.A., & Brown, G.K. (1996). *BDI-II: Beck Depression Inventory manual* (2nd ed.). San Antonio, TX: The Psychological Corporation.
- Beck, A.T., Steer, R.A., & Garbin, M.G. (1988). Psychometric properties of the Beck Depression Inventory: 25 years of evaluation. *Clinical Psychology Review*, 8, 77-100.
- Beck, A.T., & Weishaar, M. (1989). Cognitive Therapy. In R. Corsini & D. Wedding (Eds). *Current Psychotherapies* (4th ed) (pp.285-320). USA: F.E. Peacock Publishers, Inc.
- Beevers, C.G., & Miller, I.V. (2005). Unlinking negative cognition and symptoms of depression: Evidence of a specific treatment effect for cognitive therapy. *Journal of Consulting and Clinical psychology*, 73 (1), 68-77.
- Blackburn, I.M., Eunson, K.M., & Bishop, S. (1986). A two-year naturalistic follow-up of depressed patients treated with cognitive therapy, pharmacotherapy, and a combination of both. *Journal of Affective Disorders*, 10, 67-75.

- Blackburn, I.M., Jones, S., Lewin, R.J.P. (1986). Cognitive style in depression. *British Journal of Psychology*, 94, 140-153.
- Blackburn, I.M., & Smyth, P. (1985). A test of cognitive vulnerability in individuals prone to depression. *British Journal of Clinical Psychology*, 24, 61-62.
- Blaney, P.H., Behar, V., & Head, R. (1980). Two measures of depressive cognitions: Their association with each other. *Journal of Abnormal psychology*, 89, 678-682.
- Blatt, S.J. (1974). Level of object representation in anaclitic and introjective depression. *Psychoanalytic Study of the Child*, 29, 107-157.
- Blatt, S.J., Quinlan, D., Chevron, E., McDonald, C., & Zuroff, D. (1982). Dependency and self-criticism: Psychological dimensions of depression. *Journal of Consulting and Clinical Psychology*, 50, 113-124.
- Bosson, J.K., Swann, W.B., Jr., & Pennebaker, J.W. (2000). Stalking the perfect measure of implicit self-esteem. The blind man and the elephant revisited? *Journal of Personality and Social Psychology*, 79, 631-643.
- Bouwer, C., & Stein, D.J. (1998). Use of selective serotonin reuptake inhibitor citalopram in the treatment of generalized social phobia. *Journal of Affective Disorders*, 49, 79-82.
- Bower, G.H. (1981). Mood and memory. *American Psychologist*, 36, 129-148.
- Bowers, W.A. (1990). Treatment of depressed inpatients: cognitive therapy plus medication, relaxation plus medication, and medication alone. *British journal of Psychiatry*, 156, 73-78.
- Bowlby, J. (1969). *Attachment and loss: Vol. I: Attachment*. Hogarth Press/Institute of Psychoanalysis: London (Pimlico edition, 1998).
- Bowlby, J. (1973). *Attachment and loss: Vol. II: Separation*. Hogarth Press/Institute of Psychoanalysis: London (Pimlico edition, 1998).
- Bowlby, J. (1980). *Attachment and loss: Vol. III: Loss*. Hogarth Press/Institute of Psychoanalysis: London (Pimlico edition, 1998).
- Boyer, W.F., McFadden, G.A., & Feighner, J.P. (1996). Clinical use of selective serotonin reuptake inhibitors in depression. In J.P. Feighner & W.E. Boyer (Eds.), *Selective serotonin reuptake inhibitors* (pp.153-160). Wiley, London.
- Bradley, B.P., & Mathews, A. (1988). Memory bias in recovered clinical depressives. *Cognition and Emotion*, 2, 235-245.
- Brebner, J.T., & Welford, A.T. (1980). Introduction: an historical background sketch. In A.T. Welford (Ed.), *Reaction Times* (pp.309-320). Academic Press, New York.
- Brendl, M., Markman, A., & Messner, C. (2001). How do indirect measures of evaluation work? Evaluating the interference of prejudice in the Implicit Association Test. *Journal of Personality and Social Psychology*, 81, 760-773.

- Brewerton, M.D., Brandt, H.A., Lesem, M.D., Murphy, D.L., & Jimersen, D.C. (1990). Serotonin in eating disorders, In E.F. Coccaro (Ed.), *Serotonin in major psychiatric disorders* (pp.155-184). American Psychiatric Press, London.
- Brewin, C.R. (1988). *Cognitive foundation of clinical psychology*. Hove: Lawrence Erlbaum Associates Ltd.
- Brown, J.D. (1993). Self-esteem and self-evaluation: Feeling is believing. In J. Suls (Ed.), *Psychological Perspectives on the Self, Vol 4* (pp.22-58). Hillsdale, NJ: Erlbaum.
- Brown, S.L. (1991). *The role of serotonin in psychiatric disorders*. Brunner/Mazel, New York.
- Brown, G.R., & Anderson, B. (1991). Psychiatric morbidity in adult inpatients with childhood histories of sexual abuse. *American Journal of Psychiatry*, 148, 55-61.
- Brown, G.W., & Harris, T.O. (1978) *Social origins of depression. A study of psychiatric disorder in women*. London: Tavistock.
- Bryer, J.B., Nelson, B.A., Miller, J.B., & Kroll, P.A. (1987). Childhood sexual physical abuse as factors in adult psychiatric illness. *American Journal of Psychiatry*, 144, 1426-1430.
- Bucci, W. (2000). The need for a psychoanalytic psychology in the cognitive science. *Psychoanalytical psychology*, 17, 203-224.
- Burns, D., & Nolen-Hoeksema, S. (1992). Therapeutic empathy and recovery from depression in cognitive-behavioral therapy: A structural equation model. *Journal of Consulting and Clinical Psychology*, 60, 441-449.
- Calvete, E., Estevez, A., de Arroyabe, E.L., & Ruiz, P. (2005). The Schema Questionnaire-Short-Form: Structure and relationship with automatic thoughts and symptoms of affective disorders. *European Journal of Psychological Assessment*, 21(2), 90-99.
- Carlson, N.R. (1994). *Physiology of behaviour (5th edition)*. Needham Heights, MA: Allyn & Bacon.
- Chadwick, P., Trower, P., & Dagnan, D. (1999). Measuring negative person evaluations: The Evaluative Beliefs Scale. *Cognitive Therapy and Research*, 23, 549-559.
- Charney, D.S., Heninger, G.R., Sternberg, D.E. (1983). Alpha-2 adrenergic receptor sensitivity and the mechanism of action of antidepressant therapy. *British Journal of Psychiatry*, 142, 265-275.
- Claesson, K., & Sohlberg, S (2002). Internalized shame and early interactions characterized by indifference, abandonment and rejection. Replicated findings. *Clinical Psychology and Psychotherapy*, 9, 277-284.
- Clark, D.M., & Teasdale, J.D. (1985). Constraints of the effects of mood and memory. *Journal of Personality and Social Psychology*, 48, 1595-1608.
- Clark, L.A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology*, 103, 116.

- Coccaro E.F., Siever L.J., Owen K.R., & Davis, K.L. (1990). Serotonin in mood and personality disorder. In E.F. Coccaro, & D.L. Murphy, (Eds.), *Serotonin in major psychiatric disorder* (pp.71-97). American Psychiatric Press, London.
- Cox, J.L. (1992) Depression and childbirth. In E.S. Paykel (Ed.), *Handbook of affective disorders* (569-584). New York: Guilford Press.
- Cox, B.J., Enns, M.W., Borger, S.C., & Parker, J.D.A., (1999). The nature of the depressive experience in analogue and clinically depressed samples. *Behaviour Research and Therapy*, 37, 15-24.
- Cox, B.J., Enns, M.W., & Larson, D.K. (2001). The continuity of depression symptoms: Use of cluster analysis for profile identification in patient and student samples. *Journal of Affective Disorders*, 65, 67-73.
- Coyne, J.C., & Gotlib, I.H. (1983). The role of cognition in depression: A critical appraisal. *Psychological Bulletin*, 94, 472-505.
- Coyne, J.C., & Whiffen, V.E. (1995). Issues in personality as diathesis for depression: the case of sociotropy-dependency and autonomy self-criticism. *Psychological Bulletin*, 118, 358-378.
- Crandell, C.J., & Chambless, D.L. (1986). The validity of an inventory for measuring depressive thoughts: The Crandell Cognitions Inventory. *Behaviour Research and Therapy*, 24, 403-411.
- Crowne, D.P., & Marlowe, D.A. (1960). A new scale of social desirability independent of psychopathology. *Journal of Consulting Psychology*, 24, 349-354.
- Davidson, G.C., Neale, J.M. (1986). *Abnormal Psychology*. New York. John Wiley & Sons.
- De Houwer, J. (2001). A structural and process analysis of the IAT. *Journal of Experimental-Social Psychology*, 37, 443-451.
- De Houwer, J. (2002). The Implicit Association Test as tool for studying dysfunctional associations in psychopathology: strengths and limitation. *Behavior Therapy and Experimental Psychiatry*, 33, 115-133.
- De Houwer, J. (2003). The extrinsic affective simon task. *Experimental Psychology*, 50, 77-85.
- De Houwer, J. (2006). What are implicit measures and why are we using them. In R. W. Wiers & A. W. Stacy (Eds.), *The handbook of implicit cognition and addiction* (pp. 11-28). Thousand Oaks, CA: Sage Publishers.
- de Jong, P.J. (2000). Implicit self-esteem and social anxiety: differential self-favouring effects in high and low anxious individuals. *Behaviour research and therapy*, 40(5), 501-8.
- de Jong, P.J., Van Den Hout, M.A., Merckelbach, H. (1995). Covariation bias and the return of fear. *Behavior Research and Therapy*, 33 (2), 211-213.

- de Jong, P.J., Van den Hout, M.A., & Rietbroek, H. (2003). Dissociations between implicit and explicit affect in response to phobic stimuli. *Cognition and Emotion*, 17, 521-545.
- De Raedt, R., Schacht, R., Franck, E., & De Houwer, J. (2006). Self-esteem and depression revisited: Implicit positive self-esteem in depressed patients? *Behavior Research and Therapy*, 44 (7), 1017-1028.
- De Monbreun, B.G., & Craighead, W.E. (1977). Distortion of perception and recall of positive and neutral feedback in depression. *Cognitive Therapy and Research*, 1, 311-329.
- Depue, R., & Monroe, S. (1978). Learned helplessness in the perspective of the depressive disorders: Conceptual and definitional issues. *Journal of Abnormal Psychology*, 87, 3-20.
- Dent, J. & Teasdale, J.D. (1988). Negative cognition and the persistence of depression. *Journal of Abnormal Psychology*, 97, 29-34.
- Diagnostic and Statistical Manual of mental Disorders*, 1994, Fourth Edition, American Psychiatric Association.
- Dinan, T. (1994). Glucocorticoids and the genesis of depressive illness: a psychobiological model. *British Journal of Psychiatry*, 164, 365-371.
- Dobson, K.S., & Breiter, H.J. (1983). Cognitive assessment of depression: Reliability and validity of three measures. *Journal of Abnormal Psychology*, 92, 107-109.
- Dobson, K.S., Shaw, B. (1986). Cognitive assessment with major depressive disorders. *Cognitive Therapy and Research*, 10, 13-29.
- Dobson, K., & Shaw, B. (1987). Specificity and stability of self-referent encoding in clinical depression. *Journal of Abnormal Psychology*, 96, 34-40.
- Dohr, K.B., Rush, A.J., & Bernstein, I.H. (1989). Cognitive biases in depression. *Journal of Abnormal Psychology*, 98, 263-267.
- Dorne, J.L.C.M., Walton, K., Slobb, W., & Renwick, A.G. (2004). Human variability in polymorphic CYP2D6 metabolism: is the kinetic default uncertainty factor adequate? *Food and Chemical Toxicology*, 40, 1633-1656.
- Downing-Orr, K. (1998). *Rethinking depression: Why current treatments fail*. Plenum Press, New York.
- Dozois, D. J. A., & Dobson, K. S. (2001). A longitudinal investigation of information processing and cognitive organization in clinical depression: Stability of schematic interconnectedness. *Journal of Consulting and Clinical Psychology*, 69, 914-925.
- Dozois, D.J.A., Dobson, K.S., & Ahnberg, J.L. (1998). A psychometric evaluation of the Beck Depression Inventory II. *Psychological Assessment*, 10, 83-89.
- Drew, M., Dobson, K., & Stam, H. J. (1999). The negative self-concept in clinical depression: A discourse analysis. *Canadian Psychology*, 40, 192-204.

- Dreyfus, H. (1989). *Alternative philosophical conceptualizations of psychopathology*. Retrieved September 8, 2004, from, http://ist-socrates.berkeley.edu/~hdreyfus/html/paper_alternative.html
- Dryman, A., & Eaton, W.W. (1991). Affective symptoms associated with onset of major depression in the community: Findings from the U.S. National Institute of Mental Health Epidemiological Catchment Area program. *Acta Psychiatrica Scandinavica*, 84, 1-5.
- Dykman, B.M. (1997). A test of whether negative emotional priming facilitates access to latent dysfunctional attitudes. *Cognition & Emotion*, 11(2), 197-222.
- Dykman, B.M., Abramson, L.Y., Alloy, L.B., Hartledge, S. (1989). Processing of ambiguous feedback by depressed and non-depressed college students: schematic biases and their implications for depressive realism. *Journal of Personality and Social Psychology*, 56 (3), 431-445.
- Eaves, G., Rush, A.J. (1984). Cognitive patterns in symptomatic and remitted unipolar major depression. *Journal of Abnormal Psychology*, 93, 31-40.
- Edwards, A.L. (1957). *The social desirability variable in personality assessment and research*. New York, NY: Holt, Rinehart, & Winston.
- Ellis, A. (1962). *Reason and emotion in psychotherapy*. New York: Lyle Stuart.
- Ende, R.N. (1999). *Moving ahead: Integrating influences of affective processes for development and for psychoanalysis*. Paper presented at the 41st Congress of the International Psychoanalytical Association in Santiago.
- Epstein, S., & Morling, B. (1995). Is the self motivated to do more than enhance and/or verify itself? In M.H. Kernis (Ed.), *Efficacy, agency, and self-esteem* (pp. 9-29). New York: Plenum.
- Evans, M.D., Hollon, S.D., DeRubeis, R.J., Piasecki, J.M., Grove, W.M., Garvey, M.J., & Tuason, V.B. (1992). Differential relapse following cognitive therapy and pharmacotherapy for depression. *Archives of General Psychiatry*, 49, 802-808.
- Eysenck, H. J. (1991). Dimensions of personality: 16, 5, or 3? Criteria for a taxonomic paradigm. *Personality and Individual Differences*, 12, 773-790.
- Farnham, S.D, Greenwald, A.G., & Banaji, M.R. (1999). Implicit self-esteem. In D. Abrams & M.A. Hogg (Eds.), *Social identity and social cognition* (pp. 230-248). Oxford, UK: Blackwell.
- Fava, G., Grandi, S., Zielesny, M., Rafanelli, C., & Canestrari, R. (1996). Four-year outcome for cognitive behavioural treatment of residual symptoms in major depression. *American Journal of Psychiatry*, 153, 945-947.
- Fennell, M.J., & Campbell, E.A. (1984). The Cognitions Questionnaire: Specific thinking errors in depression. *British Journal of Clinical Psychology*, 23, 81-92.

- Ferguson, T., Rule, B., & Carlson, D. (1983). Memory for persistently relevant information. *Journal of Personality and Social Psychology*, 44, 251-261.
- First, M.B., Gibbon, M., Spitzer, R.L., & Williams, J.B.W. (1996). *Structured Clinical Interview for DSM Axis I Disorders*. Research Version (SCID-I, Version 2.0, February 1996, final version). New York: Biometrics Research.
- Fisk, S.T., & Linville, P.W. (1980). What does the schema concept buy us? *Personality and Social Psychology Bulletin*, 6, 543-557.
- Frank, J.D., & Frank, J.B. (1991). *Persuasion and healing: a comparative study of psychotherapy: 3rd Edition*. John Hopkins University Press, Baltimore.
- Freeman, W.J. (2001). *How the brain makes up its mind*. Columbia University Press.
- Freud, S. (1973). *Introductory lectures on psychoanalysis*. Penguin Books Ltd, Hammondsworth, Middlesex, England.
- Garber, J., & Hollon, S.D. (1991) What can specificity designs say about causality in psychopathology research? *Psychological Bulletin*, 110, 129-136.
- Gemar, M.C., Segal, Z.V., Sagrati, K., Kennedy, S.J. (2001) Mood-induced changes on the Implicit Association Test in recovered depressed patients. *Journal of Abnormal Psychology*. 110(2), 282-289.
- Gershon, E.S., & Nurnberger, J. (1982). Inheritance of major psychiatric disorders. *Trends in Neuroscience*, 5 (7), 241-242.
- Gilbert, D.T., Giesler, R.B., & Morris, D.A. (1995). When comparisons arise. *Journal of Personality and Social Psychology*, 69, 227-236.
- Gladstone, G., Parker, G. (2002). Depressogenic cognitive schemas: Enduring beliefs or mood state artifacts? *Australian & New Zealand Journal of Psychiatry*, 35, 210-216
- Gleitman, H. (1994). *Psychology (4th Edition)*. London: Routledge.
- Gold, P.W., Goodwin, F.K., & Charousos, G.P. (1998). Clinical and biochemical manifestations of depression: relation to the neurobiology of stress. *New England Journal of Medicine*, 320, (13), 869-870.
- Goodwin, F.K., & Jaminson, K.R. (1990). *Manic-depressive illness*. New York: Oxford University Press.
- Gotlib, I.H. (1983). Perception and recall of interpersonal feedback. *Cognitive Therapy and Research*, 7, 399-412.
- Gotlib, I.H. (1984). Depression and psychopathology in university students. *Journal of Abnormal Psychology*, 93, 19-30.
- Gotlib, I.H., & Cane, C.B. (1987). Construct accessibility and clinical depression: A longitudinal investigation. *Journal of Abnormal Psychology*, 96, 199-204.
- Gotlib, I.H. & Krasnoperova, E. (1998). Biased information processing as a vulnerability factor for depression. *Behavior Therapy and Research*, 29, 603-617.

- Gotlib, I.H., Mount, J.H., Cordy, N.I., & Whiffen, V.E. (1988). Depression and perceptions of early parenting: A longitudinal investigation. *British Journal of Psychiatry*, 152, 24-27.
- Govern, J.M., & Marsch, L.A. (1997). Inducing positive mood without demand characteristics. *Psychological Reports*, 81 (3 part 1), 1027-1034.
- Greene, J.G. (1980). Life stress and symptoms at the climacterium. *British Journal of Psychiatry*, 136, 486-491.
- Greenberg, M.S., & Alloy, L.B. (1989). Depression versus anxiety: Processing of self-and other-referent information. *Cognition and Emotion*, 3, 207-223.
- Greenfield, S. (1998). How might brains generate consciousness? In S. Rose (Ed), *From Brains to Consciousness: Essays on the new sciences of the mind* (pp.210-227). London, Penguin.
- Greenwald, A. G. (1997). Validity concerns and usefulness of student ratings. *American Psychologist*, 52, 1182-1186.
- Greenwald, A.G., & Banaji, M.R. (1995). Implicit social cognition: Attitudes, self-esteem, and stereotypes. *Psychological Review*, 35, 603-618.
- Greenwald, A.G., Banaji, M.R., & Schwartz, J.L.K. (1998). Measuring individual differences in implicit cognition: The Implicit Association Test. *Journal of Personality and Social Psychology*, 74, 1464-1480.
- Greenwald, A.G., & Farnham, S.D. (2000). Using the Implicit Association Test to measure self-esteem and self-concept. *Journal of Personality and Social Psychology*, 79, 1022-1038.
- Gross, R. (1999). *Key Studies in Psychology*. London: Routledge.
- Grossberg, J.M., & Grant, B.F. (1978). Clinical psychophysics: Applications of ratio scaling and signal detection methods to research on pain, fear, drugs, and medical decision making. *Psychological Bulletin*, 85, 1154-1176.
- Hagga, D. A. F, Dyck, M.J., & Ernst, D. (1991). Empirical status of the cognitive theory of depression. *Psychological Bulletin*, 110, 215-316.
- Hamilton, E.W., & Abramson, L.Y. (1983). Cognitive patterns and major depressive disorder: A longitudinal study in a hospital setting. *Journal of Abnormal Psychology*, 92, 173-184.
- Hammen, C.L. (1980). Depression in college students: Beyond the Beck Depression Inventory. *Journal of Consulting Psychology*, 48, 126-128.
- Hammen, C.L., & Krantz, S.E. (1985). Measures of psychological processes in depression. In Beckham, E.E. & Leber, W.R. (Eds), *Handbook of depression: Treatment, assessment, and research* (pp. 408-444). Homewood, IL: Dorsey.
- Hammen, C.L., Marks, T., Mayol, A., & DeMayo, R. (1985). Depressive self-schemas. Life stress, and vulnerability to depression. *Journal of Abnormal Psychology*, 94, 308-319.

- Harrell, T.H., & Ryon, N.B. (1983). Cognitive-behavioural assessment of depression: Clinical validation of the Automatic Thoughts Questionnaire. *Journal of Consulting and Clinical Psychology*, 51, 721-725.
- Harris, A.E., & Curtin, L. (2002). Parental perceptions, early maladaptive schemas, and depressive symptoms in young adults. *Cognitive Therapy and Research*, 26 (3), 405-416.
- Hartledge, S., Alloy, L.B., Vazquez, C., & Dykman, B. (1993). Automatic and effortful processing in depression. *Psychological Bulletin*, 113, 247-278.
- Hawton, K, Salkovskis, J., & Clark, D. (1989). *Cognitive behaviour therapy for psychiatric problems*. A practical guide. United States. Oxford University Press.
- Healy, D. (2003). Lines of evidence on the risks of suicide with selective serotonin reuptake inhibitors. *Psychotherapy and Psychosomatics*, 72, 71-79
- Hedlund, S., & Rude, S.S. (1995). Evidence of latent depressive schemas in formerly depressed individuals. *Journal of Abnormal Psychology*, 104, 517-525.
- Heidegger, M. (2001). *Zollikon Seminars. Protocols-conversations-letters*. M. Boss (Ed.). Translated by F. Mayer & R. Askay. USA: Northwestern University Press.
- Hendren, R.I. (1983). Depression in anorexia nervosa. *Journal of the American Academy of Child Psychiatry*, 22, 59-62.
- Hensley, P.L., Nadiga, D., & Uhlenhuth, E.H. (2004). Long-term effectiveness of cognitive therapy in major depressive disorder. *Depression and Anxiety*, 20, 1-7.
- Hersen, M., & Ammerman, R.T. (1995). *Advanced Abnormal Child Psychology*. New Jersey: Lawrence Erlbaum Associates, Inc.
- Hertal, P.T., & Rude, S.S. (1991). Depressive deficits in memory: Focusing attention improves subsequent recall. *Journal of Experimental Psychology*, 20, 301-309.
- Higgins, E.T., & King, G.A. (1981). Accessibility of social constructs: Information processing consequences of individual and contextual variability. In N. Cantor & J.F. Kihlstrom (Eds.), *Personality, cognition, and social interaction* (pp. 69-121). Hillsdale, NJ: Erlbaum.
- Hollon, S.D., Evans, M.D., & De Rubeis, R.J. (1990). Cognitive mediation of relapse prevention following treatment for depression: Implications of differential risk. In R.E. Ingram (Ed.), *Contemporary psychological approaches to depression: Theory, research, and treatment* (pp.117-136). New York: Guilford Press.
- Hollon, S.D., & Kendall, P.C. (1980). Cognitive self-statements in depression: Development of an automatic thoughts questionnaire. *Cognitive Therapy and Research*, 4, 383-395.
- Hollon, S.D., & Kriss, M.R. (1984). Cognitive factors in clinical research and practice. *Clinical Psychology Review*, 4, 35-76.
- Hsieh, S. (2002). Task shifting in dual-task settings. *Perceptual and Motor Skills*, 94 (2), 407-415.

- Illardi, S.S., & Craighead, W.E. (1994). The role of neuroscientific factors in cognitive-behaviour therapy for depression. *Clinical Psychology: Science and Practice*, 1, 138-156.
- Ingram, R.E. (1984). Toward an information processing analysis of depression. *Cognitive Therapy and research*, 8, 443-478.
- Ingram, R.E. (1990). Self-focused attention in clinical disorders: review and a conceptual model psychological model. *Psychological Bulletin*, 107, 156-176.
- Ingram, R.E., Brenet, C. Z., & McLaughlin, S. C. (1994). Attentional allocation processes in individuals at risk for depression. *Cognitive Therapy and research*, 18, 317-322.
- Ingram, R.E., Lumery, A.E., Cruet, D., & Sieber, W. (1987). Attentional processes in depressive disorders. *Cognitive Therapy and Research*, 11, 351.
- Ingram, R.E., Miranda, J., Segal, Z.V. (1998). *Cognitive vulnerability to depression*. The Guilford Press. New York.
- Ingram, R.E., & Ritter, J. (2000). Vulnerability to depression: Cognitive reactivity and parental bonding in high-risk individual. *Journal of Abnormal Psychology*, 109, 588-596.
- Ingram, R.E., & Wisnicki, K.S. (1991). Cognition and depression. In P.A. Magaro (Ed.). *Annual Review of Psychopathology, (Vol1)*, (pp.187-230). Newbury Park, CA: Sage.
- Jacobson, N.S., & Anderson, E.A. (1982). Interpersonal skills and depression in college students: An analysis of the timing of self-disclosures. *Behaviour Therapy*, 13, 271-282.
- Jacobson, N.S., Dobson, K.S., Truax, P.A., Addis, M.E., Koerner, K., Gollan, J.K., Gortner, E.T., & Prince, S.E. (1996). A component analysis of cognitive-behavioral treatment for depression. *Journal of Consulting and Clinical Psychology*, 64, 295-304.
- Jacobson, N. S., & Gortner, E. T. (2000). Can depression be de-medicalized in the 21st century: Scientific revolutions, counter-revolutions and the magnetic field of normal science. *Behaviour Research & Therapy*, 38, 103-117.
- James, O. (1988). *Britain on the couch: Treating a low serotonin society*. Arrow Books Limited, London.
- Jarrett, R.B., Kraft, D., Doyle, J., Foster, B.M., Eaves, G., & Silver, P. (2001). Preventing recurrent depression. *Archives of General Psychiatry*, 58, 381-388.
- Jones, E.E., & Pulos, S.M. (1993). Comparing the process in psychodynamic and cognitive-behavioural therapies. *Journal of Consulting and Clinical Psychology*, 61(2), 306-316.
- Jordan, M.I., & Rosenbaum, D.A. (1989). *Action*. In M.I. Posner (Ed.), *The foundations of cognitive science* (pp. 727-768). Cambridge: MIT Press.
- Judd, L.L. (1997). Pleomorphic expressions of unipolar depressive disease: Summary of the 1996 CINP President's workshop. *Journal of Affective Disorders*, 45, 109-116.
- Just, N, & Alloy, L.B. (1997). The responses style theory of depression: tests and an extension of the theory. *Journal of Abnormal Psychology*, 106, 221-229.
- Kant, E. (1963). *Critique of pure reason (2nd Edn.)*. London: Macmillan.

- Kaplan, H.I., & Sadock, B.J. (1995). *Comprehensive textbook of Psychiatry*. Williams & Williams, New York.
- Kendall, P.C., Hollon, S.D., Beck, A.T., Hammen, C.L., & Ingram, R.E. (1987). Issues and recommendations regarding use of the Beck Depression Inventory. *Cognitive Therapy and Research*, 11, 280-299.
- Kendall, P.C., & Lipman, A.J. (1991). Psychological and pharmacological therapy: methods and modes for comparative outcome research. *Journal of Consulting and Clinical Psychology*, 59, 78-87.
- Kendall-Tackett, K.A., William, L.M., & Finkelhor, D. (1993). Impact of sexual abuse on children: A review and synthesis of recent empirical studies. *Psychological Bulletin*, 113, 164-180.
- Kendler, K.S., Kessler, R.C., & Neale, M.C. (1993). The prediction of major depression in women: Toward an integrated etiologic model. *American Journal of Psychiatry*, 150, 1139-1148.
- Kendler, K.S., Neale, M.C., Kessler, R.C., & Heath, A.C. (1992). Familial influences on the clinical characteristics of major depression: A twin study. *Acta Psychiatrica Scandinavica*, 86, 371-378.
- Kessler, R.C., McGonagle, K.A., Zhao, S., Nelson, C.B., Hughes, M., Elshman, S., Wittchen, H.U., & Kendler, K.S. (1994). Lifetime and 12 month prevalence of results from the National Comorbidity Survey. *Archives of General Psychiatry*, 51, 8-19.
- Kirsch, I. (1985). Response expectancy as a determinant of experience and behaviour. *American Psychologist*, 40, 1189-1202.
- Kirsch, I., & Lynn, S.J. (1999). Automaticity in clinical psychology. *American Psychologist*, 54, 504-515.
- Klein, D., Harding, K., Taylor, E.B., & Dickstein, S. (1988). Dependency and self-criticism in depression: Evaluation in a clinical population. *Journal of Abnormal Psychology*, 97, 399-404.
- Klerman, G.L., & Weissman, M.M. (1992). The course, morbidity, and costs of depression. *Archives of General Psychiatry*, 49, 831-834.
- Kovacs, M., & Beck, A.T. (1978). Maladaptive cognitive structures in depression. *American Journal of Psychiatry*, 135, 525-533.
- Kuiper, N.A., Olinger, L.J., & McDonald, M. (1988). Vulnerability and episodic cognitions in a self-worth contingency model of depression. In L.B. Alloy (Ed.), *Cognitive processes in depression* (pp.289-309). New York: Guilford Press.
- Laing, R.D. (1967). *The Politics of Experience and the Bird of Paradise*. Harmondsworth, Middlesex: Penguin Books.

- Lavelle, T.L., Metalsky, G.I., & Coyne, J.C. (1979). Learned helplessness, test anxiety, and acknowledgement of contingencies. *Journal of Abnormal Psychology*, 121, 133-143.
- Leuchter, A.F., Cook, I.A., Witte, E.A., Morgan, M., & Abrams, M. (2002). Changes in Brain Function of Depressed Subjects. *American Journal of Psychiatry*, 159 (1), 117-122.
- Le Doux, J. (1998). *The Emotional Brain*. London: Orion Books.
- Lewinsohn, P.M., Hoberman, H.M., Teri, L., & Hautzinger, M. (1985). An integrated theory of depression. In S. Reiss & R.R. Bootzin (Eds.), *Theoretical issues in behaviour therapy* (313-359). Orlando, FL: Academic Press.
- Lewinsohn, P.M., Mischel, W., Chaplin, W., & Barnton, R. (1980). Social competence and depression: The role of illusory self-perceptions. *Journal of Abnormal Psychology*, 89, 203-212.
- Lewinsohn, P.M., Steinmetz, J.L., Larson, D.W., & Franklin, J. (1981). Depression related cognitions: antecedents or consequences? *Journal of Abnormal Psychology*, 90, 213-219.
- Linnoila, M., de Jong, J., & Virkkunen, M. (1989). Monoamines, glucose metabolism, and impulse control. *Psychopharmacology Bulletin*, 25, 404-406.
- Logan, G.D. (1979). On the use of a concurrent memory load to measure attention and automaticity. *Journal of Experimental Psychology: Human Perception and Performance*, 5, 189-207.
- Luxton, D.D., & Wenzlaff, R.M. (2005). Self-esteem uncertainty and depression, *Cognition and Emotion*, 19 (4), 611-622.
- Lynch, T. (2004). *Beyond Prozac*. PCCS Books, Ross-on-Wye.
- Malinoskky-Rummell, R., & Hansen, D.J. (1993). Long-term consequences of childhood physical abuse. *Psychological Bulletin*, 114, 68-79.
- Martin, M. (1990). On the induction of mood. *Clinical Psychology Review*, 10, 669-697.
- Martin, S.D., Martin, E., Rai, S.S., Richardson, M.A., & Royall, R. (2001). Brain blood flow changes in depressed patients treated with interpersonal psychotherapy or venlafaxine hydrochloride: preliminary findings. *Archives of General Psychiatry*, 58, 641-648.
- Mayberg, H.S., Liotti, M., Brannan, S.K., McGinnis, S., Mahurin, R.K., Jerabeck, P.A., Silva, J.A., Tekell, J.L., Martin, C.C., Lancaster, J.L., & Fox, P.T. (1999). Reciprocal limbic-cortical function and negative mood: conveying PET findings in depression and normal sadness. *American Journal of Psychiatry*, 156 (5), 675-682.
- McCabe, S., & Gotlib, I.H. (1993). Attentional processing in clinically depressed subjects: A longitudinal investigation. *Cognitive Therapy and Research*, 17, 359-377.
- McClain, L., & Abramson, L.Y. (1995). Self-schemas, stress, and depressed mood in college students. *Cognitive Therapy and Research*, 19, 419-432.
- McCranie, E.W., & Bass, J.D. (1984). Childhood family antecedents of dependency and self-criticism: Implications for depression. *Journal of Abnormal Psychology*, 93, 3-8.

- McEwen, B.S., & Magarinos, A.M. (2001). Stress and hippocampal plasticity: Implications for the pathophysiology of affective disorders. *Human Psychopharmacology*, 16, 7-19.
- McGuffin, P., Katz, R., & Rutherford, J. (1991). Nature, nurture, and depression: A twin study. *Psychological Medicine*, 21, 329-335.
- McNeal, E.T., & Cimbolich, P. (1986). Antidepressants and biochemical theories of depression. *Psychological Bulletin*, 99, 361-374.
- Merleau-Ponty, M. (1962). *The Phenomenology of Perception*. London, Routledge & Kegan Paul.
- Metalsky, G.I., Joiner, T.E., Hardin, T.S., Abramson, L.Y. (1987). Vulnerability to depressive mood reactions: Toward a more powerful test of the diathesis-stress and causal mediation components of the reformulated theory of depression. *Journal of Personality and Social Psychology*, 52, 386-393.
- Miranda, J., Pearsons, J.B. (1988). Dysfunctional attitudes are mood-state dependent. *Journal of Abnormal Psychology*, 97, 76-79.
- Miranda, J., Pearsons, J.B., Byers, C. (1990). Endorsement of dysfunctional beliefs depends on current mood state. *Journal of Abnormal Psychology*, 99, 237-241.
- Moncrieff, J. (2003). A comparison of antidepressant trials using active and inert placebos. *International Journal of Medicine*, 12, 117-127.
- Mulrow, C.D., Williams, J.W., Trivedi, M., Chiquette, E., Aguilar, C., Cornell, J.E., Badgett, R., Noel, P.H., Lawrence, V., Lee, S., Luther, M., Ramirez, G., Richardson, W.S., & Stamm, K. (1999). Treatment of depression: Newer pharmacotherapies. *Evidence Report/Technology Assessment*, 7, 1-4.
- Murphy, H., Wittkower, E., & Chance, N. (1964). Cross-cultural inquiry into the symptomatology of depression. *Transcultural Psychiatric Research Review*, 1, 5-21.
- Murray, L., Fiori-Cowley, R., Hooper, P.J., & Cooper, P.J. (1996). The impact of postnatal depression and associated adversity on early mother-infant interactions and later infant outcome. *Child Development*, 67, 2515-2526.
- Musson, R.F., & Alloy, L.B. (1988). Depression and self-directed attention. In L.B. Alloy (Ed.), *Cognitive processes in depression* (pp. 193-220). New York: Guilford Press.
- Mrazek, P.J., & Haggerty, R.J. (1994). *Reducing risks for mental disorders: Frontiers for preventive intervention research*. National Academy Press, Washington, DC.
- Nelson, L.D., Stern, S.L., & Cicchetti, D.V. (1992). The Dysfunctional Attitude Scale: How well can it measure depressive thinking? *Journal of Psychopathology and Behavioral Assessment*, 14 (3), 217-223.
- Nisbett, R.E., & Wilson, T.D. (1977). Telling more than we can know: Verbal reports on mental processes. *Psychological Review*, 84, 231-259.

- Nolen-Hoeksema, S. (1987). Sex differences in unipolar depression: Evidence and theory. *Psychological Bulletin*, 101, 259-282.
- Nolen-Hoeksema, S., Girgus, J.S., & Seligman, M.E.P. (1992). Learned helplessness in children: A longitudinal study of depression, achievement, and explanatory style. *Journal of Personality and Social Psychology*, 101, 405-422.
- Nolen-Hoeksema, S., & Morrow, J. (1991). A prospective study of depression and posttraumatic stress symptoms after a natural disaster: The 1989 Loma Prieta earthquake. *Journal of Abnormal Psychology*, 100, 569-582.
- Nolen-Hoeksema, S., Parker, L., & Larson, J. (1994). Ruminative coping with depressed mood following loss. *Journal of Personality and Social Psychology*, 67, 92-104.
- Nurcombe, B. (1992). The evolution and validity of the diagnosis of major depression in childhood and adolescence. In D. Cicchetti & S.L. Toth (Eds.), *Developmental perspectives on depression* (pp.1-27). Rochester, NY: University of Rochester Press.
- Nuttin, J.M. (1985). Narcissism beyond gestalt and awareness: The name letter effect. *European Journal of Social Psychology*, 15, 353-361.
- O'Hara, M.W., Rehm, L., & Campbell, S. (1982). Predicting depressive symptomatology: Cognitive-behavioural models of postpartum depression. *Journal of Abnormal Psychology*, 91, 457-461.
- O'Hara, M.W., Zekoski, E.M., Philips, L.H., Wright, E.J. (1990). Controlled prospective study of postpartum mood disorders: Comparison of childbearing and non-childbearing women. *Journal of Abnormal Psychology*, 99, 3-15.
- O'Keane, V. (1992). Blunted prolactin response to d-Fenfluramine in sociopathy. *British Journal of Psychiatry*, 160, 643-646.
- Oliver, J.M., & Baumgart, E.P. (1985). The Dysfunctional Attitude Scale: Psychometric properties and relation to depression in an unselected adult population. *Cognitive Therapy and Research*, 9, 161-168.
- Oltmans, T.F., & Emery, R.E. (1998). *Abnormal Psychology*, 2nd Edition. New Jersey, Prentice-Hall.
- O'Toole, S., & Johnson, D.A. (1997). Psychobiology and psychopharmacology of unipolar major depression: a review. *Archives of Psychiatric Nursing*, 11 (6), 306-408.
- Parker, G., Gladstone, G., Mitchell, P., Wilhelm, K., & Roy, K. (2000). Do early adverse experiences establish a cognitive vulnerability to depression on exposure to mirroring life events in adulthood? *Journal of Affective Disorders*, 57, 209-215.
- Paykel, E.S. (2001). The evolution of life events research in psychiatry. *Journal of Affective Disorders* 62(3), 141-149.

- Paykel, E.S., Scott, J., Teasdale, J.D., Johnson, A.L., Garland, A., Moore, R., Jenaway, A., Cornwall, P.L., Hayhurst, H., Abbott, R., & Pope, M. (1999). Prevention of relapse in residual depression by cognitive therapy. *Archives of General Psychiatry*, 56 (9), 829-835.
- Pelham, B.W., & Hetts, J.J. (1999). Implicit self-evaluation. (Unpublished manuscript).
- Perry, B.D., Pollard, R.A., Blakley, T.L., Baker, W.L., & Vigilante, D. (1995). Childhood trauma, the neurobiology of adaptation and use-dependent development of the brain: How states become traits. *Infant Mental Health Journal*, 16, 271-291.
- Piaget, J. (1926). *The language and thought of the child*. New York: Basic Books.
- Piccinelli, M., & Wilkinson, G. (1994). Outcome of depression in Psychiatric Settings. *British Journal of Psychiatry*, 164, 297-304.
- Pielage, S., Gerlsma, C., & Schaap, C. (2000). Insecure attachment as a risk factor for psychopathology: The role of stressful events. *Clinical Psychology and Psychotherapy*, 7, 296-302.
- Pilkonis, P., & Frank, E. (1988). Personality pathology in recurrent depression: Nature, prevalence, and relationship to treatment response. *American Journal of Psychiatry*, 145, 435-441.
- Pollatsek, A., & Rayner, K. (1989). Effects of background information on object perception. *Journal of Experimental Psychology: Human Perception & Performance*, 15 (3), 556-566.
- Posner, M. I., & Snyder, C. R. R. (1975). Attention and cognitive control. In R. Solso (Ed.), *Information processing and cognition* (pp. 55-85). Hillsdale, NJ: Erlbaum.
- Power, M.K., Katz, R., McGuffin, P., Duggan, C.F., Lam, D., & Beck, A.T (1994). The Dysfunctional Attitude Scale (DAS): A comparison of forms A and B and proposals for a new subscaled version. *Journal of Research in Personality*, 28, 263-276.
- Pyszczynski, T., & Greenberg, J. (1992b). Putting cognitive constructs in their place: Is depression really just a matter of interpretation? *Psychological Inquiry*, 3, 255-258.
- Radloff, L., & Rae, D.S. (1979). Susceptibility and precipitation factors in depression: Sex differences and similarities. *Journal of Abnormal Psychology*, 88, 174-181.
- Rector, N.A., Segal, S.V., & Gemar, M. (1998). Schema Research in Depression: A Canadian Perspective. *Canadian Journal of Behavioural Science*, 30, 213- 224.
- Rehm, L.P. (1995). Psychotherapies for depression. In K.D. Craig & K.S. Dobson (Eds.), *Anxiety and depression in adults and children* (pp. 183-208). Thousand Oaks, CA; Sage.
- Reynolds, W.M. (1991b). Psychometric characteristics of the Adult Suicidal Questionnaire with college students. *Journal of Personality Assessment*, 56, 289-307.
- Roberts, J.E., & Kasel, J.D. (1996). Mood state dependence in cognitive vulnerability to depression: The roles of positive and negative affect. *Cognitive Therapy and Research*, 20, 1-12.

- Roberts, J.E., & Monroe, S.M. (1992). Vulnerable self-esteem and depressive symptoms: Prospective findings comparing three alternative conceptualizations. *Journal of Personality and Social Psychology*, 62, 804-812.
- Roberts, J.E., & Monroe, S.M. (1994). A multidimensional model of self-esteem in depression. *Clinical Psychology Review*, 14, 161-181.
- Robins, L.N., Heltzer, J.E., Croughan, J., & Radcliff, K.S. (1981). National Institute of mental health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry*, 38, 381-389.
- Robinson, L.A., Berman, J.S., & Neimeyer, R.A. (1990). Psychotherapy for the treatment of depression: a comprehensive review of controlled outcome research. *Psychological Bulletin*, 108(1), 30-49.
- Rosenblum, L.A., Coplan, J.D., Friedman, S., Bassoff, T., Gorman, J.M., & Andrews, M.W. (1994). Adverse early experience affects noradrenergic and serotonergic functioning in adult primates. *Biological Psychiatry*, 35, 221-227.
- Rosenthal, D. (1970) *Genetic theory and abnormal behaviour*. New York: McGraw-Hill.
- Roy, A., Virkkunen, M., & Linnoila, M. (1990). Serotonin in suicide, violence, and alcoholism. In E.F. Coccaro, & D.L. Murphy (Eds.), *Serotonin in major psychiatric disorders* (pp.187-208). Washington DC, American Psychiatric Press.
- Rude, S.S., Covich, J., Jarrold, W., Hedlund, S., & Zentner, M. (2001). Detecting depressive schemata in vulnerable individuals: Questionnaires versus laboratory tasks. *Cognitive Therapy and Research*, 1, 103-116.
- Rudman, L.A. (2004). Social justice in our minds, homes, and society: The nature, causes, and consequences of implicit bias. *Social Justice Research*, 17, 129-142.
- Rudman, L.A., Greenwald, A.G., Mellott, D.S., & Schwartz, J.L.K. (1999). Measuring the automatic components of prejudice: Flexibility and generality of the Implicit Association Test. *Social Cognition*, 17, 437-465.
- Sargeant, J.K., Bruce, M.L., Florio, L.P., & Weissman, M.M. (1990). Factors associated with 1-year outcome of major depression in the community. *Archives of General Psychiatry*, 47, 519-526.
- Sartorius, N., Jablensky, A., Gulbinat, W., & Ernberg, G. (1980). WHO collaborative study: Assessment of depressive disorders. *Psychological Medicine*, 10, 743-749.
- Schacter, D.L. (1992) Understanding implicit memory: A cognitive neuroscience approach. *American Psychologist*, 47, 559-569.
- Scheier, M.F., & Carver, C.S. (1992). Effects of optimism on psychological and physical well-being: Theoretical overview and empirical update. *Cognitive Therapy and Research*, 16 (2), 201-228.

- Schmidt, N.B., Joiner, T.E., Young, J., & Telch, M.J. (1995). The Schema Questionnaire: Investigation of psychometric properties and the hierarchical structure of a measure of maladaptive schemas. *Cognitive Therapy and Research*, 19, 295-321.
- Schmidt, P.J., Nieman, L.K., Grover, G.N., Muller, K.L., Merriam, G.R., & Rubinow, D.R. (1991). Lack of effect of induced menses on symptoms in women with premenstrual syndrome. *New England Journal of Medicine*, 324, 1174-1179.
- Schukit, M. (1986). Primary men alcoholics with histories of suicide attempts. *Journal for the Study of Alcoholism*, 47, 78-81
- Scott, J., Palmer, S., Paykel, E.S., Teasdale, J.D., & Hayhurst, H. (2003). Use of cognitive therapy for relapse prevention in chronic depression: Cost-effectiveness study. *British Journal of Psychiatry*, 182, 221-227.
- Segal, Z.V. (1988). Appraisal of the self-schema construct in cognitive models of depression. *Psychological Bulletin*, 103, 87-105.
- Segal, Z.V., & Dobson, K.S. (1992). Cognitive models of depression: report from a consensus conference. *Psychological Inquiry*, 3, 225-229.
- Segal, Z.V., Gemar, M., & Williams, S. (1999). Differential response to a mood challenge following successful cognitive therapy or pharmacotherapy for unipolar depression. *Journal of Abnormal Psychology*, 108, 3-10.
- Segal, Z.V., & Ingram, R.E. (1994). Mood priming and construct activation in tests of cognitive vulnerability to unipolar depression. *Clinical Psychology Review*, 14, 663-695.
- Segal, Z.V., & Shaw, B.F. (1986) Cognition in depression: A reappraisal of Coyne's and Gotlib's critique. *Cognitive Therapy and Research*, 10, 671-694.
- Segal, Z.V., & Swallow, S.R. (1994). Cognitive assessment of unipolar depression: Measuring products, processes, and structures. *Behaviour, Research, and Therapy*, 32 (1), 147-158.
- Segal, Z.V., Williams, M.J., Teasdale, J.D. (2002). *Mindfulness Based Cognitive Therapy for Depression: A New Approach to Preventing Relapse*. New York, the Guilford Press.
- Seligman, M.E.P. (1975). *Helplessness: On depression, development, and death*. San Francisco: Freeman.
- Seligman, M.E.P. (1990). Why is there so much depression today? In Ingram, R.E. (Ed.). (1990). *Contemporary Psychological Approaches to Depression: Theory, Research and Treatment*, (pp.1-9). New York: Plenum Publishing.
- Seligman, M.E.P., Castellon, C., Cacciola, J., Schulman, P., Luborsky, L., Ollove, M., & Downing, R. (1988). Explanatory style changes during cognitive therapy for unipolar depression. *Journal of Abnormal Psychology*, 93, 235-241.
- Seligman, M.E.P., Semmel, A., & von Baeyer, C. (1979). Depressive attributional style. *Journal of Abnormal Psychology*, 88, 242-248.

- Shapiro, D.A., Barkham, M., Rees, A., Hardy, G.E., Reynolds, S., & Startup, M. (1994). Effects of treatment duration and severity of depression on the effectiveness of cognitive-behavioral and *psychodynamic-interpersonal* psychotherapy. *Journal of Consulting and Clinical Psychology*, 62, 522-534.
- Shapiro, D.A., & Shapiro, D. (1982). Meta-analysis of comparative therapy outcome studies: A replication and refinement. *Psychological Bulletin*, 92, 581-604.
- Shea, T., Glass, D., Hollon, S.D., Pilkonis, P., Watkins, J., & Docherty, J. (1987) Frequency and implications of personality disorders in a sample of depressed outpatients. *Journal of Personality Disorders*, 1, 27-42.
- Shedler, J., Mayman, M., & Manis, M. (1993). The illusion of mental health. *American Psychologist*, 48, 1117-1131.
- Sheppard, L.C., & Teasdale, J.D. (2004). How does dysfunctional thinking decrease during recovery from major depression? *Journal of Abnormal Psychology*, 113 (1), 64-71.
- Shiffrin, R.M., & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, attending and a general theory. *Psychological review*, 84, 127-190.
- Simons AD, Lustman PJ, Wetzel RD, Murphy GE. (1985). Predicting response to cognitive therapy of depression: the role of learned resourcefulness. *Cognitive Therapy and Research*, 9(1), 79-89.
- Simons, A.D., Murphy, G.E., Levine, J.L., & Wetzel, R.D. (1986). Cognitive therapy and pharmacotherapy for depression: Sustained improvement over one year. *Archives of general Psychiatry*, 43, 43-48.
- Skeat, W.W. (1993). *The concise dictionary of English etymology*. Wordsworth Editions Ltd. Ware.
- Slapp, J.W. (2000). *Neuroscience and models of the mind: A response to Dr. Yovell's paper*. Retrieved April 22, 2001, from, <http://neuro-psa.com/slapp.htm>.
- Smith, E.E. (1989). *Concepts and Induction*. In M.I. Posner (Ed.), *The foundations of cognitive science* (pp.501-526). Cambridge: MIT.
- Smith, M., Glass, G., & Miller, T. (1980). *The benefits of psychotherapy*. Baltimore, MD: John Hopkins University Press.
- Smith, A. L. & Weissman (1992). *Epidemiology. Handbook of Affective Disorders*. In E.S. Paykel (Ed.), *Handbook of Affective Disorders* (pp. 111-129). New York: Guilford Press.
- Southall, D., & Roberts, J.E. (2002). Attributional style and self-esteem in vulnerability to adolescent depressive symptoms following life stress: A 14 week prospective study. *Cognitive Therapy and Research*, 26 (5), 563-579.

- Spalding, L.R., & Hardin, C.D. (1999). Unconscious unease and self-handicapping: Behavioral consequences of individual differences in implicit and explicit self-esteem. *Psychological Science, 10*, 535-539.
- Spasojevic, J., & Alloy, L.B. (2001). Rumination as a common mechanism relating depressive risk factors to depression. *Emotion, 1*, 25-37.
- Stapel, D.A., & Blanton, H. (2004). From seeing to being: Subliminal comparisons affect implicit and explicit self-evaluations. *Journal of Personality and Social Psychology, 87* (4), 468-481.
- Steer, R.A., Ball, R., Ranieri, W.F., & Beck, A.T. (1997). Further evidence for the construct validity of the Beck Depression Inventory-II with psychiatric outpatients. *Psychological Reports, 80*, 443-446.
- Steer, R.A., Ball, R., Ranieri, W.F., & Beck, A.T. (1999). Dimensions of the Beck Depression Inventory-II in clinically depressed outpatients. *Journal of Clinical Psychology, 55*, 117-128.
- Steer, R.A., Beck, A.T., & Garrison, B. (1986). Applications of the Beck Depression Inventory. In T.A. Ban & N. Sartorius (Eds.), *Assessment of depression* (pp. 123-142). New York, Springer-Verlag.
- Steer, R.A., Kumar, G., Ranieri, W.F., & Beck, A.T. (1998). Use of the Beck Depression Inventory-II with psychiatric outpatients. *Journal of Psychopathology & Behavioural Assessment, 20*, 127-137.
- Steer, R.A., Rissmiller, D.J., & Beck, A.T. (2000). Use of the Beck Depression Inventory-II with depressed geriatric inpatients. *Behavior Research & Therapy, 38*, 311-318.
- Stein, D.J. (1992) Schemas in the Cognitive and Clinical Sciences: An Integrative Construct. *Journal of Psychotherapy Integration, 2*, 45-63.
- Stern, D.N. (1985). The interpersonal world of the infant. New York: Basic Books.
- Stiles, W.B., Shapiro, D.A., & Elliot, R. (1990). Are all psychotherapies equivalent? *American Psychologist, 41*, 165-180.
- Stokes, P., & Mass, J.B. (1987). Biogenic amine and metabolite levels in depressed patients with high versus normal hypothalamic-pituitary-adrenocortical activity. *American Journal of Psychiatry, 144*, 868-872.
- Stopa, L., Thorne, P., Waters, A., & Preston, J. (2001). Are the short and long forms of young schema questionnaire comparable and how well does each version predict psychopathology scores? *Journal of Cognitive Psychotherapy, 15*, 253-272
- Stroop, J.R. (1935). Studies of interference in the serial verbal reactions. *Journal of Experimental Psychology, 18*, 643-662.

- Sturt, E., Kumakura, N., & Der, G. (1984). How depressing life is: Life-long morbidity risk for affective disorder in the general population. *Journal of Affective Disorders*, 7, 109-122.
- Swann, W. B., Jr. (1990). To be adored or to be known? The interplay of self-enhancement and self-verification. In E. T. Higgins & R. M. Sorrentino (Eds.), *Handbook of motivation and cognition: Foundations of social behavior* (Vol. 2, pp. 408-448). New York: Guilford Press.
- Taft, M. (1985). The decoding of words in lexical access: A review of the morphological approach. In D. Besner T.G. Waller, & G.E. MacKinnon (Eds.), *Reading Research: Advances in Theory and Practice*, (Vol. 5, pp. 83-124). New York: Academic Press.
- Tanaka-Matsumi, J., & Kameoka, V.A. (1986). Reliabilities and concurrent validities of popular self-report measures of depression, anxiety, and social desirability. *Journal of Consulting and Clinical Psychology*, 54, 328-333.
- Tanner, R.J., Stopa, L., & De Houwer, J. (in press). Implicit views of the self in social anxiety. *Behavior, Research, and Therapy*.
- Taylor, S.E., & Brown, J. (1988). Illusion and well-being: A social psychological perspective on mental health. *Psychological Bulletin*, 103, 193-210.
- Taylor, S.E., & Crocker, J. (1981). Schematic bases of social information processing. In E.T. Higgins, P. Herman, & M.P. Zanna (Eds.), *The Ontario Symposium in Personality and Social Psychology* (Vol.1, pp.81-134). Hillsdale, NJ: Erlbaum.
- Taylor, L., & Ingram, R.E. (1999). Cognitive reactivity and depressotypic information processing in the children of depressed mothers. *Journal of Abnormal Psychology*, 108, 202-210.
- Teasdale, J.D., Barnard, P.J. (1993). *Affect Cognition and Change*. Hove: Lawrence Erlbaum Associates Ltd.
- Teasdale, J.D., & Dent, J. (1987). Cognitive vulnerability to depression: An investigation of two hypotheses. *British Journal of Clinical Psychology*, 26, 113-126.
- Teasdale, J.D., Moore, R.G., Hayhurst, H., Pope, M., Williams, S., & Segal, Z.V. (2002). Metacognitive awareness and prevention of relapse in depression: Empirical evidence. *Journal of Consulting and Clinical Psychology*, 70, 275-287.
- Teasdale, J.D., Scott, J., Moore, R.G., Hayhurst, H., Pope, M., & Paykel, E.S. (2001). How does cognitive therapy prevent relapse in residual depression? Evidence from a controlled trial. *Journal of Consulting and Clinical Psychology*, 69, 347-357.
- Teasdale, J.D., Segal, Z.V., & Williams, J.M.G. (1995). How does cognitive therapy prevent relapse and why should attentional control (mindfulness) training help? *Behaviour Research and Therapy*, 33, 25-39.

- Teasdale, J.D., Segal, Z.V., Williams, J.M.G., Ridgeway, V.A., Soulsby, J.M., & Lau, M.A. (2000). Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy. *Journal of Consulting and Clinical Psychology*, 68, 615-623.
- Tennant, C., Smith, L.A., Bebbington, P., & Hurry, J. (1980). Parental death in childhood and risk of adult depressive disorders: A review. *Psychological Medicine*, 10, 289-299.
- Thase, M.E. (1990). Major depression in adulthood. *Handbook of Child and Adult Psychopathology: A longitudinal perspective*. New York: Pergammon Press.
- Thase, M.E. (2002). *Antidepressant effects: The suit may be small, but the fabric is real*. *Prevention & Treatment*, 5, Article 32. Retrieved August 24, 2003, from, <http://www.journals.apa.org/prevention/volume5/pre0050032c.html>
- Thase, M.E., Simons, A.D., Mcgeary, J., Cahalane, J.F., Hughes, C., Harden, T., & Friedman, E. (1992). Relapse after cognitive behaviour therapy of depression: Potential implications for longer courses of treatment. *American Journal of Psychiatry*, 149, 1046-1052.
- Townsend, J.T., & Ashby, F.G. (1983). *The stochastic modeling of elementary psychological processes*. New York: Cambridge University Press.
- Tracy, A.B. (1994). *Prozac: Panacea or Pandora?* Cassia Publications, Salt Lake City, USA.
- Trimble, M.R. (1996). *Biological Psychiatry*. New York. John Wiley & Sons Ltd.
- Van Lehn, K. (1989). Problem solving and cognitive skill acquisition. In M.I. Posner (Ed.), *Foundations of Cognitive Science* (pp.527-580). Cambridge: MIT Press.
- Vazquez, C. (1987). Judgment of contingency: cognitive biases in depressed and non-depressed subjects. *Journal of Personality and Social Psychology*, 52, 419-431.
- Venarde, D.F. (1999). Medication and meaning: Psychotherapy patients' subjective experiences of taking selective serotonin reuptake inhibitors (SSRIs). Dissertation Abstracts International: Section B: *The Sciences and Engineering*, Vol. 60, (4-B):1874.
- Von Knorring, I. (1987). Personality traits in subtypes of alcoholics. *Journal for the Study of Alcohol*, 48, 523-527.
- Vredenburg, K., Flett, G.L., & Krames, L. (1993). Analogue versus clinical depression: A critical reappraisal. *Psychological Bulletin*, 113, 327-344.
- Watkins, J.T., & Rush, A.J. (1983). Cognitive response test. *Cognitive Therapy and Research*, 1, 425-436.
- Weissmann, A. (1979). *The Dysfunctional Attitude Scale: A validation study*. Unpublished doctoral dissertation, University of Pennsylvania.
- Weissmann, A., & Beck, A.T. (1978). *Development and validation of the Dysfunctional Attitudes Scale: a preliminary investigation*, unpublished paper, read to American Educational Research Association.

- Wells, K.B., Stewart, A., Burnam, M.A., Rogers, W., Daniels, M., Berry, S. Greenfield, S., & Ware, J. (1989). The functioning and well-being of depressed patients: Results from the Medical Outcomes Study. *Journal of the American Medical Association*, 262, 914-919.
- Wenzlaff, R.M. (1988, May). *Automatic information processing in depression*. Paper presented at the International Conference on Self-Control, Hags Head, NC.
- Williams, J.B.W. (1988). A structured interview guide for the Hamilton Depression Rating Scale. *Archives of General Psychiatry*, 45, 742-747.
- Williams, J.M.G., Nulty, D.D. (1986). Construct accessibility, depression and the emotional stroop task: Transient mood or stable structure. *Personality and Individual differences*, 7, 485-491.
- Wilson, T.D., Lindsey, S., & Schooler, T.Y. (2000). A model of dual attitudes. *Psychological Review*, 107 (1), 101-126.
- Wheeler, M. (2006). *Reconstructing the cognitive world: The next step*. MIT Press: London.
- World health organisation report (2001). Retrieved July 5, 2002, from, <http://www.int/whr/2001/main/en/media/disorders.html>
- Young, J.E. (1990). *Cognitive therapy for personality disorders: a schema focused approach*. Sarasota, FL: Professional Resource Exchange.
- Young, J.E., & Brown, G. (1994). The Young Schema Questionnaire. In J.E. Young (Ed.). *Cognitive therapy for personality disorders: A schema based approach*. 3rd Edition. Professional Resource Press, Sarasota, FL, USA.
- Zemore, R., Fischer, D.G., Garrett, L.S., & Miller, C. (1990). The Depression Proneness Rating Scale: Reliability, validity, and factor structure. *Current Psychology: Research and reviews*, 9, 255-263.
- Zis, A.P., & Goodwin, F.K. (1979). Major affective disorder as a recurrent illness: a critical review. *Archives of General Psychiatry*, 36, 835-839.
- Zuckerman, M. (1984). Sensation-seeking: a comparative approach to a human trait. *Behavioural Brain Science*, 7, 413-471.
- Zurroff, D.C., Mongrain, M., & Santor, D.A. (2004). Conceptualizing and measuring personality and vulnerability to depression: Comment on Coyne and Whiffen (1995). *Psychological Bulletin*, 130, 489-511.
- Zurroff, D.C., Pilkonis, P.A., Blatt, S.J., Sainslow, C.A., Biondi, C.M. (1999). Vulnerability to depression: Re-examining state dependence and relative stability. *Journal of Abnormal Psychology*, 108, 76-89.

Appendix I: An overview of depression

Introduction

This appendix chapter is intended provide the reader with a general overview of the syndrome of depression and the non-cognitive models of depression. Depression is a very complex disorder with much disagreement amongst depression researchers regarding what constitutes the disorder and what causes the disorder (James, 1998). Fortunately, there are aspects of depressive disorders that can be routinely identified and for the most part agreed upon by psychopathologists. There is also compelling research that implicates factors other than cognitions in the causation of depression. However, many of the non-cognitive models fall short in providing both a comprehensive and valid theory of depression and addressing the issue of treatment of depression. This chapter will give an overview of the research findings on the phenomenology, epidemiology, course, risk factors, and cross-cultural factors implicated in the etiology and maintenance of depression. Following this, a discussion of the biological, behavioural, and psychoanalytical models of depression will focus on the merits and weaknesses of these models. This will involve firstly the neurotransmitter theories of depression. Following this, a consideration of the genetic influence on depression will be discussed. The influence of hormonal abnormalities in depression is the next topic and deals with the complex interaction between neurotransmitters and hormones. This will then lead us to a review of two well-known psychological models of depression: the psychoanalytic and the behavioural models of depression.

Phenomenology of depression

Depression is defined by DSM-IV (Diagnostic and Statistical Manual of Mental Disorders, 1994) as a common yet serious debilitating disorder. There are a variety of different sub-types of depression that are recognised. The first, Major Depressive Disorder (MDD), can present itself as a single episode or as a recurrent disorder. Approximately 50% of individuals who have one episode of depression will have a subsequent episode. An episode of major depression is defined as a period of two weeks during which there is either a depressed mood present or a loss of interest or pleasure in most activities. As sad or low moods are very common in people in general, at least four additional symptoms must be present for an individual to meet the diagnostic criteria for major depression. These additional four symptoms include changes in appetite, weight, sleep, and psychomotor activities which include: decreased energy, feelings of worthlessness or guilt, difficulty thinking, concentrating, or making plans, recurrent thoughts of death or suicidal ideation, plans, or attempts at suicide.

The second subtype of depression, dysthymia, is a less intense, but more chronic form of depression. DSM-IV describes dysthymia as a pervasive negative mood or lack of interest or pleasure in most activities that occurs for most of the day, for most days. Additional diagnostic criteria include two or more symptoms such as sleep or appetite disturbance, and problems with concentration, libido, and energy. To qualify for a diagnosis of dysthymia an individual must have experienced no more than two months of symptom relief over a 2-year period.

A variety of other subtypes of depression have been proposed by DSM-IV. Atypical depression is characterised by two atypical symptoms, overeating and over sleeping, as well as a long-standing pattern of interpersonal rejection. Melancholia is a subtype of depression that requires three or more of the following symptoms: a distinct quality of negative mood that differs from intense sadness, symptoms that worsen in the morning as compared to evening, early morning awakenings, marked psychomotor retardation or agitation, significant anorexia or weight loss, and excessive or inappropriate guilt. The psychotic subtype of depression requires additional psychotic symptomatology (e.g. hallucinations, delusions). Seasonal affective disorder is a subtype of depression with a regular temporal relationship with a particular time of the year, typically during the winter months. These subtypes of depression generally show different treatment responses (DSM-IV, 1994; Gleitman, 1994).

One final point regarding subtypes of depression must refer to the distinction between bipolar and unipolar depression. Depressive disorders that occur with manic phases (bipolar depression) and without manic periods (unipolar depression) should be viewed as two distinct disorders. Manic periods in bipolar depression involve elation or excessive irritability, heightened activity and energy levels, increased self-esteem, racing thoughts, distractibility, impulsive behaviour, and a decreased need for sleep. In some individuals, depression is intermittent with periods of mania and is then classified as bipolar disorder. Bipolar depression seems to be strongly determined by genetic factors. Therefore, bipolar depression is clearly distinguishable from unipolar major depression and should be considered an altogether different disorder (Gleitman, 1994). For the purposes of this thesis, Major Depressive Disorder is the type of depression that is focused upon. However, it is important to recognise that different sub-types of depression do exist.

There is considerable data to support the distinction between depression and other diagnoses. For example, depression can be clearly distinguished from disorders such as panic disorder or schizophrenia. There are, however, problems with aspects of the diagnostic criteria for depression. For example, social withdrawal is a frequent concomitant of depression but is not listed in DSM-IV diagnostic criteria. Furthermore, a clear rationale regarding the length of time required for symptoms to be present, or the number of symptoms necessary for a diagnosis of depression, does not exist. Some researchers have argued that the

diagnostic criteria for depression are too restrictive. Wells et al. (1989) found that medical patients suffering from depressive symptomatology, but who did not reach diagnostic criteria for major depression, incurred as much disability from depression as those who did meet diagnostic criteria. The diagnostic criteria for depression are likely to evolve in the future as DSM criteria have undergone, and will no doubt continue, to undergo revision (Garber & Hollon, 1991).

Epidemiology of depression

Prevalence of depression

Prevalence figures for depressive disorders vary substantially between surveys (Smith and Weissman, 1992). These differences are likely to result from differences in the methodology used, the criteria for diagnosis, the sampling method, and the study design.

In relation to other psychiatric conditions, depressive disorders are extremely common. Lifetime estimates have ranged from 4.9%- 20% for women and 2.3%-12% for men (Sturt, Kumakura, & Der, 1984; Robins, Helzer, Croughan, & Radcliff, 1981). A ten-year prospective study in Zurich (Agnst, 1986) found a lifetime prevalence of 16% for major depression, with a 6-month prevalence of 6%. The National Comorbidity Survey (Kessler et al., 1994) found a lifetime prevalence for major depression of 17%. A recent World Health Organisation report (WHO, 2001) estimated the prevalence of depression in men at 5.8% and 9.5% in women. It is estimated that 121 million people worldwide suffer from depression. Most studies have found a twofold greater prevalence of depressive disorders in women than in men (WHO, 2001). This gender difference regarding the prevalence of depression will be discussed later.

First onset episodes of depression tend to occur mostly in early adulthood, although depression does occur in children and adolescents (Nurcombe, 1992). The available epidemiological data shows that 20% of cases of major depressive disorder occurred in individuals who were under 25 years old, and 50% of cases occurred before the age of 39 (Dryman & Eaton, 1991). Therefore, depression seems to be a problem that particularly afflicts young people.

This early onset phenomenon is relatively new and there appears to be a cohort effect for depressive disorder. For example, cohorts born in the 20th century show a higher prevalence of depression for each decade (Klerman & Weissman, 1992). Recent birth cohorts seem to be at an increased risk of developing depression. This is shown by comparisons of prevalence rates for cohorts born early in the 20th century compared to cohorts born in the middle of the 20th century. People born early in the 20th century demonstrate a prevalence rate of approximately 1%. However, people born in the middle of the 20th century have an 8-9% risk. Artifact explanations for these differences are possible (e.g., depression becoming more widely recognised by the public and medical profession than it was in the past and thus more

people seek/receive treatment), but very few appear plausible. Seligman (1990) reported that older people, when surveyed, were cautious in reporting depressive symptoms. However, they did not appear to be cautious in reporting psychotic symptoms or substance abuse problems. However, the factors creating vulnerability to depression seem to be increasingly prevalent with each decade. It has been suggested that the rising rates of depression are due to improved recognition and diagnosis of the disorder, and/or to negative environmental factors. The increasingly stressful way of life of western cultures, with input from high pressure media influences that have been argued to cultivate a feeling of failure and hopelessness, has been blamed for the rise of depression (James, 1998). However, the reasons for the rise in depression are far from clear. Nonetheless, it is clear that depression and other associated psychiatric and medical complications are becoming more prevalent in this period of history (Lynch, 2004).

Course of depression

Depression is either a chronic or an episodic and recurrent disorder for a large proportion of patients. A review of long-term studies showed that approximately 34% of people have a single episode of a depressive disorder (Zis & Goodwin, 1979). An untreated depressive episode usually lasts between 6 and 13 months (Kaplan & Sadock, 1995). Other estimates have suggested that patients with an untreated depressive episode can remain symptomatic for as long as 24 months (Goodwin & Jaminson, 1990). In two-thirds of cases of untreated depression, symptoms remit and functioning returns to premorbid level. In the remainder of cases, the depressive episode may last for more than two years (5-10%) or recovery between episodes may only be partial (20-25%). Around 25% of individuals who have recurrent depression may develop chronic dysthymia. Furthermore, relapse rates in depression are high, and for those who do relapse there is a 20% chance of developing chronic depression or dysthymia (Kaplan & Sadock, 1995).

From the perspective of treated depression, the Zurich study (Angst, 1986) investigated 173 unipolar depressed patients admitted to a psychiatric institution. These patients spent approximately 20% of their time experiencing depressive symptomatology, and experienced an average of four depressive episodes with 40% suffering from 1-3 episodes. 25% experienced 6 or more episodes. The median length of treated episodes was 23 weeks.

Piccinelli & Wilkinson (1994), who examined 16 studies with follow-up periods of 6 months to 10 years looking at the outcome of depression, estimated that 64% of people recovered from depression. Recovery was defined as a limited period where the patients did not meet the criteria for the disorder. Sustained recovery, which required patients to recover from an index episode of depression and to be well during the whole follow up period, was estimated at between 26% and 43% at one year, and between 24% and 76% at ten years or more. The percentage of patients with sustained index episodes of depression was estimated

at 15.5% at 1 year and 12% at 10 years or more. Judd (1997) argues that 8 out of 10 people who experience a major depressive episode will have at least one more during their lifetime and that this would approximate to 100% if minor or subsyndromal depression were included. This evidence supports the idea that depression is a chronic and recurrent condition. Indeed, individuals who experience multiple depressions tend to have more frequent episodes that last longer as the disorder progresses (Kaplan & Sadock, 1995).

Risk factors for depression

An understanding of risk factors in depression is important in the management of depression and its prevention. From a public health perspective, the modification of reversible risk factors has the potential to reduce the incidence and prevalence of depression (WHO, 2001). The association of risk factors with depression does not imply that any given factor has a causal role in the development of depression. However, Mrazek & Haggerty (1994) list the following points as possible indications that risk factors may play a contributory role: (i) there is a statistical association between the risk factors and the incidence/prevalence of the disorder; (ii) the risk predates the disorder; (iii) there is an association between the strength of the risk factor and the severity of the disorder; and (iv) the process by which the factor is linked to the disorder can be described.

Several groups of childhood and familial antecedents of depression have been identified as possible risk factors for the development of depression: childhood onset of major depression and dysthymia, a family history of affective disorders, alcoholism and a social learning factor (e.g., negative reinforcement), adverse early life experience involving parental loss, sexual or physical abuse, attention-deficit disorder (Hersen & Ammerman, 1995), temperament (Thase, 1990), and the presence of certain personality or behavioural traits (Zemore et al., 1990).

Another notable risk factor for the development of depression is the fact that women seem to have a higher risk of developing depression. This ratio is approximately 2:1 (Gleitman, 1994). Although males and females develop depression at the same age, and the chronicity and recurrence rate are similar for both males and females, females may be more likely to seek help and/or be diagnosed with depression (James, 1998). Another hypothesis is that women may encounter more critical events that initiate depressive episodes (Radloff & Rae, 1979). There is clear evidence that women do in fact encounter more critical events. For example, caring for a small child, the stresses of a typical housewife role, and media pressures of body-image have been identified as critical events associated with an increased risk for women developing depression (Radloff & Rae, 1979; James, 1998). However, these differences are probably not sufficient to account for gender differences in depression. A biological hypothesis implies that women may be more vulnerable to depression due to endocrinological differences (Akiskal et al., 1987). Alternatively, changes associated with

hormones, including the menstrual cycle (Schmidt, Nieman, Grover, Muller, Merriam, & Rubinow, 1991), menopause (Greene, 1980), and the postpartum period (Cox, 1992), may predispose women to depression. There is, however, opposing evidence which suggests that hormones do not play a role in predisposition to depression. For example, O'Hara, Zekoski, Phillips, & Wright (1990) showed that there was no difference in the rates of depression for postpartum women when compared to non-child bearing women of the same age. Therefore the support for biological differences as a risk factor for increased rates of depression in women is not strong.

Rumination in response to a low mood has been investigated as a potential factor in the different prevalence rates for depression between men and women. Nolen-Hoeksema (1987) argued that women are far more likely to ruminate in response to a low mood and to focus their attention on themselves. It may be that these cognitive differences result from social background factors (e.g., the environment and critical events) or biological factors.

Cross-cultural aspects of depression

The experience of struggling with a low mood and the associated symptoms of depression appears to be very common across all cultures (Oltmanns & Emery, 1998). There are however some differences between the experience of depression in western cultures compared to non-western cultures. In some non-western cultures there seems to be a reduced frequency or absence of the psychological factors of depression, but a dominance of the somatic aspects (Oltmanns & Emery, 1998). Murphy, Wittkower, & Chance (1964) completed two surveys involving 30 countries and found that a cluster of symptoms including depressed mood, diurnal variations, insomnia, and loss of interest, were common in western cultures. However, in non-western cultures fatigue, anorexia, weight loss, and low libido were the most common cluster of symptoms to emerge. The World Health Organisation Collaborative study of Depression (Sartorius, Jablensky, Gulbinat, & Ernberg, 1980) found similar patterns of depressive disorder in western and non-western cultures (Canada, India, Iran, Japan, and Switzerland). However, they also found cultural variations in the frequency of different symptoms. Suicidal ideation was observed in 70% of the Canadian sample compared to only 40% of the Japanese sample. Therefore, cross-cultural research suggests that depressive symptomatology in western countries is more psychological while in non-western countries depressive symptomatology is more somatic. Indeed, a recent World Health Organisation Report (WHO, 2001) confirms that in developing countries, mental distress is expressed somatically rather than psychologically. These findings have implications both for the developmental and theoretical models that one uses to understand depression and for its treatment.

Non-cognitive models of depression

Neurotransmitter theories of depression

According to neurotransmitter theories of depression, the basic biological causes of depression are strongly linked to abnormalities in the functioning of certain key neurotransmitters. Contemporary biochemical theories of depression have focused on the monoamines: norepinephrine and serotonin. Both of these neurotransmitters are localised in the limbic system and hypothalamus. It is hypothesised that these two areas are involved with the regulation of emotion. The biological hypothesis is that depression is associated with abnormalities in one or both of these neurotransmitter systems. However, the evidence for their involvement in depression is indirect and is based on the effects of drugs on certain neurotransmitters (Trimble, 1996).

Several lines of converging evidence implicate norepinephrine and serotonin in depression. The first main finding was the result of research looking at the effect of reserpine on mood. Reserpine is an alkaloid derived from the Indian medicinal herb *rauwolfia serpentina*. Initially used as a treatment for schizophrenia in the 1950s and then later as a hypertensive agent, it was found to induce depressive symptoms in individuals. During the time that reserpine was used as a hypertensive agent, up to 15% of the people who took it became depressed. Reserpine acts on the membrane of the synaptic vesicles in the terminal buttons of the monoaminergic neurons, making the membranes leak, thus creating a situation where neurotransmitters are lost from the vesicles and are destroyed by a substance called monoamine oxidase (MAO). MAO is an enzyme that inactivates molecules of norepinephrine and serotonin, converting these molecules into biologically inactive compounds. Therefore, pharmacologically, reserpine acts to deplete presynaptic supplies of monoamines within the central nervous system. As a result, it was suggested that depression might be caused by depletion of one or more of the monoamines (Trimble, 1996).

Due to the findings that monoamines may be implicated in depression, three main types of drugs were developed and used to treat depression, each of which worked in different ways to increase levels of the monoamines. Monoamine Oxidase Inhibitors block the activity of an enzyme that can destroy norepinephrine and serotonin, and thus increase the concentration of these two neurotransmitters in the brain. The tricyclic antidepressants prevent reuptake of both neurotransmitters, while the serotonin reuptake inhibitors are selective in that they only block the reuptake of serotonin (Trimble, 1996).

Neuroscientific research investigating neurotransmitters in the brain and the long-term effects of antidepressants on postsynaptic receptors has discovered that antidepressant drugs take time to be therapeutically effective (Carlson, 1994). All classes of antidepressants take between 1-3 weeks to exert their therapeutic effects of reducing depressive symptoms. This fact does not fit with the finding that when these drugs are first taken, increases in norepinephrine or serotonin levels occur only temporarily, and after several days the neurotransmitters return to their previous levels. Therefore, an increase in neurotransmitters

cannot be the mechanism that relieves depression per se. Evidence suggests that antidepressants increase the sensitivity of the neurotransmitter's postsynaptic receptors. The time frame in which this occurs corresponds well with the course of the drugs' action on symptoms (Charney, Heninger, & Sternberg, 1984). Thus, even though levels of neurotransmitters have returned to low levels, it might be the case that these neurotransmitters are more effective because the receptors receiving them have become more sensitive (Trimble, 1996).

The neurotransmitter systems that influence mood and cognition are complex, and comprehension of their role is still at an early stage of development. Several neurotransmitter systems, acting alone or in combination, may be responsible for producing depressive symptoms. There is, however, no doubt that depression involves biochemical changes. The unresolved issue is whether physiological changes are the cause or the result of psychological changes or some kind of dynamic interaction (McNeal & Cimbolich, 1986). Indeed, recent evidence seems to implicate the enzyme (CYP2D6), produced in the liver, in the etiology of depression. This enzyme is involved in the metabolism of serotonin. It has been found that the levels of this enzyme vary from individual to individual, and thus the ability to metabolise antidepressant medication varies from individual to individual. In other words, some individuals are more effective in metabolising antidepressants than others due to the presence of this liver enzyme. This finding may explain why the same dosage of an antidepressant has a differing efficacy in the reduction of symptoms of depression in different individuals, and why some serious cases of serotonin syndrome (dangerously high brain levels of serotonin due to poor serotonin metabolism) occur (Dorne et al., 2004).

Genetics of depression

The tendency to develop depression appears to have a genetic basis (Trimble, 1996). Rosenthal (1970) found that close relatives of people who suffer from affective psychoses were ten times more at risk of developing an affective disorder compared to people with no afflicted relatives. Gershon & Nurnberger (1982) discovered that if one member of a set of monozygotic twins was affected by an affective disorder, the other twin had a 69% chance of developing an affective disorder. The rate for dizygotic twins was only 13%. Further, the concordance rate for monozygotic twins seems to be the same whether the twins lived apart or were raised together. Since 1990 there have been more recent studies investigating genetic influences on depression. Andrews, Nelson, Hunt, & Stewart (1990) found that concordance rates for major depression were low, but greater in monozygotic than dizygotic twins. McGuffin, Katz, & Rutherford (1991) found a concordance rate of 58% in monozygotic twins and 28% in dizygotic twins. Kendler, Neale, Kessler, & Heath (1992) found rates of 44% in monozygotic twins and 19% in dizygotic twins. In a follow-up to their study one year later, Kendler, Kessler, & Neale (1993) found moderate genetic influence in prevalence rates of

depression over one year, indicating stressful life-events/environmental factors may have some part to play.

Indeed, Kendler et al., (1993) found that environmental factors do play a part in the etiology of depression, but their effects seem to be transient. In other words, genetic factors predispose individuals to depression during their lifespan, whereas environmental influences lead to the onset of depression, but do not predispose an individual to depression beyond a one-year period. Further, it has not been established in genetic research whether one single gene is responsible for the development of depression. Indeed, there have been several genes posited as responsible for the development of depression (Oltmans & Emery, 1998).

It is unclear how genetics play a role in depression. However, it appears that there is some genetic component. The question, however, remains: in what way does genetic make-up influence vulnerability to depression and how does the interaction of environment and genetics influence depression?

Hormone theories of depression

Hypercortisolism is recognised as being an essential part of normal adaptation to stress (Kaplan & Sadok, 1995). Cortisol secretion is thought to counter-regulate the effects of stress. This is managed by the hypothalamic-pituitary-adrenal axis (HPA). When a threat to physical or psychological well-being is detected, the hypothalamus amplifies production of corticotrophin-releasing factor (CRF), which induces the pituitary gland to secrete adrenocorticotrophic hormone (ACTH). ACTH then instructs the adrenal gland, situated in the kidneys, to release cortisol. This mechanism prepares the body for flight or fight. Since the 1960s and 1970s, research has reported increased activity in the HPA axis in unmedicated depressed patients, as shown by elevated levels of cortisol in the urine, blood and cerebrospinal fluid (Trimble, 1996).

One of the most consistent findings regarding hormonal abnormalities and depression is that individuals with depression often have hypercortisolism (Trimble, 1996). Despite evidence that shows that only 50% of patients with depression show hypercortisolism, there exists some evidence for the role played by endocrine function abnormalities in the etiology of depression. Stokes & Mass (1987) have suggested that cortisol induces alterations in the neurotransmitter systems involved in depression. O'Toole & Johnson (1997) believe that the direction of causality is not clear as the monoamines also affect neuroendocrine activity. However, Dinan (1994) found support for the proposal that monoamine abnormalities are secondary to hypothalamic pituitary over-activity. When individuals recover from depression, cortisol function returns to normal. However, even if clinical recovery has taken place, abnormal cortisol activity can still occur in around 45-60% of patients, which could be indicative of vulnerability for a depressive relapse (Trimble, 1996).

It is therefore proposed that this hormonal mechanism that deals with stress becomes “switched on” for longer in depressed individuals and becomes a generalised stress response that escapes the usual counter regulatory restraint. The observation that depression has a genetic component means that certain genetic traits lower the threshold for development of depression. It is perhaps conceivable that the genetic features directly or indirectly diminish monoamine levels in the synapses or increase reactivity of the HPA axis to stress. The exact causal biological antecedents of depression are not clear, and issues that cloud this area are that HPA hyperactivity is present in a number of other mental disorders (Trimble, 1996).

The psychoanalytic perspective of depression

Psychoanalytic theories interpret depression as a reaction to loss. Whatever the nature of the loss (rejection by a loved one, loss of status, loss of moral support, loss of support of friends), the depressed person reacts intensely to the loss because the current situation (episode of depression) brings back embodied fears of an earlier loss that occurred in childhood. This loss, it is argued, is the loss of parental affection. The reason for the onset of depression is that an individual’s needs for affection were not satisfied in childhood. Therefore a loss in later life causes the individual to regress to his or her helpless dependent state when the original loss occurred. The depressed person’s behaviour therefore represents a cry for love, and an appeal for affection and security (Blatt, 1974).

An individual’s reaction to loss is complicated by angry feelings toward the person who has deserted him or her. The underlying assumption of psychoanalytic theory is that people who are vulnerable to depression have learned to repress their hostile feelings because they are afraid of alienating those people upon whom they depend for support. When things do go wrong, depressed individuals turn their anger inward and blame themselves. For example, a man may feel hostile towards his wife who left him for another man. His anger toward his wife arouses anxiety. He then internalises his anger: he is not angry, rather it is his wife who is angry with him. He assumes that his wife had a reason for leaving him. The man therefore regards himself as unlovable and worthless (Davidson & Neale, 1986).

Psychoanalytic theories propose that a depressed individual’s low self-esteem and feelings of worthlessness stem from a childlike need for approval. A child’s self-esteem depends on the affection and approval of caregivers (parents). The self-esteem of a person vulnerable to depression depends primarily on external sources. When approval and support from others is not forthcoming, the individual may be thrown into a state of depression (Brewin, 1988). However, this theory can be criticised from a behavioural point of view (see behavioural perspective below). It could be argued that theoretically, as a child grows up, feelings of worth also should also be derived from the individual’s own accomplishments and effectiveness (Seligman, 1975).

Psychoanalytic theories of depression focus on loss, over-dependence on approval from others, and internalisation of anger. They are reasonable hypotheses for behaviours exhibited by depressed individuals. They are however difficult to prove or refute. Some studies have indicated that individuals who are vulnerable to depression are more likely than normal to have lost a parent early in life (Brown & Harris, 1978). But parental loss is found in people who suffer from other mental disorders, and most people who suffer from parental loss do not develop emotional problems in adulthood (Tennant, Smith, Bebbington, & Hurry 1980). To deal with this theoretical problem, psychoanalytic theorists have invoked the concept of “symbolic loss”, whereby the loss is shown as withdrawal of love or lack of affection from a parent, which seems more consistent with clinical practice (Davidson & Neale, 1986). In essence, however, little research has been generated by psychoanalytic formulations of depression. The little information that has been gleaned from research does not support the theory (Beck, 1967). Psychoanalytic ideas, however, have found their way into more recent theorising, notably irrational self-statements of need for love and approval, and Beck’s (1967) idea that early life negative experiences may impinge on well-being later in life.

A behavioural perspective of depression

Learning theorists assume that a lack of reinforcement plays a major role in depression. The inactivity of the depressed individual and feelings of sadness are due to a low rate of positive reinforcement and/or a high rate of unpleasant experiences (Lewinsohn et al., 1980). Many of the events that precipitate depression (e.g. loss of a job or death of a loved one) reduce accustomed reinforcement. Further, individuals who are vulnerable to depression may lack social skills either to attract positive reinforcement or the ability to cope with aversive events. When people become depressed and inactive, their main source of reinforcement is the sympathy that they receive from relatives and friends. This attention may at first reinforce behaviours that are maladaptive (crying, complaining). However, it is tiresome to be with someone who is depressed and the depressed person’s behaviour may push people away, which results in less reinforcement of positive experiences, increasing the depressed person’s social isolation and feelings of sadness.

A major behavioural theory of vulnerability to depression is the learned helplessness theory of depression, which has its roots in learning theory (Seligman, 1975). Seligman observed that when animals were unable to control negative stimuli (electric shocks) they often developed behaviour consistent with depressive behaviour in humans. Further, dogs that were unable to control the electric shocks became helpless and did not try to escape them, even when escape was available. Seligman observed the similarity between animals exposed to helplessness conditions and people with depressive symptoms. Seligman argued that people suffering from depression show very little curiosity and spend a great deal of time

doing nothing. Often they do not take action that could improve their circumstances because they cannot be bothered or because they see any action as pointless. Thus, the helplessness theory of depression focuses on how depression prone people have an expectation that they are helpless in controlling aversive outcomes, and behave in ways that are in accordance with their expectations. The learned helplessness theory of depression has generated much interest but, due to certain shortcomings, a reformulated theory was put forward by Abramson, Seligman, & Teasdale (1978), as it had become apparent there were certain oversights in the original theory. It was recognised that many of life's misfortunes are beyond the control of individuals, but they do not sadden everyone to the extent that they develop a negative self-image and depression. If people regard themselves as helpless, how can they blame themselves? In other words, why do depressed people have such a low self-esteem if events are out with their control? Recognition of events being uncontrollable should not have an effect on self-esteem as such, but may cause a low mood in general due to the poor circumstances. The revised theory focuses on individuals' attributions about the causes of negative events. Therefore attributional style was posited as the key causal factor in depression. Specifically, making global, stable, internal attributions for negative events and, conversely, making specific, unstable, external attributions for positive events were presumed to lead to depression. As one can observe, the learned helplessness theory of depression is somewhat of a halfway house between learning theory and cognitive theory. The role of cognitions is assigned a crucial role in this theory.

The learned helplessness theory of depression has received support in research. For example, when individuals are depressed, cross-sectional research shows that they do indeed make the kinds of attributions posited by the theory. Also, research supports the notion that the tendency to make these kinds of attributions precedes negative mood reactions by university students in response to negative events (Gleitman, 1994). Two prospective studies found that college students who attributed negative achievement events (e.g., a low grade in an exam) to stable and global causes experienced more enduring depressive mood in response to low midterm grades than did students without this attributional style (Metalsky, Joiner, Hardin, & Abramson, 1987).

Although the learned helplessness theory of depression has received wide support, some issues still need to be addressed. The first is which type of depression is being modeled by this theory? It could be that this model is only applicable to reactive depression and/or resembles the depressive phase of someone who suffers from bi-polar disorder rather than unipolar depression (Davidson & Neale, 1986). Secondly, is the learned helplessness model only applicable to depression? Lavelle et al. (1979) found that the model could also be applied to anxiety. However, negative reinforcement and aversive events do affect

neurochemicals (e.g., low serotonin/norepinephrine). Therefore, the model has some aspects that do correlate with other theories of depression (James, 1998)

Summary

Major depressive disorder is recognised as a serious, recurrent and debilitating disorder. It is distinguishable from other psychiatric disorders by a recognised cluster of signs and symptoms that most clinicians largely agree upon. In relation to other disorders, depression is extremely prevalent, is increasing in prevalence at this time in history, and is becoming more frequently associated with an earlier onset.

Evidence supports the idea that depression is a recurrent and chronic condition in approximately 80% of individuals. Risk factors associated with the development of depression include adverse life experiences, a family history of depression, cognitive vulnerability and dysfunctional ruminative thought patterns, and being female. Cross-cultural research shows that depressive symptomatology in western countries is more psychological in nature, while in non-western countries depressive symptomatology is more somatic but that, overall, the experience of depression is common to all cultures.

Neurotransmitter theories of depression have provided much promise in linking abnormalities of serotonin and other neurotransmitters with the development of depression. However, it is far from clear how much one neurotransmitter is implicated in depression or how much the dynamic interaction of several neurotransmitters is responsible for susceptibility to the disorder. Furthermore, new research has implicated serotonin metabolism in the etiology of depression. It is also unclear how much genetics play a role and how much adverse environmental influences interact with genetic predisposition to depression. Hypercortisolism or increased activity in the HPA axis has also been connected with the development of depression. However, the exact role of a hyperactive HPA is unclear as HPA hyperactivity is present in a number of other mental disorders.

Psychoanalytic theories of depression interpret the development of depression as a reaction to loss (symbolic or real). However, loss is associated with a number of other mental disorders other than depression. In essence, little research has generated or confirmed psychoanalytic formulations of depression.

The learning theorists have tried to explain depression in terms of dysfunctional positive reinforcement contingencies, which have been compelling, very successful, and helped many people in the treatment derived from the results of learning research. The main problem with a purist learning approach to depression is that it dismisses the role of personal agency on the part of an individual or the role of attributions as being a key causal factor in the development of depression. The revised learned helplessness model of depression does however acknowledge some aspect of personal agency in-built within an individual, but does confuse the issue of self-esteem and low mood associated with helplessness in a negatively

reinforcing environment. In other words, low self-esteem should not be a result of negative circumstances out of one's control, but a low mood might be. Further, the revised learned helplessness model can be generalised to other psychopathologies. There is also the problem as to which kind of depression is being modeled by the theory: reactive depression rather than other subtypes of depression.

Appendix II: Other cognitive theories that deal exclusively with cognitive products or cognitive processes in depression

Cognitive theories that deal exclusively with cognitive contents in depression

Other theories that focus on cognitive products place prime importance on the cognitions that an individual experiences and views such cognitive products as causal factors in the development of depression. One well-known theory is Albert Ellis' irrational beliefs model (Ellis, 1962). This model arose around the same time as Beck's ideas and added to the development of cognitive theories of depression in the 1960s alongside Beck's theory. Ellis' theory derives from the assumption that errors in thinking lead to depression. Ellis argues that irrational beliefs lead to psychological disorders. Depression prone individuals are hypothesised to hold very rigid standards with which they judge their life. These standards are applied to the individual's performance, the performance of others, and general events in life. The consequence of these rigid standards is that an individual may expect too much of him/herself, others, or life in general, and is likely to become disappointed, and ultimately become depressed.

Although research has found some support for Ellis' theory, there are problems with the model with regard to the measurement of irrational beliefs, which are confounded by the presence of negative mood. In other words, irrational beliefs seem to be present only during the depressed episode. However, because rigid beliefs are proposed by Ellis to be causal and precipitating factors for depression, and appear only when someone is depressed, they may only constitute symptoms of depression rather than causal factors. Indeed, rigid beliefs have been shown to subside in remission while other factors, argued to be linked to the precipitation of depression, have been implicated. Numerous depression researchers have argued that it is biased negative information processing, or abnormal cognitive processes, that may be the key causal or precipitating factors for depression onset (e.g., Teasdale & Branard, 1993; Segal et al, 1999; Ingram et al., 1998; Segal, 1988; Hedlund & Rude, 1995; Rude et al., 2001; Gemer et al., 2001). There is some confusion over whether Ellis' model overlaps with the concepts of cognitive errors, dysfunctional beliefs, or faulty cognitive processes that constitute the key concepts in Beck's model. Indeed, in Beck's model (1967, 1976), dysfunctional assumptions or beliefs (e.g., "I will be a failure if I do not gain top marks in the exam"), are proposed as the factors that cause vulnerability to depression and, once activated, give rise to negative cognitive products, namely negative automatic thoughts such as "I am a failure". In the hierarchical system of Beck's model, the triad consists of cognitive products that are mediated by faulty cognitive processes which ultimately are the result of the operation of an underlying cognitive structure or schema that guides information processing at an implicit level (Hollon & Kriss, 1984).

The main weakness of a theory or empirical research study that only focuses on the products of cognition in depression, is a lack of etiological specificity or explanatory account of depression. Many measures of depressive products (e.g., negative automatic thoughts) are as a result of a depressive episode, and return to normal levels once the episode of depression has remitted (Hagga et al., 1991). Many studies using psychometric measures of depressive cognition in the treatment outcomes of depression falsely attribute treatment success to reduction in self-reported depressive cognition, assuming the products of cognition are integral to the onset, or even maintenance of the depression. In essence, products of cognition in depression fail to yield any insight into the developmental origins of depression and only provide a descriptive account of depression (Rector et al., 1998). This is why Beck's model of depression (See Chapter 1), which incorporates the three cognitive variables (products, processes, and structure), is more useful for explaining etiology as well as providing a comprehensive descriptive account of depression and explaining its persistence.

Cognitive theories focusing exclusively on cognitive processes in depression

Research has also found that self-focused attention, representing aspects of Beck's idea of faulty cognitive processes or errors (e.g., magnification, dichotomous thinking, personalisation etc), is an integral part of depression (Musson & Alloy, 1988). There are two theoretical accounts of self-focused attention in depression, the first being Lewinsohn, Hobermanm, Teri, & Hautzinger's (1985) theory. Lewinsohn et al. argue that disruptions in the life of individuals who are vulnerable to depression (e.g., marriage breakdown) affect self-concept or self-esteem (one's conscious perception of oneself) and initiate a heightened sense of self-awareness. The result of this process is that there may be a reduction in an individual's behavioural and social competencies, as one may become overly self-focused. This self-focus can be to the detriment of other issues that otherwise need to be focused upon (e.g., social relationships). These competencies are needed if one is to improve the negative life events that precipitated the depressive episode. Jacobson & Anderson (1982) found that depressed individuals talk about themselves more in conversations. This is interpreted as a form of self-preoccupation that prevents depressed individuals from being able to engage in effective interpersonal interaction, and this leads to a disruption in interpersonal relationships as a whole. This in turn contributes to the formation of a negative self-concept, which leads to greater self-focused attention (Jacobson & Anderson, 1982).

A more detailed theory of self-focused attention was proposed by Pyszczynski & Greenberg (1992b), who also argue that a fundamental part of a depressed person's experience is a dysfunction in self-focused attention. They assert that when an individual experiences some

kind of a negative disruptive life event (e.g., romantic rejection) that is relevant to his or her conceptions of self-worth, self-focus increases. This triggers a self-evaluation process.

Depression prone people engage more in this type of mental behaviour, which in turn affects task performance and self-esteem, and triggers the onset of symptoms of depression. If individuals experience subsequent disruptive life events, this leads them to further increase their self-focus. Conversely, for positive events individuals reduce their self-focus as self-worth has not been challenged, but also because positive conceptions of self-worth are less pronounced in depressed prone individuals compared to non-prone individuals. Therefore, depressed people have limited cognitive access to the positive effects of positive events.

Evidence for the exclusivity of self-focused attention in depression has been inconclusive as data suggests that self-focused attention is not unique to depression. Self-focus is present in generalised anxiety disorder and social phobia (Ingram, 1990). Therefore, self-focus perhaps can only be considered as a generalised factor in psychopathology. It may be that the comprehension of other variables (e.g., the content of the self-focus) is what differentiates self-focus in different psychopathologies. In other words, in depression there may be aspects of the self under scrutiny when engaging in self-focus, such as the self as being depressed, while in anxiety the self in danger may be the content of self-focus (Ingram, 1990). Further, the amount of time engrossed in self-focus or the behaviours associated with self-focus (e.g., inability to distract oneself from a negative self-focus with alternative behaviours) may be crucial in differentiating between distinct psychopathologies (Nolen-Hoeksema, Parker, & Larson, 1994).

A cognitive model that addresses these issues and which gives pathological self-focus an important role in depression, whilst also taking into account the amount of time associated with self-focus, content of self-focus, and behaviours associated with self-focus, is Nolen-Hoeksema's (1987) ruminative response style model. This model proposes that individuals who are vulnerable to depression are more liable to maintain a depressed mood and are different in the way they self-focus, when compared with those who do not maintain a low mood or who are not vulnerable to depression. In other words, individuals who are in a low mood and think about the low mood, and the thoughts associated with the low mood, are more likely to maintain their depressive mood and spiral into clinical depression. Furthermore, these individuals are less likely to try to distract themselves from their low mood. However, individuals who experience the same low mood, but think less about the thoughts associated with the low mood, and who distract themselves, are less likely to maintain the depressed mood or develop depression. Therefore the ruminative response and behaviours associated with the low mood are hypothesised to be causative factors in the maintenance and severity of low moods leading to depression. This theory is similar to the

aforementioned theories of self-focused attention in that pathological self-focus is given an important role in depression. The ruminative theory, however, identifies the importance of the self-focus on depressive symptoms associated with the low mood and behaviours that occur during the low mood and time spent in the low mood. Other theories implicating pathological self-focus in depression are more generalised as the focus of internal attention is identified as only the self, but this can be attributed to other psychopathologies (e.g., Jacobson and Anderson, 1982; Pyszczynski & Greenberg, 1992b).

Research has supported the notion that depressed people ruminate more than non-depressed people. However, it is difficult to ascertain if a negative mood precipitates rumination, or rumination precipitates the maintenance of a negative mood (Ingram et al., 1998). Research that focuses on faulty cognitive processing or abnormal processes like rumination still fails to articulate and account for the etiology of depression. Vulnerability to depression, therefore, cannot be measured in the absence of a challenging (negative) event being present and the subsequent activation of the ruminative style. It is possible that such cognitive processes are traits in people vulnerable to depression, but that these processes are only evident in a depressed mood, and are mediated by structural differences (the schema) as articulated by Beck's model (1967). However, the notion of a schema being implicated in the etiology of depression is one of the most problematic facets of depression research.

Appendix III

In the following order¹:

Beck Depression Inventory (BDI II; Beck et al., 1979). Pages 235-236

Beck Anxiety Inventory (BAI; Beck, 1980). Page 237

Young Schema Questionnaire (short-form) (YSQ; Young & Brown, 1990). Pages 238-241.

Automatic thoughts Questionnaire (ATQ; Hollon & Kendall, 1980). Page 242.

Dysfunctional Attitudes Scale (Form A)-(DAS-A; Weissman & Beck, 1978). Pages 243-247.

Dysfunctional Attitudes Scale (Form B)- (DAS-B; Weissman & Beck, 1978). Pages 248-252.

Evaluative Beliefs Scale (EBS; Chadwick et al., 1999). Page 253.

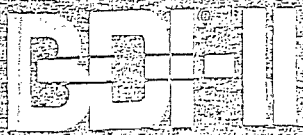
Depression Proneness Rating Scale (DPRS; Zemore et al., 1990). Pages 254-255.

The Ruminative Responses subscale of the Response to Depression Questionnaire (RDQ; Nolen-Hoeksema & Morrow, 1991). Page 256.

Schedule Clinical Interview for DSM-IV Axis I Disorders (SCID I; First et al., 1996). Pages 257-261.

Visual Analogue Scale (VAS; Grossberg & Grant, 1978). Page 262.

¹ Consent was given to the Author to reproduce these questionnaires for the purposes of this thesis by their respective Author's.



Date: _____

Name: _____ Marital Status: _____ Age: _____ Sex: _____
Occupation: _____ Education: _____

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the one statement in each group that best describes the way you have been feeling during the past two weeks, including today. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness

- 0 I do not feel sad.
- 1 I feel sad much of the time.
- 2 I am sad all the time.
- 3 I am so sad or unhappy that I can't stand it.

2. Pessimism

- 0 I am not discouraged about my future.
- 1 I feel more discouraged about my future than I used to be.
- 2 I do not expect things to work out for me.
- 3 I feel my future is hopeless and will only get worse.

3. Past Failure

- 0 I do not feel like a failure.
- 1 I have failed more than I should have.
- 2 As I look back, I see a lot of failures.
- 3 I feel I am a total failure as a person.

4. Loss of Pleasure

- 0 I get as much pleasure as I ever did from the things I enjoy.
- 1 I don't enjoy things as much as I used to.
- 2 I get very little pleasure from the things I used to enjoy.
- 3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings

- 0 I don't feel particularly guilty.
- 1 I feel guilty over many things I have done or should have done.
- 2 I feel quite guilty most of the time.
- 3 I feel guilty all of the time.

6. Punishment Feelings

- 0 I don't feel I am being punished.
- 1 I feel I may be punished.
- 2 I expect to be punished.
- 3 I feel I am being punished.

7. Self-Dislike

- 0 I feel the same about myself as ever.
- 1 I have lost confidence in myself.
- 2 I am disappointed in myself.
- 3 I dislike myself.

8. Self-Criticalness

- 0 I don't criticize or blame myself more than usual.
- 1 I am more critical of myself than I used to be.
- 2 I criticize myself for all of my faults.
- 3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

- 0 I don't have any thoughts of killing myself.
- 1 I have thoughts of killing myself, but I would not carry them out.
- 2 I would like to kill myself.
- 3 I would kill myself if I had the chance.

10. Crying

- 0 I don't cry anymore than I used to.
- 1 I cry more than I used to.
- 2 I cry over every little thing.
- 3 I feel like crying, but I can't.

Subtotal Page 1

Continued on Back

11. Agitation

- 0 I am no more restless or wound up than usual.
- 1 I feel more restless or wound up than usual.
- 2 I am so restless or agitated that it's hard to stay still.
- 3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

- 0 I have not lost interest in other people or activities.
- 1 I am less interested in other people or things than before.
- 2 I have lost most of my interest in other people or things.
- 3 It's hard to get interested in anything.

13. Indecisiveness

- 0 I make decisions about as well as ever.
- 1 I find it more difficult to make decisions than usual.
- 2 I have much greater difficulty in making decisions than I used to.
- 3 I have trouble making any decisions.

14. Worthlessness

- 0 I do not feel I am worthless.
- 1 I don't consider myself as worthwhile and useful as I used to.
- 2 I feel more worthless as compared to other people.
- 3 I feel utterly worthless.

15. Loss of Energy

- 0 I have as much energy as ever.
- 1 I have less energy than I used to have.
- 2 I don't have enough energy to do very much.
- 3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

- 0 I have not experienced any change in my sleeping pattern.
- 1a I sleep somewhat more than usual.
- 1b I sleep somewhat less than usual.
- 2a I sleep a lot more than usual.
- 2b I sleep a lot less than usual.
- 3a I sleep most of the day.
- 3b I wake up 1-2 hours early and can't get back to sleep.

17. Irritability

- 0 I am no more irritable than usual.
- 1 I am more irritable than usual.
- 2 I am much more irritable than usual.
- 3 I am irritable all the time.

18. Changes in Appetite

- 0 I have not experienced any change in my appetite.
- 1a My appetite is somewhat less than usual.
- 1b My appetite is somewhat greater than usual.
- 2a My appetite is much less than before.
- 2b My appetite is much greater than usual.
- 3a I have no appetite at all.
- 3b I crave food all the time.

19. Concentration Difficulty

- 0 I can concentrate as well as ever.
- 1 I can't concentrate as well as usual.
- 2 It's hard to keep my mind on anything for very long.
- 3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

- 0 I am no more tired or fatigued than usual.
- 1 I get more tired or fatigued more easily than usual.
- 2 I am too tired or fatigued to do a lot of the things I used to do.
- 3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

- 0 I have not noticed any recent change in my interest in sex.
- 1 I am less interested in sex than I used to be.
- 2 I am much less interested in sex now.
- 3 I have lost interest in sex completely.

Subtotal Page 2

Subtotal Page 1

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Total Score



NAME _____

DATE _____

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

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

THE PSYCHOLOGICAL CORPORATION
HARCOURT BRACE JOVANOVICH, INC.

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13 14 15 16 17 A B C D E

YSQ - S1

Name _____ Date _____

INSTRUCTIONS:

Listed below are statements that a person might use to describe himself or herself. Please read each statement and decide how well it describes you. When there you are not sure, base your answer on what you emotionally feel, not on what you think to be true. Choose the **highest rating from 1 to 6** that describes you and write the number in the space before the statement.

RATING SCALE:

- 1 = Completely untrue of me
- 2 = Mostly untrue of me
- 3 = Slightly more true than untrue
- 4 = Moderately true of me
- 5 = Mostly true of me
- 6 = Describes me perfectly

1. _____ Most of the time, I haven't had someone to nurture me, share him/herself with me, or care deeply about everything that happens to me.
2. _____ In general, people have not been there to give me warmth, holding, and affection.
3. _____ For much of my life, I haven't felt that I am special to someone.
4. _____ For the most part, I have not had someone who really listens to me, understands me, or is tuned into my true needs and feelings.
5. _____ I have rarely had a strong person to give me sound advice or direction when I'm not sure what to do.
- *ed
6. _____ I find myself clinging to people I'm close to, because I'm afraid they'll leave me.
7. _____ I need other people so much that I worry about losing them.
8. _____ I worry that people I feel close to will leave me or abandon me.

9. _____ When I feel someone I care for pulling away from me, I get desperate.

10. _____ Sometimes I am so worried about people leaving me that I drive them away.

*ab

11. _____ I feel that people will take advantage of me.

12. _____ I feel that I cannot let my guard down in the presence of other people, or else they will intentionally hurt me.

13. _____ It is only a matter of time before someone betrays me.

14. _____ I am quite suspicious of other people's motives.

15. _____ I'm usually on the lookout for people's ulterior motives.

*ma

16. _____ I don't fit in.

17. _____ I'm fundamentally different from other people.

18. _____ I don't belong; I'm a loner.

19. _____ I feel alienated from other people.

20. _____ I always feel on the outside of groups.

*si

21. _____ No man/woman I desire could love me once he/she saw my defects.

22. _____ No one I desire would want to stay close to me if he/she knew the real me.

23. _____ I'm unworthy of the love, attention, and respect of others.

24. _____ I feel that I'm not lovable.

25. _____ I am too unacceptable in very basic ways to reveal myself to other people.

*ds

26. _____ Almost nothing I do at work (or school) is as good as other people can do.

27. _____ I'm incompetent when it comes to achievement.

28. _____ Most other people are more capable than I am in areas of work and achievement.

29. _____ I'm not as talented as most people are at their work.

30. _____ I'm not as intelligent as most people when it comes to work (or school).

*fa

31. _____ I do not feel capable of getting by on my own in everyday life.

32. _____ I think of myself as a dependent person, when it comes to everyday functioning.

33. _____ I lack common sense.

34. _____ My judgment cannot be relied upon in everyday situations.

35. ____ I don't feel confident about my ability to solve everyday problems that come up.

*di

36. ____ I can't seem to escape the feeling that something bad is about to happen.

37. ____ I feel that a disaster (natural, criminal, financial, or medical) could strike at any moment.

38. ____ I worry about being attacked.

39. ____ I worry that I'll lose all my money and become destitute.

40. ____ I worry that I'm developing a serious illness, even though nothing serious has been diagnosed by a physician.

*vh

41. ____ I have not been able to separate myself from my parent(s), the way other people my age seem to.

42. ____ My parent(s) and I tend to be overinvolved in each other's lives and problems.

43. ____ It is very difficult for my parent(s) and me to keep intimate details from each other, without feeling betrayed or guilty.

44. ____ I often feel as if my parent(s) are living through me--I don't have a life of my own.

45. ____ I often feel that I do not have a separate identity from my parent(s) or partner.

*em

46. ____ I think that if I do what I want, I'm only asking for trouble.

47. ____ I feel that I have no choice but to give in to other people's wishes, or else they will retaliate or reject me in some way.

48. ____ In relationships, I let the other person have the upper hand.

49. ____ I've always let others make choices for me, so I really don't know what I want for myself.

50. ____ I have a lot of trouble demanding that my rights be respected and that my feelings be taken into account.

*sb

51. ____ I'm the one who usually ends up taking care of the people I'm close to.

52. ____ I am a good person because I think of others more than of myself.

53. ____ I'm so busy doing for the people that I care about, that I have little time for myself.

54. ____ I've always been the one who listens to everyone else's problems.

55. ____ Other people see me as doing too much for others and not enough for myself.

*ss

56. ____ I am too self-conscious to show positive feelings to others (e.g., affection, showing I care).

57. ____ I find it embarrassing to express my feelings to others.

58. ____ I find it hard to be warm and spontaneous.

59. ____ I control myself so much that people think I am unemotional.

60. ____ People see me as uptight emotionally.

*ei

61. ____ I must be the best at most of what I do; I can't accept second best.

62. ____ I try to do my best; I can't settle for "good enough."

63. ____ I must meet all my responsibilities.

64. ____ I feel there is constant pressure for me to achieve and get things done.

65. ____ I can't let myself off the hook easily or make excuses for my mistakes.

*us

66. ____ I have a lot of trouble accepting "no" for an answer when I want something from other people.

67. ____ I'm special and shouldn't have to accept many of the restrictions placed on other people.

68. ____ I hate to be constrained or kept from doing what I want.

69. ____ I feel that I shouldn't have to follow the normal rules and conventions other people do.

70. ____ I feel that what I have to offer is of greater value than the contributions of others.

*et

71. ____ I can't seem to discipline myself to complete routine or boring tasks.

72. ____ If I can't reach a goal, I become easily frustrated and give up.

73. ____ I have a very difficult time sacrificing immediate gratification to achieve a long-range goal.

74. ____ I can't force myself to do things I don't enjoy, even when I know it's for my own good.

75. ____ I have rarely been able to stick to my resolutions.

*is

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Automatic Thoughts Questionnaire.

Listed below are a variety of thoughts that pop into people's heads. Please read each thought and indicate how frequently, if at all, the thought occurred to *over the last week*. Please read each item carefully and fill in the appropriate circle on the answer sheet in the following fashion (1= "not at all," 2= "sometimes," 3= "moderately often," 4= "often," and 5= "all the time").

RESPONSES

- 1 2 3 4 5 – I feel like I'm up against the world.
- 1 2 3 4 5 – I'm no good.
- 1 2 3 4 5 – Why can't I ever succeed?
- 1 2 3 4 5 – No one understands me.
- 1 2 3 4 5 – I've let people down.
- 1 2 3 4 5 – I don't think I can go on.
- 1 2 3 4 5 – I wish I were a better person.
- 1 2 3 4 5 – I'm so weak.
- 1 2 3 4 5 – My life's not going the way I want it to.
- 1 2 3 4 5 – I'm so disappointed in myself.
- 1 2 3 4 5 – Nothing feels good anymore.
- 1 2 3 4 5 – I can't stand this anymore.
- 1 2 3 4 5 – I can't get started.
- 1 2 3 4 5 – What's wrong with me.
- 1 2 3 4 5 – I wish I were somewhere else.
- 1 2 3 4 5 – I can't get things together.
- 1 2 3 4 5 – I hate myself.
- 1 2 3 4 5 – I'm worthless.
- 1 2 3 4 5 – Wish I could just disappear.
- 1 2 3 4 5 – What's the matter with me?
- 1 2 3 4 5 – I'm a loser.
- 1 2 3 4 5 – My life is a mess.
- 1 2 3 4 5 – I'm a failure.
- 1 2 3 4 5 – I'll never make it.
- 1 2 3 4 5 – I feel so helpless.
- 1 2 3 4 5 – Something has to change.
- 1 2 3 4 5 – There must be something wrong with me.
- 1 2 3 4 5 – My future is bleak.
- 1 2 3 4 5 – It's just not worth it.
- 1 2 3 4 5 – I can't finish anything.

DASFORM A

This Inventory lists different attitudes or beliefs which people sometimes hold. Read EACH statement carefully and decide how much you agree or disagree with the statement.

For each of the attitudes, show your answer by placing a checkmark (✓) under the column that BEST DESCRIBES HOW YOU THINK. Be sure to choose only one answer for each attitude. Because people are different, there is no right answer or wrong answer to these statements.

To decide whether a given attitude is typical of your way of looking at things, simply keep in mind what you are like MOST OF THE TIME.

EXAMPLE:

ATTITUDES	TOTALLY AGREE	AGREE VERY MUCH	AGREE SLIGHTLY	NEUTRAL	DISAGREE SLIGHTLY	DISAGREE VERY MUCH	TOTALLY DISAGREE
1. Most people are O.K. once you get to know them.			✓				

Look at the example above. To show how much a sentence describes your attitude, you can check any point from totally agree to totally disagree. In the above example, the checkmark at "agree slightly" indicates that this statement is somewhat typical of the attitudes held by the person completing the inventory.

Remember that your answer should describe the way you think MOST OF THE TIME.

NOW TURN THE PAGE AND BEGIN

DAS

ATTITUDES	TOTALLY AGREE	AGREE VERY MUCH	AGREE SLIGHTLY	NEUTRAL	DISAGREE SLIGHTLY	DISAGREE VERY MUCH	TOTALLY DISAGREE
REMEMBER, ANSWER EACH STATEMENT ACCORDING TO THE WAY YOU THINK <u>MOST OF THE TIME</u> .							
1. It is difficult to be happy unless one is good looking, intelligent, rich and creative.							
2. Happiness is more a matter of my attitude towards myself than the way other people feel about me.							
3. People will probably think less of me if I make a mistake.							
4. If I do not do well all the time, people will not respect me.							
5. Taking even a small risk is foolish because the loss is likely to be a disaster.							
6. It is possible to gain another person's respect without being especially talented at anything.							
7. I cannot be happy unless most people I know admire me.							
8. If a person asks for help, it is a sign of weakness.							

ATTITUDES	TOTALLY AGREE	AGREE VERY MUCH	AGREE SLIGHTLY	NEUTRAL	DISAGREE SLIGHTLY	DISAGREE VERY MUCH	TOTALLY DISAGREE
9. If I do not do as well as other people, it means I am an inferior human being.							
10. If I fail at my work, then I am a failure as a person.							
11. If you cannot do something well, there is little point in doing it at all.							
12. Making mistakes is fine because I can learn from them.							
13. If someone disagrees with me, it probably indicates he does not like me.							
14. If I fail partly, it is as bad as being a complete failure.							
15. If other people know what you are really like, they will think less of you.							
16. I am nothing if a person I love doesn't love me.							
17. One can get pleasure from an activity regardless of the end result.							
18. People should have a reasonable likelihood of success before undertaking anything.							

ATTITUDES	TOTALLY AGREE	AGREE VERY MUCH	AGREE SLIGHTLY	NEUTRAL	DISAGREE SLIGHTLY	DISAGREE VERY MUCH	TOTALLY DISAGREE
19. My value as a person depends greatly on what others think of me.							
20. If I don't set the highest standards for myself, I am likely to end up a second-rate person.							
21. If I am to be a worthwhile person, I must be truly outstanding in at least one major respect.							
22. People who have good ideas are more worthy than those who do not.							
23. I should be upset if I make a mistake.							
24. My own opinions of myself are more important than other's opinions of me.							
25. To be a good, moral, worthwhile person, I must help everyone who needs it.							
26. If I ask a question, it makes me look inferior.							
27. It is awful to be disapproved of by people important to you.							
28. If you don't have other people to lean on, you are bound to be sad.							

ATTITUDES	TOTALLY AGREE	AGREE VERY MUCH	AGREE SLIGHTLY	NEUTRAL	DISAGREE SLIGHTLY	DISAGREE VERY MUCH	TOTALLY DISAGREE
29. I can reach important goals without slave driving myself.							
30. It is possible for a person to be scolded and not get upset.							
31. I cannot trust other people because they might be cruel to me.							
32. If others dislike you, you cannot be happy.							
33. It is best to give up your own interests in order to please other people.							
34. My happiness depends more on other people than it does on me.							
35. I do not need the approval of other people in order to be happy.							
36. If a person avoids problems, the problems tend to go away.							
37. I can be happy even if I miss out on many of the good things in life.							
38. What other people think about me is very important.							
39. Being isolated from others is bound to lead to unhappiness.							
40. I can find happiness without being loved by another person.							

This Inventory lists different attitudes or beliefs which people sometimes hold. Read EACH statement carefully and decide how much you agree or disagree with the statement.

For each of the attitudes, show your answer by placing a checkmark (✓) under the column that BEST DESCRIBES HOW YOU THINK. Be sure to choose only one answer for each attitude. Because people are different, there is no right answer or wrong answer to these statements.

To decide whether a given attitude is typical of your way of looking at things, simply keep in mind what you are like MOST OF THE TIME.

EXAMPLE:

ATTITUDES	TOTALLY AGREE	AGREE VERY MUCH	AGREE SLIGHTLY	NEUTRAL	DISAGREE SLIGHTLY	DISAGREE VERY MUCH	TOTALLY DISAGREE
1. Most people are O.K. once you get to know them.			✓				

Look at the example above. To show how much a sentence describes your attitude, you can check any point from totally agree to totally disagree. In the above example the checkmark at "agree slightly" indicates that this statement is somewhat typical of the attitudes held by the person completing the inventory.

Remember that your answer should describe the way you think MOST OF THE TIME

NOW TURN THE PAGE AND BEGIN

DAS

ATTITUDES	TOTALLY AGREE	AGREE VERY MUCH	AGREE SLIGHTLY	NEUTRAL	DISAGREE SLIGHTLY	DISAGREE VERY MUCH	TOTALLY DISAGREE
REMEMBER, ANSWER EACH STATEMENT ACCORDING TO THE WAY YOU THINK <u>MOST OF THE TIME</u> .							
1. You can be a happy person without going out of your way in order to please other people.							
2. I have to impress new acquaintances with my charm, intelligence, or wit or they won't like me.							
3. If I put other peoples' needs before my own, they should help me when I want them to do something for me.							
4. It is shameful for a person to display his weaknesses.							
5. People will like me even if I am not successful.							
6. People who have the marks of success (good looks, fame, wealth) are bound to be happier than people who do not.							
7. I should try to impress other people if I want them to like me.							
8. If a person I love does not love me, it means I am unloveable.							

ATTITUDES	TOTALLY AGREE	AGREE VERY MUCH	AGREE SLIGHTLY	NEUTRAL	DISAGREE SLIGHTLY	DISAGREE VERY MUCH	TOTALLY DISAGREE
9. I ought to be able to solve my problems quickly and without a great deal of effort.							
10. If a person is indifferent to me, it means he does not like me.							
11. I should be able to please everybody.							
12. Others can care for me even if they know all my weaknesses.							
13. If people whom I care about do not care for me, it is awful.							
14. Criticism need not upset the person who receives the criticism.							
15. My life is wasted unless I am a success.							
16. People should prepare for the worst or they will be disappointed.							
17. I must be a useful, productive, creative person or life has no purpose.							
18. A person should think less of himself if other people do not accept him.							
19. I do not need other people's approval for me to be happy.							
20. I can enjoy myself even when others do not like me.							

ATTITUDES	TOTALLY AGREE	AGREE VERY MUCH	AGREE SLIGHTLY	NEUTRAL	DISAGREE SLIGHTLY	DISAGREE VERY MUCH	TOTALLY DISAGREE
21. My value as a person depends greatly on what others think of me.							
22. If I make a foolish statement, it means I am a foolish person							
23. If a person has to be alone for a long period of time, it follows that he has to feel lonely.							
24. A person should be able to control what happens to him.							
25. If a person is not a success, then his life is meaningless.							
26. A person doesn't need to be well liked in order to be happy.							
27. If someone performs a selfish act, this means he is a selfish person.							
28. I should always have complete control over my feelings.							
29. I should be happy all the time.							
30. If people consider me unattractive it need not upset me.							
31. Whenever I take a chance or risk I am only looking for trouble.							
32. A person cannot change his emotional reactions even if he knows they are harmful to him.							

ATTITUDES	TOTALLY AGREE	AGREE VERY MUCH	AGREE SLIGHTLY	NEUTRAL	DISAGREE SLIGHTLY	DISAGREE VERY MUCH	TOTALLY DISAGREE
33. I may be able to influence other people's behavior but I cannot control it.							
34. People will reject you if they know your weaknesses.							
35. People should be criticized for their mistakes.							
36. One should look for a practical solution to problems rather than a perfect solution.							
37. If I do well, it probably is due to chance; if I do badly, it is probably my own fault.							
38. The way to get people to like you is to impress them with your personality.							
39. Turning to someone else for advice or help is an admission of weaknesses.							
40. A person should do well at everything he undertakes.							

EB SCALE

Name: _____

Date: _____

Below is a list of beliefs people sometimes report. Please read each one and tick in one of the boxes to indicate how much you believe it is true. Please give your 'gut' response.

	Agree Strongly	Agree Slightly	Unsure	Disagree Slightly	Disagree Strongly
Other people are worthless					
I am a total failure					
People think I am a bad person					
Other people are inferior to me					
People see me as worthless					
I am worthless					
Other people are total failures					
Other people are totally weak & helpless					
People see me as a total failure					
Other people are bad					
I am totally weak and helpless					
People see me as unlovable					
I am a bad person					
People see me as totally weak & helpless					
Other people are unlovable					
Other people look down on me					
I am an inferior person					
I am unlovable					

8. Compared to others, how often did you have difficulty concentrating or making a decision?

1 _____ 2 _____ 3 _____ 4 _____ 5 _____ 6 _____ 7 _____ 8 _____ 9 _____
Much About Much
less often the same more often

9. Compared to others, how often did you feel tired and lacking energy?

1 _____ 2 _____ 3 _____ 4 _____ 5 _____ 6 _____ 7 _____ 8 _____ 9 _____
Much About Much
less often the same more often

10. Compared to others, how often did you feel disappointed in yourself?

1 _____ 2 _____ 3 _____ 4 _____ 5 _____ 6 _____ 7 _____ 8 _____ 9 _____
Much About Much
less often the same more often

11. Compared to others, how often did you feel sad or blue?

1 _____ 2 _____ 3 _____ 4 _____ 5 _____ 6 _____ 7 _____ 8 _____ 9 _____
Much About Much
less often the same more often

12. Compared to others, how often did you think seriously about suicide?

1 _____ 2 _____ 3 _____ 4 _____ 5 _____ 6 _____ 7 _____ 8 _____ 9 _____
Much About Much
less often the same more often

13. Compared to others, how often did you suffer from lack of appetite?

1 _____ 2 _____ 3 _____ 4 _____ 5 _____ 6 _____ 7 _____ 8 _____ 9 _____
Much About Much
less often the same more often

RESPONSES TO DEPRESSION

People think and do many different things when they feel depressed. Please read each of the items below and indicate whether you never, sometimes, often, or always think or do each one when you feel down, sad, or depressed. Please indicate what you *generally* do, not what you think you should do by circling the appropriate number.

1= almost never, 2= sometimes, 3= often, 4= almost always.

1 2 3 4 – think about how alone you feel.

1 2 3 4 - think "I won't be able to do my job/work because I feel so bad."

1 2 3 4 – think about your feelings of fatigue and achiness.

1 2 3 4 - think about how hard it is to concentrate.

1 2 3 4 – think about how passive and unmotivated you feel.

1 2 3 4 - analyse recent events to try to understand why you are depressed.

1 2 3 4 – think about how you don't seem to feel anything anymore.

1 2 3 4 - think "Why can't I get going?"

1 2 3 4 – think "Why do I always react this way?"

1 2 3 4 - go away by yourself and think about why you feel this way.

1 2 3 4 – write down what you are thinking about and analyse it.

1 2 3 4 - think about a recent situation, wishing it would have gone better.

1 2 3 4 – think "Why do I have problems other people don't have?"

1 2 3 4 - think about how sad you feel.

1 2 3 4 – think about all your shortcomings, failings, faults, mistakes.

1 2 3 4 - think about how you don't feel up to anything.

1 2 3 4 – analyse your personality to try to understand why you are depressed.

1 2 3 4 - go someplace alone to think about your feelings.

1 2 3 4 – think about how angry you are with yourself.

1 2 3 4 - listen to sad music.

1 2 3 4 – isolate yourself and think about the reasons why you feel sad.

1 2 3 4 - try to understand yourself by focusing on your depressed feelings.

A. MOOD EPISODES

IN THIS SECTION, MAJOR DEPRESSIVE, MANIC, HYPOMANIC EPISODES, DYSTHYMIC DISORDER, MOOD DISORDER DUE TO A GENERAL MEDICAL CONDITION, SUBSTANCE-INDUCED MOOD DISORDER, AND EPISODE SPECIFIERS ARE EVALUATED. MAJOR DEPRESSIVE DISORDER AND BIPOLAR DISORDERS ARE DIAGNOSED IN MODULE D.

CURRENT MAJOR DEPRESSIVE
EPISODE

MDE CRITERIA

Now I am going to ask you some more questions about your mood.

A. Five (or more) of the following symptoms have been present during the same two-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood, or (2) loss of interest or pleasure.

In the last month...

...has there been a period of time when you were feeling depressed or down most of the day nearly every day? (What was that like?)

IF YES: How long did it last? (As long as two weeks?)

...what about losing interest or pleasure in things you usually enjoyed?

IF YES: Was it nearly every day? How long did it last? (As long as two weeks?)

(1) depressed mood most of the day, nearly every day, as indicated either by subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful). Note: in children and adolescents can be irritable mood.

(2) markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated either by subjective account or observation made by others).

? 1 2 3

A1

? 1 2 3

A2

IF NEITHER
ITEM (1)
NOR ITEM
(2) IS
CODED "3,"
GO TO
*PAST MAJOR
DEPRESSIVE
EPISODE,*
A. 12

NOTE: WHEN RATING THE FOLLOWING ITEMS,
CODE "1" IF CLEARLY DUE TO A GENERAL MED-
ICAL CONDITION, OR TO MOOD-INCONGRUENT
DELUSIONS OR HALLUCINATIONS

?=inadequate information

1=absent or false

2=subthreshold

3=threshold or true

FOR THE FOLLOWING QUESTIONS, FOCUS ON THE WORST TWO WEEKS IN THE PAST MONTH (OR ELSE THE PAST TWO WEEKS IF EQUALLY DEPRESSED FOR ENTIRE MONTH)

During this (TWO-WEEK PERIOD)...

...how was your appetite? (What about compared to your usual appetite?) (Did you have to force yourself to eat?) (Eat [less/more] than usual?) (Was that nearly every day?) (Did you lose or gain any weight?) (How much?) (Were you trying to [lose/gain] weight?)

(3) significant weight loss when not dieting, or weight gain (e.g., a change of more than 5% of body weight in a month) or decrease or increase in appetite nearly every day. Note: in children, consider failure to make expected weight gains.

? 1 2 3

A3

Check if:

___ weight loss or decreased appetite

A4

___ weight gain or increased appetite

A5

...how were you sleeping? (Trouble falling asleep, waking frequently, trouble staying asleep, waking too early, OR sleeping too much? How many hours a night compared to usual? Was that nearly every night?)

(4) insomnia or hypersomnia nearly every day

? 1 2 3

A6

Check if:

___ insomnia

A7

___ hypersomnia

A8

...were you so fidgety or restless that you were unable to sit still? (Was it so bad that other people noticed it? What did they notice? Was that nearly every day?)

(5) psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down)

? 1 2 3

A9

IF NO: What about the opposite -- talking or moving more slowly than is normal for you? (Was it so bad that other people noticed it? What did they notice? Was that nearly every day?)

NOTE: ALSO CONSIDER BEHAVIOR DURING THE INTERVIEW

Check if:

___ psychomotor retardation

A10

___ psychomotor agitation

A11

...what was your energy like? (Tired all the time? Nearly every day?)

(6) fatigue or loss of energy nearly every day

? 1 2 3

A12

?=inadequate information

1=absent or false

2=subthreshold

3=threshold or true

During this time...

...how did you feel about yourself? (Worthless?)
(Nearly every day?)

(7) feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)

? 1 2 3

A13

...what about feeling guilty about things you had done or not done? (Nearly every day?)

NOTE: CODE "1" OR "2" IF ONLY LOW SELF-ESTEEM

Check if:

— worthlessness
— inappropriate guilt

A14

A15

...did you have trouble thinking or concentrating? (What kinds of things did it interfere with?) (Nearly every day?)

(8) diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others)

? 1 2 3

A16

IF NO: Was it hard to make decisions about everyday things?
(Nearly every day?)

Check if:

— diminished ability to think
— indecisiveness

A17

A18

...were things so bad that you were thinking a lot about death or that you would be better off dead? What about thinking of hurting yourself?

(9) recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide

? 1 2 3

A19

IF YES: Did you do anything to hurt yourself?

NOTE: CODE "1" FOR SELF-MUTILATION W/O SUICIDAL INTENT

Check if:

— thoughts of own death
— suicidal ideation
— specific plan
— suicide attempt

A20

A21

A22

A23

AT LEAST FIVE OF THE ABOVE
SXS [A (1-9)] ARE CODED "3"
AND AT LEAST ONE OF THESE
IS ITEM (1) OR (2)

1 3

A24

GO TO
*PAST
MAJOR
DEPRES-
SIVE
EPI-
SODE,*
A. 12

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

IF UNCLEAR: Has (DEPRESSIVE EPISODE/OWN WORDS) made it hard for you to do your work, take care of things at home, or get along with other people?

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

? 1 2 3

A25

NOTE: DSM-IV criterion B (i.e., does not meet criteria for a Mixed Episode) has been omitted from the SCID.

GO TO
*PAST
MAJOR
DEPRES-
SIVE
EPI-
SODE,*
A. 12

Just before this began, were you physically ill?

D. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, medication) or to a general medical condition

? 1 3

A26

IF YES: What did the doctor say?

Just before this began, were you using any medications?

IF YES: Any change in the amount you were using?

Just before this began, were you drinking or using any street drugs?

IF THERE IS ANY INDICATION THAT THE DEPRESSION MAY BE SECONDARY (I.E., A DIRECT PHYSIOLOGICAL CONSEQUENCE OF A GMC OR SUBSTANCE, GO TO *GMC/SUBSTANCE,* A.43, AND RETURN HERE TO MAKE A RATING OF "1" OR "3."

DUE TO SUB-
STANCE USE
OR GMC.
GO TO *PAST
MAJOR DEP-
RESSIVE
EPISODE*
A. 12

PRIMARY
MOOD
EPISODE

Etiological general medical conditions include: degenerative neurological illnesses (e.g., Parkinson's disease), cerebrovascular disease (e.g., stroke), metabolic conditions (e.g., Vitamin B-12 deficiency), endocrine conditions (e.g., hyper- and hypothyroidism, hyper- and hypoadrenocorticism); viral or other infections (e.g., hepatitis, mononucleosis, HIV), and certain cancers (e.g., carcinoma of the pancreas).

Etiological substances include: alcohol, amphetamines, cocaine, hallucinogens, inhalants, opioids, phencyclidine, sedatives, hypnotics, anxiolytics. Medications include antihypertensives, oral contraceptives, corticosteroids, anabolic steroids, anticancer agents, analgesics, anticholinergics, cardiac medications.

CONTINUE
BELOW

?=inadequate information

1=absent or false

2=subthreshold

3=threshold or true

(Did this begin soon after someone close to you died?)

E. The symptoms are not better accounted for by Bereavement, i.e., after the loss of a loved one, the symptoms persist for longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

1

3

SIMPLE
BEREAVE-
MENT

NOT SIM-
PLE
BEREAVE-
MENT

GO TO
*PAST
MAJOR
DEPRES-
SIVE
EPISODE*
A. 12

CONTINUE
BELOW

A27

MAJOR DEPRESSIVE EPISODE
CRITERIA A, C, D, AND E ARE
CODED "3"

1

3

GO TO
*PAST
MAJOR
DEPRES-
SIVE
EPI-
SODE,*
A. 12

CUR-
RENT
MAJOR
DE-
PRES-
SIVE
EPI-
SODE

A28

How many separate times in your life have you been (depressed/ OWN WORDS) nearly every day for at least two weeks and had several of the symptoms that you described, like (SXS OF WORST EPISODE)?

Total number of Major Depressive Episodes, including current (CODE 99 IF TOO NUMEROUS OR INDISTINCT TO COUNT)

NOTE: TO RECORD DETAILS OF PAST EPISODES, GO TO J. 9 (OPTIONAL).

— —

A29

?=inadequate information

1=absent or false

2=subthreshold

3=threshold or true

Could you please mark a straight line through the line below on how you feel **right now**

Marking a mark on the centre of the line will indicate you are neither happy or sad. The closer you make a mark on the line towards the words sad or happy is indicative of how happy or sad you feel right now

e.g., SAD HAPPY
 -----I-----

This means someone is happy

Or SAD HAPPY
 -----I-----

This means someone is sad

Please mark on the line below HOW YOU FEEL RIGHT NOW

SAD HAPPY

Appendix IV

Words used for IAT and EAST tasks in Study 1

Positive words (EAST & IAT)

Enchanting, exotic, humorous, miracle, victory, adorable, courageous, ecstatic.

Negative words (EAST & IAT)

Helpless, desolate, despair, pessimistic (low mood words), defective, unworthy, insecure, abandonment (schema words).

Self-words for EAST task

First name, surname, hometown, month of birth, place of birth, nationality, and subject of study (or occupation), region living in.

Other person words for EAST task

Cuthbert, Smithers, a different month of birth from each participant, Arizona, Canadian (amended if participant was Canadian).

Self-words for IAT task

Me, mine, I, and first name of the participant.

Other person words for IAT task

They, them, his, Bruce (amended if same as the participant).

Table 17

Attributes of Words Used in the EAST & IAT Tasks-Study 1: Mean Scores (SD in brackets) of Groups of Words with F value from the Result of a 1-Way ANOVA

Variable	Positive words	Schema words	Low mood words	F
Letters	8 (1.25)	9 (1.41)	8.5 (2.08)	.536
Frequency	15.85 (20.66)	6.25 (2.98)	11 (8.66)	.292
Emotionality	6.95 (0.60)	7.37 (.83)	6.86 (0.86)	2.427
Pleasantness	8.08 (0.33)	2.61 (0.46)	2.65 (0.21)	36.75*
Syllables	3 (0.57)	3.25 (0.50)	3 (1.0)	.389

* $p < .001$, positive words were rated significantly more pleasant than schema words and low mood words; low mood and schema words were not rated significantly different from each other on pleasantness.

Chi-Square for test of word-type: $\chi^2 (4) = 4.52, p > .05$

Words used for IAT task in Study 2

Self-words

Me, mine, I, and first name of the participant.

Other person words

They, them, his, Bruce (amended if same as a participant).

Negative schema words

Insecure, abandonment, rejection, shameful, defective, unworthy, inferior, isolated.

Positive words

Enchanting, exotic, humorous, miracle, victory, adorable, courageous, ecstatic.

Table 18

Attributes of Words used in the IAT Schema Task-Study 2: Mean Scores (SD in brackets) and t from Result of Independent t -tests

Variable	Positive schema words	Negative schema words	t result
Letters	8 (1.41)	8.62 (1.06)	1
Frequency	15.85 (20.66)	10.3 (1.01)	.706
Emotionality	7.34 (0.60)	7.17 (0.83)	.443
Pleasantness	8.08 (0.33)	2.69 (0.39)	29.65*
Syllables	3 (0.57)	3.25 (0.46)	1

* $p < .001$, positive schema words were rated significantly more pleasant than negative schema words.

Chi-square test for word-type on IAT Schema: $\chi^2(2) = 1.50, p > .05$

Appendix IV: Details of words in experimental studies

Words used for IAT and EAST tasks in Study 3

EAST task

Positive words. Enchanting, exotic, humorous, miracle, victory, adorable, courageous, ecstatic.

Negative words. Helpless, desolate, despair, pessimistic (low mood words), defective, unworthy, insecure, abandonment (schema words).

Self-words. First name, surname, hometown, month of birth, place of birth, nationality, and subject of study (or occupation), region living in.

Other person words. Cuthbert, Smithers, a different month of birth from each participant, Arizona, Canadian (amended if participant was Canadian).

Table 19

Attributes of Words Used in the EAST Task- Study 3: Mean Scores (SD in brackets) of Groups of Words with F Value from the Result of a 1-Way ANOVA

Variable	Positive words	Schema words	Low mood words	<i>F</i>
Letters	8 (1.25)	9 (1.41)	8.5 (2.08)	.536
Frequency	15.85 (20.66)	6.25 (2.98)	11 (8.66)	.292
Emotionality	6.95 (0.60)	7.37 (0.83)	6.86 (0.86)	2.427
Pleasantness	8.08 (0.33)	2.61 (0.46)	2.65 (0.21)	36.75*
Syllables	3 (0.57)	3.25 (0.50)	3 (1.0)	.389

* $p < .001$, positive words were rated significantly more pleasant than schema words and low mood words; low mood and schema words were not rated significantly different from each other on pleasantness.

Chi-Square for test of word-type: $\chi^2 (4) = 4.52, p > .05$

IAT Mood task

Positive words. Enchanting, exotic, humorous, miracle, victory, adorable, courageous, ecstatic.

Low mood words. Misery, desolate, despair, pessimistic, suicide, hopeless, wretched, pitiful.

Appendix IV: Details of words in experimental studies

Self-words. Me, mine, I, first name of participant.

Other person words. They, them, his, Bruce (amended if the same as a participant).

Table 20

Attributes of Words Used in the IAT Mood Task-Study 3: Mean Scores (SD in brackets) and t from Result of Paired t-test

Variable	Positive mood words	Negative mood words	t result
Letters	8 (1.41)	7.75 (1.48)	.334
Frequency	15.85 (20.66)	11.0 (6.25)	.602
Emotionality	7.34 (0.60)	7.26 (0.79)	.225
Pleasantness	8.08 (0.33)	2.52 (0.45)	28.13*
Syllables	3 (0.57)	2.87 (0.64)	.424

*p<.001, positive mood words were rated significantly more pleasant than negative mood words.

Chi-square test for word-type on IAT Mood: $\chi^2 (2) = 2.42, p > .05$.

IAT Schema task

Positive words. Enchanting, exotic, humorous, miracle, victory, adorable, courageous, ecstatic.

Negative schema words. Insecure, abandonment, rejection, shameful, defective, unworthy, inferior, isolated.

Self-words. Me, mine, I, first name of participant.

Other person words. They, them, his, Bruce (amended if the same as a participant).

Table 21

Attributes of Words Used in the IAT Schema Task-Study 3: Mean Scores (SD in brackets) and t from Result of Independent t -tests

Variable	Positive schema words	Negative schema words	t result
Letters	8 (1.41)	8.62 (1.06)	1
Frequency	15.85 (20.66)	10.3 (1.01)	.706
Emotionality	7.34 (0.60)	7.17 (0.83)	.443
Pleasantness	8.08 (0.33)	2.69 (0.39)	29.65*
Syllables	3 (0.57)	3.25 (0.46)	1

* $p < .001$, positive schema words were rated significantly more pleasant than negative schema words.

Chi-square test for word-type on IAT Schema: $\chi^2 (2) = 1.50, p > .05$

Words used in IAT task Study 4.

Self-words

Me, mine, I, first name of participant.

Other person words

They, them, his, Bruce (amended if the same as a participant).

Negative schema words

Defective, flawed, inferior, unworthy, imperfect, worthless, inadequate, undesirable.

Positive schema words

Competent, loveable, successful, confident, thoughtful, genuine, deserving, admirable.

Table 22

Attributes of Words Used in the IAT Schema Task-Study 4: Mean Scores (SD in brackets) and t from Result of Independent t -tests

Variable	Positive schema words	Negative schema words	t result
Letters	8.87 (.99)	8.75 (1.48)	.198
Frequency	23.37 (31.0)	10.62 (9.69)	1.11
Emotionality	6.85 (0.97)	6.76 (1.39)	.145
Pleasantness	8.87 (0.99)	2.58 (0.30)	26.67*
Syllables	2.87 (0.35)	3.12 (0.64)	.966

* $p < .001$, positive schema words were rated significantly more pleasant than negative schema words.

Words used in IAT Mood task, IAT Schema task, and EAST task Study 5

IAT Mood task

Self-words. Me, mine, I, first name of participant.

Other person words. They, them, his, Bruce (amended if the same as a participant).

Positive mood words. Jolly, blissful, contented, joyful, glad, pleased, happy, satisfied.

Negative mood words. Sad, miserable, dismal, dejected, forlorn, glum, cheerless, somber.

Table 23

Attributes of Words Used in the IAT Mood Task-Study 5: Mean Scores (SD in brackets) and t from Result of Independent t -tests

Variable	Positive mood words	Negative mood words	t result
Letters	6.62 (1.92)	6.5 (2.20)	.121
Frequency	23.25 (34.71)	7.75 (11.90)	1.19
Emotionality	6.66 (0.84)	6.66 (1.00)	.000
Pleasantness	8.02 (0.49)	3.13 (0.56)	18.48*
Syllables	2.12 (0.64)	1.87 (0.83)	.672

* $p < .001$, positive mood words were rated significantly more pleasant than negative mood words.

IAT Schema task

Self-words. Me mine, I, first name of participant.

Other person words. They, them, his, Bruce (amended if the same as a participant).

Negative schema words. Defective, flawed, inferior, unworthy, imperfect, worthless, inadequate, undesirable.

Positive schema words. Competent, loveable, successful, confident, thoughtful, genuine, deserving, admirable.

Table 24

Attributes of Words Used in the IAT Schema Task-Study 5: Mean Scores (SD in brackets) and t from Result of Independent t-tests

Variable	Positive schema words	Negative schema words	<i>t result</i>
Letters	8.87 (.99)	8.75 (1.48)	.198
Frequency	23.37 (31.0)	10.62 (9.69)	1.11
Emotionality	6.85 (0.97)	6.76 (1.39)	.145
Pleasantness	8.87 (0.99)	2.58 (0.30)	26.67*
Syllables	2.87 (0.35)	3.12 (0.64)	.966

*p<.001, positive schema words were rated significantly more pleasant than negative schema words.

EAST task

Self-words. First name, surname, hometown, month of birth, place of birth, nationality, and subject of study (or occupation), region living in.

Other person words. Cuthbert, Smithers, a different month of birth from each participant, Arizona, Canadian (amended if participant was Canadian).

Positive schema words. Deserving, loveable, successful, confident.

Negative schema words. Defective, flawed, inferior, unworthy.

Positive mood words. Jolly, blissful, contented, cheerful.

Negative mood words. Sad, miserable, dismal, dejected.

Table 25

Attributes of Words Used in the EAST Task-Study 5: Mean Scores (SD in brackets) of Groups of Words with F Value from the Result of a 1-Way ANOVA

Variable	Positive schema words	Negative schema words	Low mood words	Positive mood words	F
Letters	9.0 (.81)	7.75 (1.25)	6.50 (2.64)	7.50 (1.73)	1.38
Frequency	27.75 (45.46)	9.0 (5.41)	14.0 (14.98)	4.5 (4.12)	.694
Emotionality	6.67 (0.73)	6.45 (1.66)	7.12 (0.97)	6.67 (0.73)	.230
Pleasantness	8.35 (0.73)	2.47 (0.32)	3.0 (0.74)	8.22 (0.41)	121.21*
Syllables	3.0 (0.00)	3.0 (0.00)	2.25 (0.95)	2.25 (0.50)	2.57

* $p < .001$, positive words (+ve mood & +ve schema) were rated significantly more pleasant than negative schema words and low mood words; low mood and negative schema words were not rated significantly different from each other on pleasantness.

Appendix V
Additional statistical results from experimental studies

Study 1: EAST reaction time- word-type (negative & positive) by person (self & other) by group (high BDI and low BDI) ANOVA.

Group: $F(1, 58) = .324, p = .571$
Word-type: $F(1, 58) = 3.17, p = .080$
Person: $F(1, 58) = .719, p = .40$
Person by group: $F(1, 58) = .972, p = .328$
Word-type by group: $F(1, 58) = 2.04, p = .158$

Study 1: EAST reaction time- word-type (negative schema & low mood words) by person (self & other) by group (high BDI and low BDI) ANOVA.

Group: $F(1, 58) = 1.12, p = .293$
Word-type: $F(1, 58) = .024, p = .878$
Person: $F(1, 58) = 6.25, p = .015$
Word-type by person: $F(1, 58) = .005, p = .941$
Word-type by group: $F(1, 58) = .082, p = .776$

Study 1: EAST error rates- word-type (negative & positive) by person (self & other) by group (high BDI and low BDI) ANOVA.

Group: $F(1, 58) = .482, p = .490$
Person: $F(1, 58) = .021, p = .884$
Person by group: $F(1, 58) = .021, p = .884$
Word-type by group: $F(1, 58) = 2.82, p = .098$
Word-type by person: $F(1, 58) = 4.40, p = .040$

Study 1: EAST error rates- word-type (negative schema & low mood words) by person (self & other) by group (high BDI and low BDI) ANOVA.

Group: $F(1, 58) = .006, p = .939$
Word-type: $F(1, 58) = .011, p = .915$
Person: $F(1, 58) = 1.18, p = .281$
Word-type by group: $F(1, 58), p = 1.37, p = .245$
Word-type by person: $F(1, 58) = .007, p = .935$

Study 1: IAT reaction time- compatibility (self with positive, other with negative /self with negative, positive with other) by group (high BDI and low BDI) ANOVA.

Group: $F(1, 58) = .681, p = .413$

Study 1: IAT error rates- compatibility (self with positive, other with negative /self with negative, positive with other) by group (high BDI and low BDI) ANOVA.

Group: $F(1, 58) = .950, p = .334$

Study 3: EAST reaction time- word-type (negative & positive) by person (self & other) by group (high-trait and low-trait groups) ANOVA- BDI not added as a covariate.

Group: $F(1, 64) = 3.42, p = .069$
Word-type: $F(1, 64) = 1.64, p = .838$
Person: $F(1, 64) = .901, p = .346$
Word-type by person: $F(1, 64) = 3.15, p = .199$
Person by group: $F(1, 64) = .676, p = .414$
Word-type by group: $F(1, 64) = 2.90, p = .093$

Study 3: EAST reaction time- word-type (negative & positive) by person (self & other) by group (high-trait and low-trait groups) ANOVA- BDI added as a covariate.

Group: $F(1, 63) = 1.04, p = .312$
Word-type: $F(1, 63) = .022, p = .822$
Person: $F(1, 63) = .346, p = .558$
Person by group: $F(1, 63) = 2.15, p = .664$
Word-type by person: $F(1, 63) = .339, p = .562$
Word-type by group: $F(1, 63) = 2.03, p = .159$

Study 3: EAST error rates- word-type (negative & positive) by person (self & other) by group (high-trait and low-trait groups) ANOVA- BDI not added as a covariate.

Group: $F(1, 64) = 1.97, p = .165$
Word-type: $F(1, 64) = .104, p = .742$
Word-type by group: $F(1, 64) = 1.17, p = .284$
Person by group: $F(1, 64) = .071, p = .791$
Word-type by person: $F(1, 64) = 3.64, p = .061$

Study 3: EAST error rates- word-type (negative & positive) by person (self & other) by group (high-trait and low-trait groups) ANOVA- BDI added as a covariate.

Group: $F(1, 63) = 2.89, p = .094$
Word-type: $F(1, 63) = .745, p = .391$
Word-type by group: $F(1, 63) = .002, p = .269$
Word-type by person: $F(1, 63) = 2.45, p = .122$
Person by group: $F(1, 63) = .988, p = .324$

Study 3: IAT Mood reaction time- compatibility (self with positive, other with negative /self with negative, positive with other) by group (high-trait and low trait groups) ANOVA-BDI not added as a covariate.

Group: $F(1, 64) = 1.86, p = .177$

Study 3: IAT Mood reaction time- compatibility (self with positive, other with negative /self with negative, positive with other) by group (high-trait and low trait groups) ANOVA-BDI not added as a covariate.

Group: $F(1, 63) = .541, p = .465$

Study 3: IAT Schema reaction time- compatibility (self with positive, other with negative /self with negative, positive with other) by group (high-trait and low trait groups) ANOVA-BDI not added as a covariate.

Group: $F(1, 64) = .521, p = .473$

Study 3: IAT Schema reaction time- compatibility (self with positive, other with negative /self with negative, positive with other) by group (high-trait and low trait groups)
ANOVA-BDI added as a covariate.

Group: $F(1, 63) = .571, p = .453$

Study 3: IAT Mood error rates- compatibility (self with positive, other with negative /self with negative, positive with other) by group (high-trait and low trait groups)
ANOVA-BDI not added as a covariate.

Group: $F(1, 64) = .479, p = .492$

Study 3: IAT Mood error rates- compatibility (self with positive, other with negative /self with negative, positive with other) by group (high-trait and low trait groups)
ANOVA-BDI added as a covariate.

Group: $F(1, 63) = 2.17, p = .146$

Study 3: IAT Schema error rates- compatibility (self with positive, other with negative /self with negative, positive with other) by group (high-trait and low trait groups)
ANOVA-BDI not added as a covariate.

Group: $F(1, 64) = .236, p = .629$

Study 3: IAT Schema error rates- compatibility (self with positive, other with negative /self with negative, positive with other) by group (high-trait and low trait groups)
ANOVA-BDI added as a covariate.

Group: $F(1, 63) = 1.83, p = .181$

Study 3: EAST specific schema content reaction time- word-type (negative schema & low mood words) by person (self & other) by group (high-trait and low-trait groups)
ANOVA- BDI not added as a covariate.

Word-type: $F(1, 64) = 1.32, p = .255$

Person: $F(1, 64) = 3.10, p = .088$

Person by group: $F(1, 64) = 2.00, p = .162$

Word-type by person: $F(1, 64) = 2.15, p = .148$

Word-type by group: $F(1, 64) = .017, p = .897$

Study 3: EAST specific schema content reaction time- word-type (negative schema & low mood words) by person (self & other) by group (high-trait and low-trait groups)
ANOVA- BDI added as a covariate.

Group: $F(1, 63) = 1.38, p = .245$

Word-type: $F(1, 63) = .602, p = .441$

Person: $F(1, 63) = .042, p = .838$

Word-type by group: $F(1, 63) = .095, p = .759$

Person by group: $F(1, 63) = 2.07, p = .155$

Word-type by person: $F(1, 63) = .007, p = .932$

Study 3: EAST specific schema content error rates- word-type (negative schema & low mood words) by person (self & other) by group (high-trait and low-trait groups) ANOVA- BDI not added as a covariate.

Group: $F(1, 64) = .889, p = .349$
Word-type: $F(1, 64) = .753, p = .389$
Word-type by group: $F(1, 64) = .058, p = .810$
Word-type by person: $F(1, 64) = .217, p = .643$
Person by group: $F(1, 64) = .048, p = .828$

Study 3: EAST specific schema content error rates- word-type (negative schema & low mood words) by person (self & other) by group (high-trait and low-trait groups) ANOVA- BDI added as a covariate.

Group: $F(1, 63) = 2.26, p = .114$
Word-type: $F(1, 63) = .218, p = .642$
Word-type by group: $F(1, 63) = .652, p = .422$
Person by group: $F(1, 63) = 1.51, p = .224$
Word-type by person: $F(1, 63) = .591, p = .445$

Study 2: IAT Effect reaction time- IAT-type (IAT Schema & IAT Mood) by group (high-trait and low trait groups) ANOVA-BDI not added as a covariate.

Group: $F(1, 64) = 3.63, p = .061$

Study 3: IAT Effect reaction time- IAT-type (IAT Schema & IAT Mood) by group (high-trait and low trait groups) ANOVA-BDI added as a covariate.

Group: $F(1, 63) = 1.29, p = .261$

Study 4: DAS form (DAS-A & DAS-B) by group (AB & BA) ANOVA.

Group: $F(1, 52) = 2.02, p = .162$

Study 4: IAT reaction time- compatibility (self with positive, other with negative /self with negative, positive with other) by time (pre and post negative mood induction) by group (high-trait and low trait groups) ANOVA-no covariate added.

Group: $F(1, 52) = .077, p = .782$
Time by group: $F(1, 52) = .002, p = .961$
Compatibility by group: $F(1, 52) = .040, p = .782$

Study 4: IAT reaction time- compatibility (self with positive, other with negative /self with negative, positive with other) by time (pre and post negative mood induction) by group (high-trait and low trait groups) ANOVA-VAS added as a covariate.

Group: $F(1, 51) = .000, p = 1.00$
Time: $F(1, 51) = .000, p = .993$
Time by group: $F(1, 51) = .234, p = .631$
Compatibility by group: $F(1, 51) = .000, p = .998$

Study 4: IAT reaction time- compatibility (self with positive, other with negative /self with negative, positive with other) by time (pre and post negative mood induction) by group (high-trait and low trait groups) ANOVA-BDI added as a covariate.

Group: $F(1, 51) = .001, p = .973$
Time by group: $F(1, 51) = .083, p = .774$
Compatibility by time: $F(1, 53) = .121, p = .729$

Study 4: IAT error rates- compatibility (self with positive, other with negative /self with negative, positive with other) by time (pre and post negative mood induction) by group (high-trait and low trait groups) ANOVA-no covariate added.

Group: $F(1, 52) = .834, p = .365$
Compatibility by time: $F(1, 52) = .031, p = .860$

Study 4: IAT reaction time- compatibility (self with positive, other with negative /self with negative, positive with other) by time (pre and post negative mood induction) by group (high-trait and low trait groups) ANOVA-VAS added as a covariate.

Group: $F(1, 51) = .302, p = .585$
Time: $F(1, 51) = 2.50, p = .120$
Compatibility by time: $F(1, 51) = 1.10, p = .300$

Study 4: IAT reaction time- compatibility (self with positive, other with negative /self with negative, positive with other) by time (pre and post negative mood induction) by group (high-trait and low trait groups) ANOVA-BDI added as a covariate.

Group: $F(1, 51) = .809, p = .373$
Time: $F(1, 51) = .495, p = .485$
Compatibility by time: $F(1, 51) = .069, p = .793$

Study 5: YSQ: Time (1, 2, 3) by subscale (15 subscales) by group (control and SSRI) ANOVA.

Time: $F(2, 58) = 1.38, p = .255$
Time by subscale: $F(28, 812) = 1.08, p = .379$

Study 5: EAST reaction time- word-type (negative & positive) by person (self & other) by time (1, 2, 3) by group (control and SSRI) ANOVA.

Group: $F(1, 29) = .199, p = .659$
Word-type: $F(1, 29) = .075, p = .787$
Person: $F(1, 29) = .375, p = .545$
Word-type by group: $F(1, 29) = .985, p = .329$
Person by group: $F(1, 29) = .217, p = .645$
Time by group: $F(2, 58) = .211, p = .734$
Word-type by person: $F(1, 29) = .236, p = .630$
Word-type by time: $F(2, 58) = .268, p = .766$
Person by time: $F(2, 58) = 2.29, p = .111$
Word-type by time by group: $F(2, 58) = 1.95, p = .151$
Person by time by group: $F(2, 58) = .342, p = .712$
Word-type by person by time by group: $F(2, 58) = 2.50, p = .090$

Study 5: EAST error rate- word-type (negative & positive) by person (self & other) by time (1, 2, 3) by group (control and SSRI) ANOVA.

Group: $F(1, 29) = .010, p = .921$
Word-type: $F(1, 29) = .075, p = .787$
Person: $F(1, 29) = .173, p = .881$
Time: $F(2, 58) = .124, p = .884$
Time by group: $F(2, 58) = 1.95, p = .151$
Word-type by person: $F(1, 29) = 1.58, p = .219$
Word-type by time: $F(2, 58) = .197, p = .822$
Person by time: $F(2, 58) = 5.28, p = .008$
Word-type by time by group: $F(2, 58) = .112, p = .894$
Person by time by group: $F(2, 58) = .451, p = .639$
Word-type by person by time: $F(2, 58) = .461, p = .921$

Study 5: IAT Mood reaction time- compatibility (self with positive, other with negative /self with negative, positive with other) by time (1,2,3) by group (control and SSRI) ANOVA.

Group: $F(1, 29) = 1.43, p = .242$
Time by group: $F(2, 58) = .094, p = .910$
Compatibility by time by group: $F(2, 58) = 1.34, p = .269$

Study 5: IAT Schema reaction time- compatibility (self with positive, other with negative /self with negative, positive with other) by time (1,2,3) by group (control and SSRI) ANOVA.

Group: $F(1, 29) = .028, p = .869$
Time by group: $F(2, 58) = 1.15, p = .323$
Compatibility by time by group: $F(2, 58) = .883, p = .365$

Study 5: IAT Mood error rates- compatibility (self with positive, other with negative /self with negative, positive with other) by time (1,2,3) by group (control and SSRI) ANOVA.

Group: $F(1, 29) = .019, p = .892$
Time by group: $F(2, 58) = 1.38, p = .260$

Study 5: IAT Schema error rates- compatibility (self with positive, other with negative /self with negative, positive with other) by time (1,2,3) by group (control and SSRI) ANOVA.

Group: $F(1, 29) = .365, p = .550$
Time: $F(2, 58) = 1.06, p = .352$
Time by group: $F(2, 58) = .032, p = .968$
Compatibility by time: $F(2, 58) = .390, p = .679$

Study 5: IAT Effect reaction time-specific schema content- time (1, 2, 3) by IAT-type (schema and low mood IAT) by group (control and SSRI) ANOVA.

Group: $F(2, 58) = 2.16, p = .152$
Time by group: $F(2, 58) = 2.09, p = .132$
Time by IAT-type: $F(2, 58) = .027, p = .973$

Study 5: IAT Effect error rates-specific schema content- time (1, 2, 3) by IAT-type (schema and low mood IAT) by group (control and SSRI) ANOVA.

Time by group: $F(2, 58) = .459, p = .634$

Time by IAT-type: $F(2, 58) = 1.22, p = .304$

Study 5: EAST specific schema content reaction time- word-type (negative schema, positive schema, positive mood, & low mood words) by person (self & other) by time (1, 2, 3) by group (control and SSRI) ANOVA.

Group: $F(1, 29) = .279, p = .602$

Word-type: $F(3, 87) = .831, p = .480$

Person: $F(1, 29) = .434, p = .515$

Person by time: $F(2, 58) = 2.29, p = .110$

Person by group: $F(1, 29) = .191, p = .666$

Word-type by group: $F(3, 87) = .731, p = .536$

Time by group: $F(2, 58) = .330, p = .721$

Word-type by person: $F(3, 87) = .058, p = .981$

Word-type by time: $F(6, 174) = 1.35, p = .239$

Word-type by time by group: $F(6, 174) = 1.417, p = .211$

Person by time by group: $F(2, 58) = .349, p = .707$

Word-type by person by time: $F(6, 174) = 1.61, p = .146$

Word-type by person by time by group: $F(6, 174) = 1.17, p = .324$

Study 5: EAST specific schema content error rates- word-type (negative schema, positive schema, positive mood, & low mood words) by person (self & other) by time (1, 2, 3) by group (control and SSRI) ANOVA.

Group: $F(1, 29) = .002, p = .967$

Word-type: $F(3, 87) = .533, p = .661$

Person: $F(1, 29) = .296, p = .590$

Time: $F(2, 58) = .044, p = .957$

Word-type by group: $F(3, 87) = 2.13, p = .102$

Time by group: $F(2, 58) = 1.76, p = .181$

Word-type by person: $F(3, 87) = 2.54, p = .084$

Word-type by time: $F(6, 174) = 1.27, p = .269$

Word-type by time by group: $F(6, 174) = 1.08, p = .375$

Person by time by group: $F(2, 58) = .531, p = .591$

Word-type by person by time: $F(6, 174) = .573, p = .751$

Word-type by person by time by group: $F(6, 174) = .488, p = .721$

Appendix VI: Ethics forms

Consent and information sheet for Study 1 (page 279)

Debriefing form for Study 1 (page 280)

Consent and information sheet for Study 2 (page 281)

Debriefing form for Study 2 (page 282)

Consent and information sheet for Study 3 (page 283)

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Consent from Study 5 (page 291)

Debriefing form Study 5 (page 292)

Consent form and Information sheet for Study 1

A study investigating self-perception, mood, beliefs, and information processing

Consent Form and Information for Research Participants

Information sheet

I am Bruce Scott a PhD student in psychology from the University of Southampton. I am requesting your participation in a study regarding self-perception and information processing. This will involve taking part in three computer tasks and filling out some questionnaires. This will take approximately 1 hour. The computer tasks involve classifying words presented on a computer screen while the questionnaires will assess moods and beliefs. Personal information will not be released or viewed by anyone other than researchers involved in this project. Results of this study will not include your name or any other identifying characteristics, your participation is voluntary and you may withdraw your participation at any time. For those participants (psychology students from the University of Southampton) who are taking part as part of credits for participations scheme, if you choose to withdraw from the study at any time, there will be no consequences to your grade or to your treatment as a student in the psychology department. If you have any questions please ask them now, or contact me Bruce Scott on 0238-0594594 or email me at b.scott@soton.ac.uk.

Signature of
Researcher.....Date.....

Name _____

Statement of Consent

Ihave read the above information and terms of consent.

I understand that I may withdraw my consent and discontinue participation at any time without penalty or loss of benefit to myself. I understand that data collected as part of this research project will be treated as confidential, and that the published results of this research project will maintain my confidentiality. In signing this consent sheet, I am not waiving my legal claims, rights, or remedies.

I give consent to participate in the above study (please circle Yes or No)

YES NO

Signature _____ Date _____

Name

I understand that if I have questions about my rights as a participant in this research, or if I feel that I have been placed at risk, I can contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, SO17 1BJ, tel: 023-8059-3995

Study 1 debriefing statement

A study investigating self-perception, mood, beliefs, and information processing

Debriefing statement.

The aim of this research was to see if individuals who score higher on measures of depressive symptomatology, thinking and beliefs (the questionnaires) have a bias to process negative information faster and more efficiently (faster reaction time and fewer errors to negative words on the computer tasks) when it is associated with words associated with the self (self-words). Further this study was also investigating whether people who score low on measures of depressive symptomatology, thinking and beliefs process positive information more efficiently when it is associated with self-words. People who score higher on measures of depressive symptomatology, thinking and beliefs may be more likely to process negative information more efficiently when it is associated with the self, whereas those who score low on measures of depressive symptomatology, thinking and beliefs may be more likely to process positive information more efficiently when it is associated with the self. Your data will help our understanding of how differences in information processing are related to people who differ on levels of depressive symptomatology, thinking and beliefs. This may provide valuable insight into the way people who are “clinically depressed” process information related to the self. Once again the results of this study will not include your name or any other identifying characteristics. The experiment did not use deception. You may have a copy of the summary of research findings once the project is completed. If you have any further questions please contact me (Bruce Scott) on 023-8059-4594 and/or email me at b.scott@soton.ac.uk

Thank you for your participation in this research

Signature.....Date.....

Name.....

If you have any questions about your rights as a participant in this research, or if I feel that you have been placed at risk, you may contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ, tel: 023-8059-3995

Consent form and Information sheet for Study 2**A study investigating self-perception and information processing****Consent Form and Information for Research Participants**Information sheet

I am Bruce Scott a PhD student in psychology from the University of Southampton. I am requesting your participation in a study regarding self-perception and information processing. This will involve taking part in a computer task. This will take approximately 30 minutes. The computer task involves classifying words presented on a computer screen. Personal information will not be released or viewed by anyone other than researchers involved in this project. Results of this study will not include your name or any other identifying characteristics, your participation is voluntary and you may withdraw your participation at any time. For those participants (psychology students from the University of Southampton) who are taking part as part of credits for participations scheme, if you choose to withdraw from the study at any time, there will be no consequences to your grade or to your treatment as a student in the psychology department. If you have any questions please ask them now, or contact me Bruce Scott on 0238-0594594 or email me at b.scott@soton.ac.uk.

Signature of

Researcher.....Date.....

Name _____

Statement of Consent

Ihave read the above information and terms of consent.

I understand that I may withdraw my consent and discontinue participation at any time without penalty or loss of benefit to myself. I understand that data collected as part of this research project will be treated as confidential, and that the published results of this research project will maintain my confidentiality, In signing this consent sheet, I am not waiving my legal claims, rights, or remedies.

I give consent to participate in the above study (please circle Yes or No)

YES NO

Signature

Date

Name

I understand that if I have questions about my rights as a participant in this research, or if I feel that I have been placed at risk, I can contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, SO17 1BJ, tel: 023-8059-3995

Study 2 debriefing statement

A study investigating self-perception and information processing

Debriefing statement.

The aim of this research was to see if individuals are more efficient (faster reaction time and fewer errors on the computer tasks) when positive words are associated with the self and less efficient when negative information is associated with the self. People may be generally quicker and make fewer errors when positive information is associated with the self and slower when and make more errors when negative information associated with the self. Your data will help our understanding of how and why people automatically have a bias towards processing positive information when it is associated with the self. In other words how and why people have an unconscious self-serving bias. This data may provide valuable information that can be compared to other samples of individuals (e.g., individuals suffering from depression) and how they process negative and positive information when it is associated with the self. Once again the results of this study will not include your name or any other identifying characteristics. The experiment did not use deception. You may have a copy of the summary of research findings once the project is completed. If you have any further questions please contact me (Bruce Scott) on 023-8059-4594 and/or email me at b.scott@soton.ac.uk

Thank you for your participation in this research

Signature.....Date.....

Name.....

If you have any questions about your rights as a participant in this research, or if I feel that you have been placed at risk, you may contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ, tel: 023-8059-3995

Consent form and Information sheet for Study 3**Self-perception, mood, personality and information processing****Consent Form and Information for Research Participants**Information sheet

I am Bruce Scott a PhD student in psychology from the University of Southampton. I am requesting your participation in a study regarding self-perception and information processing. This will involve taking part two computer tasks and filling out some questionnaires. This will take approximately 1 hour. The computer task involves classifying words presented on a computer screen, while the questionnaires will assess mood, beliefs and personality traits. Personal information will not be released or viewed by anyone other than researchers involved in this project. Results of this study will not include your name or any other identifying characteristics, your participation is voluntary and you may withdraw your participation at any time. For those participants (psychology students from the University of Southampton) who are taking part as part of credits for participations scheme, if you choose to withdraw from the study at any time, there will be no consequences to your grade or to your treatment as a student in the psychology department. If you have any questions please ask them now, or contact me Bruce Scott on 0238-0594594 or email me at b.scott@soton.ac.uk.

Signature of

Researcher.....Date.....

Name _____

Statement of Consent

Ihave read the above information and terms of consent.

I understand that I may withdraw my consent and discontinue participation at any time without penalty or loss of benefit to myself. I understand that data collected as part of this research project will be treated as confidential, and that the published results of this research project will maintain my confidentiality, In signing this consent sheet, I am not waiving my legal claims, rights, or remedies.

I give consent to participate in the above study (please circle Yes or No)

YES NO

Signature

Date

Name

I understand that if I have questions about my rights as a participant in this research, or if I feel that I have been placed at risk, I can contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, SO17 1BJ, tel: 023-8059-3995

Study 3 debriefing statement

A study investigating self-perception, mood, personality and information processing

Debriefing statement.

The aim of this research was to see if individuals who score higher on a measure of trait depression (behaviours and experiences indicative of traits of depression), have a bias to process negative information faster and more efficiently (faster reaction time and fewer errors to negative words on the computer tasks) when it is associated with self words. Further this study was also investigating whether people who score lower on a measure of trait depression process positive information more efficiently when it is associated with self-words. People who score higher on a measure of trait depression may be more likely to process negative information more efficiently when it is associated with the self, whereas those who score lower on a measure of trait depression may be more likely to process positive information more efficiently when it is associated with the self. Your data will help our understanding of how differences in information processing are related to people who are more or less prone to depression. This may provide valuable insight into the reasons why some vulnerable individuals develop clinical depression. Once again the results of this study will not include your name or any other identifying characteristics. The experiment did not use deception. You may have a copy of the summary of research findings once the project is completed. If you have any further questions please contact me (Bruce Scott) on 023-8059-4594 and/or email me at b.scott@soton.ac.uk

Thank you for your participation in this research

Signature.....Date.....

Name.....

If you have any questions about your rights as a participant in this research, or if I feel that you have been placed at risk, you may contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ, tel: 023-8059-3995

Consent Form and Information for Research Participants

Study 4 debriefing statement

A study investigating information processing, thoughts and beliefs.

Debriefing statement.

The aim of this research was to see if individuals who score higher on a measure of trait depression (behaviours and experiences indicative of traits of depression), have a bias to process negative information faster and more efficiently (faster reaction time and fewer errors to negative words on the computer tasks) when it is associated with self words. Further this study was also investigating whether people who score lower on a measure of trait depression process positive information more efficiently when it is associated with self-words. People who score higher on a measure of trait depression may be more likely to process negative information more efficiently when it is associated with the self after listening to sad music, whereas those who score lower on a measure of trait depression may be more likely to process positive information more efficiently when it is associated with the self before and after listening to sad music. Your data will help our understanding of how differences in information processing are related to people who are more or less prone to depression. This may provide valuable insight into the reasons why some vulnerable individuals develop clinical depression. Once again the results of this study will not include your name or any other identifying characteristics. The experiment did not use deception. You may have a copy of the summary of research findings once the project is completed. If you have any further questions please contact me (Bruce Scott) on 023-8059-4594 and/or email me at b.scott@soton.ac.uk

Thank you for your participation in this research

Signature.....Date.....

Name.....

If you have any questions about your rights as a participant in this research, or if I feel that you have been placed at risk, you may contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ, tel: 023-8059-3995

Control group information sheet-Study 5

Direct Line: 023-8033-7390 or 023-8059-6897

Email: lusia@soton.ac.uk

or b.scott@soton.ac.uk

The effects of depression and its treatment on thinking and beliefs.

INFORMATION SHEET

We are conducting a study to look at the thoughts and beliefs of people who are depressed and undergoing treatment for depressions and people who are not depressed. We know that depression influences the way people think and that this changes as they recover. This study uses a combination of questionnaires and simple computer tasks to investigate thinking and beliefs in people who are depressed and undergoing treatment for their depression and in people **who are not** depressed.

We are looking for healthy, non-depressed adult volunteers to take part in this study.

Here is some information to help you decide whether or not to take part. Please take time to read the following information carefully and discuss it with friends and relatives if you wish. Please do not hesitate to ask if there is anything that is not clear or if you would like more information. **I (Bruce Scott) can be contacted on 023-8033-7390 or 077888-51769.**

You are under no obligation to take part in this study. You can take part, but if you wish, can leave at any time. All records of your participation will be identifiable only from an identification code, and all data will be securely stored during and after the study has been completed.

What taking part involves:

- There will be three testing sessions, which will last approximately 1-1.5 hours. After the first session, the second session will take place approximately 3 months after the first, and the third will take place approximately 3 months after the second visit. The study will be conducted in the Department of Psychology at the University of Southampton.
- Part of your travelling expenses you incur will be reimbursed (£5 per visit).
- During the sessions you will be asked:
 - i) to complete some computer tasks,

- ii) to complete some questionnaires.
- Your participation in this study is strictly confidential. Your name will not be used when analysing the data obtained.
- If you decide to take part you will be asked to sign a consent form:
 - i) You can decide to withdraw from the study at any time without giving a reason.
 - ii) Signing the consent form does not mean that you must complete the tests.

**For further information please contact Bruce Scott at The University of
Southampton, Department of Psychology on
023-8033-7390 or 07788-851769
or Dr. Lusía Stopa, The University of Southampton, Department of Psychology
on
023-8059-6897**

SSRI group information sheet-Study 5

Direct Line: 023-8033-7390 or 023-8059-6897

**Email: lusia@soton.ac.uk
or b.scott@soton.ac.uk**

The effects of depression and its treatment on thinking and beliefs.

INFORMATION SHEET

We are conducting a study to look at people's thoughts and beliefs about themselves and other people when they are depressed. We know that depression influences the way people think and that this changes as they recover. This study uses a combination of questionnaires and simple computer tasks to explore how and when these changes take place.

We are looking for adult volunteers who have been diagnosed as depressed by their GP and who have been prescribed certain kinds of anti-depressants (e.g. Prozac, Sertraline, Seoroxat, Cipramil etc).

Here is some information to help you decide whether or not to take part. Please take time to read the following information carefully and discuss it with friends and relatives if you wish. Please do not hesitate to ask if there is anything that is not clear or if you would like more information. **I (Bruce Scott) can be contacted on 023-8033-7390 or 077888-51769.**

You are under no obligation to take part in this study. If you decide not to take part in the study, or to take part and then leave at any time, your normal medical care. All records of your participation will be identifiable only from an identification code, and all data will be securely stored during and after the study has been completed.

What taking part involves:

- There will be three testing sessions, which will last approximately 1-1.5 hours. The first session will be within the first two weeks of the commencement of treatment, the second approximately 3 months into treatment, and the third approximately 6 months into treatment. The study will be conducted in the Department of Psychology at the University of Southampton.
- Part of your travelling expenses you incur will be reimbursed (£5 per visit).
- During the sessions you will be asked:
 - i) to complete some computer tasks,

- ii) to complete some questionnaires.
- Your participation in this study is strictly confidential. Your name will not be used when analysing the data obtained.
- Although you are unlikely to receive any direct personal benefit from participating in this study, we hope the results will improve our understanding of how depression and its treatment with anti-depressants affects thinking and beliefs. We will share useful findings with our colleagues through publication.
- If you decide to take part you will be asked to sign a consent form:
 - i) You can decide to withdraw from the study at any time without giving a reason.
 - ii) Signing the consent form does not mean that you must complete the tests.
 - iii) Deciding not to take part in the study or withdrawing during the study will not affect your treatment in the National Health Service.

**For further information please contact Bruce Scott at The University of
Southampton, Department of Psychology on
023-8033-7390 or 07788-851769
or Dr. Lusia Stopa, The University of Southampton, Department of Psychology
on
023-8059-6897**

Consent form for Study 5

The effects of depression and its treatment on thinking and beliefs.

Consent Form for Research Participants

I am Bruce Scott a PhD student in psychology from the University of Southampton. I am requesting your participation in a study regarding the effects of depression and its treatment on thinking and beliefs. This will involve filling out some questionnaires and completing three computer based tasks on three occasions. These sessions will take approximately 1 hour. Personal information will not be released or be viewed by anyone other than the researchers involved in this project. Results of this study will not include your name or any other identifying characteristics. Your participation is voluntary and you may withdraw your participation at any time. If you have any questions please ask them now, or contact me Bruce Scott on 0238-0594594 or email me at b.scott@soton.ac.uk.

Signature of
Researcher.....Date.....

Name _____

Statement of Consent

Ihave read the attached information sheet and the above information and terms of consent.

I understand that I may withdraw my consent and discontinue participation at any time without penalty or loss of benefit to myself. I understand that data collected as part of this research project will be treated as confidential, and that the published results of this research project will maintain my confidentiality. In signing this consent sheet, I am not waiving my legal claims, rights, or remedies.

I give consent to participate in the above study (please circle Yes or No)

YES NO

Signature

Date

Name

I understand that if I have questions about my rights as a participant in this research, or if I feel that I have been placed at risk, I can contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, SO17 1BJ, tel: 023-8059-3995

Study 5 debriefing statement**A study investigating the effects of depression and its treatment on thinking and beliefs****Debriefing statement.**

The aim of this research was to investigate the effects of SSRI antidepressant treatment on conscious (e.g., the questionnaires) and implicit or unconscious (e.g., the computer tasks) thinking and beliefs in individuals who were diagnosed as suffering from depression and compare this data to individuals who were not diagnosed as suffering from depression. People who have been diagnosed as suffering from depression and who take SSRI anti-depressant's usually show a reduction over time in self-reported symptoms of depression, thinking and beliefs (e.g., the questionnaires) associated with depression to levels comparable to non-depressed people. Further, people who are depressed (e.g., before treatment) usually have a bias to be more efficient (faster and fewer errors) when negative information is associated with the self, whereas non-depressed people usually have a bias to be more efficient when positive information is associated with the self (e.g., the computer tasks). However, very little is known how SSRI antidepressant medication over time affects the implicit or unconscious processing of negative and positive information when it is associated with the self. This study will hopefully shed light on how individuals who are undergoing SSRI antidepressant process negative and positive information when associated with the self at different stages in treatment, and how this compares with the usual observed reduction in self-reported symptoms of depression, thinking and beliefs. We will be able to use this information to see how it compares to the information processing biases observed in non-depressed people. This may provide valuable insight into how SSRI anti-depressants affect different levels of thinking (e.g., conscious and unconscious) and may help clinicians develop improved treatments for depression. Once again the results of this study will not include your name or any other identifying characteristics. The experiment did not use deception. You may have a copy of the summary of research findings once the project is completed. If you have any further questions please contact me (Bruce Scott) on 023-8059-4594 and/or email me at b.scott@soton.ac.uk

Thank you for your participation in this research

Signature.....Date.....

Name.....

If you have any questions about your rights as a participant in this research, or if I feel that you have been placed at risk, you may contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ, tel: 023-8059-3995