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Depression and Cognitive Vulnerability

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FRA EN ANNEN VIRKELIGHET…

"Jeg holder et menneskes hånd,
ser inn i et menneskes øyne,
men jeg er på den andre siden
der mennesket er en tåke av ensomhet og angst.
Å, om jeg var en sten
som kunne romme denne tomhetens tyngde,
om jeg var en stjerne
som kunne drikke denne tomhetens smerte,
men jeg er et menneske kastet ut i grenselandet,
og stillheten hører jeg bruse,
stillheten hører jeg rope
fra dypere verdner enn denne."

Gunvor Hofmo

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1. GENERAL AIMS OF THE STUDY

In 1996, when the study was planned, the overall aim of the study was to investigate how cognitive processing of emotionally valenced information may be a vulnerability factor to depression. To do so, we decided to compare previously depressed individuals with clinically depressed individuals and non-depressed controls on several cognitive functions including preference for information, visual attention, reaction time, free recall and recognition.

In the course of the study several factors have influenced how we have chosen to analyse and interpret the data. First of all, the data collection was comprehensive and lasted for several years. In the meantime the research literature on cognitive vulnerability factors to depression were considerably increased, especially with regard to the question whether a depressive episode by itself may function as a vulnerability factor. Also, there was a new tendency in the field to integrate different cognitive approaches to depression, including self-regulation theories and meta-cognitive perspectives. Finally, the quantity of collected data was great and this gave us the opportunity to generate and explore new hypotheses, and to develop a theoretical model to explain recurrent depression.

Accordingly, in the course of the study the general aim of the study changed from a more general approach to the question of cognitive vulnerability factors in depression, to a larger focus on how depression may act as a vulnerability factor by itself. With this alteration of the focus, the main aim of the study has been to review the research literature and to integrate different cognitive theories to explain how cognitive processes may contribute to recurrent episodes of depression. Results from the present study have contributed to the development of the Cognitive battle model of recurrent depression presented in paper 1.

Also, a general aim of the study has been to increase the knowledge of how self-regulation, such as approach motivation, may influence cognitive processes and be related to cognitive vulnerability factors such as dysfunctional attitudes (paper 2). Finally, we wanted to increase our understanding of how automatic and effortful processes, respectively, are involved in the processing of emotionally valenced information (paper 3). Findings from previous research on cognitive processes in depression have been inconclusive regarding this issue.

2. INTRODUCTION

"That's the thing I want to make clear about depression: It's got nothing at all to do with life. In the course of life, there is sadness and pain and sorrow, all of which, in their right time and season, are normal - unpleasant, but normal. Depression is in an altogether different zone because it involves a complete absence: absence of affect, absence of feeling, absence of response, absence of interest. The pain you feel in the course of a major clinical depression is an attempt on nature's part (nature, after all, abhors a vacuum) to feel up the empty space. But for all intents and purposes, the deeply depressed are just the walking, waking dead.

And the scariest part is that if you ask anyone in the throes of depression how he got there, to pin down the turning point, he'll never know. There is the classic moment in The Sun Also Rises when someone ask Mike Campbell how he went bankrupt, and all he can say in response is, "Gradually and then suddenly." When someone asks how I lost my mind, that is all I can say too."

Elizabeth Wurtzel, 1994 (p. 22)

2.1 Background

As early as 1975, Seligmann described major depression as the "common cold" of psychiatry. Today, thirty years later, the situation has become even worse. Depression is currently affecting about 121 million people worldwide (World Health Organization; WHO, 2001a), and the incidence of depressive symptoms increases in all groups of age and in all western cultures (Klerman et al., 1985; Klerman & Weissman, 1992; Sartorius, Jablensky, Gulbinat, & Ernberg, 1980). According to the WHO (2001b) depression is today the leading cause of disability. Also, the WHO predicts that, of all diseases, in 2020 depression will impose the second-largest burden of ill health worldwide (Murray & Lopez, 1998).

In a relatively recent epidemiological study from six European countries it was found that about 17 % of the population reported some experience with depression in the last six months, whereas major depression accounted for 7 % (Lepine, Gastpar, Mendelwicz, & Tylee, 1997). These findings are comparable to rates reported in Canadian (Parikh, Wasylenki, Goering, & Wong, 1996) and U.S. (Weissman, Bruce, & Leaf, 1990) samples, as well as in a Norwegian sample (Kringlen, Torgersen, & Cramer, 2001). In the U.S. population, at any one time, 10% had experienced clinical depression in the past year, and between 20-25% of the women and 7-12% of the men would suffer a clinical depression during their lifetime. In the Norwegian study it was found that at any one time 3-5% of the population are experiencing a clinical depression, and that during their life time, 24% of the women and 10% of the men have experienced a major depression (Kringlen et al., 2001).

In the broadest sense, the term depression includes a number of meanings from the description of everyday moods of feeling down or blue, to the description of serious depression with psychotic symptoms and increased risk of suicide. Depression includes emotional (e.g. depressed mood), motivational (e.g. loss of interest or pleasure), cognitive (e.g. negative thoughts, feelings of hopelessness), and somatic (e.g. loss of energy, sleep disturbances) symptoms (American Psychiatric Association, 1980). Almost everybody will experience some of these symptoms in more or less degree. This is just as normal as experiencing happiness, interest and motivation, and thinking good about oneself and the future. However, when the symptoms have just been too many, too intense and are lasting too long in such a way that it interfere with social life, studies and working life, the depression is described as a clinical depression or a major depression, i.e., a depression that requires treatment.

In depression research, an important question concerns whether a clinical depression is a qualitatively different experience from the experience of just feeling down for some days. In other words, is there also a qualitative threshold or are there just quantitative differences in symptoms, which distinguish these two conditions? The answer to this question has important implications for how clinical depression is defined and studied, i.e., as a cut-off point on self-rating scales or in accordance with categorical diagnostic criteria as outlined by diagnostic systems such as the International Classification of Diseases (ICD-10; World Health Organization, 1992) and the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1980).

An important difference between diagnostic systems and self-rating scales is how diagnostic systems emphasize the duration of depressive symptoms (i.e., at least two weeks) in addition to the amount, and intensity of symptoms. Also, the clinical diagnoses are made by interviews and not by questionnaires. Such differences between the diagnostic criteria for a clinical depression and cut-off points on self-rating scales may possibly grasp the qualitative difference between the experience of a clinical depressive episode compared to passing high depression scores on a self-rating scale. While much of previous depression research have relied on depression defined according to high scores on self-rating scales, the "gold standard" for defining population for study has now been clinical depression as defined according to diagnostic criteria (Gotlib & Hammen, 2002).

The DSM-IV distinguishes between different diagnostic categories and divides mood disorders into two main categories, which include depressive disorders (major depressive disorder, dysthymic disorder, depressive disorder NOS) and bipolar disorders (bipolar I, bipolar 2, cyclothymic disorder, bipolar disorder NOS). Also, the disorders may be specified according to the severity of the last episode, the recurrence of episodes and to whether the disorder has become chronic. More recently, however, researchers have argued that major depression may not be an episodic, but a chronic disorder in the sense of long-term vulnerability (Segal, Williams, & Teasdale, 2002). The reason is that at least 50% of patients who recover from an initial episode of

depression will have at least one subsequent depressive episode (Paykel, et al., 1995). Moreover, patients with two or more past episodes will have a 70-80% likelihood of recurrence in their lives (Consensus Development Panel, 1985).

The findings that the risk of getting new depressive episodes increases with number of previous episodes, also question whether the additional numbers of recurrent episodes is a vulnerability factor for experiencing new episodes (Coyne, Flynn, & Pepper, 1999; Solomon et al., 2000). Furthermore, while the first depressive episode seems to be associated with negative life events, this association does not seem to be as strong for the recurrent episodes of depression (Zuckerman, 1999). Accordingly, it is nearby to conclude that depressive episodes by itself may act as a vulnerability factor. The important question to ask, then, is why? An answer to this question is essential when planning clinical interventions to prevent relapse and recurrence in depression. Obviously, preventing relapse and recurrence in depression will have the potential to reverse the trends of depression as the leading cause of disability in the world.

2.2 Cognitive vulnerability to depression

The first cognitive approaches to depression developed in the late 1960s and the early 1970s. The new approaches were inspired from the rise of information-processing approaches in psychology, especially from the study of social cognition (e.g. Nissbett & Ross, 1980), and by Lazarus' research (1966), which demonstrated that specific contents of thoughts (e.g. threat and danger) mediated emotional reactions (e.g. anxiety). Also, it was particularly appealing for investigators on depression that cognitive models were testable, which had not been the case for the psychoanalytic approach to depression. A common feature of the cognitive approaches was the emphasis placed on cognitive processes in etiology, maintenance, and treatment of depression (Abramson et al., 2002).

Aaron Beck's (1967) theory of negative automatic thoughts as triggers of depressive symptoms and Martin Seligman's (1975) theory of learned helplessness and depression were two of the first cognitive models of depression. Both of them have strongly influenced the cognitive approach. During the last decades, however, several cognitive theories have been developed and partly in competition with each other. Today, however, there is a tendency present to integrate the different models in the sense that they cover different aspects of the cognitive approach. For example, Ingram, Miranda and Segal (1998) distinguish between cognitive models that focus on cognitive structures, cognitive operations, and cognitive products. Also, researchers have tried to understand how cognitive aspects of depression may be understood in a self-regulatory and psychobiological context (Abramson et al., 2002), in relation to frontal brain asymmetry (Tomarken & Keener, 1998), and in an interpersonal context (Joiner & Coyne, 1999).

2.2.1 Vulnerability and the diathesis-stress relationship

People are vulnerable to the extent that they are susceptible to being hurt or wounded. In psychological domains this may imply an increased susceptibility to emotional pain and the occurrence of psychopathology of some type (Ingram et al., 1998). Vulnerability can consist of both inherited (e.g. genetic, family history of psychopathology, temperament, personality) and acquired (e.g. trauma, family experiences, peer interactions, other life events) factors (Zubin & Spring, 1977). However, whether a social experience will become a vulnerability factor will often depend on the interaction between inherited factors and social events.

Several researchers distinguish between trait and state variables (Hollon, Evans, & DeRubeis, 1990; Hollon & Cobb, 1993). Whereas vulnerability is mostly regarded as an enduring trait, which predisposes individuals to a disorder, but do not initiate the disorder per se, the state variables represent the occurrence of the symptoms that reflect the onset of the disorder. The stability of trait variables may differ according to the genetic component making some trait variables very stable and almost permanent (e.g. bipolar disorder), whereas other trait variables are more easily changed through correcting learning experiences. A trait may be either strengthened or weakened through new experiences (Ingram et al., 1998).

Vulnerability predisposes individuals to a disorder, but do not initiate the disorder. The diathesis-stress relationship explains how latent vulnerability becomes activated through events perceived as stressful by the individual. The diathesis refers to the predisposition to illness, whereas stress refers to the individual's subjective perception of an event as stressful.

Whether an individual experiences an event as stressful depends on the individual's previous experience with similar events, which in turn have contributed to the vulnerability. Accordingly, as Ingram et al. (1998) state: "psychopathology is thus the interactive effect of the diatheses and events perceived as stressful (p. 78)".

2.2.2 How to study cognitive vulnerability?

Ingram and Siegle (2002) distinguish between distal and proximal vulnerability factors. While developmental antecedents (e.g. negative self-schemata) are defined as distal vulnerability factors, proximal vulnerability factors may be cognitive dispositions (e.g. negative automatic thoughts), which are the result of the distal vulnerability, but which appear just before the onset of the depressive episode. Different research designs are available depending on whether the focus of research is on distal or proximal vulnerability factors. Methodological strategies in the study of distal vulnerability include high-risk research and longitudinal designs which try to decide whether factors in childhood determine depressive disorder later in life. In the study of proximal vulnerability, research has made use of cross-sectional, remission, and priming designs.

The focus of the present study is on proximal vulnerability factors and the design of the study is a cross-sectional remission design. Accordingly, the literature review below will also be limited to proximal factors. The intent of most remission studies is to examine the stability of potentially, causative factors, i.e., cognitive vulnerability traits. The assumption is that such traits should be stable, and if so, empirically detectable. However, and as discussed above, in the last decade, investigators on depression have been increasingly aware that a depressive episode, by itself, may be a vulnerability factor to recurrent episodes. Accordingly, by examining previously depressed individuals it may be difficult to distinguish between vulnerability factors that may be the consequence of having experienced a previous depressive episode, and vulnerability factors that contributed to the first depressive episode.

2.3 Negative self-schemata and negative knowledge structures

Beck's (1967, 1976) cognitive theory of depression suggests that individuals who have experienced loss or adversity in childhood will develop negative self-schemata. Negative self-schemata are thought to be relatively stable across time, situations, and mood-states but also to be relatively dormant and inaccessible during non-depressive states. Clark and Beck (1999) suggest that negative self-schemata can be activated by a wide range of negative and stressful life events or situations, but especially by stress which reminds the individual of the experience when the negative self-schemata was established. When activated, negative self-schemata tend to generate negative automatic thoughts and to negatively bias the individual's self-referent information processing. This will in turn trigger depressive symptoms. Also, Beck (1976, 1987) assumed that negative self-schemata contain dysfunctional attitudes concerning loss, failure and abandonment. Dysfunctional attitudes include beliefs such as one's happiness depends on being perfect, being in control, or on other people's approval (Beck, Hallon, Young, Bedrosian, & Budenz, 1985). When activated, dysfunctional attitudes will negatively influence the individual's coping style and automatic compensatory strategies.

Beck's cognitive theory is a model that places the key emphasis on putative depressogenetic cognitive structures. Also, incorporated in Beck's model is other cognitive elements such as thoughts and cognitions and various cognitive processes, but this is not the central organizing principle of this approach (Ingram et el., 1998). However, cognitive structures can only be tested through the content or the processes they generate. Numerous of studies have reported that currently depressed individuals report more evidence of dysfunctional cognitive structures, than those who are not currently depressed (Clark & Beck, 1999).

2.3.1 The processing of emotionally valenced information in currently depressed individuals

Early studies examining postulates and hypothesis derived from cognitive theories relied in large part on self-report methodologies to assess the negativity of the *content*

of the cognitive structure of depressive individuals. From this research there is a large literature showing that depressed individuals have significantly higher levels of self-reported dysfunctional attitudes and negative automatic thoughts, more negative attribution style, and more negative autobiographical memories (Clark & Beck, 1999; Gotlib & Abramson, 1999; Gotlib & Krasnoperova, 1998; Gotlib, Kurtzman, & Blehar, 1997).

More recently, investigators have employed experimental methodologies to assess cognitive *processes* in depression such as attention, encoding, retrieval, and interpretation of emotionally valenced information. An advantage with these methodologies, as opposed to self-report methodology, is that they allow the assessment of more automatic, as opposed to strategic and controlled functioning (Gotlib & Neubauer, 2000). This is important because the cognitive structure models of depression emphasize that negative self-schemata (Beck, 1967, 1976) and associative networks (e.g. Bower, 1981, 1987; Teasdale, 1988) are activated automatically and outside awareness.

There has been extensive research on cognitive processes in depression, which have generated enormous amount of data, especially on selective attention and memory. Although much of the research support the hypothesis derived from cognitive theories that currently depressed individuals, compared to non-depressed controls, are negatively biased in information processing, there are also discrepancies across studies (for a review; Clark & Beck, 1999; Gotlib & Neubauer, 2000). One of the discrepancies has been whether depressed individuals process information in a negatively biased direction or if they have just "lost" the positivity bias, or the 'illusory optimism', which has been found to characterize non-depressed individuals (for a review on this area, see Alloy & Abramson, 1988). Several explanations of the discrepant findings have been suggested, including the relevance of the stimuli to the concerns of depressed individuals, the method of the study, the co-occurrence of anxiety symptoms, the severity of the depressive symptoms, and whether the different cognitive processes are differently affected by negative self-schemata and negative knowledge structures (Clark & Beck, 1999; Gotlib & Neubauer, 2000).

With regard to cognitive processes, two research groups have reviewed the research literature and have come up with different conclusions (i.e., Clark & Beck, 1999; Williams, Watts, MacLeod, & Mathews, 1997). The difference may be related to how the research groups have defined cognitive processes according to the continuum of automatic to effortful processing. For example, Clark and Beck (1999) argue that cognitive processes seldom are purely automatic, or purely strategic, and that it may be better to consider them in terms of the defining characteristics of automatic and strategic processing. For example, according to schemata theory of depression it is reasonable to assume that cognitive processes involving more integrative and elaborative processes will be more affected by negative self-schemata than earlier perceptually based processes. However, the earlier, more perceptually-based processes may also be more affected by negative self-schemata because they are more automatic

according to the defining characteristics of automatic/strategic processes (Beck & Clark, 1997; Hartlage et al., 1993; Williams et al., 1997). Furthermore, Clark and Beck (1999) argue that in depression positive self-schemata have low resting activation levels, requiring effortful processing to reach threshold. Because research has shown that depression interferes with effortful processing, but not with automatic processing (Hartlage et al., 1993), Clark and Beck (1999) suggest that depression would be characterized by enhanced automatic processing of negative self-referent information, but decreased effortful processing of positive stimuli.

Also, Williams et al. (1997) conclude that emotional disorders may affect different aspects of cognitive processing and distinguish between two main processes that operate upon mental representation, priming and elaboration. In contrast to Clark and Beck (1999) they argue that depression is primarily characterized by a bias in elaboration, i.e., in the strategic and conceptually based memorial processing of negative self-referent information. A negativity bias in priming, which they describe as an automatic and selective perceptual encoding of information, they conclude is present in anxiety, but not in depression. Their argument is based on empirical findings and on theoretical viewpoints. For example, to avoid threatening stimuli, anxious individuals need to be alert to negative information and this will negatively bias priming processes, but not elaboration processes. Depressed individuals, on the other hand, have no need to be especially attentive to negative stimuli, accordingly no bias in priming processes will occur. However, after this first stage of passive intake of partial information from the environment (i.e., priming), the information is mapped on to internal representations or schemata. These schemata will then accommodate the information and direct further processing resources during the next intake cycle towards the most salient stimulus. For individuals with negative self-schemata, this will be negative self-referent information. Accordingly, Williams et al. (1997) suggest that depression affects the active strategic element of memory retrieval, enhancing the recall of negative material.

2.3.2 Studies supporting depressogenetic cognitive structures in previously depressed individuals

Along with the research on cognitive processes in currently depressed individuals, there has also been an extensive examination of individuals supposed to be cognitively vulnerable to depression (Clark & Beck, 1999; Ingram et al., 1998). However, a serious problem with Beck's theory is that depressogenetic cognitive structures have shown to be less detectable when the depressive episode remits, indicating that dysfunctional structures may rather be a state than a trait characteristic.

According to Beck's theory of latent negative self-schemata, these findings are not very surprising. The problem has been to find an adequate priming procedure that is able to activate the depressogenetic cognitive structures. Because negative self-schemata are thought to be established as a result of *personally* experienced negative events, experiences that may vary greatly from person to person, it has been a major

methodological problem to find relevant primers to activate negative self-schemata (Clark & Beck, 1999).

The help came from an information processing approach to depression, also with the focus on cognitive structures. In 1981 Bower developed a model of mood and memory and argued that associative networks are developed between mood nodules and memory nodules. As a result, mood can precipitate changes in thinking and changes in thinking can precipitate changes in mood (Ingram et al., 1998). Based on Bower's model of mood and memory, Ingram (1984) and Teasdale (1988) have, independently of each other, developed two similar information-processing models of depression. Generally, a common focus of these two models is that vulnerability is conceptualised as the availability of cognitive networks, or structures, that are associated with affective structures of sadness. A consequence of these models is the suggestion that dysphoric mood may be involved in the activation of negative thinking.

This mood-state hypothesis of the activation of negative thinking has been tested in several studies. For example, Miranda, Gross, Persons, and Hahn (1998) experimentally induced dysphoric mood in their subjects and found that previously depressed individuals who reported increased negative mood also reported increased dysfunctional attitudes, whereas less vulnerable individuals who reported increased negative mood, reported decreased dysfunctional attitudes. Other studies have found a positive correlation between dysphoric mood and dysfunctional thinking in previously depressed individuals, but not in individuals who had never been depressed (Miranda & Persons, 1988; Miranda, Persons, & Byers, 1990; Roberts & Kassel, 1996).

Central to the mood-state hypothesis is the suggestion that dysphoric mood work as a primer of depressogenetic cognitive structures. Hartlage et al. (1993) argue, however, that dysphoric mood contributes to depression by decreasing the effortful processing of information making the more automatic processing of well-learned negative thinking in cognitively vulnerable individuals more accessible. Accordingly, dysphoric mood may be involved either as a primer or by decreasing effortful information processing. And, as discussed above, according to Clark and Beck (1999), decreased effortful processing will also involve decreased processing of positive information in addition to increased processing of negative self-referent information.

2.4 Self-regulation, coping strategies and meta-cognition

Another explanation of what may regulate negative thinking in vulnerable individuals come from cognitive models which focus more on cognitive *operations* (Ingram et al., 1998) or the way people deal with dysphoric mood or negative situations (Segal et al., 2002). For example, from an extensive research on mental control in depression, Wenzlaff and colleagues argue that previously depressed individuals are characterized by actively suppressing dysfunctional thinking in an attempt to ward off the depressive thoughts that threaten their emotional well-being. Wenzlaff, Rude, and West (2002) argue that the mood-state hypothesis (Miranda & Persons, 1988) is vague concerning

how dysfunctional attitudes become dormant and it seems that the prevailing explanation is that "when external circumstances improve, negative cognitions ebb and eventually become dormant, thereby facilitating a return to a normal state (p. 535)". As an alternative explanation, Wenzlaff and colleagues suggest thought suppression. However, thought suppression will have the ironic consequence of triggering the automatic processing system to be especially alert on the negative information to be suppressed. Consequently, when effortful processing is decreased by dysphoric mood, the ability to suppress dysfunctional thinking will decrease and the ironic processing of negative stimuli will dominate the individuals information-processing (Wenzlaff & Wegner, 2000).

Wenzlaff et al. (2002) suggest that formerly depressed individuals in an attempt to *maintain* their emotional well-being are cognitively characterized by continuously suppressing dysfunctional thinking. Self-regulation theories of depression are, however, emphasizing more what happens to cognitively vulnerable individuals when confronted with a stressful situation. In those theories, a negative event, or an experience of "discrepancy" in a situation, initiates a shift in attention to evaluate the current situation (Gray, 1994; Higgins, 1987; Pyszczynski & Greenberg, 1987). This shift in attention begins with attention directed internally to focus on the self (Carver & Scheier, 1998), which allows individuals to compare their current state with their desired state and to initiate behaviour to reduce the discrepancy. Such a shift in attention is generally an adaptive response because people switch their attention to the problem in an attempt to resolve it (Abramson et al., 2002). In other words, this is a normal, healthy coping strategy. However, while less vulnerable individuals are able to disengage from this self-focused attention, cognitively vulnerable individuals seem to become stuck in this checking process. For example, holding the dysfunctional attitudes of rigid and perfectionist standards may both make it more difficult to solve problems, and to adjust one's goals in the face of thorny problems.

Nolen-Hoeksema (1991) describes the cognitive condition of being stuck in a checking process as depressive rumination. According to Nolen-Hoeksema, rumination is the cause of why individuals who maintain depressive mood states differ from individuals who are able to cope effectively with passing dysphoric mood. More recently, depressive rumination has been understood from a meta-cognitive perspective (Segal et al., 2002; Wells, 2000). Papageorgiou and Wells (2001) argue that individuals who are cognitively vulnerable to depression both held positive and negative beliefs about rumination. Positive beliefs reflect themes concerning rumination as a coping strategy and motivate individuals to engage in sustained rumination (i.e., "I need to think about things in this way to find answers to my depression and reduce my distress"). However, thinking about negative aspects of the self or the negative situation rather serves to perpetuate than to resolve the negative feelings (Segal et al., 2002). As a consequence, negative beliefs about rumination will arise and be reflected in themes concerning the uncontrollability and the harm of rumination, and its interpersonal and social consequences. Papageorgiou and Wells (2003) suggest that it is especially the activation of negative beliefs that contributes to

the experience of depression. Accordingly, Papageorgiou and Wells argue that rumination is a coping strategy, which ultimately backfires.

Self-regulation may also be understood in a psychobiological perspective. Gray (1994) has proposed two systems that are critical in the regulation of behaviour: The Behavioural Approach System (BAS) and the Behavioural Inhibition System (BIS). While the BAS is sensitive to signals of reward, non-punishment, and escape from punishment; the BIS is sensitive to signals of punishment and non-reward (e.g. failure). While the activation of BAS causes the person to begin movement toward goals, the activation of BIS inhibits behaviour that may lead to negative or painful outcomes. Signals activating BIS or BAS can either be external events or internal cognitions. While BAS is assumed to be associated with positive emotions such as hope, elation, and happiness, BIS is assumed to be associated with negative affect and anxiety (Carver & White, 1994).

From the self-regulation perspective, it is the BIS that will be activated when the individual experience a negative event or a "discrepancy" in the situation. When less vulnerable individuals are able to disengage from the self-focused attention and resume goal-seeking activity, this will probably be reflected in a deactivation of BIS and a reactivation of BAS. In cognitively vulnerable individuals, however, who are not able to disengage from the ruminative checking, one may expect a large-scale deactivation of BAS, which may, if strong enough result in a depressive episode (Abramson et al., 2002).

Crowe and Higgins (1997) distinguish between promotion and prevention strategies for self-regulation and suggest that people differ in degree to whether they are prevention or promotion focused. Promotion focus, or nurturance-related regulation, is concerned with ideals, advancement, aspiration, and accomplishment, whereas prevention focus, or security-related regulation, is concerned with oughts, protection, safety and responsibility. Also, recent personality research has found that people may differ with respect to approach or avoidance focus in the conceptualisation of personal goals (Elliot & Sheldon, 1997; Elliot, Sheldon, & Church, 1997). Having negative self-schemata containing dysfunctional attitudes may possibly contribute to such differences between people. For example, Abramson et al. (2002) argue that individuals characterized by a combination of perfectionism and high self-efficacy may show relatively high approach motivation. On the other hand, individuals with a combination of perfectionism and low self-efficacy may probably show a relatively high prevention motivation. Accordingly, high levels of dysfunctional attitudes may possibly be associated with a prevention or avoidance focus.

The suggestion that activated negative self-schemata will negatively bias the information processing in depression is central to Beck's cognitive theory of depression. However, and as discussed above, Clark and Beck (1999) also assume that decreased effortful processing, as a result of dysphoric mood, will involve decreased processing of positive stimuli. This assumption is in line with self-regulation theory

that suggests that confronted with a negative event or discrepant situation, cognitively vulnerable individuals may exhibit decreased approach motivation to positive information. Also one may assume that along with the deactivation of BAS, vulnerable individuals will move from having a positivity bias in their information processing, to be fifty-fifty, to finally be negatively biased in self-referent information processing. This viewpoint may resemble that of other researchers who have suggested that it may be the equality between positive and negative thoughts that puts people at risk for depression (Ingram & Smith, 1984; Kendall & Hollon, 1981). Also, it resembles that of the self-regulation perspective of depression, which holds that the core problem in depression may be the regulation between BIS and BAS.

2.5 A depressive episode as a vulnerability factor for depression

As discussed above, research has found that the risk of getting another depressive episode increases with number of previous episodes (Consensus Development Panel, 1985). Furthermore, while the first depressive episode is associated with negative life events, this association is not that strong for recurrent depression (Post, 1992; Zuckerman, 1999), indicating that the depressive episode by itself has been a vulnerability factor (Coyne, et al., 1999; Solomon et al., 2000). Several researchers have tried to explain why this is the case.

From a neurobiological perspective, Post (1992; Post & Weiss, 1995), has proposed a kindling-sensitization model which suggests that with each episode of depression, the neurotransmitter systems become more easily dysregulated. While a strong stressor is needed for the dysregulation initiating the first episode, only mild stressors are required for the subsequent episodes. This neurobiological model is in accordance with the cognitive information processing models (Ingram, 1984; Teasdale, 1988). For example, Ingram et al. (1998) suggest that because the depressive knowledge structures, for each depressive episodes, are deployed in a growing number of contexts, this will lead to a situation where even small changes in mood would be sufficient to activate them. Consequently, Ingram et al. (1998) conclude that the relapse and recurrence of depressive episodes can be viewed as the "retriggering" of the patterns of biological and information-processing activity that characterize the initial episode.

Also, Segal et al. (2002) emphasize how small changes in mood are sufficient to activate depressive knowledge patterns, but they also recognize how the tendency in cognitively vulnerable individuals to ruminate about the easily accessible negative material, actually worsen the situation. It seems obvious that ruminating about the negative material only will strengthen the depressive knowledge pattern. While the biological and information processing models, and also the rumination model as outlined by Nolen-Hoeksema (1991), emphasize the relatively passive contribution of the cognitively vulnerable individual, Papageorgiou and Wells (2003) more strongly emphasize how the individual, by the use of dysfunctional coping strategies, actively

contributes to the escalation process of dysphoric mood into depressive relapse or recurrence. For, as discussed above, according to Papageorgiou and Wells (2003) rumination is a coping strategy, which ultimately backfires. Also, Wenzlaff et al. (2002) are more emphasizing how mental control strategies, such as suppression (the ironic processing hypothesis), actively contribute to the escalation process. Interestingly then, two qualitatively different coping strategies, i.e., suppression and rumination, are suggested to be responsible for the escalation process of dysphoric mood to clinical depression.

Central to theories of depression is the assumption that previous life events, primarily experienced in childhood, make individuals vulnerable to depression (i.e., distal vulnerability factors). One of their common features is how they emphasize the class of experiences, which are associated with the loss of emotional care, social reinforcement, self-worth, and feelings of control (for a review; Gotlib & Hammen, 2002). For example, Martin Seligman's (1975) theory of learned helplessness and depression, which later was reformulated to the theory of learned hopelessness (Abramson, Seligman, & Teasdale, 1978), was developed from research which had shown the deleterious effects of uncontrollable aversive events.

It is striking, however, how little attention that has actually been paid to the fact that a clinical depressive episode by itself may be experienced as a highly uncontrollable and traumatic life event. In the clinic, it is quite common that clients express such experiences with their depressive episode. In the research on cognitive vulnerability factors to depression, this approach has been almost absent. Certainly, several researchers have discussed how the depressive condition may result in social rejection and lowered self-worth and thereby reinforce the depressive symptoms (for a review; Joiner & Coyne, 1999). However, limited attention has been devoted to increase our understanding of how previously depressed individuals react and cope when faced with a situation that reminds them of a previous depressive episode. For example, do they feel anxious of again loosing control, i.e., turning into a new depressive episode? And do they try to cope with such a situation by avoiding and suppressing negative thinking and dysphoric symptoms? Or are they *continuously* suppressing depressive thinking in an effort to maintain emotional well-being as Wenzlaff and colleagues have suggested?

The present study has placed these questions regarding the mechanisms of recurrent depression on the agenda and has gathered some data, which may support a Cognitive battle model of recurrent depression. Also, the present study has investigated whether decreased approach motivation may be a vulnerability factor to depression, and how automatic and effortful processes are involved in the processing of emotionally valenced information.

3. SPECIFIC AIMS OF THE STUDY

The general aims of the study have been to: (1) develop a cognitive model of recurrent depression; (2) investigate whether decreased approach motivation may be a vulnerability factor to depression; and, to (3) investigate how automatic and effortful processes are involved in the processing of emotionally valenced information in depression.

In addition to the general aims of the study, there were several more specific aims:

- To examine how dysfunctional attitudes and dysphoric symptoms are related to each other in each group of participants, respectively (paper 1).

- To examine how dysfunctional attitudes and dysphoric symptoms are related to the cognitive processing of emotionally valenced information in two different information processing tasks, including listening preferences for tape-recorded self-statements and choice preferences in a visual attention task (paper 1, paper 2).

- To examine how increased reaction time in a visual attention task may be a state-independent measure of decreased approach motivation in individuals cognitively vulnerable to depression (paper 2).

- To examine how dysfunctional attitudes and dysphoric symptoms are related to reaction time to positively and negatively valenced words in a visual attention task (paper 2).

- To examine how dysfunctional attitudes and dysphoric mood are related to automatic and effortful processing of positive and negative self-statements (paper 3).

- To examine how automatic and effortful processing are related either to an increased or to a decreased processing of positive or negative self-statements (paper 3).

4. METHOD

4.1 Participants

The study included 149 participants who were either clinically depressed, had experienced a depressive episode in the past, or had never been clinically depressed. All the participants took part in the whole procedure of the study. Accordingly, the participants are the same in all three papers. Demographic and clinical characteristics of subjects are presented in Table 1.

Table 1

Demographic and Clinical Characteristics of Subjects

	Clinically depressed (n = 61)		Previously depressed (n = 42)		Never depressed (n = 46)	
Variables	*M*	*SD*	*M*	*SD*	*M*	*SD*
Sample (patients/students)	36/25		17/25		18/28	
Sex (f/m)	52/9		35/7		35/11	
Age	30.9	10.3	27.0	8.3	26.9	9.5
BDI_1	22.4	9.9	11.2	5.7	1.8	1.7
BDI_2	19.0	7.5	7.4	4.8	1.6	1.7
BDI_3	15.7	8.5	6.4	4.1	1.1	1.7
DAS	133.6	38.3	116.6	30.4	93.9	21.4
Medication:						
Antidepressant	9		1			
Neuroleptics	1		1			

The participants were recruited from two different populations, i.e., undergraduate students at the University of Tromsø, and patients consulting their general practitioner, also in Tromsø. The decision to recruit subjects from these two populations was three-folded: Firstly, because we wanted to include in the study both clinically, previously and never depressed individuals, we had to find populations were it would be possible to get this three categories of individuals. Furthermore, the decision to recruit participants from *two* different outpatient populations was pure practical, i.e., in a limited period of time we needed a large number of participants to get enough subjects

to each group. Finally, we decided to include only clinically depressed individuals who were *outpatients*, i.e., not hospitalised. This decision was both practically and theoretically founded.

The study was designed with a procedure of about six hours administrated on two different days of testing. We thought that it would be difficult to motivate more severely depressed individuals to take part in a study in an order of that size. From a more theoretical point of view, however, it would be especially interesting to examine individuals with less severe depressive symptoms, but who were still diagnosed with a clinical diagnostic interview. After a review of previous research on depression, we found that such a sample of outpatient-depressed individuals was relatively rare in the literature. Research had either focused on students with depressive symptoms as measured with different self-rating scales, but who were not clinically diagnosed, or on clinically diagnosed inpatients with more severe depressive symptoms and often on medication.

4.2 Procedures

In an initial screening procedure a questionnaire including items about current (Beck Depression Inventory: BDI_1; Beck Rush, Shaw, & Emery, 1979) and previous depression (Previous Depression Questionnaire: PDQ; Wang, 1996) was administered to approximately 800 undergraduate students and to approximately 600 patients consulting their general practitioner. From the sample of individuals who returned the questionnaire by mail, subjects were invited to participate if their answers to the questionnaire indicated that they were: (1) clinically depressed; (2) were not clinically depressed but had experienced a previous depression; or (3) were neither clinically depressed nor had ever experienced a depressive episode (cf. paper 1). The invitation to participate in the study was made by telephone and an appointment for the first of two testing days was made. However, all the potential participants were informed about a second screening, i.e., initially they knew that some of the participants would finish after a clinical interview and some would continue the study.

4.2.1 The first day of testing

Before the second screening procedure, all participants answered the Depression Adjective Check List ($DACL_1$; Lubin, 1965) and then completed the BDI_2. Based on information from the clinical diagnostic interview (Structured Clinical Interview for *DSM-IV*, Axis I disorders; SCID-CV; First, Spitzer, Gibbon, & Williams, 1997), several individuals were excluded from the study because they either failed to meet the full criteria for a current or a previous depression, and for some other reasons (cf. paper 1). Accordingly, the final group assignment was made according to the clinical interview and not according to self-reported symptoms on a self-rating scale. The group-classification reliability was tested and a highly satisfactory reliability was found (cf. paper 1).

For the individuals who did not meet the requirements, the participation in the study was finished after the SCID-interview. The remaining others continued the procedure of the first day by answering several questionnaires (those with reference, not reported on in the present papers) and doing one experimental task in the following order: the Hope Scale (Snyder et al., 1991), the Unrealistic Optimism Scale (Weinstein, 1980), the $DACL_2$, the Crowson's Auditory Forced Choice Device, the Tonal Quality (Crowson & Cromwell, 1995), the $DACL_3$, the Post-Experimental Questionnaire, measures of free recall and recognition, and finally the $DACL_4$.

4.2.2 The second day of testing

Like the first day of testing, the procedure of the second day included several questionnaires and an experimental task (those with reference, not reported on in the present papers), and the participants were tested in the following order: BDI_3, the Ivac Core Check (Tympanic Thermometer System, Model 2090), the Deployment of Attention Task, the California Verbal Learning Test (Delis et al., 1987), the Dysfunctional Attitude Scale (DAS), the Schema Questionnaire (Young, 1990), and finally the Seasonal Pattern Assessment Questionnaire (Rosenthal, Bradt, & Wehr, 1984).

4.3 Methods

4.3.1 Measurements

The Beck Depression Inventory (BDI; Beck et al., 1979) was included in the study to measure the severity of depressive symptoms on the two separate days of testing (BDI_2 & BDI_3), and to select potentially participants to the study (BDI_1). BDI-scores are classified as follows: normal range, 0-9; mild-moderate depression, 10-18; moderate-severe depression, 19-29; and serious depression, 30-63 (Beck & Steer 1987). The means and standard deviations for each group are presented in Table 1.

The Previous Depression Questionnaire (PDQ; Wang, 1996) was developed to, in the initial screening, identify currently nondepressed individuals who had previously been depressed and to identify individuals who had never experienced a depressive episode. The PDQ was constructed using DSM-IV criteria for a past major depressive episode.

The Structured Clinical Interview for *DSM-IV*, Axis I disorders (SCID; First et al., 1997) is a semi-structured interview administered individually by a trained interviewer. It is designed to identify diagnosis as outlined in the Diagnostic and Statistical Manual of Mental Disorders, Axis I disorders (American Psychiatric Association, 1994).

The Dysfunctional Attitude Scale (Form A)(DAS; Weissman & Beck, 1978) was included in the study to measure the presence of dysfunctional attitudes that may relate to cognitive vulnerability to depression (Oliver & Baumgart, 1985). The content

of items concerns the need for approval, dependency, perfectionism performance standards, and rigid ideas about the world. Scores on the DAS can range from 40 to 280, with higher scores indicating more dysfunctional attitudes. Scores above 125 are considered as high. The means and standard deviations for each group are presented in Table 1.

The Depression Adjective Check List (Form E)(DACL; Lubin, 1965) was included in the study to measure rapid mood changes during the first day of testing. Especially we wanted to test whether the listening preference task, described below, had any impact on mood.

The Post-Experimental Questionnaire (PEQ; Crowson & Cromwell, 1995) was included to the study to assess the participants' impressions after the listening preference task.

Recognition was measured with a questionnaire to measure recognition of self-statements from the listening preference task (Wang & Holte, 1995), whereas free recall and fabrication were measured with an empty page with an instruction to write all the sentences and phrases the participants could remember from the task.

4.3.2 Apparatuses and materials

The Crowson's Auditory Forced Choice Device (Crowson & Cromwell, 1995) is equipment designed to measure listening preferences for positive and negative tape-recorded self-statements where the dependent variable is total listening time to each tape. The negative self-statements characterize depressive self-talk (Holon & Kendall, 1980), whereas the positive self-statements characterize positive self-talk among depressed as well as non-depressed individuals (Ingram & Wisnicki, 1988).

The Deployment of Attention Task (Gotlib, McLachlan, & Katz, 1988; Kakolewski, Crowson, Sewell, & Cromwell, 1999; McCabe & Gotlib, 1995) is a task designed to measure visual attention where the dependent variables are choice and reaction time. The stimulus material consists of positive, negative and neutral words.

5. SUMMARY OF PAPERS

Paper I

Wang, C. E., Brennen, T., & Holte, A. (2005). Mechanisms of recurrent depression: A cognitive battle model and some preliminary results. Clinical Psychology and Psychotherapy, 12, 427- 442.

Theoretical models of cognitive mechanisms assumed to be involved in recurrent depression are discussed and a cognitive battle process between compensatory coping strategies and the automatic processing of negative information is suggested. Preliminary support for the model comes from a study that investigated preferences for positive and negative tape-recorded self-statements in clinically depressed (CD), previously depressed (PD), and never depressed individuals (ND). The results showed: 1) A positive correlation between dysfunctional attitudes and dysphoric symptoms in CDs and PDs, but not in NDs; 2) NDs preferred positive self-statements, whereas CDs preferred neither positive nor negative self-statements; 3) PDs exhibited different patterns of preference depending on the levels of dysfunctional attitudes and dysphoric symptoms. For example, simultaneous high levels of both dysfunctional attitudes and dysphoric symptoms in PDs resulted in a preference for positive self-statements. This finding is discussed as a possible compensatory strategy of avoiding negative information in PDs. Clinical implications for treatment and prevention of depression are discussed.

Paper II

Wang, C. E., Brennen, T., & Holte, A. (2005). Decreased approach motivation in depression. Scandinavian Journal of Psychology, in press.

The present study examined relations between choice preference and reaction time to emotionally valenced words, dysphoric symptoms (BDI), and dysfunctional attitudes (DAS) in clinically depressed (CD; n = 61), previously depressed (PD; n = 42), and never depressed controls (ND; n = 46). The results showed: 1) NDs and PDs exhibited a choice preference for the relatively more positive words and differed significantly from CDs; 2) PDs and CDs exhibited longer reaction time and differed significantly from NDs; and 3) BDI and DAS were positively associated with reaction time to positively valenced words, whereas no associations were found for reaction time to negatively valenced words. The increased reaction time, in PDs and CDs, is discussed as a possible vulnerability factor to depression, which may be related to decreased approach motivation.

Paper III

Wang, C. E., Brennen, T., & Holte, A. (2006). Automatic and effortful processing of self-statements in depression. Cognitive Behaviour Therapy, 35, 117-124.

Clark and Beck (1999) and Williams et al. (1997) have come up with quite different conclusions regarding which cognitive processes are most affected by negative self-schemata and negative knowledge structures. In order to increase the understanding of differences in effortful and automatic processing in depression, we compared never depressed (ND), previously depressed (PD) and clinically depressed (CD) individuals on free recall, recognition and fabrication of positive and negative self-statements. The results showed that: 1) overall NDs and PDs recalled more positive self-statements than CDs, whereas CDs correctly recognized more negative self-statements than NDs and PDs; and, 2) CDs and PDs fabricated more negative than positive self-statements, whereas no difference was obtained for NDs. The results seem to be in line with Clark and Beck's suggestions. However, there are several aspects of the present findings that make the picture more complicated.

6. GENERAL DISCUSSION

6.1 Summary of results

A Cognitive Battle Model

The main result of the present study is the development of a Cognitive battle model to explain mechanisms involved in recurrent depression (paper 1; figure 1). The model proposes an escalation process of dysphoric mood to clinical depression where compensatory coping strategies, such as avoidance and suppression, may be responsible for a "cognitive decomposition" with an increased risk for a new episode of clinical depression. The model emphasizes that suppression and avoidance are coping strategies which are situation-released to cope with the anxiety of again loosing control, i.e., turning into a new depressive episode. This model contrasts Wenzlaff and Wegner's model (2000), which propose that previously depressed individuals are *continuously* suppressing dysfunctional thinking in order to maintain their emotional well-being. Also, the model explains how it may be possible that two qualitatively different coping strategies, such as suppression and rumination, may be present at the same time, i.e., we suggest that anxiety of again loosing control will trigger self-instructions such as: "I *must not* think about it" or "*I have to* keep on going". Accordingly, our suggestion is that such self-instructions are "rumination of the necessity of avoiding negative self-referent information".

In order to examine how formerly depressed individuals would react and cope when presented with information that remind them of a previous depressive episode, we gave the participants the opportunity to choose to listen to either positive or negative self-statements. We found a listening pattern among previously depressed individuals that was different from the rest of the findings (see below) and which may support a possible compensatory strategy of avoiding negative information, i.e., simultaneously high levels of both dysfunctional attitudes and dysphoric symptoms resulted in a preference for positive self-statements. Also, findings from self-reported preferences for positive and negative self-statements, and mood changes during the procedure, gave preliminary support for the Cognitive battle model of recurrent depression.

Decreased approach motivation

Another main result of the present study was the finding of increased reaction time to visually presented emotionally valenced words in both previously depressed and in clinically depressed individuals. Also we found that dysphoric symptoms and dysfunctional attitudes were positively associated with reaction time to positively valenced words, whereas no associations were found for reaction time to negatively valenced words. These findings were interpreted as decreased approach motivation in previously depressed and in clinically depressed individuals, indicating that this may be a vulnerability trait to depression. However, in choice preference, previously

depressed and non-depressed controls exhibited a positivity bias whereas the clinically depressed individuals chose about fifty-fifty, indicating that this may be a state variable that reflects the onset of the clinical depression.

Automatic and effortful processing

The final aim of the study was to increase our understanding of how automatic and effortful processes are involved in the processing of emotionally valenced information. To do so, we compared the three groups on three different memory processes assumed to be more or less automatic or effortful. For methodological reasons, these findings are difficult to interpret. In conclusion, however, we found support for the suggestion that in depression one would find decreased effortful processing of positive self-referent information as suggested by Clark and Beck (1999), but no bias in the more automatic processing of emotionally valenced information as suggested by Williams et al. (1997). Also, we found that clinically depressed and previously depressed individuals fabricated more negative self-statements than positive self-statements, indicating that this may be a vulnerability trait to depression.

The main findings in each group of participants

In the group of *non-depressed controls* we found a positivity bias both in choice preference for visually presented emotionally valenced words, and in preference for tape-recorded self-statements. We did not find any association between dysphoric symptoms and dysfunctional attitudes, indicating that mood and attitudes do not have any impact on each other in individuals who are not cognitively vulnerable to depression. These findings are in line with previous research (Miranda & Persons, 1988; Miranda, Persons, & Byers, 1990; Roberts & Kassel, 1996).

Also, in the group of *previously depressed individuals* we found a positivity bias in choice preference for visually presented emotionally valenced words. However, in preference for tape-recorded self-statements high levels of dysfunctional attitudes in combination with different levels of dysphoric symptoms lead to different cognitive processing of self-statements: While high levels of dysfunctional attitudes alone decreased the preference for positive self-statements, simultaneously high levels of dysfunctional attitudes and dysphoric symptoms increased the preference for positive self-statements. Finally, we found a positive association between dysfunctional attitudes and dysphoric symptoms, decreased approach motivation to emotionally valenced words, and more fabrication of negative self-statements compared to positive self-statements.

In the group of *clinically depressed individuals* we found an equal processing of positive and negative information in choice preference to visually presented emotionally valenced words, and in preference to tape-recorded self-statements, which may indicate that these variables are state-dependent measures of depression.

However, and similar to the previously depressed group, we found a positive association between dysfunctional attitudes and dysphoric symptoms, decreased approach motivation to emotionally valenced words, and more fabrication of negative self-statements compared to positive self-statements. Accordingly, this may indicate that these variables are trait-measures of vulnerability to depression.

6.1.1 Dysphoric symptoms and dysfunctional attitudes as vulnerability factors to depression

The participants in the present study may possibly differ from cognitively vulnerable individuals who have participated in previous research on depression. Firstly, we defined depression in terms of clinical diagnoses as provided by a structural interview (i.e., SCID) and not according to a cut-off point on a self-rating scale (e.g. BDI). As discussed in the introduction section, it may be important qualitative differences between subjects who have been defined as depressed according to self-rating scales, and subjects who have been defined as depressed by a diagnostic interview. Secondly, even if the depressed individuals in the present study were depressed according to clinical diagnosis, they were outpatients and thus possibly not as severely depressed as in many other studies. Accordingly, a sample of diagnosed clinically depressed outpatients is rare in the research literature and our findings on cognitive deviations in this group may be valuable to increase our understanding of how mild-moderate major depression may escalate in to a more serious depression.

Furthermore, by defining depression in terms of clinical diagnoses, it is possible that we have grasped some specific characteristics of formerly depressed individuals, which may have been lost in previous research. For example, in our study we found that previously depressed individuals had relatively high degree of dysphoric symptoms and dysfunctional attitudes. This contrasts with previous studies, which have found that dysfunctional attitudes do not persist beyond recovery from the depressive state (Clark & Beck, 1999; Ingram et al., 1998). However, these studies have only included previously depressed individuals with a depression score within the normal range as measured with self-rating scales. A problem with defining previous depression by this criterion is, however, that if dysfunctional attitudes persist in vulnerable individuals, but are inaccessible until they have become activated by negative mood, then it will be impossible to detect this association. Also, if previously depressed individuals generally exhibit higher levels of dysphoric symptoms and dysfunctional attitudes than what is normal, this may be a vulnerability factor to depression that has been previously overlooked.

Consequently, our findings of a positive association between dysphoric symptoms and dysfunctional attitudes in clinically depressed and in previously depressed individuals, but not in non-depressed controls, may indicate that mood and cognition are largely, functionally separate and hence may reflect a causal relationship in depression. The findings are consistent with previous research (Miranda & Persons; 1988; Miranda et al., 1990; Roberts & Kassel, 1996). To summarize, dysfunctional attitudes may be a

vulnerability factor to depression, which may be activated by dysphoric mood. Compared to less vulnerable individuals, previously depressed individuals may generally be more dysphoric and exhibit higher levels of dysfunctional attitudes, making them more vulnerable to recurrent episodes of depression.

6.1.2 The relationship between dysfunctional attitudes and the cognitive processing of positive and negative information

Central to cognitive structure models of depression are the suggestion that activated negative self-schemata, or negative knowledge structures, will bias the information processing negatively. However, although much research has supported this suggestion, there are also findings, which indicate that the main problem in depression is the loss of positivity bias. Results from the present study seem to support this latter suggestion. By looking more thoroughly at the methods we have used, the cognitive processes we have studied, and the characteristics of the participants, we suggest that the loss the positivity bias is the consequence of high levels of dysfunctional attitudes. This is an interesting hypothesis because dysfunctional attitudes have usually been assumed to negatively bias the individual's information processing (Beck, 1967, 1976). The loss of positivity bias, we suggest, will in turn contribute to the escalation process of dysphoric mood into a clinical depression.

For example, in the listening preference task we found in the clinically depressed group a negative correlation between listening time to positive self-statements and dysfunctional attitudes. Because the previously depressed and the never depressed group were positively biased, whereas the clinically depressed group was not, this may indicate that dysfunctional attitudes in the clinically depressed group decreased the positivity bias which was present in the other two groups. In the previously depressed group there was different patterns of listening preferences depending on the levels of dysfunctional attitudes and dysphoric symptoms (see above). These findings indicate that also in the previously depressed group, dysfunctional attitudes decreased the listening preferences to positive self-statements when the previously depressed were only mildly dysphoric. In the Cognitive battle model, however, we actually argue that increased processing of negative self-referent information (in this case because of decreased processing of positive information), in turn will be responsible for the use of coping strategies to avoid negative self-referent information. Accordingly, the positivity bias found in previously depressed individuals might actually be an avoidance reaction.

In choice preference to visually presented emotionally valenced words, we found for the whole sample, a negative correlation between choice preference to positive words and dysfunctional attitudes. Also in choice preference, we found that the previously depressed and the never depressed group were positively biased, whereas the clinically depressed group was not. Accordingly, also in this visual attention task it seems that dysfunctional attitudes were responsible for decreasing the positivity bias. However, it is striking that the previously depressed individuals who, compared to the never

depressed individuals, exhibited more dysfunctional attitudes and more dysphoric symptoms, still were positively biased in choice preference. A possible explanation may be that there exists a "threshold" where the positivity bias disappears. This explanation may be supported by the findings that it was only in the clinically depressed group that dysphoric symptoms were negatively correlated with choice preference.

To summarize, findings from both the listening preference task and the visual attention task seem to indicate that dysfunctional attitudes may impair the ability to attend to positive information and by this wipe out the 'illusory optimism' that often characterize non-depressed individuals. An exception from this is the positivity bias found in previously depressed individuals with simultaneously high levels of dysfunctional attitudes and dysphoric symptoms. However, and as discussed above, we suggest that this is not a genuine attraction to positive information, but rather an avoidance reaction to negative self-referent information.

The strongest support for the suggestion that dysfunctional attitudes may decrease the processing of positive information seems to be the finding that dysfunctional attitudes, in the visual attention task, were related to increased reaction time to relatively more positive words whereas no relation was found between dysfunctional attitudes and the reaction time to relatively more negative words. In visual attention tasks, reaction time has usually been regarded as a measure of selective attention either to positively or to negatively valenced information. However, in the present study we argue that there is several reasons to claim that reaction time is better conceptualised as a measure of approach motivation. For example, researchers have discussed whether the method used in the present study is a pure measure of visual attention or whether it implicitly encourage the use of guessing strategies in a discrepant situation. Also, we found that reaction time was unrelated to choice preference and to dysphoric symptoms. Accordingly, it is not reasonable to suggest that reaction time in the present study is a measure of selective attention, neither a measure of motoric retardation which often follows depressive symptoms.

Finally, we found that dysfunctional attitudes were negatively related to free recall of positive self-statements and therefore may be involved in the decreased effortful processing of positive self-statements which was found in clinically depressed individuals. Interestingly, however, the difference obtained, was primarily in the *effort* to recall positive self-statements, i.e., it was only when a looser criterion of correct recall was used that the difference was present. Also, we found that the fabrication of more negative self-statements than positive self-statements in clinically depressed and previously depressed individuals, was obtained as a consequence of decreased fabrication of positive self-statements. Altogether, these findings may indicate that it was actually the 'illusory optimism', or the 'self-enhancing illusion', which was lost in the information processing of positive self-statements. These findings support Clark and Beck's (1999) assumption that depression is characterized by decreased effortful

processing of positive stimuli. Also, the findings are in line with self-regulation theories of decreased approach motivation to positive stimuli.

In the present study we used recognition as a measure of automatic processing and found that dysfunctional attitudes also were related to increased recognition of negative self-statements. However, we did not find increased recognition of negative self-statements in the clinically depressed individuals as suggested by Clark and Beck (1999). Possible explanations may be that recognition is not an appropriate measure of automatic processing and that the clinically depressed individuals in the present study were only mildly to moderately depressed.

6.1.3 The escalation process of dysphoric mood to a clinical depression; the consequence of an imbalance in positive and negative information processing?

Why are dysfunctional attitudes capable of wiping out the 'illusory optimism', or decreasing the positivity bias, normally present in non-depressed individuals? Our suggestion is that dysfunctional attitudes, when activated will make the cognitively vulnerable individual more motivated to prevent unfortunate outcome rather than taking the risk of approaching new goals. For example, holding an attitude that: "If I fail partly, it is as bad as being a complete failure" (i.e., an item in DAS), will change the focus from the desired positive outcomes, to the possible negative outcomes of the situation, to be sure that it will not happen. However, by changing the focus to the possible negative outcomes, the approach motivation to positive stimuli will decrease and the processing of negative information will increase. Accordingly, when dysfunctional attitudes are activated, it seems likely that the first part of an escalation process of dysphoric mood in to a clinical depression may be the decreased approach motivation to positive stimuli, which in turn will increase the processing of negative self-referent information. This explanation is in line with the self-regulation perspective of depression suggesting that the core problem in depression may be the regulation between BIS and BAS (Carver & White, 1994; Gray, 1994). It also resembles the viewpoint of Ingram and Smith (1984), and Kendall and Hollon (1981), who suggest that it may be the equality between positive and negative thoughts that put people at risk for depression.

In the present study, increased reaction time to positive stimuli was unrelated to choice preference. This may indicate that decreased approach motivation does not immediately decrease the processing of positive stimuli. However, one may argue that decreased approach motivation to positive stimuli is an expression of uncertainty, or ambivalence, about how to cope in a discrepant or stressful situation. Furthermore, we assume that it is actually this ambivalence that is the core problem in individuals who are cognitive vulnerable to depression. This ambivalence may be measured by increased self-focused attention (Pyszczynski & Greenberg, 1987) and rumination (Nolen-Hoeksema, 1991; Papagerorgiou & Wells, 2003). Accordingly, decreased approach motivation may be an important part of an escalation process of dysphoric

mood to a clinical depression. In such an escalation process we suggest that the balance between the processing of positive and negative information will be gradually displaced in the favour of the processing of negative information. And, when the processing of negative information becomes as strong as the processing of positive information, we suggest that the consequence will be a "cognitive decomposition" where the information processing will be predominantly negative.

A cognitive decomposition may constitute the qualitative difference between just having incidentally high depression scores on a self-rating scale and the presence of a clinical depression. An experience of loosing control may be the expression of stepping over this threshold. Individuals who have experienced a clinical depression have described this threshold in the following way: "Gradually and then suddenly" (Wurtzel, 1994). And, also they have described the clinically depressive state as being a "walking, waking dead", a different zone, which involves a complete *absence*, an absence of interest, response, feeling, and affect (Solomon, 2001; Wurtzel, 1994).

To summarize, by including previously depressed individuals with dysphoric symptoms, and clinically depressed individuals who were not hospitalised, the present study made it possible to collect data and generate hypotheses about possible vulnerability factors contributing to the escalation process of dysphoric mood into a clinical depression. By including more severe clinically depressed individuals, we might probably have uncovered the presence of a negativity bias in the processing of emotionally valenced information.

6.2 Clinical implications and further research

Clinical implications of the present study may be related to several findings. Firstly, if previously depressed individuals generally exhibit higher levels of dysphoric symptoms and dysfunctional attitudes than what is normal, it is reasonable to assume that these individuals will be more vulnerable to depression. Accordingly, an important goal for future research is to replicate our findings and to design treatments, which ensure that previously depressed individuals will get help in bringing down their dysphoric symptoms and dysfunctional attitudes. However, also the findings of higher levels of dysphoric symptoms in previously depressed individuals, question whether formerly depressed individuals have problems with their previous depressive experience (e.g. worrying about the risk of loosing control again, and feeling continuously stressed by trying to avoid such a risk). Results from a study using the Rorschach method seem to support these suggestions (Hartmann, Wang, Berg, & Sæther, 2003). The authors found a tendency in previously depressed individuals, as compared to never depressed individuals, towards increased levels of anxiety, low stress tolerance, low self-esteem, rigid and maladaptive coping strategies and feelings of hopelessness. Also, these findings are in line with research, which has found that people exposed to chronic stress may develop poorly regulated neuroendocrine systems. Researchers have argued that when these people later in life are exposed to even a minor stressor, the hypothalamic-pituitary-adrenal axis overreacts, which in

turn creates changes in the functioning of the monoamine neurotransmitters in the brain. This may possibly cause depressive episodes (Holsboer, 1992; Young & Korzun, 1998; Weiss, 1991). Accordingly, future research on cognitive processes in recurrent depression should include measures of neuroendocrine activity such as for example the hormone cortisol.

The demonstration of increased levels of dysphoric symptoms and dysfunctional attitudes in previously depressed individuals, also has contributed to the development of the Cognitive battle model. As we have discussed above, dysphoric symptoms, dysfunctional attitudes and decreased information processing of positive stimuli, may remind the formerly depressed individual of the previous depressive episode and evoke anxiety of again losing control. This may in turn trigger the use of maladaptive coping such as avoidance and suppression. Accordingly, different relapse preventive treatment should be designed dependent of the dysphoric state of the previously depressed individual. For example, similarly to traditional cognitive therapy for depression (Beck, 1995), previously depressed individuals without dysphoric symptoms should learn to reduce their amount of dysfunctional attitudes, not to engage in sustained rumination and actively to select positive self-referent information. However, as dysphoric symptoms arise, it may be important not to avoid or suppress depressive cognitions, but rather to explore and reality tests them. Meta-cognitive interventions may be important to bring to conscious how coping strategies intended to prevent depressive relapse, may actually have the opposite effect by increasing the possibility of experiencing a new depression episode.

Finally, depending upon their affective and dysfunctional attitude profile, and their coping style, previously depressed individuals may be regarded as having experienced a "traumatic life event", which has led to impairment of their information processing and thereby loss of control. Accordingly, our understanding of depressive patients may profit from knowledge accumulated from the treatment of posttraumatic stress disorder. A crucial point in relapse prevention of depression, then, could be, at the end of a depressive episode, to treat this experience as much as the treatment of the depressive symptoms and the presumed causal factors behind these depressive symptoms. This may be a focus of further research on prevention treatment for recurrent depression.

6.3 Limitations of the study and methodological challenges

The design of the present study was a cross-sectional remission design and the focus of the study was on proximal vulnerability factors. Accordingly, the present study was not designed to explain distal vulnerability factors such as childhood experiences. In the last decade, however, several researchers have actually made distal vulnerability factors the focus of their research. From longitudinal research, findings generally support the suggestion that interpersonal experiences in childhood influence the development of the child's self-regulation and cognitive style (for a review; Goodman, 2002). Also it has been found in infants of depressed mothers, atypical patterns of

frontal EEG asymmetry, i.e., reduced left frontal brain activity, which are associated with positive emotions of joy and interest (Davidson & Fox, 1982; Dawson et al., 1999). This finding may indicate that there exist neurobiological correlates of reduced approach motivation in depression (Tomarken & Keener, 1998). Furthermore, high-risk research has provided compelling support for the cognitive theories of depression (Abramson et al., 1999; Alloy, Abramson et al., 1999; Alloy et al., 1997).

By having this more recent research on development antecedents as a backcloth for the present study, our focus was on cognitive vulnerability factors that may be caused by previous experiences, but which appear just before the onset of the depressive episode. Because of methodological limitations of the present study, there are potentially two main problems related to the interpretation of the results. Firstly, the intent of most remission studies has been to examine the stability of potentially, causative factors predicting first-episodes of depression. However, by being aware that the depressive episode by itself may act as a vulnerability factor, a core problem with the remission design is to distinguish between these two types of vulnerability. In the present study we have discussed vulnerability factors that may be primarily caused by development antecedents, (i.e., dysfunctional attitudes; decreased approach motivation), and vulnerability factors that may primarily be the consequence of the depressive experience (cf. the Cognitive battle model). However, due to the cross-sectional research design, we cannot be absolutely certain about the origin of these proximal vulnerability factors. Furthermore, when interpreting the results from the present study, we chose to focus on possible escalation processes of dysphoric mood into a clinical depression. However, to obtain more conclusive results, it had been an advantage to use a longitudinal design to, after a mood induction procedure, follow the participants in the presumed escalation process. Of ethical reasons, of course, this opportunity is excluded. Accordingly, if one is just aware of the methodological limitations, cross-sectional designs may be useful to examine potentially escalation processes in depression.

In the present study we have challenged some common understanding of what experimental methods are actually measuring. For example, in the Deployment of Attention Task (paper 2), we argued that the task is more like a guessing task in a discrepant situation, rather than a pure visual attention task, which the task has usually been regarded as (Gotlib, McLachlan, & Katz, 1988; Kakolewski et al., 1999; McCabe & Gotlib, 1995). The findings on reaction time from the present study supported this suggestion. Also, we have discussed whether experimental set-ups with good internal validity are good models of real-world situations that confront us in daily life (paper 3). Experimental methods to investigate cognitive processes in depression are of great value, but may be useless if the external or clinical validity is poor. For example, in research on memory processes in depression one should be aware that recall and recognition is functionally dependent of earlier stages of information processing including how people choose the information they will attend to. In fact, it is a crucial point in cognitive theory of depression, that vulnerable individuals select and interpret emotionally ambiguous information differently to non-vulnerable individuals.

Accordingly, if the present study in an effort to increase the internal validity had given the participants exactly the same amount of listening time to positive and negative self-statements, the finding may have been less useful in clinical purposes.

Finally, there are several limitations with the Cognitive battle model and with the findings supporting this model (paper 1). Firstly, the model was primarily developed on the basis of reviewing theories and previous research on cognitive vulnerability to depression. By doing so, we were aware that little attention had been paid to the formerly depressed individuals experience of the depressive episode itself. Accordingly, the present study was carried out in order to explore and increase such knowledge. The findings gave some support to our model, but must of course be interpreted cautiously due to small sample sizes and marginally significant results. Also, each step of the model was not tested in the present study. Alternative interpretations of the findings may be present. However, the main intention with this article was not to strictly test the Cognitive battle model, but to contribute to generate a basis of hypotheses testing and clinical interventions. Future research will decide if the Cognitive battle model will increase our understanding and treatment of relapse and recurrence in depression.

7. CONCLUSIONS

The present study has developed a Cognitive battle model to explain possible mechanisms involved in recurrent depression. The model has several methodological limitations and there may be alternative interpretations of the findings that have been taken as support to the model. The model has, however, generated a basis of future hypothesis testing and clinical interventions.

Also, the study has contributed with findings which suggest that previously depressed individuals, in general, may be more in psychological distress than the part of the population that have never experienced a clinical depression. More dysphoric symptoms in previously depressed individuals may be a vulnerability factor to recurrent depression and future research should be carried out to replicate this finding.

Furthermore, findings from the study seem to indicate that dysfunctional attitudes, in mildly to moderately clinically depressed individuals, may have the potential to decrease the effortful processing of positive stimuli. Decreased processing of positive stimuli may result in an imbalance in the processing of positive and negative information, which possibly will contribute to an escalation process of dysphoric mood into a clinical depression.

Finally, the present study has contributed with results, which indicate that decreased approach motivation to positive stimuli may be a vulnerability factor to depression. Future research should be aimed to examine the relationship between parenting (e.g. insecure attachment; inadequate affect regulation), frontal lobe development (i.e., hemispheric asymmetry), cognitive vulnerability factors (e.g. uncertainty; rumination), and decreased approach motivation to positive stimuli.

REFERENCES

Abramson, L.Y., Alloy, L. B., Hankin, B. L., Haeffel, G. J., MacCoon, D. G., & Gibb, B. E. (2002). Cognitive vulnerability-stress models of depression in a self-regulatory and psychobiological context. In: I. H. Gotlib, & C. L. Hammen (Eds.), Handbook of depression (pp. 268-294). New York: Guilford Press.

Abramson, L.Y., Alloy, L. B., Hogan, M. E., Whitehouse, W. G., Donovan, P., Rose, D., Panzarella, C., Raniere, D. (1999). Cognitive vulnerability to depression: Theory and evidence. *Journal of Cognitive Psychotherapy: An International Quarterly,* 13, 5-20.

Abramson, L. Y., Seligman, M. E. P., & Teasdale, J. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology, 87,* 49-74.

Alloy, L.B., & Abramson, L.Y. (1988). Depressive realism: four theoretical perspectives. In: L.B. Alloy (Eds.), *Cognitive processes in depression* (pp. 223-265). New York: Guilford Press.

Alloy, L. B., Abramson, L. Y., Murray, L. A., Whitehouse, W. G., & Hogan, M. E. (1997). Self-referent information-processing in individuals at high and low cognitive risk for depression. *Cognition and Emotion, 11,* 539-568.

Alloy, L. B., Abramson, L. Y., Whitehose, W. G., Hogan, M. E., Tashman, N., Steinberg, D., Rose, D. T., & Donovan, P. (1999). Depressogenetic cognitive styles: Predictive validity, information processing and personality characteristics, and development origins. *Behaviour Research and Therapy, 37,* 503-531.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders (4th ed.).* Washington, DC: Author.

Beck, A. T. (1967). *Depression: Clinical, experimental, and theoretical aspects.* New York: Harper & Row.

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders.* New York: International Univversities Press.

Beck, A. T. & Clark, D. A. (1997). An information processing model of anxiety: Reconsidering the role of automatic and strategic processes. *Behaviour Research and Therapy, 35,* 49-58.

Beck, A.T., Hollon, S.D., Young, J.E., Bedrosian, R.C., & Budenz, D. (1985). Treatment of depression with cognitive therapy and amitriptyline. *Archives of General Psychiatry, 42*, 142-148.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression.* New York: Guilford Press.

Beck, A. T., & Steer, R. A. (1987). *Beck Depression Inventory Manual.* New York: The Psychological Corporation Harcourt Brace Jovanovich, Inc.

Beck, J. S. (1995). *Cognitive therapy: basics and beyond.* New York: Guilford Press.

Bower, G. H. (1981). Mood and memory. *American Psychologist, 36,* 129-148.

Bower, G. H. (1987). Commentary on mood and memory. *Behaviour Research Therapy, 25,* 443-455.

Carver, C. S., & Scheier, M. F. (1998). *On the self-regulation of behavior.* New York: Cambridge University Press.

Carver, C.S., & White, T.L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology, 67,* 319-333.

Clark, D. A., & Beck, A. T. (1999). *Scientific foundation of cognitive theory and therapy of depression.* New York: John Wiley & Sons, Inc.

Consensus Development Panel (1985). NIMH/NIH consensus development conference statement: Mood disorders – Pharmacologic prevention of recurrence. *American Journal of Psychiatry, 142, 469-476.*

Coyne, J. C., Flynn, H., & Pepper, C. M. (1999). Significance of prior episodes of depression in two patient populations. *Journal of Consulting and Clinical Psychology, 67,* 76-81.

Crowe, E., & Higgins, E. T. (1997). Regulatory focus and strategic inclinations: Promotion and prevention in decision-making. *Organizational Behaviour and Human Decision Processes, 69,* 117-132.

Crowson, J. J., & Cromwell, R. L. (1995). Depressed and normal subjects differ both in selection and in perceived tonal quality of positive/negative messages. *The Journal of Abnormal Psychology, 104,* 305-311.

Davidson, R. J., & Fox, N. A. (1982). Assymetrical brain activity discriminates between positive versus negative affective stimuli in human infants. *Science, 218,* 1235-1237.

Dawson, G., Frey, K., Self, J., Panagiotides, H., Hessl, D., Yamada, E., & Rinaldi, J. (1999). Frontal brain electrical activity in infants of depressed and nondepressed mothers: Relation to variations in infant behavior. *Development and Psychopathology, 4,* 67-80.

Delis, D. C., Kramer, J. H., Kaplan, E. & Ober, B. A.(1987). *California Verbal learning test.* Cleveland, OH: Psychological Corporation.

Elliot, A. J., & Sheldon, K. M. (1997). Avoidance achievement motivation: A personal goals analysis. *Journal of Personality and Social Psychology, 73,* 171-185.

Elliot, A. J., Sheldon, K. M., & Church, M. A. (1997). Avoidance personal goals and subjective well-being. *Personality and Social Psychology Bulletin, 23,* 915-927.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1997). *The structured clinical interview for DSM-IV, axis I disorders – clinician version (SCID-CV).* Washington, DC, American Psychiatric Press.

Goodman, S. H. (2002). Depression and early adverse experiences. In: I. H. Gotlib, & C. L. Hammen (Eds.), *Handbook of depression* (pp. 245-267). New York: Guilford Press.

Gotlib, I. H., & Abramson, L. Y. (1999). Attributional theories of emotion. In: T. Dalgleish & M. Power (Eds.), *Handbook of cognition and emotion (pp. 613-636).* Chichester, UK: Wiley.

Gotlib, I. H., & Krasnoperova, E. (1998). Biased information processing as a vulnerability factor for depression. *Behavior Therapy, 29,* 603-617.

Gotlib, I. H., & Hammen, C. L. (Eds.) (2002). *Handbook of depression*. New York: Guilford Press.

Gotlib, I. H., Kurtzman, H. S., & Blehar, M. C. (1997). Cognition and depression: Issues and future directions. *Cognition and Emotion, 11,* 663-673.

Gotlib, I.H., McLachlan, A.L., & Katz, A.N. (1988). Biases in visual attention in depressed and nondepressed individuals. Special issue: Information processing and emotional disorders. *Cognition and Emotion, 2,* 185-200.

Gotlib, I.H. & Neubauer, D.L. (2000). Information-processing approaches to the study of cognitive biases in depression. In S. L. Johnson, A. M. Hayes, T. M. Field, N. Schneiderman, & P. M. McCabe (Eds.), *Stress, coping and depression* (pp. 145-176). New Jersey: Lawrence Erlbaum Associates, Inc.

Gray, J. A. (1994). Three fundamental emotions systems. In P. Ekman, & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions (pp. 243-247).* New York: Oxford University Press.

Hartlage, S., Alloy, L. B., Vazquez, C., & Dykman, B. (1993). Automatic and effortful processing in depression. *Psychological Bulletin, 113*: 247-278.

Hartmann, E., Wang, C. E., Berg, M. & Sæther, L. (2003). Depression and vulnerability as assessed by the Rorschach method. *Journal of Personality Assessment, 81,* 242-255.

Higgins, E.T. (1987). Self-discrepancy: A theory relating self and affect. *Psychological Review, 94,* 319-340.

Hofmo, G. (1948). *Fra en annen virkelighet.* Oslo: Gyldendal.

Hollon, S. D., & Cobb, R. (1993). Relapse and recurrence in psychopathological disorders. In: C. G. Costello (Eds.), *Basic issues in psychopathological.* New York: Guilford Press.

Hollon, S. D., Evans, M. D., & DeRubeis, R. J. (1990). Cognitive mediation of relapse prevention following treatment for depression: Implications of differential risk. In R. E. Ingram (Eds.), *Contemporary psychological approaches to depression: Theory, research, and treatment.* New York: Plenum 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.

Holsboer, F. (1992). The hypothalamic-pituitary-adrenocortical system. In E.S. Paykel (Eds.), *Handbook of affective disorders* (pp. 267-287). New York: Guilford Press.

Ingram, R. E. (1984). Toward an information processing analysis of depression. *Cognitive Therapy and Research, 8,* 443-478.

Ingram, R. E., Miranda, J., & Segal, Z. V. (1998). *Cognitive vulnerability to depression.* New York: Guilford Press.

Ingram, R. E., & Siegle, G. J. (2002). Contemporary methodological issues in the study of depression: not your father's oldsmobile. In: I. H. Gotlib, & C. L. Hammen (Eds.), *Handbook of depression* (pp. 86-114). New York: Guilford Press.

Ingram, R.E. & Smith, T.W. (1984). Depression and internal versus external focus of attention. *Cognitive Therapy and Research, 8,* 139-159.

Ingram, R. E., & Wisnicki, K. S. (1988). Assessment of positive automatic cognition. *Journal of Consulting and Clinical Psychology, 56,* 898-902.

Joiner, T., & Coyne, J. C. (Eds.)(1999). *The interactional nature of depression.* Washington: American Psychological Association.

Kakolewski, K. E., Crowson Jr., J. J., Sewell, K. W., & Cromwell, R. L. (1999). Laterality, word valence, and visual attention: a comparison of depressed and non-depressed individuals. *International Journal of Psychophysiology 34,* 283-292.

Kendall, P. C., & Hollon, S. D. (1981). Assessing self-referent speech; methods in the measurement of self-statements. In: P. C. Kendall, & S. D. Hollon (Eds.), *Assessment strategies for cognitive-behavioral interventions* (pp. 85-118). New York: Academic Press.

Klerman, G. L., Lavori, P. W., Rice, J., Reich, T., Endicott, J., Andreasen, N. C., Keller, M. B., & Hirchfeld, R. M. A. (1985). Birth-cohort trends in rates of major depressive disorder among relatives of patients with affective disorder. *Archives of General Psychiatry, 42,* 689-693.

Klerman, G. L., & Weissman, M. M. (1992). The course, morbidity, and costs of depression. *Archives of General Psychiatry, 49,* 831-834.

Kringlen, E., Torgersen, S., & Cramer V. A. (2001). Norwegian psychiatric epidemiological study. *Archives of General Psychiatry, 158,* 1091-1098.

Lazarus, R. S. (1966). *Psychological stress and the coping process.* New York: McGraw-Hill.

Lepine, J. P., Gastpar, M., Mendlewicz, & J., Tylee, A. (1997). Depression in the community: The first pan-European study DEPRES. *International Clinical Psychopharmacology, 12,* 19-29.

Lubin, B. (1965). Adjective checklists for measurement of depression. *Archives of General Psychiatry, 12,* 57-62.

McCabe, S. B., & Gotlib, I. H. (1995). Selective attention and clinical depression: Performance on a deployment-of-attention task. *Journal of Abnormal Psychology, 104,* 241-245.

Miranda, J., Gross, J. J., Persons, J. B., & Hahn, J. (1998). Mood matters: Negative mood induction activates dysfunctional attitudes in women vulnerable to depression. *Cognitive Therapy and Research, 22,* 363-376.

Miranda, J., & Persons, J. B. (1988). Dysfunctional attitudes are mood-state dependent. *Journal of Abnormal Psychology, 97,* 76-79.

Miranda, J., Persons, J. B. & Byers, C. N. (1990). Endorsement of dysfunctional beliefs depends on current mood state. *Journal of Abnormal Psychology, 99,* 237-241.

Murray, C. L., & Lopez, A. D. (1998). *The global burden of disease: A comprehensive assessment of mortality and disability from disease, injuries and risk factors in 1990 and projected to 2020.* Boston: Harvard University Press.

Nissbett, R. E., & Ross, L. (1980). *Human interference: Strategies and shortcomings of social judgment.* Englewood Cliffs, NJ: Prentice-Hall.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100,* 569-582.

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, 11,* 25-40.

Papageorgiou, C., & Wells, A. (2001). Metacognitive beliefs about rumination in recurrent major depression. *Cognitive and Behavioral Practice, 8,* 160-164.

Papageorgiou, C., & Wells, A. (2003). An empirical test of a clinical metacognitive model of rumination and depression. *Cognitive Therapy and Research, 27, 3,* 261-273.

Parikh, S. V., Wasylenki, D., Goering, P., & Wong, J. (1996). Mood disorders: Rural/urban differences in prevalence, health care utilization and disability in Ontario. *Journal of Affective Disorders, 38,* 57-65.

Paykel, E. S., Ramana, R., Cooper, Z. Hayhurst, H., Kerr, J., & Barocka, A. (1995). Residual symptoms after partial remission: An important outcome in depression. *Psychological Medicine, 25,* 1171-1180.

Post, R. M. (1992). Transduction of psychsocial stress into the neurobiology of recurrent affective disorder. *American Journal of Psychiatry, 149,* 999-1010.

Post, R. M., & Weiss, S. R. B. (1995). The neurobiology of treatment-resistant mood disorders. In F. E. Bloom & D. J. Kupfer (Eds.), *Psychopharmacology: The fourth generation of progress* (pp.1155-1170). New York: Raven Press.

Pyszczynski, T., & Greenberg, J. (1987). Self-regulatory preservation and the depressive self-focusing style: A self-awareness theory of reactive depression. *Psychological Bulletin, 102,* 122-138.

Roberts, J. E., & Kassel, 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.

Rosenthal, N. E., Bradt, G. H., & Wehr, T. A. (1984). *Seasonal Pattern Assessment Questionnaire.* Bethesda Maryland: National Institute of Health.

Sartorius, N., Jablensky, A., Gulbinat, W., & Ernberg, G. (1980). WHO collaborative study: Assessment of depressive disorders. *Psychological Medicine, 10,* 743-749.

Segal, Z. V., Williams, J. M. G., & 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*. San Francisco: Freeman.

Snyder, C. R., Harris, C., Anderson, J. R., Holleran, S. A., Irving, L. M., Sigmon, S. T., Yoshinobu, L., Gibb, J., Langelle, C., & Harney, P. (1991). The will and the ways: Development and validation of an individual-differences measure of hope. *Journal of Personality and Social Psychology, 60,* 570-585.

Solomon, A. (2001). *The Noonday Demon.* London: Chatto and Windus.

Solomon, D. A., Keller, M. B., Leon, A. C., Mueller, T. I. et al. (2000). Multiple recurrences of major depressive disorder. *American Journal of Psychiatry, 157,* 229-233.

Teasdale, J. D. (1988). Cognitive vulnerability to persistent depression. *Cognition and Emotion, 2,* 247-274.

Tomarken, A. J., & Keener, A. D. (1998). Frontal brain asymmetry and depression: A self-regulatory perspective. *Cognition and Emotion, 12* (3), 387-42.

Wang, C. E (1996). *Spørreskjema om tidligere depressive episoder. [Questionnaire about Previous Depressive Episodes (PDQ)].* Unpublished manuscript. The University of Tromsø, the Department of Psychology, Norway.

Wang, C. E., & Holte, A. (1995). *Spørreskjema om gjenkjenning av selv-refererende budskap. (Self-referent Statement Recognition Questionnaire).* Unpublished manuscript. University of Tromsø, Department of Psychology, Norway.

Weinstein, N.D. (1980). Unrealistic optimism about future life events. *Journal of Personality and Social Psychology, 39,* 806-820.

Weiss, J. M. (1991). Stress-induced depression: Critical neurochemical and electrophysiological changes. In J. I. Madden (Ed.), *Neurobiology of learning, emotion, and affect* (pp. 123-154). New York: Raven Press.

Weissman, A., & Beck, A. T. (1978). *Development and validation of the Dysfunctional Attitude Scale: A preliminary investigation.* Paper presented at the meeting of the American Educational research Association, Toronto, Canada.

Weissman, M. M., Bruce, L. M., & Leaf, P. J. (1990). Affective disorders. In: Robins L. N., & Regier, D. A. (Eds.), *Psychiatric disorders in America: The Epidemiologic Catchment Area study* (53-80). New York: Free Press.

Wells, A. (2000). *Emotional disorders and metacognition: innovative cognitive therapy.* New York: John Wiley & Sons, Inc.

Wenzlaff, R. M., & Wegner, D. M. (2000). Thought suppression. *Annual Review of Psychology, 51,* 59-91.

Wenzlaff, R. M., Rude, S. S., & West, L.M. (2002). Cognitive vulnerability to depression: The role of thought suppression and attitude certainty. *Cognition and Emotion, 16,* 533-548.

Williams, J. M. G., Watts, F. N., MacLeod, C., & Mathews, A. (1997). *Cognitive psychology and emotional disorders.* Chichester, England: Wiley

World Health Organization (1992). *International Classification of Diseases (10th ed.).* Geneva, DC: Author.

World Health Organization (2001a). *Mental and neurological disorders.* Retrieve November 1, 2004, from http://www.who.int/mediacentre/factsheets/fs265/en/

World Health Organization (2001b). *The world health report 2001- mental health new understanding, new hope.* Retrieved November 1, 2004, from http://www.who.int/whr/2001/en/index.html

Wurtzel, E. (1994). *Prozac Nation.* New York: Riverhead Books.

Young, J. E. (1990). *Cognitive therapy for personality disorders: A schema-focused approach.* Sarasota, FL: Professional Resource Press.

Young, E., & Korzun, A. (1998). Psychoneuroendocrinology of depression: Hypothalamic-pituitary-gonodal axis. *Psychiatric Clinics of North America, 21,* 309-323.

Zubin, J., & Spring, B. (1977). *Vulnerability - A new view of schizophrenia. Journal of Abnormal Psychology, 86,* 103-126.
Zuckerman, M. (1999). *Vulnerability to psychopathology: A biosocial model.* Washington, DC: American Psychological Association.