

Cognition in Depression:

What is the Relationship between Negatively Biased
Cognitive Mediators and Clinical Unipolar Depression?

Tiivistelmä

Tämän tutkielman tarkoitus on selvittää, millainen suhde on negatiivisten kognitiivisten mediaattorien ja kliinisen yksisuuntaisen depression välillä. Tavoitteena on määritellä, aiheuttavatko negatiiviset mediaattorit depressiota, vai ovatko ne depression oireita tai mahdollisesti seurauksia. Mikäli negatiiviset mediaattorit aiheuttavat depressiota, selvitetään, voivatko ne laukaista depression ilman ulkoista ärsykettä, vai onko ulkoinen ärsyke välttämätön. Beckin kognitiivista teoriaa sekä muita vaihtoehtoisia teorioita tarkastellaan kriittisestä näkökulmasta ja niihin liittyvien tutkimusten tuloksia arvioidaan.

Beckin kognitiivisen teorian (Beck, 1970) mukaan negatiiviset mediaattorit aiheuttavat depressiota, mutta vain jos ulkoinen ärsyke on aktivoinut ne. Toisaalta Parryn ja Brewinin mukaan (Parry & Brewin, 1988) negatiiviset mediaattorit voivat aiheuttaa depressiota täysin ilman ulkoista ärsykettä. Lisäksi Lewinsohn *et al.* (1981) huomasivat, että negatiiviset mediaattorit voivat olla myös depression oireita tai seurauksia. Koska teoriat ovat lähtökohdiltaan vastakkaisia, on tarpeen tukia asiaa empiirisesti. Joidenkin tutkimusten voidaan katsoa tukevan mitä tahansa teoriaa, jonka mukaan negatiiviset mediaattorit aiheuttavat depressioita, kun taas toiset tutkimukset tukevat vain Beckin teoriaa. Lisäksi on myös tutkimuksia, jotka tukevat hypoteesia, jonka mukaan negatiiviset mediaattorit ovat depression oireita tai seurauksia. Koska kaikki nämä teoriat ovat saaneet tukea tutkimuksista, ehdotetaan, että ne ovat kaikki tietyssä asteessa toimivia teorioita ja muut asiat, kuten kulttuuri ja sukupuoli, määräävät minkälainen suhde yksilön depression ja negatiivisten mediaattorien välillä on. Analyysin pohjalta voidaan todeta, että negatiivisten kognitiivisten mediaattorien ja kliinisen depression välinen suhde onkin kaksisuuntainen, eli depressio voi johtaa negatiivisiin kognitiivisiin prosesseihin, mutta usein negatiiviset kognitiiviset mediaattorit johtavat depression.

Abstract

This paper examines the nature of the relationship between negatively biased cognitive mediators and clinical unipolar depression. It aims at determining whether negatively biased cognitive mediators are a cause, a symptom or a consequence of major depression, and if they are a cause, whether they necessitate an environmental stressor to produce the depressed affect. Beck's tripartite model of depression and alternative theories, such as the alternate aetiologies model and the symptom model, are discussed from a critical perspective and studies supporting or challenging these theories are evaluated.

The tripartite model (Beck, 1970) proposes that negatively biased cognitive mediators cause depression but only after being activated by an environmental stressor, whereas the alternate aetiologies model (Parry and Brewin, 1988) postulates that negative cognitions can cause depression in the absence of environmental stimuli. Furthermore, as identified by Lewinsohn *et al.* (1981), negative cognitive biases could also be a symptom or a consequence of the depressed affect. As these models evidently contradict each other, there is a need for empirical validation. Some studies, such as the Evans *et al.* (2005) study, could be accepted as supporting any of the theories that propose negatively biased cognitive mediators to have a causal role in depression, while other studies specifically support Beck's theory. However, there are also studies supporting the hypothesis that cognitions are merely a symptom or a consequence of depressed affect. As all of these theories have received considerable support, it is suggested that they may all be valid to an extent and it is other factors, such as culture, gender and personality, which determine the specific nature of the relationship between negatively biased cognitive mediators and major depression for an individual. The analysis concludes that this relationship is in fact bidirectional, as already identified by Oei, Hibbert and O'Brien (2005).

Table of Contents

Introduction.....2
Cognitive Theories of Depression.....3
Evidence from Psychological Studies.....7
Conclusion.....11
References.....13

Introduction

Clinical unipolar depression is a serious mental disorder characterized by an extremely low affective state, a negative self-concept, self-punitive wishes and retardation (Beck, 1970). The formal DSM-IV-TR diagnosis of major depression requires low mood and four additional symptoms, such as loss of appetite and sleep disturbance, to have persisted for at least two weeks (American Psychiatric Association, 1944). Individuals suffering of major depression often have very low motivation, which may lead to decreased work productivity, problems in relationships and inability to carry on their daily lives. With lifetime prevalence rates of depression reaching 20-33% for women and 12-18% for men within Finland at the present (Räsänen, 2006), clearly there are many important social, economical and ethical reasons for trying to understand the causes of depression as well as developing effective methods of treatment and prevention. It is therefore not surprising that this issue has been among the targets of scientific investigation for decades, yielding many biological, genetic, behavioral as well as cognitive theories of the disorder. Since the 1960's revolution of cognitive psychology, many theorists have focused on the role of negatively biased cognitive mediators in explaining the onset of depression. The mediators, the internal processes that come between the stimuli and the response, that are generally hypothesized as having a causal or a consequential role in the etiology of depression include dysfunctional schemata as well as negative biases in attention, memory, perception and attributions. In this essay, cognitive structures, processes and products are all considered as mediators, since they all operate within the individual, between the stimuli and the response.

Although the notion that negatively biased cognitive mediators are a characteristic of depression is widely accepted and supported by many studies (e.g. Rude *et al.*, 2003, Rinck *et al.*, 2005), the nature of the relationship between these biases and the depressed affect remains uncertain. Among other theorists, Beck (1970) hypothesizes that negative schemata interact with negative life events to produce depressive thoughts and biases in cognitive mediational processes, which all together lead to depression. However, Parry and Brewin (1988) propose that negatively biased cognitive mediators are independent risk factors for depression, meaning that they may produce the depressed affect without an external stimulus. Furthermore, as

Lewisohn *et al.* (1981) pointed out, negative cognitions could also be a symptom or a consequence of depression, rather than being a causal factor.

What is the nature of the relationship between negatively biased cognitive mediators and clinical unipolar depression? The aim of this paper is to examine the cause-effect relationship between negative biases in cognitive functioning and major depression from both a theoretical and an empirical viewpoint. The tripartite model of depression (Beck, 1970) and alternative theories will be discussed from a critical perspective, and studies supporting and challenging these theories will be evaluated.

Cognitive Theories of Depression

The tripartite model of depression developed by A.T. Beck in the late 1960s is still one of the most influential cognitive theories of depression, although newer models have been developed incorporating Beck's ideas. According to Beck (1970), the underlying cause of major depression is dysfunctional schemata, which the individual has acquired through several negative experiences, such as loss of a loved one or being bullied, in the early childhood. Schemata are stable cognitive structures that organize information in a systematic way and are involved in "--screening, coding, and evaluating the stimuli that impinge on the organism" (Beck, 1970, pg. 283). If these schemata are negatively biased, the individual is likely to interpret incoming information in an idiosyncratic way, leading to biases in attention, memory and perception, which in turn lead to negative automatic thoughts and consequently the depressive symptomatology (Beck, 1970). An important feature of Beck's theory is that the depressive schemata remain latent until an environmental stressor, such as being fired from a job, activates them. Once these idiosyncratic schemata are activated, they dominate the information processing sequence, causing automatic thoughts that "--force the individual to see himself, his world and his future in an idiosyncratic way" (Beck, 1970, pg. 225). Beck (1970) refers to these negative views of self, the world and the future as a negative cognitive triad, which is the ultimate cause of depression.

Beck (1970) identified many different types of cognitive distortions of reality that demonstrate the operation of dysfunctional schemata, including selective abstraction, arbitrary inference, overgeneralization and exaggeration. Arbitrary inference refers to drawing negative conclusions in the absence of real evidence, which could be linked to biases in perception. Selective abstraction refers to conclusions drawn based on only one aspect of a situation, demonstrating selective attention affecting the process of interpretation. Overgeneralization means drawing major conclusions based on only one event and exaggeration describes how the importance of negative events is magnified and the significance of positive events is minimized. In addition to these negative biases in the processing of incoming information, Beck (1970) noted that depressed patients tend to remember more negative events than positive events, and may often remember only the negative aspects of a past occurrence. This is easily understood if we consider that many cognitive mediators are often interlinked, for example the perceptual process is likely to be affected by other processes such as selective attention, memory and mental schemata. Thus if biases occur in one cognitive process, other processes are likely to be affected as well.

Kwon and Oei (1994) revised and elaborated Beck's cognitive theory of depression to produce the integrated cognitive model of depression, which postulates that "--negative life events interact with dysfunctional attitudes to increase the frequency of automatic thoughts which, in turn, affect depressive symptoms" (Kwon & Oei, 1994, pg. 341). These theorists distinguished between surface and deep level cognitions, between stable and transient cognitions, and between cognitive moderators and mediators in the different phases of depression. They considered dysfunctional attitudes to reflect the stable, activated depressogenic schemata, and automatic thoughts to reflect relatively unstable surface level cognitions. The stable, deep level cognitions act as cognitive moderators, which are defined as the variables that change the causal relationship between an independent and a depended variable (Baron & Kenny, 1986, as cited in Kwon & Oei, 1994). The unstable, surface level cognition is considered as a mediator, the variable that "--changes the effects of an independent variable on a dependent variable by various transformation processes inside an active organism" (Kwon & Oei, 1994, pg. 336). Kwon and Oei (1994) postulated automatic thoughts to act as the cognitive mediators between the interaction of negative life events and dysfunctional attitudes and the depressed affect.

As can be identified, both Beck's cognitive theory and the integrated cognitive model of depression hypothesize negatively biased cognitive mediators to have a causal role in the development of depression. Both theories propose that negative schemata are stable cognitive structures, which the individual will always have. This raises arguments within psychologists due to the evidence suggesting that cognitive styles can change (Hollon, DeRubeis & Evans, 1996, as cited in Abramson *et al.*, 2005). Furthermore, these theories propose that negative cognitive structures need to be activated by an environmental event in order to produce the depressed affect, implying that cognitions do not act as independent risk factors but rather pose a vulnerability to specific stressors. Due to this aspect in the theories, both are capable of explaining why some people may go through extremely negative life events without developing clinical depression, i.e. these people do not have negative schemata, but neither of these models explains why others might develop clinical depression in the absence of negative life events. If negative schemata need to be activated by a stimulus, what is the stimulus for successful people who develop depression when everything in their lives seems to be well? This consideration slightly decreases the power of the theories that have been discussed, however, as Beck (1970) implied, another person simply might not understand the environmental stressors that caused the activation of latent depressive schemata. In this case the diathesis-stress theories could still explain the onset of depression, and the problem of identifying the stimulus is in our lack of understanding of others' thinking patterns.

There are also theories proposing that cognitive factors can cause depression in the total absence of an environmental stressor. The alternate aetiologies model proposes that "--depression can be precipitated either by a severely stressful event or alternatively by pre-existing cognitive vulnerability, both operating as independent provoking factors" (Parry and Brewin, 1988, pg. 26). This model is better suited to explaining the case where an individual develops depression in the absence of observable stimuli, i.e. the cognitions alone caused the depressed affect. However, human behaviour and emotions in all their complexity are unlikely to be the result of any single factor, and thus this theory is rather reductionist. In addition, it does not explain why some people might have negatively biased cognitive mediators and do not get depressed. Evidently it challenges the well established diathesis-stress models of

depression, although it is very difficult to distinguish which of these models are supported by given empirical data due to the difficulty of identifying environmental stressors.

The models already discussed quite convincingly propose that negative cognitions play a causal role in depression, but there is also a model that proposes negatively biased cognitive mediators to be a consequence of the depressed affect. Parry and Brewin (1988) describe the symptom model of depression, which “-- holds that maladaptive cognitions are merely *symptom-related*” (pg. 25). This means that negative life-events directly cause depressive symptomatology, which in turn causes automatic thoughts that lead to dysfunctional attitudes (Oei, Hibbert, & O’Brien, 2005). Evidently, this model can not explain cases where people have developed depression without the occurrence of a negative life event. Furthermore, it does not specify whether the negative cognitions are a persisting consequence of the depressed affect, or change towards the positive once the affect improves.

Other noteworthy cognitive models of depression include the revised learned-helplessness model and the differential activation model. The revised learned-helplessness model (Abramson, Seligman, & Teasdale, 1978) proposes that a depression prone individual has learned to expect that the outcomes of situations are not dependent on his responses, and makes global, stable, and external attributions for his personal success. Thus this model assumes a causal role of negative cognitions in depression. The differential activation model (Teasdale, 1988) also assumes a causal role, however, it proposes that depressed mood is directly caused by negative events and it is the cognitive framework of the individual that determines whether the depressed affect develops into clinical depression. Thus we may identify that there are several different models proposing that negatively biased cognitive mediators have differential causal roles in depression, whereas there is only one model proposing that negative cognitions are consequences of depressed affect.

Review of Evidence from Psychological Studies

The cognitive diathesis-stress models, the first one of them being Beck's cognitive theory of depression, originated from Beck's clinical observations. From reviews of case records of patients at a psychiatric outpatient clinic and conducting interviews and therapy sessions, Beck (1970) noted that depressed patients tend to distort their experiences in a negative way, and formulated his theory on the basis of this. Therefore, it could be said that the origin of this theory seems justified. However, since the patients were Beck's own, the theory might not be very objective. There is also a problem of generalizability from one clinical population to another, and more importantly, from one culture to another. In addition, since Beck observed already depressed patients, it is ambiguous whether the negative cognitions preceded the depressed affect or *vice versa*. This theory has been tested in various studies since its formulation, yielding controversial results.

Abramson *et al.* (2005) conducted a large-scale correlational study among college students to test the hypothesis that a negative cognitive style is a stable vulnerability factor for clinical depression. The participants were divided into two groups, recovered depressed and never depressed students, and their cognitive styles were assessed using self-report questionnaires. The researchers expected the recovered depressed to show more negative cognitive styles, since negative schemata are considered a persisting vulnerability, which does not change upon recovery. The results supported their hypothesis. However, a major factor that should be taken into account when discussing this study is that it can not be accepted as supporting the notion that negative cognitive styles precede the depressed affect. As Coyne and Gotlib (1981) identified, the cognitive styles of recovered depressed subjects in studies using the retrospective design could in fact be a persisting consequence of the previous depressed affect. However, this study has received support from a longitudinal study by Evans *et al.* (2005), in which a prospective design was used. This means that the investigators first assessed the cognitive styles of the participants and subsequently followed for depressive symptoms. In this study, the participants were pregnant women and self-report measures were used to assess both depressive symptoms and cognitive styles. The results supported the notion that depressogenic schemata pose a vulnerability to major depression (Evans *et al.*, 1999).

Although both these studies offer support to a well established theory, there are several methodological considerations that should be taken into account. First of all, the accuracy of self-report measures can be questioned, since the participants have volitional control over their answers (Rude *et al.*, 2003). There is no way for the researcher to know if the participants are being honest, or if they are showing social desirability effects by answering in a way that they think is expected of them. Evidently, the risk of demand characteristics is high using this methodology, although it is lower than in diagnostic interviews. In addition, self-report measures assume that participants can readily access their mental schemata and report them (Rude *et al.*, 2001). This might not be the case, especially considering the notion in Beck's theory that negative schemata remain latent until an external stimulus activates them. In addition, Rude *et al.* (2001) carried out research investigating this issue, and found that the operation of negative schemata may be observed in laboratory tasks involving information processing, but not in self-report measures.

Other methodological considerations include generalizability. The results of the Evans *et al.* study can only be generalized to pregnant women, whereas the results of the Abramson *et al.* study can only be generalized to college students. Most importantly, the results of both these studies may not be applicable to clinical populations or to other cultures. However, the study by Evans *et al.* was conducted in England and the study by Abramson *et al.* was conducted in the U.S.A., wherefore the similar results of the studies imply that the conclusions can be generalized across some cultures, although perhaps not to non-western cultures. Another positive factor in both these studies is that the sample sizes were relatively large and may be considered representative. Also, both these studies had people fill the questionnaires in their natural environment i.e. at home or in another familiar place, which increases the ecological validity of the results. Furthermore, the researchers controlled for confounding variables, such as major depression at the time of assessment of cognitive styles. Neither of these studies is subject to major ethical criticism, although very slight deception was involved.

Studies using self-report measures alone would be a rather weak support for the notion that negatively biased cognitive mediators are a vulnerability factor for major depression. In support of the previously mentioned studies, there are several studies that used laboratory

measures to assess cognitive styles, for example the longitudinal study by Rude *et al.* (2003) in which a prospective design was used. These investigators used the Scrambled Sentence Test (SST) to assess cognitive styles and subsequently conducted diagnostic interviews with a subset of the participants 18-28 months later to see whether high scores on the SST predicted the development of depression. The results of the study showed that negative biases in information processing predicted subsequent depression (Rude *et al.*, 2003). However, the cognitive styles of men did not predict subsequent depression unless volitional control was reduced by cognitive load procedures, although the cognitive styles of women clearly predicted subsequent depression in both the no-load and the load condition (Rude *et al.*, 2003). This finding by Rude *et al.* has important implications with regard to the effect of gender on the cognitive framework. However, it should be noted that the ecological validity of this study may be criticised with regard to the unrealistic task that the participants were asked to perform.

Due to the investigators not controlling for stressful events, the findings of the studies by Evans *et al.* (2005) and Rude *et al.* (2003) do not specify whether these negative cognitions need to be activated by external stimuli. Therefore, the results of these studies could be accepted as supporting any of the cognitive models discussed in this paper, except the symptom model. More specific research was carried out by Joiner *et al.* (1999), in which the interaction between negative life events and negative cognitions, as hypothesized by Beck's cognitive theory, was tested. The results of this study, in which self-report measures were used, support the notion that negative life events interact with negative cognitive styles to predict subsequent depressive symptoms (Joiner *et al.* 1999). This implies that a specific stressor is required to activate the negatively biased cognitive mediators to produce depression.

There is also evidence supporting the view that negative cognitions are a symptom or a consequence of depression. Lewinsohn *et al.* (1981) used a prospective design and self-report measures in a longitudinal investigation, in which they aimed to find out whether negative cognitions are a cause, a symptom or a consequence of depression. These investigators concluded that negative cognitions are merely symptom-related, meaning that they do not pose a vulnerability factor for depression or persist long after the depressed affect improves, and that "-- people who are vulnerable to depression are not characterized by stable patterns of

negative thinking of the type postulated by the cognitive theorists” (Lewinsohn *et al.*, 1981, pg. 218). However, again the reliability of self-report questionnaires can be doubted and the risk of demand characteristics is relatively high. In addition, Lewinsohn *et al.* (1981) identified that their sample might not be representative of the North American population from which it was drawn. However, this study is fairly free from ethical criticism and it can be accepted as support for the symptom model of depression. On the other hand, this study is challenged by studies such as the Gemar *et al.* (2001) study, in which mood induction procedures were used to detect latent depressive schemata in recovered depressed patients. Gemar *et al.* (2001) concluded that “-- even a mild negative mood in formerly depressed individuals can reinstate some of the cognitive features observed in depression itself ” (pg. 282). Evidently the Gemar *et al.* (2001) study could be accepted as supporting the notion that negative cognitions are a persisting consequence of the depressed affect, or that they are stable vulnerability factors as postulated by Beck (1970).

Strengthening the results of the study by Lewinsohn *et al.* are the results of a more recent investigation by Oei, Hibbert and O’Brien (2005). These investigators used a correlational method with self-report measures to test different cognitive models of depression, including the alternate aetiologies model, the integrated cognitive model and the symptom model of depression, among Latin-Americans living in Australia. The results of the study only support the symptom model of depression, and the researchers concluded that “--depression symptoms can have an impact on negative cognitions” (Oei, Hibbert & O’Brien, 2005, pg. 937). However, as Oei, Hibbert and O’Brien (2005) identified, the correlational method is rather weak in providing evidence specifically for the symptom model of depression, and more detailed longitudinal investigation is needed for the validation of this model. In addition, this finding contradicts the findings of many other studies already mentioned, as well as the investigation by Parry and Brewin (1988), in which strong support for the alternate aetiologies model and only limited support for the symptom model was found. Furthermore, Oei, Hibbert and O’Brien (2005) used self-report measures that had been translated from English to Spanish, wherefore it is questionable whether the exact wording remained the same or was slightly changed, which may have affected the results.

An important implication of the study by Oei, Hibbert and O'Brien (2005) is that the relationship between depression and negative cognitions might be mediated by the cultural background of the individual. Oei, Hibbert and O'Brien (2005) identified that the integrated cognitive model might not be applicable to different cultures, which was the initial reason for carrying out this research among Latin-Americans. The notion that cultural background plays a role in the relationship between negative cognitions and depression is supported by the fact that a research by Catteau and Chadral (2005), which was carried out in France, supports Beck's cognitive theory of depression, whereas the Oei, Hibbert and O'Brien (2005) study does not. Further strengthening the power of the hypothesis that in western cultures negative cognitions pose at least some kind of a risk factor for depression are the results of a very recently finished long term project conducted in U.S.A., namely the Cognitive Vulnerability to Depression-project (Abramson *et al.*, 2006). This project found that negatively biased cognitive mediators can act as independent risk factors for major depression (Abramson *et al.*, 2006).

Conclusion

The results of the studies by Evans *et al.* (2005), Rude *et al.* (2003), Joiner *et al.* (1999) and Abramson *et al.* (2006) support the hypothesis that negatively biased cognitive mediators are causal factors in depression. On the contrary, the results of studies by Lewinsohn *et al.* (1981) and Oei, Hibbert and O'Brien (2005) clearly propose these biases to have a consequential role. Thus, it is suggested that the relationship between negatively biased cognitive mediators and clinical depression is bidirectional, as already identified by Oei, Hibbert and O'Brien (2005). This means that negatively biased cognitive mediators may produce a depressed affect, or alternatively the depressed affect may lead to negatively biased mediators. Furthermore, from the comparison of the results of studies that were conducted in different cultures, such as the Oei, Hibbert & O'Brien (2005) and the Catteau & Chadral (2005) studies, it could be suggested that culture mediates the relationship between negative cognitions and depression. In western cultures, the causal relationship seems to operate more frequently from cognitions to affect, whereas in Latin-Americans it might more frequently be the other way round. However, due to the difficulty of identifying latent depressive schemata, more research is needed to clarify this

issue. Gender might also play a mediational role in the relationship between negative cognitions and depression, as suggested by the Rude *et al.* (2003) study.

It is not yet clear whether the cognitions necessitate an external stimulus to produce the depressed affect, although the results of the Cognitive Vulnerability to Depression-project (Abramson *et al.*, 2006) add to the growing evidence that negatively biased cognitive mediators may act as independent risk-factors for major depression. However, in no way should Beck's cognitive theory be refuted, since it provides a sound basis for understanding the cognitive processes of depressed individuals and was specifically supported by the Joiner *et al.* (1999) study. In fact, there might always be an environmental stressor activating the negatively biased cognitive mediators but everyone except the individual himself fails to identify the stressor. Furthermore, it could be suggested that the sensitivity to specific stressors depends on personality, as identified by Beck (1970). The learned-helplessness model and maybe even the differential activation model may coexist with Beck's theory and the integrated cognitive model, wherefore it could be concluded that all these models are valid in western cultures, and that the exact type of relationship between negatively biased cognitive mediators and depression depends on the cultural background, gender, personality as well as biological factors that play a part in the onset of depression.

It is left unanswered whether non-depressed individuals may have negatively biased cognitive mediators and if so, why are they not depressed? Furthermore, the relationship between the biological and cognitive factors is an interesting issue for future investigation, for example: Do low serotonin levels cause negative cognitions or is there no causal relationship between the two factors present in depression? What about genetic dispositions; do they cause the cognitive vulnerability or the depressed affect or neither? It is perhaps impossible to ever establish a holistic theory of depression, however, the success of existing methods of treatment suggests that we are on the right track to discovering key issues related to depression.

References

Abramson, L. Y., Seligman, A. E. P., Teasdale, J. D. (1978). Learned Helplessness in Humans: Critique and Reformulation. *Journal of Abnormal Psychology*, 87(1), 49-74.

Abramson, L. Y., Haefel, G. J., Metalsky, G. I., Hankin, B. L., Voelz, Z. R., Halberstadt, L., et al. (2005). Negative Cognitive Styles, Dysfunctional Attitudes, and the Remitted Depression Paradigm: A Search for the Elusive Cognitive Vulnerability to Depression Factor Among Remitted Depressives. *Emotion*, 5(3), 343-348.

Abramson, L.Y., & Alloy, L. B. (2006). Cognitive Vulnerability to Depression: Current Status and Developmental Origins. In Joiner, T. E. (ed.), Brown, J. S. (ed.), Kistner, J. (ed.). *The Interpersonal, cognitive, and social nature of depression*, pp. 83-100. New Jersey, U.S.: Lawrence Erlbaum Associates Publishers.

Alloy, L. B., Whitehouse, W. G., Robinson, M. S., Abramson, L.Y., Hogan, M. E., Rose, D. T., et al. (2000). The Temple-Wisconsin Cognitive Vulnerability to Depression Project: Lifetime History of Axis I Psychopathology in Individuals at High and Low Cognitive Risk for Depression. *Journal of Abnormal Psychology*, 109(3), 403-418.

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

Beck, A. T. (1970). *Depression, Causes and Treatment*. Pennsylvania, U.S.: University of Pennsylvania Press.

Catteau, V., Chabrol, H. (2005). Étude des Relations entre les Stratégies d'Adaptation aux Sentiments Dépressifs, la Symptomatologie Dépressive et les Idées Suicidaires chez l'Adolescent. *L'année Psychologique*, 105(3), 451-476.

Coyne, J. C., & Gotlib, I. H. (1983). The Role of Cognition in Depression: A Critical Appraisal. *Psychological Bulletin*, 94(3), 472-505.

Dent, J., & Teasdale, J. D. (1988). Negative Cognition and the Persistence of Depression. *Journal of Abnormal Psychology*, 97(1), 29-34.

Evans, J., Heron, J., Lewis, G., Araya, R., & Wolke, D. (2005). Negative Self-Schemas and the Onset of Depression in Women: A Longitudinal Study. *British Journal of Psychiatry*, 186, 302-307.

Gemar, M. C., Segal, Z. V., Sagrati, S., & Kennedy, S. J. (2001). Mood-Induced Changes on the Implicit Association Test in Recovered Depressed Patients. *Journal of Abnormal Psychology*, 110(2), 282-289.

Joiner, T. E. Jr., Metalsky, G. I., Lew, A., & Klocek, J. (1999). Testing the Causal Mediation Component of Beck's Theory of Depression: Evidence for Specific Mediation. *Cognitive Therapy and Research*, 23(4), 401-412.

Lewinsohn, P. M., Steinmetz, J. L., Larson, D. W., & Franklin, J. (1981). Depression-Related Cognitions: Antecedent or Consequence? *Journal of Abnormal Psychology*, 90(3), 213-219.

Mann, J. J. (1989). *Models of Depressive Disorders: Psychological, Biological and Genetic Perspectives. The Depressive Illness series, Volume 2.* New York, U.S.: Plenum Press.

Oei, T. P. S., Kwon, S.-M. (1994). The Roles of Two Levels of Cognitions in the Development, Maintenance, and Treatment of Depression. *Clinical Psychology Review*, 14(5), 331-358.

Oei, T. P. S., Hibberd, E., & O'Brien, A. J. (2005). Study of the Integrated Cognitive Model of Depression Among Latin-Americans. *Australian and New Zealand Journal of Psychiatry*, 39, 932-939.

Parry, G., Brewin, C. R. (1988). Cognitive style and depression: Symptom-related, event-related or independent provoking factor? *British Journal of Clinical Psychology*, 27(1), 23-35.

Rinck, M., & Becker, E. S. (2005). A Comparison of Attentional Biases and Memory Biases in Women With Social Phobia and Major Depression. *Journal of Abnormal Psychology*, 114(1), 62-74.

Rude, S. S., Covich, J., Jarrold, W., Hedlund, S., & Zentner, M. (2001). Detecting Depressive Schemata in Vulnerable Individuals: Questionnaires Versus Laboratory Tasks. *Cognitive Therapy and Research*, 25(1), 103-116.

Rude, S. S., Valdez, C. R., Odom, S., & Ebrahimi, A. (2003). Negative Cognitive Biases Predict Subsequent Depression. *Cognitive Therapy and Research*, 27(4), 415-429.

Räsänen, P. (August 14th 2006). Masennus. Retrieved August 22nd 2006 from <http://www.poliklinikka.fi/?page=1539839&id=4181430>.

Teasdale, J. D. (1988). Cognitive vulnerability to persistent depression. *Cognition and Emotion*, 2(3), 247-274.