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The role of cognitive and metacognitive maladaptive beliefs in the long-term course of unipolar depression

Dissertation

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Aims and hypotheses of the study

Depression is a mental disorder with high prevalence, affecting up to one of ten people worldwide. Extensive research has yielded considerable findings concerning the aetiology of depression, but not all factors are fully understood. Maladaptive cognitive beliefs have been shown to constitute an important vulnerability factor for depression. In addition, there is evidence that maladaptive metacognitive beliefs are linked to depression and that they are an important contributing factor in its development. However, it is unclear whether metacognitive beliefs can add explanatory power to depression above and beyond maladaptive cognitive beliefs. Also, the stability of metacognitive maladaptive beliefs as compared to the stability of cognitive maladaptive beliefs and of depressive symptoms has not yet been investigated.

The aim of the present study is to investigate the course and stability of cognitive and metacognitive maladaptive beliefs in the long-term course of depression.

Eighty-four patients with diagnosed depression are assessed with the Dysfunctional Attitudes Scale (DAS) as a measure for cognitive maladaptive beliefs, three subscales of the Metacognitive Questionnaire-30 (MCQ-30) as a measure for metacognitive maladaptive beliefs, the Hamilton Depression Rating Scale, and the Beck Depression Inventory at baseline. They are reassessed after 4 weeks, 6 months and 3.5 years. The data is analyzed using a longitudinal latent growths model approach.

The main hypothesis of the present study is that metacognitive maladaptive beliefs explain additional variance of the change of depressive symptom severity from the baseline assessment to the 3.5 years follow-up assessment above and beyond cognitive maladaptive beliefs. In accordance with a vulnerability-stress model, it is postulated that the change of depressive symptoms is predicted by the presence of a critical life event. Furthermore, it is hypothesised that metacognitive maladaptive beliefs are more stable than cognitive maladaptive beliefs and more stable than depressive symptoms over the examination period.

1. Introduction

Depression is one of the most prevalent mental disorders worldwide and is assumed to concern up to one of ten individuals (World Health Organization, 2016). It contributes importantly to the global burden of disease, ranging on the 5th place of leading causes of years lived with disability worldwide, and on third place in high-income countries (Vos et al., 2017). Because of high recurrence probability and high risk for chronicity, depression produces an important strain for concerned individuals (Bukh, Andersen, & Kessing, 2016; Eaton et al., 2008).

Theoretical disease models are vital as they influence the development of therapeutic approaches (Rose, 2003). Research has yielded remarkable findings concerning vulnerability factors considered to play an important role in the aetiology of depression (Goh & Agius, 2010). Empirical investigations have shown that different cognitive vulnerability factors exist, one of them being cognitive maladaptive beliefs (Scher, Ingram, & Segal, 2005). It is known that cognitive maladaptive beliefs are malleable and they constitute an important target in Cognitive Behavioural Therapy (CBT, Beck & Dozois, 2011). In spite of the efficacy of established therapies such as CBT, amelioration of therapeutic interventions is needed, as different challenges remain to be solved such as high recurrence rates (Eaton et al., 2008). For that reason, there is need to amplify theoretical knowledge about depression in order to improve the appropriateness of models. This, in turn, could lead to the enhancement of therapies. It is suggested that metacognitive maladaptive beliefs could play an important role in the aetiology of depression (Papageorgiou & Wells, 2001). However, the role of metacognitive maladaptive beliefs and its interplay with cognitive maladaptive beliefs is not fully conclusive and until now, no longitudinal study with a clinical sample exists to elucidate this interplay.

The present study examined the course and stability of cognitive and metacognitive maladaptive beliefs and depressive symptoms in a longitudinal design with 3.5 years follow-up in individuals with diagnosed depression. The aim was to investigate the effect of cognitive and metacognitive maladaptive beliefs on the long-term course of depression. This work starts with a presentation of symptomology, epidemiology, and aetiology of depression. This is followed by an outline of the current state of research concerning cognition and metacognition in depression, continuing with methods and results. Finally, results and conclusions concerning underlying theoretical models of depression are discussed and possible conclusions for therapeutic approaches are pointed out.

2. Theoretical background

2.1 Depressive disorders

2.1.1 Diagnostic criteria of depressive disorders according to DSM-IV

This study applied the American Psychiatric Association's (APA) definition of depressive disorders (also referred to as "unipolar depression") according to the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV, APA, 2005) and description will therefore be focused on this definition. The DSM-IV definition was used to achieve consistency with baseline assessments. Nevertheless, it has to be noted that meanwhile, the APA has published a new version of the DSM, the DSM-5 (APA, 2013) and important differences to DSM-IV will be mentioned.

The DSM-IV integrates the category "depressive disorder", which is subdivided in "major depressive disorder", "dysthymic disorder" and "depressive disorder not otherwise specified", in the section of "mood disorders" besides other disorders with mood disturbances as their predominant features. "Depressive disorders" are to be distinguished from "bipolar disorders" by the absence of maniac, mixed or hypomanic episodes in lifetime.[1] According to DSM-IV, the major depressive disorder is classified as a clinical course with at least one major depressive episode which is defined by the presence of either depressed mood or the loss of interest or pleasure in nearly all activities for a period of at least two weeks. Additionally, four further symptoms have to be present including somatic symptoms such as weight loss or insomnia, or cognitive symptoms such as feeling of worthlessness or guilt, concentration impairment, or suicidal ideation (see Table 1). The severity of major depressive episodes can be rated as mild, moderate, and severe without or with psychotic features. Diagnosis criteria do not differ significantly in DSM-5. A major depressive episode is considered "in remission", when the complete diagnostic criteria of a major depressive episode have not been met for at least two consecutive months and "in partial remission" in case when some criteria are still present, but do not conform with criteria for a major depressive episode (APA, 2005). The term "full recovery" is defined as a symptom-free period for six months after remission. The term "relapse" describes a return of depressive symptoms before full recovery and "recurrence" the return of depressive symptoms after full recovery (Berger, van Calker, Brakemeier, & Schramm, 2015).

[1] The DSM-5 subclassifies the section of "mood disorders" into two distinct sections and classifies "major depressive disorder" in the section of "depressive disorders" (APA, 2013).

A dysthymic disorder can be diagnosed if depressed mood is chronically present during two years on more than half of the days and if the patient suffers of at least two further symptoms such as poor appetite or loss of self-esteem (see Table 2). Symptom-free periods of two consecutive months may not occur and criteria of a major depressive episode must not be met. If a major depressive episode occurs on top of a dysthymic disorder, a double depression can be diagnosed. The new diagnosis of persistent depressive disorder in DSM-5 includes diagnostic criteria of both chronic major depression and dysthymia and criteria differ thus from DSM-IV (APA, 2013).

2.1.2 Epidemiology of depression

According to the Word Health Organisation (WHO), depressive disorders are estimated to be the most prevalent mental disorder across all regions and cultures, affecting up to one of ten people worldwide (World Health Organization, 2016). Unipolar depressive disorders contribute importantly to the global burden of disease. They are on 5th place of the leading causes of burden of disease worldwide, measured in years lived with disability, and they figure on the third place in high-income countries (Vos et al., 2017). In the United States of America, major depressive episodes have a life-time prevalence of 17 % and a 12-months prevalence of 9 % (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). In Germany, unipolar depression is estimated to have a 12-months prevalence of 8 % (Jacobi et al., 2016). Prevalence of unipolar depression is about twice as high in women than in men (in Germany, prevalence in women of 11 % and in men of 5 %; Jacobi et al., 2016). 12-months prevalence for dysthymia is estimated 2 % in Germany of which 2 % for women and 1 % for men (Jacobi et al., 2016). Data from the United States suggest that depression often remains untreated or inadequately treated (Wang et al., 2005).

Depression might occur in people of all ages, but prevalence is significantly higher in younger than in older individuals (Kessler et al., 2010). The peak of incidence appears to be in the twenties with 50% of initial manifestations of depression occurring before the age of 30, while onset of depression in older age remains possible (APA, 2013; Berger et al., 2015). Recent data seem to suggest that the incidence of particularly mild depression increases and that the age of the first manifestation drops which is referred to as the “cohort effect” (Berger et al., 2015).

Table 1: *Criteria for Major Depressive Episode according to DSM-IV (APA, 2005): 327)*

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

Note: Do not include symptoms that are clearly due to a general medical condition, or mood-incongruent delusions or hallucinations.

(1) Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful). **Note:** In children and adolescents, can be irritable mood.

(2) Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others)

(3) Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. **Note:** In children, consider failure to make expected weight gains.

(4) Insomnia or hypersomnia nearly every day

(5) Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down)

(6) Fatigue or loss of energy nearly every day

(7) Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)

(8) Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others)

(9) Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide

B. The symptoms do not meet criteria for a Mixed Episode.

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).

E. The symptoms are not better accounted for by Bereavement, i.e., after the loss of a loved one, the symptoms persist for longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

Table 2: *Criteria for Dysthymic Disorder according to DSM-IV (APA, 2005): 349*)

A. Depressed mood for most of the day, for more days than not, as indicated either by subjective account or observation by others, for at least 2 years. **Note:** In children and adolescents, mood can be irritable and duration must be at least 1 year.
B. Presence, while depressed, of two (or more) of the following:

(1) poor appetite or overeating
(2) insomnia or hypersomnia
(3) low energy or fatigue
(4) low self-esteem
(5) poor concentration or difficulty making decisions
(6) feelings of hopelessness

C. During the 2-year period (1 year for children or adolescents) of the disturbance, the person has never been without the symptoms in Criteria A and B for more than 2 months at a time.
D. No Major Depressive Episode has been present during the first 2 years of the disturbance (1 year for children and adolescents); i.e., the disturbance is not better accounted for by chronic Major Depressive Disorder, or Major Depressive Disorder, In Partial Remission.
Note: There may have been a previous Major Depressive Episode provided there was a full remission (no significant signs or symptoms for 2 months) before development of the Dysthymic Disorder. In addition, after the initial 2 years (1 year in children or adolescents) of Dysthymic Disorder, there may be superimposed episodes of Major Depressive Disorder, in which case both diagnoses may be given when the criteria are met for a Major Depressive Episode.
E. There has never been a Manic Episode, a Mixed Episode, or a Hypomanic Episode, and criteria have never been met for Cyclothymic Disorder.
F. The disturbance does not occur exclusively during the course of a chronic Psychotic Disorder, such as Schizophrenia or Delusional Disorder.
G. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).
H. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

While the DSM-IV states that prevalence of depression does not appear to be related to ethnicity, education, income or marital status (APA, 2005), the DSM-5 (APA, 2013) omits conclusions on this topic. Contrary from evidence from DSM-IV, recent studies indeed suggest that a low socio-economic status measured by income, occupation and education is associated with a higher prevalence of depressive disorders (Busch, Maske, Ryl, Schlack, & Hapke, 2013; Hoebel, Maske, Zeeb, & Lampert, 2017). Conclusively, low socio-economic status rises the probability for the occurrence of depressive symptoms (Lampert, Kroll, Lippe, Muters, & Stolzenberg, 2013).

Another important aspect of depression are psychiatric comorbidities because comorbid depression is often accompanied by an increased risk of suicide and chronification of depression (Deutsche Gesellschaft für Psychiatrie, Psychotherapie und Nervenheilkunde & Ärztliches Zentrum für Qualität in der Medizin, 2015). Depressed patients with axis-I-comorbidities are more likely to have more severe depressive disorders with a higher morbidity and a higher risk of chronification (Rush et al., 2005).[2] Kessler et al. (2003) showed in a large study conducted in the United States that about two thirds (64 %) of the participants with a major depressive episode in the last 12 months had a psychiatric comorbidity, of which anxiety disorders were leading with the list (58 %), followed by substance use (9 %) and impulse control disorders (17 %).

2.1.3 Course and long-term remission rates in unipolar depression

This study examined the long-term course of depression. The course of depression varies strongly among different individuals (APA, 2013). Depressive episodes can be self-limiting and can recover spontaneously (Deutsche Gesellschaft für Psychiatrie, Psychotherapie und Nervenheilkunde & Ärztliches Zentrum für Qualität in der Medizin, 2015). Before the introduction of psychopharmacological therapies, the mean duration of depressive episodes was about eight months (Berger et al., 2015). Nowadays, with new therapeutic possibilities, mean episode duration is estimated to be around 16 weeks (Rovner & Casten, 2003).

Depression is a disorder with a high risk of recurrence (Solomon et al., 2000). Eaton et al. (2008) conducted a prospective population-based cohort study with 23 years of follow-up to investigate among others recurrence risk. 35% of the patients who had a first episode of depression during the study also experienced recurrence and 85 % were in remission after 10 years irrespective of treatment. Similar data were reported in a prospective 5-year follow-

[2] The term “axis-I-comorbidities” is used to denote all psychiatric diagnoses except for personality disorders as well as mental retardation.

up study including patients with a first depressive episode and treated with antidepressant. After a first depressive episode, 83.5% of the patients obtained remission after 5 years and 31.5% experienced recurrence. In addition, researchers found that after a first episode of depression, about 50 - 65% of the patients suffer another depressive episode, and that 70 - 80% of the patients with a second episode suffered a third, and the risk of relapse after three episodes might be around 90 % (Eaton et al., 2008; Katon et al., 2001). Moreover, the risk of recurrence increases by 16% for each successive depressive episode (Solomon et al., 2000). It was demonstrated that the increased risk of recurrence persists lifelong (Kessing, Hansen, Andersen, & Angst, 2004). The likelihood of remission declines with increasing duration of illness (Keller et al., 1992) and the risk of recurrence declines with increasing duration of remission (Solomon et al., 2000). Hence, effective therapeutic approaches are urgently needed to reduce the risk of long-lasting depressive periods with the ultimate goal to avoid the risk of chronification.

In terms of therapeutic success, a meta-analysis including 25 studies examined the efficacy of psychological interventions (cognitive behavioural therapy, mindfulness-based cognitive therapy and interpersonal therapy) as compared to treatment as usual (routine clinical management, assessments only, no treatment and waiting list) or antidepressant therapy in the prevention of recurrence in depression (Biesheuvel-Leliefeld et al., 2015). The meta-analysis found supporting evidence that psychological interventions prevent recurrence more effectively than treatment as usual and antidepressant therapy. Another meta-analysis supports that antidepressant therapy and psychotherapy are equivalent in short-term treatment of depression, but that psychotherapy is superior in the longer-term management of depression (Spielmans, Berman, & Usitalo, 2011). Furthermore, the favourable results of recurrence reduction have been shown to remain stable in long-term follow-ups studies (Steinert, Hofmann, Kruse, & Leichsenring, 2014), albeit the authors indicated that long-term follow-up studies of psychotherapeutic interventions are rare and that their results are to be evaluated with reservations.

Different hypotheses for the high recurrence rate in major depression have been proposed (Bockting, Hollon, Jarrett, Kuyken, & Dobson, 2015). One is the “premorbid vulnerability thesis” according to which individuals with a high risk of recurrence have all the characteristics which render them vulnerable to recurrent depression, most of them genetic, even before their first episode (Burcusa & Iacono, 2007). Another hypothesis is the “scarring hypothesis” which assumes that each episode leaves residual changes that subsequently increase vulnerability for recurrence. Different types of changes have been proposed to be

related to biological (Lok et al., 2013), cognitive (Elgersma, Glashouwer, Bockting, Penninx, & Jong, 2013), or stress-related mechanisms (Beshai, Dobson, Bockting, & Quigley, 2011). Even if mechanisms of vulnerability for recurrence and relapse remain controversial, it can be concluded that recurrence in depression is frequent, that it is less likely to occur the longer a person has recovered and that it is more likely to occur in patients with a history of depression.

2.1.4 Aetiology of depression

Depression is a complex disorder and a variety of hypotheses on its aetiology have been proposed (Hautzinger, 2010). Hypotheses concerning the aetiology of depression are inserted in distinct disease models that can usually be characterized either in biological or psychological terms. As this work focuses on cognitive and metacognitive aspects of depression, psychosocial models on the aetiology of depression will be further explained in the following. Important psychological models that will be presented are the behavioural model after Lewinsohn (1974), Beck's cognitive model (Beck, Hautzinger, & Bronder, 1992), learned helplessness and attributional style according to Seligman (1976), and, more recently, the metacognitive model as proposed by Wells (Wells & Schweiger, 2011).

Empirical data on biological influence provide sound support for the influence of genetic factors (Lacerda-Pinheiro et al., 2014), epigenetic factors (Tsankova, Renthal, Kumar, & Nestler, 2007), alteration of neurotransmitter systems (Werner & Coveñas, 2013), alteration of intracellular signal transductor systems and neuroplasticity (Manji & Duman, 2001), altered sleep patterns (Modell, Ising, Holsboer, & Lauer, 2005), as well as alteration of neuroendocrine systems (Arborelius, Owens, Plotsky, & Nemeroff, 1999; Binneman et al., 2008).

In the light of the complexity of depressive syndromes, prevailing superordinate models now integrate several disease models into multidimensional so-called "biopsychosocial models" (Berger et al., 2015). One important superordinate model is the vulnerability-stress model which was originally developed by Zubin & Spring (1977) for schizophrenia and which is also applied to depression. According to this model, depression can be triggered by stressful life events with the prerequisite that the individual is vulnerable for depression. Vulnerability can be determined through multiple psychological, social and biological factors (Goh & Agius, 2010). Though a lot of research has been conducted in order to understand the aetiology of depression, not all factors are fully explained and empirically proven

and further research has to be pursued (Berger et al., 2015). In the following, psychosocial models relevant to this work will be presented more closely.

2.1.4.1 The behavioural model

Lewinsohn (1974) developed an influential behavioural model of depression which states that depression results from low levels of response-contingent positive reinforcement, mainly in social interactions. The amount of positive reinforcement depends on three factors: a) the number of events that potentially can be reinforcing for an individual, b) the availability of those events under defined circumstances, and c) the instrumental behavioural competencies of the individual to provoke reinforcement from the environment (Dimidjian, Barrera, Martell, Munoz, & Lewinsohn, 2011). Loss of positive reinforcement can result from environmental change such as the loss of a loved person, severe physical illness, loss of work, financial troubles or other important personal failures, as well as from personality traits (Dimidjian et al., 2011). According to this model, loss of positive reinforcement subsequently leads to depressed mood and resignation that, consequently, entail diminution of behaviour likely to provoke alternative positive reinforcement. The patient shows depressed behaviour. For the short term, the social environment usually reacts to depressive behaviour with help offers and affective sympathy, which supports maintenance of depression and depressed behaviour. Behaviour that usually led to positive reinforcement is prone to extinction. On the long term, though, the social environment often withdraws from the depressed person which can lead to decreased activity and somatic as well as vegetative symptoms (Dimidjian et al., 2011). Thus, according to the behavioural model, depression results from the loss of positive reinforcement, the depressed individual's reaction to that loss, and the environmental reaction to the behavioural change. Several studies have found support for Lewinsohns hypothesis (Gotlib, 1982; Youngren & Lewinsohn, 1980).

2.1.4.2 Learned helplessness and attributional style

Experimental studies on conditioned punishment in animals and humans repeatedly showed that exposure to uncontrollable and unpleasant events can provoke uncertainty, anxiety, passivity, and apathy (Seligman, 1976). Importantly, if test persons were afterwards confronted to controllable averse stimuli, they showed the same resigned behaviour (Seligman, 1976). This led to the formulation of the model of learned helplessness to explain depression (Seligman, 1976). Helplessness is a psychological state that is often provoked if a person feels that events are uncontrollable (Seligman & Rockstroh, 1979). According to this disease model,

if a person is confronted to important life events that are experienced as aversive and uncontrollable, such as disabling disease or long-term unemployment, he/she shows similar behaviour, including passivity, apathy, and resignation. Seligman assumes that the mere confrontation to uncontrollable events is not sufficient for learned helplessness, the individual needs to expect to be helpless. Seligman (1976) calls this the cognitive aspect of learned helplessness. When confronted to uncontrollable life events or failures, individuals usually try to understand the causes of those events. According to Seligman, that so-called attributional style can be characterised on three dimensions: internal – external, stable – unstable, global – specific (Seligman, 1976). If individuals cognitively judge the cause of their failures to be internal, stable and universal, they will expect to experience helplessness and failures in future events and will be convinced that they will have no means to positively influence the course of events. As per Seligman, this cognitive set will influence the affective, motivational, cognitive, vegetative, and motoric reaction to events. For example, the individual will show less motivation to react deliberately and will exhibit loss of appetite, loss of libido, or endocrine disturbances (Seligman & Rockstroh, 1979). The model of learned helplessness states that this behaviour leads to vulnerability for depression and maintenance of depressive symptoms (Seligman, 1976). Empirical studies found support for the importance of the attributional style (Raps, Peterson, Reinhard, Abramson, & Seligman, 1982). Furthermore, the model of learned helplessness is of vital importance for biological research on depression. A large number of animal models that investigate biological factors of depression are based on the model of learned helplessness and those models are supposed to have excellent validity in mice and rats (Vollmayr & Gass, 2013).

2.1.4.3 Becks cognitive model

Beck developed a cognitive model of depression that tries to explain how cognitive factors can lead to vulnerability for depression (Beck et al., 1992). The cognitive model acts on three main concepts: 1) the cognitive triad, 2) cognitive schemata, and 3) cognitive distortions (Beck et al., 1992). The cognitive triad consists of the cognitive patterns that determine how a person perceives his-/herself, the world, and his/her future. According to Beck et al. (1992), a depressed individual often describes his-/herself in a negative way and tends to explain negative experiences with his/her own psychic, physical or moral defaults, showing low self-esteem. The depressed person also interprets the world negatively, assuming that the world is asking things he/she can't achieve or is always placing obstacles in his/her way, hindering thus his/her personal achievement. He/she expects that this won't change in his/her future.

Instead, he/she thinks that his/her future will be an interminable succession of failures, frustration and disadvantages (Beck et al., 1992). The cognitive triad is based on negative cognitive schemata. Beck understands "cognitive schemata" as stable cognitive patterns with which an individual structures his/her experiences. Different schemata are developed in the course of life and are applied in appropriate situations in order to sort and categorise different competing stimuli. According to Becks disease model, vulnerability for depression can result from early negative experiences that lead to the development of latent negative schemata. In situations similar to the original one, those latent schemata are reactivated. Beck states that whereas patients with mild depression can question their cognitive schemata, in severe depression, negative schemata are activated in an increasing extend of situations until hyperactive idiosyncratic schemata structure the individual's complete cognitive organisation (Beck et al., 1992). Finally, Beck (1979) identifies six systematic cognitive distortions that lead to the maintenance of the negative schemata and stabilise vulnerability for depression.[3]

2.1.4.5 The metacognitive model according to Wells

Wells' aetiological model of depression focuses on so-called "metacognitions" in depression, that are understood as a set of cognitions about one's own beliefs that shape one's thinking style (Fisher & Wells, 2009). Wells states that when thinking about mental disorders, it is important to investigate the cognitive processes and mechanisms that shape the content of belief (Wells, 2001). For that reason, Wells developed a generic information processing model, the Self-Regulatory Executive Function Model (S-REF model), which he consecutively applied to different psychopathologies (Wells & Matthews, 1994). According to the S-REF model, there are three interacting levels of cognition: 1) automatic stimulus-driven processing that is highly reflexive, 2) controlled processing involved in conscious appraisal of events and in conscious control of actions and thoughts and 3) self-knowledge stored in long-term memory (Wells & Matthews, 1996). All emotional processing modes or configurations available for any individual take place in this tripartite architecture of cognition. One specific emotional processing mode is the S-REF mode. Wells supposes that the S-REF mode is the predominant mode in emotional disorders including depression. In this mode, self-knowledge as stored in long-term memory directs the appraisal of external events and body signs. Also, it regulates the appraisal of the significance of the own thoughts and guides subsequent cognitions (Wells & Matthews, 1996). The S-REF mode contains two

[3] For empirical support for the impact of cognitive distortions in depression and further information concerning the cognitive distortions, see 2.2.3.

types of self-knowledge: declarative beliefs (e.g. "I am a failure") and procedural beliefs on general plans concerning processing and coping. The latter is metacognitive knowledge that directs selective attention, memory retrieval and cognitive processing in response to external stimuli (Wells & Matthews, 1996).

According to Wells, development and maintenance of depression is due to biased metacognitions leading for example to inappropriate coping strategies or misguided attention. Wells supposes that in depression, metacognitive beliefs lead to a specific thinking style in responding to negative thoughts, which Wells calls "cognitive attentional syndrome", CAS. The CAS consists of prolonged stable thinking in form of worry, rumination, focus on threat, heightened self-focus, and counterproductive coping strategies (Fisher & Wells, 2009, 11). As to the metacognitive model of depression, if triggered by negative thoughts, a depressed patient will first react with positive metacognitive beliefs about worry and rumination, such as: "My worrying helps me understand my sadness" (Wells & Schweiger,

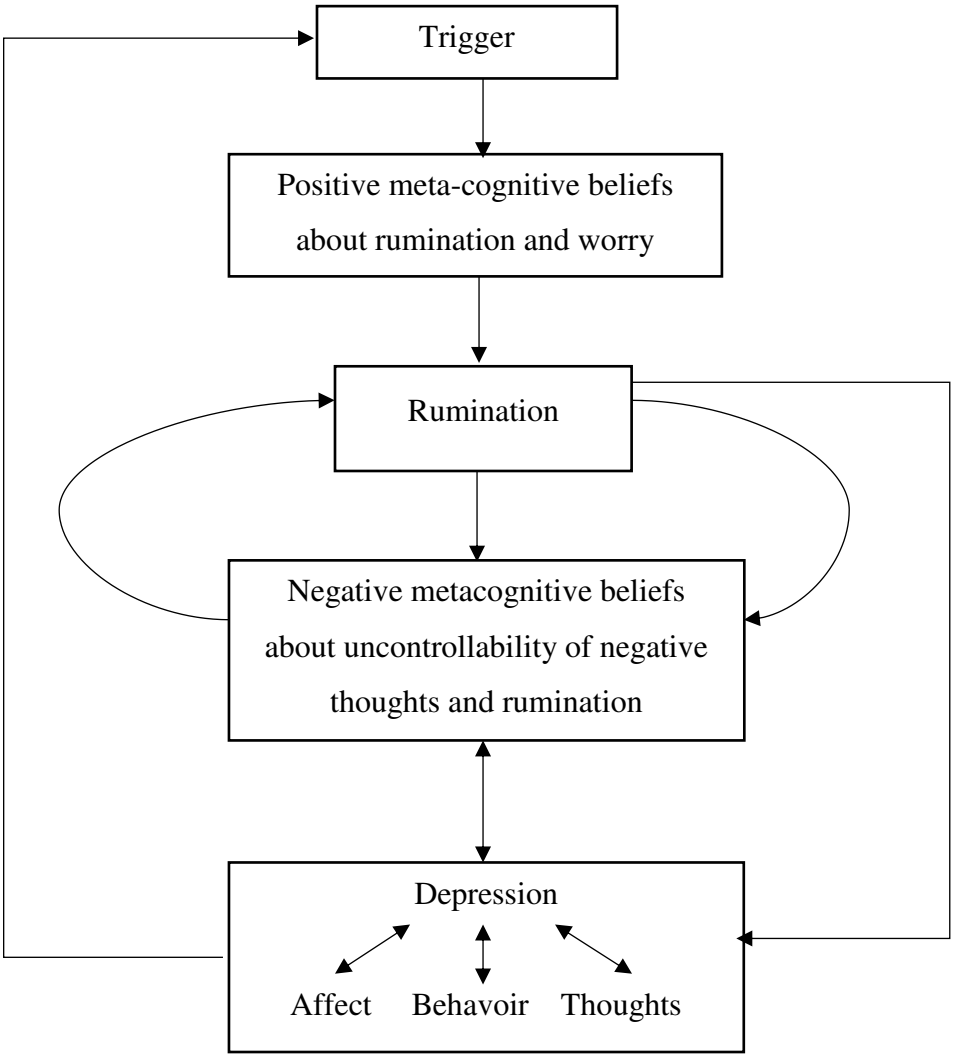

Figure 1 Adaption of Wells' metacognitive model of depression (2001, p. 47)

2011). Thus, the patient considers rumination to be a good strategy to achieve a desired state, for example alleviation of depressed feelings. In contrast, rumination will not give rise to understanding the depressed mood and alleviation but will lead to a concentration on rumination itself. This, according to Wells, will entail negative metacognitive beliefs about the uncontrollability of negative thoughts and rumination. The patient will then react with depressed behaviour such as social withdrawal, reduced activity, and hopelessness. Wells claims that constant rumination entails a loss of awareness for rumination so that the depressed patients ability to interrupt his/her maladaptive thinking style is interfered (Fisher & Wells, 2009). Thus, according to Wells, positive and negative metacognitive beliefs lead, when triggered through negative events or emotions, to the development and maintenance of depression. The metacognitive model of depression is summarised in figure 2.

2.2 Cognition and metacognition in depression

2.2.1 Cognitive distortions in depression

Cognitive distortions are an important constituent of many cognitive approaches to depression and have been subject of a huge body of empirical investigations (Scher et al., 2005). One can differentiate between cognitive maladaptive beliefs that concern salient, i.e. meaningful and depression-specific content, and more general cognitive biases and impairment of information processing that can occur in depression. Maladaptive cognitive beliefs refer to "dysfunctional attitudes" and "cognitive errors" (Beck, 1979). Beck (1979) first described a list of six maladaptive cognitive beliefs involved in onset, maintenance and recurrence of depression which was subsequently expanded by Burns (1999), Freeman and DeWolf (1992), and Freeman and Oster (1999).[4] Examples for more general cognitive biases are biased concentration and memory (Beblo, Sinnamon, & Baune, 2011), biased memory of events and false memories (Gotlib & Joormann, 2010), as well as biased emotion recognition (Liu, Huang, Wang, Gong, & Chan, 2012). As this work concentrated on the role of cognitive maladaptive beliefs in comparison to the role of metacognitive maladaptive beliefs, cognitive maladaptive beliefs will be described in more detail in the following.

Maladaptive cognitive beliefs are supposed to be developed as a consequence of latent cognitive schemata (Hautzinger & Pössel, 2017). A widely accepted definition states that cognitive schemata are constituted by "organized elements of past reactions and experience that form a relatively cohesive and persistent body of knowledge capable of guiding subsequent perception and appraisals" (Segal, 1988, 147).[5] A depressiogenic schema can be activated either directly by situations similar to the early situations which led to the development of the dysfunctional schema, or indirectly by fully active content-related schemata (Scher et al., 2005). It is generally supposed that when activated, a schema induces maladaptive cognitive beliefs and leads via negative dysfunctional attitudes and automatic thoughts to depressive symptoms (Hautzinger & Pössel, 2017). Hence, cognitive schemata, maladaptive cognitive beliefs, and dysfunctional attitudes seem to play an important role in the vulnerability for depression (Beck et al., 1992).

Labelling of maladaptive cognitive beliefs in depression is not consistent and depends on the study design and on the employed instruments (Yurica & DiTomasso, 2005).

[4] Cognitive distortions are common and can be found in different psychopathologies (Yurica and DiTomasso, 2005). This work focusses on the relevant cognitive and metacognitive distortions involved in onset, maintenance and recurrence of depression and will thus not address other cognitive distortions.

[5] The origins of the cognitive vulnerability to depression have been the subject of a substantial body of empirical studies. For a detailed review, see Ingram (2003).

Depression-specific maladaptive cognitive beliefs will briefly be introduced as they have been established in CB-T (Hautzinger & Pössel, 2017).[6] The first specific maladaptive cognitive belief, "all-or-nothing-thinking", describes the tendency to have absolute appraisals and to think in black-and-white. This is to say, judgments are not differentiated and gradual. For example, if a performance is not perfect, the exaggerated judgment "I am a total failure" is rendered. Dichotomous thinking has been demonstrated to be in many cases present in depression (Teasdale et al., 2001). Second, "overgeneralisation" refers to the maladaptive cognitive belief according to which a negative event is interpreted as an example of an infinite chain of negative events. A particularly extreme form of generalisation is so-called "labelling". One's own and other people's acting is labelled in emotional and not precise terms. For example, if an individual commits a minor misfortune, he/she is labelled a "complete idiot". Generalisation is connected to another specific maladaptive cognitive belief called "mental filtering" according to which one negative detail will negatively influence the appraisal of an event, tinting it in a negative way (Gotlib & Joormann, 2010). Another specific maladaptive cognitive belief in depression is "disqualifying the positive". This means that positive experiences are refuted by insisting that they don't count for any reason. Also, "magnification and minimization" often occurs in depressed individuals. It means that one's own faults are exaggerated whereas one's own achievements are minimized. On the opposite, other people's faults and achievements are evaluated the other way around. Effectively, studies documented reduced reward-sensitivity in depressed individuals (Eshel & Roiser, 2010). In addition, "arbitrary inference" or "jumping to conclusion" is regularly performed in depression. Negative assumptions are accepted on insufficient reasons. It includes "mindreading", which means presuming that others think negatively about oneself, and wrong predictions, which means evaluating events as predictors for negative future events and taking the predictions as facts. Pessimistic biases and predictions were shown to be common in depression (Strunk & Adler, 2009). Another maladaptive cognitive belief is "emotional reasoning". It describes the idea that emotions rather than objective facts will guide reasoning. A depressed individual will assume that negative feelings depict the reality. Berle and Moulds (2013) suppose that self-referent emotional reasoning might be associated with high levels of dysphoric symptoms, although emotional reasoning in general was found to be present in all individuals regardless of their level of depression.[7] Furthermore, so-called

[6] There is an abundance of empirical evidence for cognitive distortions in depression and exemplary studies will be cited subsequently.

[7] Berle and Moulds (2013) conclude that further investigations are needed to analyze the association between depression and emotional reasoning.

"should statements" have been identified as a typical maladaptive cognitive belief in depression. Statements on what one should, one must, or one must not, are employed as a means of motivation. Often, they express a perfectionism that cannot be met. This can lead to resignation and demotivation (for an overview concerning perfectionism as a risk factor for depression, see Egan, Wade, & Shafran, 2011). Finally, "personalization" has been closely connected to depression. It describes the fact that depressed individuals tend to feel responsible for negative events even if they are not responsible. Indeed, it has been demonstrated that depressed individuals tend to attribute negative events internally (Ball, McGuffin, & Farmer, 2008).

2.2.2 Metacognitive beliefs in depression

After presenting the concept of cognitive maladaptive beliefs in depression, the focus will now be turned to metacognitive maladaptive beliefs. Before stating most important findings concerning metacognitive maladaptive beliefs in depression, the evolution of the term will briefly be sketched.

High interest into metacognition is carried in different disciplines, including psychology, philosophy or educational sciences (Dunlosky, 2009). In philosophy, the idea that self-reflecting knowledge on one's thinking processes is essential for cognition can be traced back to antiquity (Dunlosky, 2009). Aristotle devotes important parts of *De anime* to the question how human thinking and perceiving might function and proposes a first suggestion of an architecture of mind (Caston, 2002). Nowadays, the concept of metacognition, its scope and function, is at the heart of the debate about self-consciousness and the functioning of mind (for an overview see Proust, 2010; van Gulick, 2017).

In the field of modern psychology, the idea was brought into focus by Flavell's studies concerning the development of memory in children (Bayne, Cleeremans, & Wilken, 2009). Flavell states that for the development of memory, cognitive structures for the retrieval and storage of memories as well as knowledge about mnemonic strategies are necessary: "It seems in large part to be the development of intelligent structuring and storage of input, of intelligent search and retrieval operations, and of intelligent monitoring and knowledge of these storage and retrieval operations – a kind of 'meta-memory' perhaps" (Flavell, 1971, p. 277). Beyond the investigations concerning meta-memory, the idea of meta-knowledge was expanded to different areas of cognitive processing (Flavell & Wellman, 1977). Nisbett and Wilson (1977) published an influential review which questioned the

capacity to gain real introspection into one's own higher-order cognitive processes. Thereupon, empirical investigations have focussed on metacognition, aiming at understanding three key-elements of metacognition: knowledge, control and monitoring of cognitions (Bayne et al., 2009). A further cardinal point for psychology and cognitive sciences was the introduction of a model of cognitive processing which involves two levels of processing, object-level and meta-level, by Nelson (1990).

Because of the multidisciplinary interest devoted to metacognition, the concept is complex and the term "metacognition" is not consistently defined (Semerari et al., 2012). Different definitions have been proposed. Metacognition is most basically understood as "thinking about one's thinking" (Moritz, Veckenstedt, Bohn, Köther, & Woodward, 2013, p. 359). Flavell defines metacognition as "knowledge and cognition about cognitive phenomena" and distinguishes between two subtypes: "metacognitive knowledge" and "metacognitive experiences" (Flavell, 1979, p. 906). Wells assumes that metacognition is "a multifaceted concept" which can be classified in three subtypes: metacognitive knowledge, metacognitive experiences and metacognitive control strategies (Wells, 2001, 7-9). So far, "metacognition" subsumes different cognitive functions associated with monitoring, acknowledging, and controlling thoughts on the one hand and beliefs concerning those cognitive functions on the other. The concept underlying this work is based on the broad definition given by Flavell and Wellman (1977). The term 'metacognition' is understood as cognitions about one's own thinking processes (also see Moritz & Lysaker, 2018). Regarding depression, metacognitive maladaptive beliefs are defined through the instruments used to assess them (see 2.3.5 Metacognitions Questionnaire-30).

There are further definitions to be noted. Koriat and Goldsmith (1998) stress the role of metacognition for certainty of judgment in memory processing and information retrieval. In agreement with that position, Moritz and Woodward (2006) assume that metacognition plays an important role in the impairment of memory processing, including overconfidence in memory errors as well as reduced confidence in correct memories. Gallo, Cramer, Wong, and Bennett (2012) employ the concept of metacognition in Alzheimer's research and use it to describe the awareness of cognitive declines. Lysaker et al. (2013) hypothesize that metacognition has more accurately to be understood as a spectrum of activities ranging from discrete activities to synthetic activities. Discrete activities are metacognitive processes allowing for the awareness of one's own experiences or of the accuracy of one's own judgements. Synthetic activities are involved in establishing complex representations of the own self and about other persons by integrating multiple information. According to this model,

discrete and synthetic activities are presumed to reciprocally influence one another (Aydin et al., 2016). Gumley (2011) has argued that metacognition should be conceived in a trans-diagnostic perspective, pointing to the close link between metacognition with social, affective, and interpersonal functioning in different psychopathological conditions. Pedone et al. (2017) have proposed a concept called "Metacognitive Multi-Function Modell" that brings together the concepts of metacognition, theory of mind, and mentalization.

Regarding depression, interest in metacognitions emanated from empirical findings on the role of rumination in the onset and maintenance of depression. Nolen-Hoeksema hypothesised that a ruminative response style to depressed mood which she defined as "behaviours and thoughts that focus one's attention on one's depressive symptoms and on the implications of these symptoms" would influence the duration of depressive symptoms (Nolen-Hoeksema, 1991, p. 569). In the past years, rumination has received increasing attention in empirical investigations concerning depression (Papageorgiou & Wells, 2003c). Studies supported that the induction of rumination in dysphoric patients would sustain depressed mood, whereas distraction would improve mood (Huffziger & Kuehner, 2009; Singer & Dobson, 2007). Sarin, Abela, and Auerbach (2005) demonstrated in a prospective study that the tendency to ruminate as a response to depressed mood was associated with an increase of depressive symptoms. An uncontrolled prospective study could find evidence that rumination predicted one-year relapse in depressed patients (Michalak, Hölz, & Teismann, 2011). Figueroa et al. (2015) conducted a prospective cohort study that confirmed rumination as a long-term relapse predictor in a 3.5 years interval. Rood, Roelofs, Bögels, Nolen-Hoeksema, and Schouten (2009) summarised in a meta-review that in non-clinical youths, rumination might be a vulnerability factor depression.

Wells and Matthews (1994) conjectured that a ruminative response style might be due to positive and negative metacognitive beliefs about the function and consequences of rumination when exposed to depressed mood. In an initial cross-sectional study, Papageorgiou and Wells (2001) found first evidence for their hypothesis. They conducted semi-structured interviews about rumination with 75 patients with MDE and without psychiatric axis-I-comorbidities. The interviews confirmed that all of the addressed patients shared the expected positive and negative metacognitive beliefs about rumination. As empirical findings suggest, most relevant metacognitive beliefs in depression seem to be positive metacognitive beliefs, negative metacognitive beliefs and metacognitive beliefs concerning need for control (Ruiz & Odriozola-Gonzalez, 2015). Positive metacognitive beliefs about worry and rumination (positive beliefs, PB) are those that express positive beliefs about the function of

worry and rumination as coping strategy in depressed mood. Examples would be: "Ruminating about my problems helps me to focus on the most important things", or "Ruminating about my feelings helps me to recognize the triggers for my bad things that have happened in the past" (Papageorgiou & Wells, 2003c, p. 14). Negative metacognitive beliefs (negative beliefs, NB) concern the uncontrollability, danger and negative consequences of rumination, for example: "It is impossible not to ruminate about the bad things that have happened in the past", "Ruminating will turn me into a failure", or "Ruminating makes me physically ill" (Papageorgiou & Wells, 2003c, p. 14). Metacognitive beliefs about the need for control (NFC) express the conviction that the own negative thoughts should be controlled and that it lays in one's own responsibility to make sure that rumination does not occur. Examples are: "I should be in control of my thoughts all of the time.", "It is bad to think certain thoughts", or "If I could not control my thoughts, I would not be able to function." (Cartwright-Hatton & Wells, 1997, p. 285). The findings led to the development of the Metacognitions Questionnaire-30 (MCQ-30; Wells & Cartwright-Hatton, 2004) which aims at testing the existence and intensity of metacognitive beliefs and comprises beyond the subscales for PB, NB, and NFC two further subscales called "cognitive confidence" (CC) and "cognitive self-consciousness" (CSC) (also see 2.3.5 Metacognitions Questionnaire-30). A presentation of empirical findings concerning depression-specific maladaptive cognitive belief and metacognitive beliefs in depression will be given in the next section.

The concept underlying this work is based on the broad definition given by Flavell and Wellman (1977). Regarding depression, metacognitive maladaptive beliefs are defined through the instruments used to assess them (see 2.3.5 *Metacognitions Questionnaire 30*).

2.2.3 Empirical findings concerning maladaptive cognitive and metacognitive beliefs in depression

2.2.3.1 Maladaptive cognitive beliefs in depression as vulnerability factors for depression

There is a huge body of empirical investigations concerning maladaptive cognitive beliefs in depression, often assessed in terms of "dysfunctional attitudes" via the *Dysfunctional Attitudes Scal*e (DAS; Weissman & Beck, 1978, see 2.3.4 Dysfunctional Attitudes Scale). Several longitudinal studies examined whether maladaptive cognitive beliefs predict onset or return of depressive symptoms, thus focussing on dysfunctional attitudes as cognitive vulnerability factors for depression. Relevant studies are summarised in Table 3 and most important findings will be presented in the following.

In a prospective 2.5 years follow-up study, Alloy et al. (2006) yielded evidence for the cognitive vulnerability hypothesis of depression. In their study, cognitive vulnerability predicted onset as well as recurrence of depressive symptoms over 2.5 years after controlling for age, gender, initial score in Beck Depression Inventory (Beck, Steer, & Hautzinger, 1995, see 3.3.3) and former history of depression. The risk of onset of major depression was about 7 times greater among high cognitive risk participants and risk of onset of minor depression was about 3.5 times greater. Negative life events have not, however, been considered in that report. As they seem to play an important role in the onset of depression, this could entail an important limitation of the study. Also, as high risk was defined as combination of dysfunctional attitudes and attributional style, it remains elusive which factor plays a more important role. Segal et al. (2006) conducted a longitudinal study where they followed-up patients with MDE in remission over 18 months during which relapses was investigated on a bimonthly basis. They found that dysfunctional attitudes were an important risk factor for relapse. As limiting factor, the study only investigated dysfunctional attitudes in patients in remission and did not include patients in their study that did not respond to therapy. Otto et al. (2007) confirmed the results in a large three-year prospective study with 700 never-depressed, currently depressed and formerly depressed women. The DAS score significantly predicted depressive episodes when controlling for the initial level of depression, but not when controlling for the former history of depression. Also, elevated DAS-sores were associated with a former history of depression. As limiting factor, it has to be mentioned that only women between 36 and 46 years of age where included in the study; hence generalisation might be problematic. Iacoviello, Alloy, Abramson, Whitehouse, and Hogan (2006) corroborated that maladaptive cognitive beliefs might play an important role in predicting the long-term course of depression. They conducted a 2.5 years follow-up study with 159 initially non-depressed individuals with whom diagnostic interviews and questionnaires were repeated every six weeks. Patients were grouped as per their scores in DAS and in Cognitive Styles Questionnaire composite for negative events according to their cognitive vulnerability in high and low risk groups. The high-risk group experienced more severe and more chronic courses of depression than the low-risk group. Jarrett et al. (2012) tested in a prospective study if primed or unprimed dysfunctional attitudes predict relapse in remitted patients with recurrent depressive disorder. Patients who responded to an initial cognitive therapy were followed-up during 24 months after 8 months of continuation therapy. The authors concluded that the unprimed level of dysfunctional attitudes predicts the risk of relapse in the following 32 months. The results stayed significant after controlling for the level of depression before

continuation therapy. Each additional point in the DAS score increased the risk of relapse by 1 %. The limitation of the study is that only patients who had responded to the cognitive therapy were included. Wang, Halvorsen, Eisemann, and Waterloo (2010) showed in a prospective longitudinal study with nine-years follow-up with clinically depressed, formerly depressed and never depressed individuals that early maladaptive schemata (during childhood developed body of coherent knowledge that guides appraisal and perception) are a significant prediction factor for depression (see Table 4)

Struijs, Groenewold, Oude Voshaar, and Jonge (2013) examined cognitive vulnerability in a large prospective study with one-year follow-up. Their data showed that cognitive vulnerability predicts an increase of depressive symptoms. Negative life events moderated this association. Kruijt et al. (2013) aimed to investigate the role of cognitive reactivity in the onset of major depression. They conducted a prospective study with a two-year follow-up including never depressed individuals. Their data support the hypothesis that cognitive reactivity precedes and predicts a first episode of depression. A major limitation of the study might be that the sample is recruited from a group at high risk for depression as they were chosen from mainly depressed and anxious patients. For that reason, generalization might be difficult. Importantly, Struijs et al. (2013) as well as Kruijt et al. (2013) did not measure dysfunctional attitudes using the DAS, but tested cognitive reactivity using the Leiden Index of Depression Sensitivity – revised (LEIDS-r). This might undermine comparability with the other cited studies.

The results from Beevers, Keitner, Ryan, and Miller (2003) differed slightly from the findings summarized until here. The authors examined in a prospective study with 121 depressed individuals the relation between cognitive change and prediction of relapse during one-year follow-up after hospitalization. A poor change of maladaptive cognitive beliefs during hospitalization predicted a shorter period until return of depression, while total scores of DAS after 6 months of outpatient treatment did not. As only severely depressed patients who responded to therapy were included, the sample of 53 patients was relatively small and generalisations are to be done with caution.

Moreover, there are two short-term prospective studies to be mentioned. Pedrelli, Feldman, Vorono, Fava, and Petersen (2008) tested 117 depressed or dysthymic patients who had responded to 8 weeks of pharmacotherapy after further 8 weeks. They confirmed their hypothesis that the interaction of high level of dysfunctional attitudes and high level of perceived stress would predict high level of depressive symptoms after therapy. Kuroda (2016) investigated the association between maladaptive cognitive beliefs, subjective stress

and depressive symptoms. Their findings suggest that maladaptive cognitive beliefs lead only indirectly to depressive symptoms via subjective stress, but not directly. It must be mentioned as limiting factors that 8 and 10 weeks are very short timeframes, which could limit possible conclusions.

To summarize, cognitive maladaptive beliefs seem to contribute to cognitive vulnerability for depression, increasing the risk of the onset of depression, the risk of relapse in remission and predict more severe and chronic courses. This effect could be moderated by a stressful life event or perceived stress. Furthermore, not only the level, but also the change of the level of maladaptive cognitive beliefs might play a role

Table 3*: Summary of methods and results of relevant studies concerning maladaptive cognitive beliefs as vulnerability factors for depression*

Author, year	Sample	Instruments	Follow-up	Main results
Alloy et al., 2006	- *n*=347 - healthy first semester students without psychiatric disorders - - cognitive high risk (HR, n=172) - cognitive low risk (LR, n=175)	- Cognitive Style Questionnaire (CSQ) - Dysfunctional Attitudes Scale (DAS) - Schedule for Affective Disorders and Schizophrenia—Lifetime (SADS–L) - Beck Depression Inventory (BDI)	2.5 years follow-up	Initial score in BDI and past episodes of depression predicted further depressive episodes over 2.5 years follow-up. Cognitive risk significantly predicted first onset as well as recurrences of depressive episode after controlling for gender, age, past history of depression and initial BDI.
Beevers et al., 2003	- *n*=53 - depressed inpatients who responded to combined pharmacological and psychosocial treatment	- Modified Hamilton Rating Scale for Depression (MHRSD) - Dysfunctional Attitudes Scale (DAS) - Extreme response style in DAS	- after treatment (t1) - after 6 months of continuation therapy (t2) - after 12 months (t3)	Poor change in dysfunctional attitudes and extreme response style was significantly associated with short periods to symptom recurrence. The absolute level of DAS and number of extreme responses after continuation therapy were not significantly associated with time to symptom recurrence.
Cannon et al., 1999	- *n*=138 - clinical outpatients with major depression	- Beck Hopelessness Scale - Dysfunctional Attitude Scale (DAS) - Cognitions Questionnaire (CQ) - Problem Solving Inventory (PSI) - Hamilton Rating Scale for Depression (HAMD-17)	None	Higher scores in DAS and poor problem solving significantly and non-redundantly predicted higher scores in hopelessness which seems to be associated to higher risk of suicidal tendendy in depression.

Author, year	Sample	Instruments	Follow-up	Main results
Ebrahimi, Afshar, Doost, Mousavi, & Moolavi, 2012	- *n*=130 - patients with major depressive disorder or dysthymic disorder (*n*=65) - matched healthy controls (*n*=65)	- diagnosis via clinical interview - Dysfunctional Attitude Scale-26-item (DAS-26) - General Health Questionnaire (GHQ-28)	None	Higher score in DAS-26 increased risk of depression. Individuals could be grouped in low-risk and high-risk groups on basis of their cognitive vulnerability. Likelihood of relapse in the high-risk group increased by 6.82 as compared to the low-risk group.
Halvorsen, Wang, Eisemann, & Waterloo, 2010	- *n*=149 - undergraduate students/ patients at GP: -clinically depressed (*n*=47) -formerly depressed (*n*=39) - never clinically depressed (*n*=29) -follow-up (*n*=115)	- Dysfunctional Attitude Scale (Form A) (DAS) - Young Schema Questionnaire (YSQ) - Beck Depression Inventory (BDI-I)	9 years follow-up	YSQ subscale "Undesirability" remained a significant predictor of depression 9 years later.
Iacoviello et al., 2006	- *n*=159 - initially not depressed participants who suffered a depressive episode during the study - grouped according to cognitive risk of developing a depressive episode	- Cognitive Style Questionnaire (CSQ) - Dysfunctional Attitudes Scale (DAS) - expanded Schedule for Affective Disorders and Schizophrenia-Lifetimeinterview (SADS-L) - Beck Depression Inventory I (BDI I) - SADS-Change (SADS-C)	2,5 years follow-up	Patients with high cognitive vulnerability defined on the basis of high scores in DAS and negative events subscale of CSQ had more depressive episodes with higher severity and more chronicity of depression during 2.5 years of follow-up.

Author, year	Sample	Instruments	Follow-up	Main results
Jarrett et al., 2012	- *n*=523 - patients with recurrent MDD without concurrent psychiatric axis-1 disorder - responders took part in phase (*n*=213)	- Visual Analog Scale (VAS) - Dysfunctional Attitudes Scale (DAS) - Hamilton Rating Scale for Depression (HAMD) - Longitudinal Interval Follow-up Evaluation-Psychiatric Status Ratings (LIFE-PSR)	- 8-months follow-up (after continuation therapy, t1) - 24-months follow- up (t2)	Unprimed DAS score results predicted relapse in the 32 subsequent months. Unprimed DAS score continued to predict relapse after controlling for level of depression before continuation therapy.
Kuroda, 2016	- *n*=103 - undergraduate psychology students	- Depressive Schemas Scale (DSS) - A new scale measuring subjective stress - Self-rating Depression Scale (SDS)	10 weeks follow-up	Dysfunctional attitudes at t0 led to depressive symptoms at t1 indirectly via subjective stress, but not directly.
Kruijt et al., 2013	- *n*=834 - Participants of Netherlands Study of Depression and Anxiety (NESDA) without past history of major depression or dysthymia	- Composite International Diagnostic Interview (CIDI) - Leiden Index of Depression Sensitivity – revised (LEIDS-r) - Implicit self-depressed associations (ISDA) - Lifetime version 2.1 of the Composite International Diagnostic Interview (CIDI) - Brugha Questionnaire - Inventory of Depression Symptomatology – Self Report (IDS-SR) - Neo five-factor inventory (NEO-FFI) on neuroticism	2 years follow-up	Cognitive reactivity was a significant predictor of incidence of depression, besides baseline depression and number of negative life events between t0 and t1.

Author, year	Sample	Instruments	Follow-up	Main results
Otto et al., 2007	- *n*= 750 - never-depressed women (*n*=500) - women with past history of depression without dysthymia (*n*=230) - women with current depression (*n*=20)	- Structured Clinical Interview for DSM–IV Axis I Disorders (SCID) - Dysfunctional Attitudes Scale (DAS) - Life Experience Survey (LES) - Hamilton Rating Scale for Depression (HAMD-17)	3 years follow-up	DAS scores at baseline assessment differed significantly between the groups with highest scores for currently depressed and lowest scores for never depressed participants. DAS scores significantly predicted new episodes of depression after controlling for initial levels of depression, but not when controlling for past history of depression.
Pedrelli et al., 2008	- *n*=117 - clinical patients with MDE and HAMD >15 who responded to pharmacotherapy with fluoxetine	- Dysfunctional Attitudes Scale (DAS) - Hamilton Rating Scale for Depression (HAMD-17) - Perceived Stress Scale (PSS)	-Before treatment (t0) - after 8 weeks of pharmacotherapy (t2) - 8 weeks of follow-up with two-weekly assessments (t3-t6)	Perceived stress and dysfunctional attitudes interacted significantly and predicted severity of depressive symptoms after therapy. Dysfunctional attitudes at baseline were not a significant univariate predictor of depressive symptoms.
Segal et al., 2006	- *n*=301 - outpatients with MDE at t0 - patients with MDE in remission at t1 (*n*=99)	- Dysfunctional Attitudes Scale (DAS) - Beck Depression Inventory (BDI) - Hamilton Depression Rating Scale (HAMD-17) - Mood rating via visual analogue scale (VAS)	-posttreatment evaluation (t1) - 18-months follow-up (t2)	Higher dysfunctional attitudes at t1 increased risk of relapse of depression in the 18 months following remission. Cognitive reactivity predicted relapse, also after having controlled for the number of previous depressive episodes.

Author, year	Sample	Instruments	Follow-up	Main results
Struijs et al., 2013	- *n*=2891 - patients with lifetime depressive and/or dysthymic disorder (*n*=2329) - at follow-up (*n*= 2455)	- Inventory of Depressive Symptomatology Self Report (IDS-SR) - The Leiden Index of Depression Sensitivity-Revised (LEIDS-R) - Mastery Scale - Implicit Associations Test (IAT) - List of Threatening Experiences (LTE)	1-year follow-up	Different factors of cognitive vulnerability (cognitive reactivity, external locus of control, explicit self-depressive associations) differentially predicted increase of depressive symptoms over time. This association was moderated by negative life events.

2.2.3.2 Stability of maladaptive cognitive beliefs

Several studies concerning the stability of cognitive maladaptive beliefs in depression exist. One important question that those studies address is whether those attitudes are state-like concomitants of depression or whether they are rather more stable trait-like vulnerability factors for depression. Empirical findings relevant to this work are depicted in Table 4 and the most important studies will be mentioned in what follows in more detail.

Zuroff, Blatt, Sanislow, Bondi, and Pilkonis (1999) conducted a prospective 18 months follow-up study with depressed patients. Their data, as analysed in three structural equation models, support the idea that maladaptive cognitive beliefs show significant state-dependency as well as significant stability during treatment period and follow-up time. Beevers and Millers (2004) investigation support this hypothesis. They tested 121 patients hospitalized for severe depression before inpatient therapy as well as 6 months and 18 months after admission. Their data provide support for the idea that maladaptive cognitive beliefs have trait and state characteristics as mood change influenced them while they also exposed underlying stability. The authors concluded that depressed individuals might have stable negative cognition, but that accessibility to them might depend on sad mood. As a limiting factor, a poor return rate of 65% after 18 months must be mentioned. Wang, Halvorsen, Eisemann, and Waterloo (2010) yielded similar results in their prospective longitudinal study with nine-years follow-up, showing that relative levels of dysfunctional attitudes from baseline assessment to the nine years follow-up was significantly stable, also after controlling for the level of depression at all assessments. Jarrett, Vittengl, Doyle, and Clark (2007) also confirmed that DAS scores remained relatively stable during their 2 years follow-up phase. A study from Hankin (2008) performed with children and adolescents aged from 11 to 17 also partly supports this idea, but results are more ambiguous. The study design comprised four follow-ups in 5 weeks. The mean-level stability of dysfunctional attitudes was significant for the younger aged, whereas for the overall sample, maladaptive cognitive beliefs showed mean-level change and increased over time. Test-retest correlation of dysfunctional attitudes was poor (r = .26), but the stability of maladaptive cognitive beliefs was significant in their full model fit, but with poor fit in some subgroups. The authors suggest that their data provide support for the idea that cognitive vulnerabilities exhibit trait as well as state characteristics, but acknowledge that their results are not fully conclusive.

To sum up, the presented findings support the view that dysfunctional attitudes are not a mere consequence of levels of depression, but have both state and trait characteristics.

This suggests that dysfunctional attitudes could play an important role as vulnerability factor for depression.

Table 4: *Summary of methods and results of relevant studies concerning stability of maladaptive cognitive beliefs*

Author, year	Sample	Instruments	Follow-up	Main results
Beevers & Miller, 2004	- *n*=121 - inpatients with MDE - *n*=99 at t2 - *n*=92 at t2.	- Dysfunctional Attitudes Scale (DAS) - Cognitive Bias Questionnaire (CBQ) - Beck Depression Inventory (BDI)	- after treatment (t1) -6-months follow-up (t2) -18-months follow- up(t3)	Current levels of depression as well as prior levels of dysfunctional attitudes influenced the current level of dysfunctional attitudes. Dysfunctional attitudes showed moderate test-retest correlation from t0 to t1.
Halvorsen et al., 2010	- *n*=149 - undergraduate students/ patients at GP: -clinically depressed (*n*=47) -formerly depressed (*n*=39) - never clinically depressed (*n*=29) - 115 at follow-up	- Dysfunctional Attitude Scale (Form A) (DAS) - Young Schema Questionnaire (YSQ) - Beck Depression Inventory BD (I-I)	9-years follow-up	Data suggested that DAS score might be a trait vulnerability factor for depression.
Hankin, 2008	- *n*=350 - healthy pupils aged from 11-17	- Adolescent Cognitive Style Questionnaire (ACSQ) - Children's Dysfunctional Attitudes Scale (CDAS) - Children's Response Styles Questionnaire (CRSQ) - Children's Depression Inventory (CDI)	5-months follow-up	Dysfunctional attitudes remained stable in the younger aged youths, but there were mean-level changes in the overall sample by increasing levels of dysfunctional attitudes over time.

Author, year	Sample	Instruments	Follow-up	Main results
Jarrett et al., 2007	- *n*=156 - patients with non-psychotic, recurrent, major depressive disorder and HDRS > 17 without concurrent medical disorders - ACT responders (*n*=84) - patients in follow-up phase (*n*=74)	- Structured Clinical Interview for DSM–III–R (SCID) -Inventory for Depressive Symptomatology, Clinician Version (IDSC) - Hamilton Rating Scale for Depression (HAMD-17) - Beck Depression Inventory (BDI) - Inventory for Depressive Symptomatology, Self-Report Version (IDSR) - Attributional Style Questionnaire (ASQ) - Dysfunctional Attitudes Scale (DAS) - Self-Efficacy Scale (SEF)	- after treatment (12-14 weeks of ACT, t1) - 8-months-follow-up (after continuation therapy or assessment only, t2) - 24-months follow-up (after 10 further sessions of CT or assessment only, t3)	The DAS score changed significantly during A-CT with Cohen's *d*=1.05. Depressive symptoms also changed significantly during A-CT with Cohen's *d*=1.55. Therapy-responders did not differ at pre-A-CT assessment in DAS-scores, but differed significantly from non-responders in lower DAS-scores at session 9 and 17 and 20. Patients who improved more on the DAS improved more in depressive symptoms. Early change in DAS did not significantly predict later change in depressive symptoms. The mean DAS score remained relatively stable over follow-up phase.
Wang et al., 2010	- *n*=149 - undergraduate students and patients at GP: - clinically depressed (*n*=61) - formerly depressed (*n*=42) - never clinically depressed (*n*=46)	- Dysfunctional Attitude Scale (Form A) (DAS) - Young Schema Questionnaire (YSQ) - Beck Depression Inventory (BDI-I)	9-years follow-up	The authors found significant relative stability of the dysfunctional attitudes level from baseline assessment to follow-up, also after controlling for level of depressive symptoms at both assess ments.

Author, year	Sample	Instruments	Follow-up	Main results
Zuroff et al., 1999	-*n*=250 - patients with depressive disorder -completed therapy (*n*=162) - complete data at t0 and t1 (*n*=154) - completed data at t3 (*n*=142)	- Dysfunctional Attitude Scale (DAS) - Beck Depression Inventory (BDI) - Depression subscale of Hopkins Symptom Checklist - Schedule for Affective Disorder and Schizophrenia- Change Version (SADS-C) - Hamilton Rating Depression Scale (HAMD-17)	- baseline assessment (t0) - post-treatment assessment (t1) - 6-months follow-up (t2) - 12-months follow-up (t3) - 18-months follow-up (t4)	After treatment, there were significant decreases in total the DAS-Score, in the subscale Need for Approval and Perfectionism as well as in depressive symptoms. At t1 and t3, correlation between DAS and depression were significant and moderately large. Dysfunctional attitudes showed relative stability from t0 to t1 with test-retest correlation .65 for Perfectionism, .56 for Need for Approval and. 65 for total DAS score. During follow-up, relative stability was even higher. Also, dysfunctional attitudes showed significant state dependency as well as significant stability over treatment.

2.2.3.3 Further relevant findings concerning maladaptive cognitive beliefs in depression

Concerning the mechanisms of change during therapy, a meta-analysis conducted by Garratt, Ingram, Rand, and Sawalani (2007) found that according to the majority of analysed studies, changes in cognition as function of cognitive therapies predicted a change in depression. Jarrett et al. (2007) suppose that changes in dysfunctional attitudes are a product of changes in depression, as their findings suggest that the change in depressive symptoms precedes the change in cognitive content. In their prospective RCT with two-year follow-up, decrease in DAS-score occurred early during acute phase cognitive therapy and remained stable during two-year follow-up. Higher improvement in DAS-score was correlated to higher decrease of depressive symptoms. To conclude, dysfunctional attitudes seem to be closely linked to depressive symptoms and several studies seem to support the view that they might play a role in the onset and recurrence of depression, but this role is not extensively understood.

Finally, some important meta-analyses should be mentioned. Scher et al. (2005) presented findings concerning cognitive distortions and depressogenic schemata as vulnerability factors for depression. The authors first focussed on cross-sectional priming studies, where the association between vulnerability, dysfunctional attitudes, negative mood induction and depressive symptoms was tested. The underlying hypothesis is that dysfunctional attitudes are latent in vulnerable individuals and can be activated by emotional triggers such as sad mood. This tendency to active dysfunctional attitudes when triggered is also called "cognitive reactivity". Vulnerability was mostly operationalized based on previous depressive episodes. A great number of studies showed that an endorsement of dysfunctional attitudes is associated with depressed mood states in individuals vulnerable for depression. Priming effects were consistently found in different studies, vulnerable individuals showing more dysfunctional attitudes in depressed mood states after having been primed. Next, Scher et al. (2005) analysed longitudinal studies which examined whether stressful life events in combination with vulnerability predicts depression over time. Several studies could show that dysfunctional attitudes predicted depressive symptoms within a week after confrontation to a stressful event. The results were more variable in studies where larger timeframes were investigated. The authors concluded nevertheless that the vulnerability-stress model of cognitive vulnerability based on dysfunctional cognitive attitudes is largely supported by evidence.

Further evidence for the importance of cognitive distortions in depression can be gained by the study corpus on cognitive behavioural therapy (CBT). CBT, which grounds

on the above-discussed principles, has been demonstrated to be effective in depression (Beck & Dozois, 2011; Butler, Chapman, Forman, & Beck, 2006; Clark & Beck, 2010).

2.2.3.4 Metacognitive maladaptive beliefs in depression

After describing the literature concerning cognitive maladaptive beliefs in depression, the next section will focus on the research concerning metacognitive maladaptive beliefs in depression. Cross-sectional studies provide evidence that maladaptive metacognitive beliefs are linked to depression and suggest that they could play an important role in the onset, maintenance and recurrence of depression. Most important findings are summarized in Table 5 and in the following paragraph. Maladaptive metacognitive beliefs were assessed with *Metacognitions Questionnaire 30* (MCQ-30; Wells & Cartwright-Hatton, 2004; see 2.3.5) or with *Positive Beliefs about Rumination* Scale (PBRS; Papageorgiou & Wells, 2001) and *Negative Beliefs about Rumination Scale* (NBRS, Papageorgiou & Wells, 2001). NBRS contains two subscales: negative beliefs about the uncontrollability and harm of rumination (NBRS 1) and negative beliefs about social and interpersonal consequences of ruminating (NBRS 2). PBRS and NBRS correlate significantly and positively with subscales NB, PB, CC and NFC of MCQ-30 with small to large effect sizes, where solely PBRS did not correlate significantly with CC (Roelofs, Huibers, Peeters, Arntz, & van Os, 2010). Roelofs et al. (2010) assume construct validity because of significant and positive correlations between PBRS and PB (r= .28), NBRS1 and NB and NFC (r= .61 and r=.48) as well as between NBRS2 and NB and NFC (r= .39 and r= .55). Still, the discriminatory power and construct validity of the different subscales can be questioned as PBRS also correlates significantly with NFC (r= .34) and NBRS with PB (r= .31).

Importantly, out of 11 cross-sectional studies, only four tested depressed individuals while the other studies assessed nonclinical participants. Generalizations to clinical individuals from non-clinical samples should only be carried out with precaution because findings can diverge as the Papageorgiou and Wells (2003b) study exemplifies. Cross-sectional studies with non-clinical participants established significant associations between metacognitive beliefs, depressive symptoms and rumination (Huntley & Fisher, 2016; Papageorgiou & Wells, 2003b; Roelofs et al., 2007; Solem, Hagen, Hoksnes, & Hjemdal, 2016) and a link between the subscales of MCQ-30 and negative emotions (Tajrishi, Mohammadkhani, & Jadidi, 2011). Yilmaz, Gençöz, and Wells (2015) concluded by means of their data that metacognitive beliefs are a better predictor of depression than dysfunctional schemata and cognitive maladaptive beliefs.

However, cross-sectional studies are certainly not appropriate for establishing causal relationships. Several longitudinal studies concerning the prediction of depression through metacognitive distortions exist that will be shortly presented in the following paragraph, all findings are also depicted in Table 5. It should nevertheless be noted that none of the longitudinal studies assessed clinical participants. Papageorgiou and Wells (2009) tested 164 nonclinical university students in a prospective study with 3-months follow-up. Their data showed that NB significantly predicted depressive symptoms 12 weeks later, after controlling for baseline rumination and baseline level of depression. Limiting factors of the study is the short follow-up and the young age of the participants (mean age 21.1 years; *SD*=4.2). It is not clear whether their findings can be transferred to depressed individuals in general. Also, only negative beliefs concerning uncontrollability and harm and social consequences were investigated which is only one of the assumed relevant metacognitions in depression. Yılmaz, Gençöz, and Wells (2011) examined the prediction of depressive symptoms through maladaptive metacognitive beliefs in 161 nonclinical students and adults after 6 months. They could show that metacognitions, particularly NB, predicted depressive symptoms after controlling for stressful life events and demographic variables and consequently seem to constitute an important vulnerability factor for depression. Nevertheless, because of low scores in BDI (*M*=8.43, *SD*=6.42) at intake and follow-up (*M* =6.74, *SD*=5.47), it is uncertain whether data apply for depressed individuals. Weber and Exner (2013) found in a prospective study with two-months follow-up with 60 nonclinical students that PB had a significant effect on rumination at follow-up after controlling for baseline rumination. Also, PB had an indirect effect on depressive symptoms at follow-up that was mediated through rumination. A small sample size, low age and the fact that only nonclinical students were tested are limiting factors of the findings. Ruiz and Odriozola-Gonzalez (2015) investigated in a prospective online survey with 289 nonclinical participants the relation between psychological inflexibility (key construct of Acceptance and Commitment Therapy), and maladaptive cognitive and metacognitive beliefs over nine months. Their data suggest that psychological inflexibility significantly mediated the longitudinal effect of metacognitive beliefs at baseline assessment, particularly PB, NB, and NFC, on depressive symptoms at follow-up. Only 106 participants (36,7%) completed both assessments and findings should thus be acknowledged with precaution.

A meta-review conducted by Normann, van Emmerik, Arnold, and Morina (2014) has analysed the efficacy of metacognitive therapy for depression as developed by Wells. Their data, based on 16 published and nonpublished studies of which 9 were controlled trials,

provided evidence for the efficacy of metacognitive therapy as compared to waitlist control groups (between group Hedges *g*=1.81) and cognitive behavioural therapy (between group Hedges *g*=0.97). Also, changes in metacognitions from pre- to post-treatment and follow-up were large (in-group Hedges *g*=1.18 and 1.31).[8] The efficacy of metacognitive therapy could provide support the model on which it is based.

[8] Changes in metacognition in patients after cognitive therapy were not investigated or reported.

Table 5: *Summary of methods and results of studies investigating metacognitive biases and depression*

Author (Year)	Sample	Instruments	Follow-up	Main results
Halvorsen et al., 2015	- *n*=168 - currently depressed individuals (*n*=37) - previously depressed individuals (*n*=81) - never depressed individuals (*n*=50)	- Structured Clinical Interview for DSM-IV, Axis I (SCID-I) - Beck Depression Inventory—Second Edition (BDI-II - The Beck Anxiety Inventory (BAI) - Metacognitions Questionnaire-30 (MCQ-30) - Thought Control Questionnaire (TCQ) - Ruminative Response Scale (RRS)	None	A discriminant function analysis was conducted to analyse the best indicators for discrimination between the groups. Two functions were found. The best discriminators of the first function were rumination, MCQ NB, TCQ Worry, TCQ Punishment and TCQ Distraction. The second function was best correlated with MCQ NFC, MCQ, CC, TCQ Reappraisal and MCQ PB
Huntley & Fisher, 2016	- *n*=715 - non-clinical students	-Inventory of Depressive Symptomatology-Self-Report (IDS-SR) -Ruminative Response Scale (RRS) - Dysfunctional Attitudes Scale (DAS) - Positive Beliefs about Rumination Scale (PBRS) - Negative Beliefs about Rumination Scale (NBRS): NBRS1: beliefs about the uncontrollability and harm, NBRS2: social and interpersonal consequences of ruminating	None	The study found support that metacognitive beliefs explain additional variance in depressive symptoms above and beyond dysfunctional attitudes and rumination. It also showed that the relation between BP and depressive symptoms is fully mediated by rumination and that the relation between NB and depressive symptoms was partially mediated by rumination. Negative beliefs about interpersonal and social consequences of rumination did not predict depressive symptoms.

Author (Year)	Sample	Instruments	Follow-up	Main results
Papageorgiou & Wells, 2003b Study 1	- *n*=200 - adults with Major Depressive Disorder	-Inventory to Diagnose Depression (IDD) - Ruminative Response Scale (RRS) - Positive Beliefs about Rumination Scale (PBRS) - Negative Beliefs about Rumination Scale subscales 1 and 2 (NBRS1+2) - Metacognitive Efficiency Subscale (MCQ3) of the Metacognitions Questionnaire	None	The study showed a significant association between depressive symptoms, rumination, PB, NB, and metacognitive efficiency. The study also suggests that PB are linked to rumination as a response to negative thoughts and mood. The relation between rumination and depressive symptoms seemed to be mediated by NB.
Papageorgiou & Wells, 2003b Study 2	- *n*=200 - nonclinical graduate and nongraduate psychological students (MDE as exclusion criteria)	-Inventory to Diagnose Depression (IDD) - Ruminative Response Scale (RRS) - Positive Beliefs about Rumination Scale (PBRS) - Negative Beliefs about Rumination Scale subscales 1 and 2 (NBRS1+2) - Cognitive Confidence Subscale (MCQ3) of the Metacognitions Questionnaire	None	Results differed in comparison to study 1 (depressed sample). As in group 1, there was a significant correlation between depressive symptoms, rumination, PB and NB, but there was no significant correlation with CC.
Papageorgiou & Wells, 2009	- *n*=164 - nonclinical university students	-Inventory to Diagnose Depression (IDD) - Ruminative Responses Scale (RRS) - Negative Beliefs about Rumination Scale 1+2(NBRS 1+2)	3-months follow-up	NB were found to prospectively predict depression when controlling for initial level of depression and rumination.

Author (Year)	Sample	Instruments	Follow-up	Main results
Roelofs et al., 2007	- *n*=196 - nonclinical undergraduates	- Quick Inventory of Depressive Symptomatology (QIDS) - Ruminative Response Scale (RRS) - Positive Beliefs about Rumination Scale (PBRS) - Negative Beliefs about Rumination (NBRS 1+2) -Miskimins Self-Goal Other Discrepancy Scale (MSGO)	None	The study showed a direct and an indirect link between rumination and depressive symptoms. NB mediated the indirect link. Also, a bidirectional link between rumination and NB was found.
Roelofs et al., 2010	- *n*=179 - patients with major depressive disorder	- inventory of depressive symptomatology (IDS) - Structured Clinical Interview for DSM-IV axis I (SCID-I) - Beliefs about Rumination Scale (PBRS) - Negative Beliefs about Rumination Scale 1+2(NBRS1, NBRS2) - Metacognitions Questionnaire-30 (MCQ-30), subscales 1,2,3,4 - Ruminative Response Scale (RRS) - Penn State Worry Questionnaire (PSWQ)	None	Rumination had a direct association with depressive symptoms, NB were not significantly associated to depressive symptoms. Negative metacognitive beliefs concerning the interpersonal and social consequences of rumination and uncontrollability and harm had a bidirectional relation with rumination. According to Roelofs et al., NB could contribute directly and indirectly (via rumination) to depression.

Author (Year)	Sample	Instruments	Follow-up	Main results
Ruiz & Odriozola-Gonzalez, 2015	- *n*=106 - nonclinical participants, anonymous online survey	- Acceptance and Action Questionnaire – II (AAQ-II) - Dysfunctional Attitude Scale – Revised (DAS-R) - Depression subscale of the Depression Anxiety and Stress Scales-21 (DASS-21) - Metacognitions Questionnaire-30 (MCQ-30), subscales positive beliefs, negative beliefs, need for control.	9-months follow-up	Psychological inflexibility statistically significantly mediated the relation between PB, NB and NFC at t1 and depressive symptoms at t2.
Solem et al., 2016	- *n*=1433 - Norwayan nonclinical participants	- Positive Beliefs about Rumination Scale (PBRS) - Ruminative Response Scale (RRS) - Negative Beliefs about Rumination Scale 1+2 (NBRS 1+2) - Beck Depression Inventory (BDI)	None	NBRS 1+2 and PBRS were all significantly associated with depressive symptoms and rumination. Their data suggests that PB predicted rumination that predicted NB which, in turn, predicted depressive symptoms.
Tajrishi et al., 2011	- *n*=300 - Iranian healthy students	- Metacognitions Questionnaire 30 (MCQ-30) - Hospital Anxiety And Depression Scale (HADS)	None	Four subscales of MCQ-30 were positively correlated with negative emotions: PB, NB, NFC and CC.
Weber & Exner, 2013	- *n*=60 - non-clinical undergraduate and graduate university students	- Positive beliefs about rumination scale (PBRS) - Rumination response scale (RSQ) - Beck depression scale-II (BDI-II)	2-months follow-up	The study found that PB at t1 significantly explained additional variance of depressive symptoms at t2. Also, the study showed that rumination at t2 mediated the influence of PB at t1 on the level of depressive symptoms at t2.

Author (Year)	Sample	Instruments	Follow-up	Main results
Yılmaz et al., 2011	- *n*=161 - non-clinical students and adults	- Meta-Cognitions Questionnaire-30 (MCQ-30) - Beck Anxiety Inventory (BAI) - Beck Depression Inventory (BDI) - Life Experiences Survey (LES) - Inventory of College Students' Recent life Experiences (ICSRLE)	6-months follow-up	The study showed that a higher level of NB predicted the increase of depressive symptoms independently of demographic variables and stressful life events.
Yilmaz et al., 2015	- *n*=251 - non-clinical undergraduate and postgraduate university students	-Negative Beliefs About Rumination Scale 1 and 2 (NBRS1 and NBRS2) -Positive Beliefs About Rumination Scale (PBRS) - Dysfunctional Attitude Scale-24 (DAS-24) - Beck Depression Inventory (BDI) - Beck Anxiety Inventory (BAI)	None	PB and NB explained a significant amount of variance in depressive symptoms above and beyond dysfunctional schemata. Higher levels of dysfunctional attitudes were correlated to higher levels of depressive symptoms, too, but metacognitive beliefs showed higher predictive values for depressive symptoms than the set of dysfunctional attitudes. They concluded that metacognitive beliefs make a greater contribution to depression than dysfunctional attitudes as measured by DAS.

2.3 Aims of the present study

As outlined in the introduction, depression is a chronic disease with a high recurrence probability. The present study examines a sample of initially depressed individuals over a period of 3.5 years. First, it aims at replicating findings concerning the long-term course of depression. At baseline, the examined patients had already suffered 3 depressive episodes on average. For that reason, I conjecture that the data will yield a rate of patients with a recurrent depressive disorder of around 70 - 90 % in accordance with studies in patients with a former history of depression (i.e. Hypothesis 1, H1). Next, according to established aetiological models, maladaptive cognitive beliefs are relevant in the aetiology of depression. Modification of those content-related cognitive biases seems to reduce depressive symptoms, whereas their stability seems to predict depressive symptoms. For that reason, maladaptive cognitive beliefs are supposed to be important targets in the therapy of unipolar depression. Moreover, empirical investigations have shown an association between maladaptive metacognitive beliefs and depressive symptoms. Still, only few studies concerning the association between depression and cognitive and metacognitive maladaptive beliefs in the long-term course have been published and the existing studies present limitations. Importantly, until now, no study has directly compared cognitive and metacognitive maladaptive beliefs in depressed individuals in a longitudinal setting to investigate which of them is a better predicator of the course of depressive symptoms. I conjecture that depressive symptoms and cognitive and metacognitive maladaptive beliefs decrease from baseline to 3.5 years follow-up assessment (H2). In light of the current state of empirical findings, I suppose that significant correlations between the change of both cognitive and metacognitive maladaptive beliefs and the change of depressive symptoms exist (H3a) and that metacognitive maladaptive beliefs explain additional variance of the data above and beyond cognitive maladaptive beliefs (H3b). In accordance with vulnerability-stress models, I suppose that change of depressive symptoms is also predicted by the presence of a critical life event (H3c).

Cognitive maladaptive beliefs have been shown to expose trait as well as state characteristics, which means that they correlate with depressive symptom severity while showing underlying stability. Long-term stability of maladaptive metacognitive beliefs has not yet been exhaustively examined. As metacognitive maladaptive beliefs are conceptualized as stable underlying cognitions on one's own thinking style, emotions and behaviour, I hypothesise that they are more stable than depressive symptoms as well as more stable than cognitive maladaptive beliefs which have been shown to expose a certain dependency on sad mood or stress (H4).

To sum up, the roles of cognitive and metacognitive maladaptive beliefs in long-term course of depression remain elusive. Aim of the study was to examine the role of cognitive and metacognitive maladaptive beliefs in depressed individuals over 3.5 years. The following hypotheses are to be examined:

H1) The recurrence rate is between 70 and 90 %.

H2) Self- and clinician-assessed severity of depression (HAMD, BDI), level of cognitive maladaptive beliefs (DAS) and level of metacognitive maladaptive beliefs (PB, NB, NFC) decrease from baseline (t0) to 3,5 years follow-up (t3).

H3 a) There is a positive correlation between initial status and change of cognitive (DAS), and metacognitive (BP, NB, NFC) maladaptive beliefs and initial status and change of self- and clinician assessed severity of depression (HAMD, BDI).

H3 b) The initial status and change of cognitive (DAS) and metacognitive maladaptive beliefs (PB, NB, NFC) from t0 to t3 explain additional variance of the change of depressive symptoms (DAS/BDI) from t0 to t3.

H3 c) The presence of a critical life event predicts the change of depressive symptoms from t0 to t3.

H4) Maladaptive cognitive and metacognitive beliefs (DAS, NB, PB, NFC) are more stable than depressive symptoms (DAS/BDI) and maladaptive metacognitive beliefs (NB, PB, NFC) are more stable than cognitive maladaptive beliefs (DAS) from t0 to t3.

3. Methods

3.1 Participants

Participants were recruited after their admission to a psychosomatic outpatient day clinic (RehaCentrum Hamburg). Psychologists and psychiatrists screened 171 patients shortly after their admission and 93 of them were assessed at baseline. Eighty-four patients were included in the study by previously defined in- and exclusion criteria. Inclusion criteria were the diagnosis of a depressive disorder (major depression or dysthymia) according to DSM IV and age between 18 and 65 years. Exclusion criteria were the presence of lifetime maniac or psychotic symptoms, suicidality (Suicidal Questionnaire Score Revised total score ≥ 7) or intellectual disability (estimated IQ < 70). All patients gave a written informed consent after trained staff had explained the aims of the study as well as the voluntariness of participation and the confidentiality of data. At admission, of 84 patients, 36 (43%) were diagnosed with a single episode of major depressive disorder, 47 (56%) with recurrent depression and 1 (1%) with dysthymia. Mean illness duration was 96 months (*SD*=104,96) and patients experienced on average a total of 3 depressive episodes (*SD*= 5,69) and were hospitalized for the second time (*SD*=1,13). Of 84, more than half of the patients (*n*=43; 51%) suffered of at least one comorbid psychiatric disorder, of which 38 (45%) met criteria for at least one comorbid anxiety disorder, 4 (5%) fulfilled criteria for a substance abuse disorder, and one patient (1%) met criteria for an eating disorder. At intake, 58 patients were medicated (antidepressants *n*= 54, antipsychotics *n*= 2, combination *n*=2). Nearly half of the patients (*n*=35; 42%) had received outpatient psychotherapeutic therapy prior to admission. Sociodemographic characteristics of the sample at inclusion point (t0) are displayed in Table 6.

Table 6*: Sociodemographic characteristics at inclusion time point (t0, Means (M) (standard deviations (SD)) or frequencies (percentages).*

Variable	Sample (*n*=84)
Age (years)	*M*=44.5 (*SD*=9,89)
Gender (female/male)	62 (74 %) / 22 (26 %)
Years of formal education	*M*=10.61, *SD*=1.69
Job status (working/ sick leave/ unemployed)	4 (5 %) / 47 (56 %) / 33 (39 %)

3.2 Procedure

We performed a parallel assessor-blind randomized clinical trial (RCT) about the efficacy of Metacognitive Training in Depression (D-MCT). All patients participated in diagnostic interviews including *Mini International Neuropsychiatric Interview* (M.I.N.I) and *Hamilton Depression Rating Scale* (HAMD) at baseline (t0), conducted by examiners blind to the diagnostic status. Moreover, they completed various questionnaires, including *Beck Depression Inventory* (BDI), *Dysfunctional Attitude Scale* (DAS) and *Metacognitive Questionnaire 30* (MCQ-30). Sociodemographic characteristics were investigated via a semi-structural interview. All patients participated in a standard treatment outpatient program, 5 days a week for 8 hours a day, which consisted of various psychological and physical interventions as well as medication. After the baseline assessment, patients were allocated to two different add-on therapies, D-MCT (experimental group) and health training (HT, active control group) according to a fixed computer-generated randomization plan with a 1:1 allocation ratio. After 4 weeks (post, t1) and 6 months (follow-up 1, t2), the same instruments were administered except for M.I.N.I. and sociodemographic characteristics. Three years after follow-up 1 (t3), all patients having participated in t0 were contacted by letter, email or via phone and invited to participate in a long-term follow-up (follow-up 2, t3). All patients provided once again written informed consent prior to participation in t3. All instruments from t1 were administered. Diagnostic interviews via phone were used to verify diagnostic status including HAMD. The study was approved by the Ethics Committee of the German Psychological Association (JL-SM_102015_amd_102011). The first part of the study (t0, t1, t2) was registered at the German Clinical Trials Register (No. DRKS00007907). The follow-up study (t3) was registered on the database ClinicalTrials.gov provided by the U.S. National Institutes of Health (No. NCT02771535). After having completed the follow-up 2 assessment, all patients received 30 € compensation. Figure 1 summarizes the patient flow.

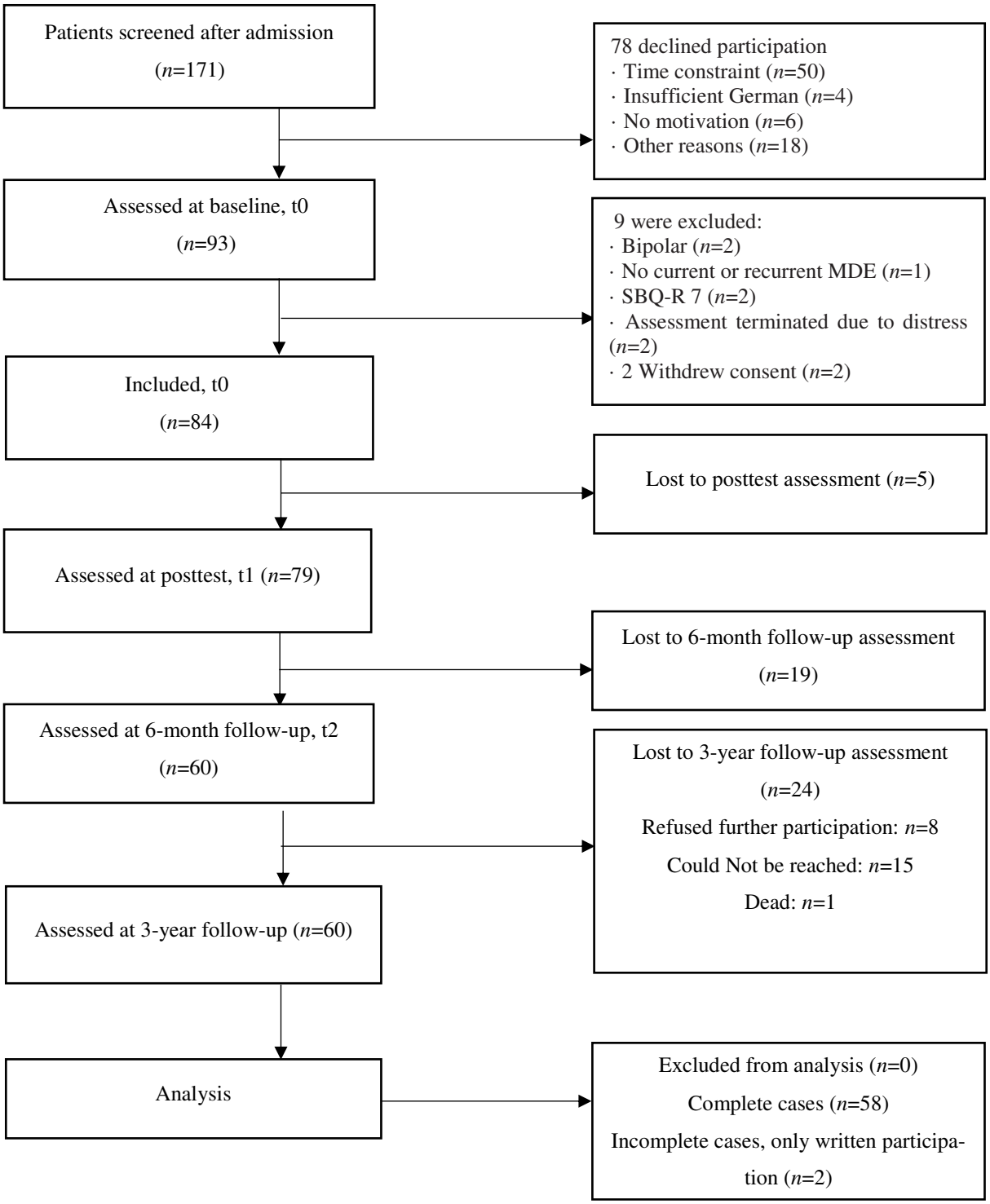

Figure 2 Flow of participants for the given study procedures and analysis.

3.3 Measurements

3.3.1 Sociodemographic variables

Sociodemographic background characteristics were investigated via a semi-structural interview. Obtained data included age, gender, educational background, drug abuse, smoking and drinking habits as well as medication. Moreover, relapses of depressive symptoms, psychiatric admission and outpatient psychotherapies since t2 were retrieved at t3.

3.3.1 Mini International Neuropsychiatric Interview

The psychopathological status and psychiatric comorbidities were assessed using *Mini International Neuropsychiatric Interview* (M.I.N.I.; Sheehan et al., 1998). The M.I.N.I. is a brief structured diagnostic interview with an administration time of about 15 minutes used to explore 15 axis-one disorders according to DSM-IV. The interview is structured in different sections, each of them investigating a different disorder. For each disorder, one or two screening questions are explored to rule out the diagnosis when negated. When answered positively, further questions regarding the main symptoms follow. Sufficiency to the criteria of good reliability and validity have been reported for the English and the French version, further studies have confirmed the results for the Italian and the Japanese version (Lecrubier et al., 1997; Rossi et al., 2004); Otsubo et al., 2005). In the present study, the German Version 5.0.0 was slightly modified in order to distinguish recurrent vs. single episode major depressive disorder as well as double depression. Suicidality was explored using the Suicide Behaviours Questionnaire-Revised (SBQ-R; Osman et al., 2001), therefore the section C of the M.I.N.I. which investigates suicidality was skipped.

3.3.2 Hamilton Depression Rating Scale

The severity of depression was measured using the 17-Item version of the *Hamilton Depression Rating Scale* (HAMD; Hamilton, 1960). The HAMD is a semi-structured interview conducted by trained interviewers. The administration time is about 20 to 30 minutes. Depressive symptoms which had occurred in the last 7 days prior to examination are investigated in 17 items. The items measure cognitive, affective, and somatic symptoms such as mood, feelings of guilt, suicidal ideation, quality of sleep, anxiety, weight loss, loss of libido, and hypochondriasis. A total score indicating the severity of depression is calculated by adding the scores of each item. Severe depression is defined as a score ≥ 25, whereas scores ≤ 8

are considered as subclinical depressive symptoms according to the German National Guidelines (DGPPN, 2015). A meta-analysis documented adequate to good reliability and good validity for the 17-version of the HAMD (Cronbach's α between .46 and .97, test-retest reliability between .81 and .98; Bagby, Ryder, Schuller, & Marshall, 2004).

3.3.3 Beck Depression Inventory

I used the *Beck Depression Inventory* (BDI; Beck et al., 1995) as second instrument to measure the severity of depression. The BDI is a self-reported questionnaire consisting of 21 items with four scalings ranging from 0 to 3. The questionnaire is used to investigate depressive symptoms that have occurred in the last 7 days prior to examination. It covers motivational, emotional, cognitive, and somatic symptoms matching the diagnosis criteria of major depression according to the DSM-IV criteria. In order to estimate the severity of depression, the scores of each item are added to a total score, where a total score of ≤ 10 is considered as subclinical depressive symptoms, 11-19 as mild, 20-29 as moderate, and a score ≥ 30 as indicator for clinically severe depressive symptoms according to the German National guidelines (DGPPN, 2015). In a meta-analysis, good reliability and validity were reported (Cronbach's α .86 for psychiatric patients; mean correlations of the BDI with HAMD .72; Beck, Street, & Garbin, 1988).

3.3.4 Dysfunctional Attitude Scale

The *Dysfunctional Attitude Scale*, Form A (DAS; Weissman & Beck, 1978; German Version: Hautzinger, Joormann, & Keller, 2005) was employed to assess dysfunctional cognitive beliefs. The DAS is a self-reported questionnaire consisting of 40 items rated by participants on a 7-point Likert scale (i.e., *totally agree, agree very much, agree slightly, neutral, disagree slightly, disagree very much, totally disagree*). The items are expected to capture the participant's self-evaluation and negative attitudes concerning the self, the world, and the future. Thus, they represent the content of typical dysfunctional cognitions in depression such as need for social approval (e.g. "I cannot be happy unless most people I know admire me"), perceived preconditions of happiness (e.g. "It is difficult to be happy unless one is good looking, intelligent, rich, and creative"), or dysfunctional attitudes concerning personal achievement or perfectionism (e.g. "If I fail partly, it is as bad as being a complete failure."). The score of each item is summed up to a total score indicating to which extent the participants' thoughts are distorted, ranging from 40 to 280. Scores of 140 and higher are considered as clinically relevant, scores of 100 and lower as inconspicuous. The original version

of the DAS contained 100 items and has been subdivided by the authors to two 40-items forms by a factor analysis. Consecutively, different factor analyses have been conducted mostly identifying one to three factors, labelled for instance "Dependency", "Achievement" or "Perfectionism", whereas no consent regarding the number and label of factors has been achieved (Cane, Olinger, Gotlib, & Kuiper, 1986; Power et al., 1994; Floyd, Scogin, & Chaplin, 2004; Beevers, Strong, Meyer, Pilkonis, & Miller, 2007; Moore, Fresco, Segal, & Brown, 2014). Still, good reliability (internal reliability measured by Cronbach α between .88 and .93 and test-retest reliability of .71; Weissman & Beck, 1978) and sufficient validity (correlation with BDI of .65 and with the depression scale of Profile of Moods States of .76; Weissman & Beck, 1978) have been reported for the 40-item version of the DAS.

3.3.5 Metacognitions Questionnaire-30

I employed three subscales of the *Metacognitions Questionnaire-30* (MCQ-30; Wells & Cartwright-Hatton, 2004) in order to assess metacognitive beliefs. The MCQ-30 is a 30-item short form of the original MCQ which comprises 65 items (Cartwright-Hatton & Wells, 1997). It is a self-reported questionnaire rated by participants on a four-point Likert scale (*do not agree, agree slightly, agree moderately, agree very much*). The MCQ-30 is composed of five distinct subscales eliciting different conceptual scopes of metacognition supposed to be relevant in the emergence and maintenance of different psychopathologies including depression (Papageorgiou & Wells, 2003a). The authors labelled the five scales as follows: (1) "positive beliefs about worry" ("positive beliefs", PB, e.g. "Worrying helps me to avoid problems in the future.", six items), (2) "negative beliefs about thoughts concerning uncontrollability and danger" ("negative beliefs", NB, e.g. "My worrying is dangerous for me.", six items), (3) "cognitive confidence" (CC, e.g. "I have little confidence in my memory for words and names", six items), (4) "negative beliefs concerning the consequences of not controlling thoughts" ("need for control", NFC, e.g. "If I did not control a worrying thought, and then it happens, it would be my fault.", six items), and (5) "cognitive self-consciousness" (CSC, e.g. "I think a lot about my thoughts.", six items). In the present study, only subscales (1), (2), and (4) were administered, as they were reported to be most related to depression (Ruiz & Odriozola-Gonzalez, 2015). For each scale, a score ranging from 6 to 24 can be calculated by adding the sums of the corresponding items. The higher the score, the higher is the extent of metacognitive distortions regarding the concerned factor, although no cut-off scores are defined. Good psychometric properties have been documented for the MCQ-30, such as good internal consistency for each subscale (Cronbach α ranging from .72-.93),

as well as for the total scale (Cronbach α .93; (Wells & Cartwright-Hatton, 2004). Previous studies showed good convergent validity, and acceptable to good test–retest reliability (Wells & Cartwright-Hatton, 2004).

3.4 Data analysis

IBM SPSS 24.0 (IBM Corp., Armonk, New York, NY) software was used for all analyses. Study completers and non-completers at t3 were compared concerning sociodemographic and psychopathological features at t0. Group comparisons were executed via t-test and Chi² test.

To test hypothesis one, recurrence data from patients participating in t3 assessment was analyzed. In line with DSM-IV criteria (APA, 2005), the diagnosis of recurrent depression was given when patients had suffered two or more Major Depressive Episodes with an interval of at least two consecutive months in which criteria for a Depressive Episode have not been met. In accordance with Zimmerman et al. (2012), remission was defined as a HAMD total score of 7 and below.

Subsequent data analysis was proceeded in two steps. First, latent growth curve models (LGMs) were calculated using Mplus (Muthén & Muthén, 2011). A latent growth model was calculated for each outcome variable (HAMD, BDI) and each predictor construct included in the theoretical model (DAS, PB, NB, NFC) to compute trajectories from baseline assessment to 3.5 years follow-up after treatment. For each variable, an intercept, indicating the status of the corresponding variable at baseline assessment, and a slope, specifying the total change from baseline to long-term follow-up 3.5 years after treatment, was estimated. The intercept was centered at baseline, the slope loading at baseline was fixed at zero, and the slope loading at the last assessment was fixed at 1.0. In order to be able to capture non-linear trajectories of change, the two intermediate slope loadings were freely estimated. Variances of the intercept and slope were allowed to covariate, but residual variances were supposed to be independent. A robust maximum likelihood was implemented to account for possible non-normality. Data were supposed to be missing at random, conditional on information in the model. The model fit was tested via goodness-of-fit indexes. The estimated intercept and slope scores for each participant were saved and used for further analysis.

Regarding hypothesis 2, the means of the slopes were calculated and one-sample T-tests were run to test whether the means of the relevant variables change significantly from t0 to t3.

In terms of hypothesis 3, bivariate relations between self- and clinician-assessed severity of depression (HAMD, BDI) and maladaptive cognitive (DAS) and metacognitive beliefs (PB, NB, NFC) were examined by calculating Pearson's correlations.

To test hypotheses 4, intercept and slope coefficients were used in multiple hierarchical regression models. First, assumptions for regression analyses were checked. Scatterplots showed linear relationships between predictor variables and outcome variable. Data exposed homoscedasticity (see appendix, Figure 2) and the residuals of the regression lines were normally distributed (see appendix, Figure 3). There was no perfect multicollinearity (Durbin-Watson d = 2,4). Though, in model A and B, values of PB, NB and NFC showed correlations between their respective slopes and intercepts (e.g. for model A: between intercept and slope of BP, r = -.85, p < .001; between intercept and slope of NP r = -.76, p < .001; between intercept and slope of NFC r = -.70, p < .001; correlations were similar for model B) suggesting multicollinearity. Multicollinearity can produce untrustworthy unstandardized regression coefficients b, because collinearity increases the standard error of b, it can limit the size of R and R^2 and it renders it difficult to assess the importance of individual predictors (Field, 2016). The variance inflation factor (*VIF*) as well as tolerance statistics (1/*VIF*) were further used to check for multicollinearity. Multicollinearity is likely if the largest VIF is larger than 5 or 10 and/or if tolerance is less than 0.2 or 0.1 (O'brien, 2007). In model A and B, those criteria were not met (model A and B: largest *VIF* = 4.4, the lowest tolerance of 0.23). For that reason, sufficient independency of predictor variables was assumed.

Models A1-3 analysed the change of depressive symptoms over time. For that reason, the slope of HAMD was entered as dependent variable. Preliminary regression analysis tested the influence of the expected risk factors (age, gender, initial status of depression, former history of depression). Significant risk factors were entered as first block predictors (model A1), intercept and slope of DAS were included as second block predictors (model A2), and intercepts and slopes of PB, NB and NFC were added as third block predictors (model A3). Model B tested the model with slope and intercept of BDI as secondary outcome parameter instead of HAMD.

To test hypothesis 5, further regression models were run to investigate whether critical life events predicted the association between vulnerability factors, i.e. cognitive and metacognitive maladaptive beliefs (DAS, PB, NB, BFC), and depressive symptom severity. To test the influence of a critical life event between t2 and t3, this variable was included in the hierarchical regression as last step (models A4 and B4).

Finally, in order to test hypothesis 6, the relative stability of the relevant variables was compared on the basis of the associated slopes and of the basis of the test-retest correlations. For each construct, a standardized positively pooled within-group effect size *d* was calculated by dividing the mean change from t0 to t3 assessment by the standard deviation of the baseline measurements. This *d* can be used to assess a constructs stability relative to the other constructs, as it measures mean change of a construct from t0 to t3: the higher *d*, the more change has occurred from t0 to t3. Relative stability was compared to test-retest correlation *r* between t0 and t3 of each construct.

Cohen's (1988) rules of thumb were applied to evaluate size of the measured effects. Standardized regression weights (β) and correlation coefficients (*r*) of .1, .3, .5 were considered as weak, medium, and strong effects. Findings with $p < .05$ were considered statistically significant.

4. Results

4.1 Sample characteristics

The sociodemographic characteristics of the patients that completed t3 assessment are displayed in Table 7. For the sample characteristics at baseline assessment, see 3.1. Sample characteristics at t1 and t2 have been described elsewhere (Jelinek et al., 2016). Completers and non-completers did not differ statistically significantly regarding age (p = .45), years of formal education (p = .97) gender (p = .35), intelligence as measured by MWTB 2 (p = .65) at t0. Furthermore, they were statistically indistinguishable concerning their psychopathologic status at t0: HAMD (p = .95), BDI (p = .59) comorbidities (p = .65). For a comparison of t3 completers and non-completers at t0, see Table 8.

Table 7*: Sociodemographic characteristics at 3,5 years follow-up (t3). Means (standard deviations) or frequencies (percentages)*

Variable	**Sample**	
Age (years)	n = 60	M=49.61 (SD=10,14)
Gender (female/male)	n=60	47 (78 %) / 13 (22 %)
Years of formal education	n=60	M=10.61, SD=1.69
Job status (employed/ education / retired / unemployed / housewife/ -husband)	n=59	25 (42 %) / 1 (2 %) / 18 (31 %) / 10 (17 %) / 5 (9 %)
Critical life event between t2 and t3 (yes / no)	n=58	41 (71 %) / 17 (29 %)

Table 8: *Sociodemographic characteristics at baseline assessment (t0) of t3 completers and non-completers. Means (standard deviations) or frequencies (percentages)*

	T3 Completers		**T3 Non-Completers**	
Variable	**Sample**		**Sample**	
Age	*n*=60	*M*=45.98, *SD*=10.18	*n*=24	*M*=44.17, *SD*=8.80
Gender (female/male)	*n*=60	46 (77 %) / 14 (23 %)	*n*=24	16 (67 %) / 8 (33 %)
Years of formal education	*n*=60	*M*=10.60, *SD*=1.73	*n*=24	*M*=10.62, *SD*=1.61
Intelligence (MWTB)	*n*=60	*M*=52.52 *SD*=8.71	*n*=23	*M*=51.52, *SD*=8.70
HAMD	*n*=60	*M*=15.46, *SD*=5.03	*n*=24	*M*=15.54, *SD*=5.71
BDI	*n*=60	*M*=25.58, *SD*=10.88	*n*=23	*M*=26.93, *SD*=7.97
Comorbidities	*n*=56	*M*=0.68 *SD*=0.79	*n*=23	*M*=1.00, *SD*=1.28

4.2 Hypothesis 1

Table 9 displays the psychopathological data of patients that completed the t3 assessment including mean total score of HAMD, classification according to severity of depression via HAMD, mean total score of BDI, classification according to severity of depression via BDI, total number of stays and mean stay duration in psychiatry since t2, ambulant psychotherapy since t2, current alcohol consume and current psychopharmacological medication. At t3, of 58 patients, 22 patients (38%) were diagnosed with an episode of major depressive disorder, 45 patients (78 %) met criteria for recurrent depression and 17 patients (29 %) for dysthymia.[9] Of 58, 13 patients (22 %) could be diagnosed with a double depression (i.e. major depressive episode and dysthymia). At t3, the majority of the patients (*n*=47; 81 %) suffered of either a recurrent depression or dysthymia or both. Patients experienced on average a total of 17.26 depressive episodes (*SD*=23.97) in their lifetime. At t3, of 58, 27 patients (47 %) had a HAMD ≤ 7 and can thus be considered in remission according to Zimmerman et al. (2012). A recurrence rate of 78 % is in line with findings from literature that state a recurrence rate of 60 – 90 % in patients with a former history of depression. Hence, hypothesis 1 can be confirmed.

[9] The diagnoses „major depressive disorder“, „recurrent depression“ and „dysthymia“ ca be given side by side. For that reason, the percentages add up to more than 100 %.

Table 9: *Psychopharmacological data at 3,5 years follow-up (t3). Means (standard deviations) or frequencies (percentages)*

Variable	**Sample**	
HAMD Total score	*n*=58	*M*=10.90 (*SD*=7.90)
HAMD (no depression/ mild depression/ middle depression / severe depression)	*n*=58	28 (48 %) / 13 (22 %) / 14 (24 %) / 3 (5 %)
BDI Total score	*n*=59	*M*=17.49 (*SD*=11.68)
BDI (no depression/ mild depression/ middle depression / severe depression)	*n*=59	20 (34 %) / 16 (27 %) / 12 (20 %) / 11 (19 %)
Number of stays at psychiatry t2-t3 (none / once / twice /more than twice)	*n*=58	51 (88 %) / 4 (7 %) / 2 (3 %) / 1 (2 %)
Mean duration of stay at psychiatry (days)	*n*=58	*M*=53.25 (*SD*=31.66)
Ambulant psychotherapy since t2 (no therapy / in search of therapist / current therapy / completed therapy between t2 and t3)	*n*=58	19 (33 %) / 4 (7 %) / 22 (38 %) / 13 (22 %)
Type of therapy (Cognitive therapy / Psychodynamic psychotherapy / other)	*n*=35	19 (54 %) / 8 (23 %) / 8 (23 %)
Alcohol Consume (g/week)	*n*=58	*M*=54.35 (*SD*=176.05)
Psychopharmacologic Medication t3 (None / antidepressant / neuroleptic / combination (antidepressant + neuroleptic)	*n*=58	34 (59 %) / 17 (29 %) / 0 (0 %) / 7 (12 %)

Note HAMD = clinician-assessed depression, BDI = self-assessed depression

4.3 Hypothesis 2

Initial status and change from t0 to t3 of self- and clinician-assessed severity of depression, cognitive (DAS) and metacognitive maladaptive beliefs (PB, NB, NFC) are depicted in Table 10. One sample t-tests showed that all slopes are significantly different from zero: HAMD (t (83) = 10.66, $p < .001$); BDI (t (83) = 9.87, $p < .001$); DAS (t (83) = 9.25, $p < .001$); PB (t (83) = 2.82, $p = .01$); NB (t (83) = 6.7, $p < .001$); NFC (t (83) = 5.54, $p < .001$). As presumed, levels of all relevant predictor and outcome variables significantly reduce from base-line to 3,5 years follow-up assessment. Hence, hypothesis 2 could be confirmed.

Table 10: *Initial status and change from t0 to t3 of self- and clinician assessed depression, cognitive and metacognitive maladaptive beliefs*

Variable	Sample	*M*	*SD*
Initial status HAMD (intercept)	*n*=84	15.53	4.49
Change of HAMD (slope)	*n*=84	-4.86	4.18
Initial status BDI (intercept)	*n*=84	25.86	10.11
Change of BDI (slope)	*n*=84	-9.11	8.46
Initial status DAS (intercept)	*n*=84	166.26	33.44
Change of DAS (slope)	*n*=84	-20.93	20.73
Initial status PB (intercept)	*n*=84	12.98	3.88
Change of PB (slope)	*n*=84	-0.96	3.14
Initial status NB (intercept)	*n*=84	17.89	3.51
Change of NB (slope)	*n*=84	-2.79	3.82
Initial status NFC (intercept)	*n*=84	13.78	3.99
Change of NFC (slope)	*n*=84	-2.14	3.55

Note HAMD = clinician-assessed depression, BDI = self-assessed depression, DAS = cognitive maladaptive beliefs, PB = positive metacognitive beliefs, NB = negative metacognitive beliefs, NFC = metacognitive beliefs about the need for control

4.4 Hypothesis 3a

The correlations between the intercepts and slopes of self- and clinician-assessed depression as well as the scores of cognitive and metacognitive maladaptive beliefs are displayed in Table 11. The initial status of HAMD and BDI (i.e. intercepts) were significantly positively correlated with the initial status of DAS, NB, NFC (i.e. intercepts) at small or medium effect sizes. This means that the higher the initial severity of depression, the higher the initial score in DAS, NB and NFC. Also, a change of HAMD (i.e. the slope) was positively associated at medium effect sizes with changes of DAS and NB and at small effect size with change of NFC (i.e. the slopes). A change of BDI was positively correlated at medium effect size with changes of DAS, PB, NB, and NFC. Thus, a change of depressive symptoms was positively correlated with a change of maladaptive cognitive beliefs and metacognitive beliefs and hypothesis 3a could be confirmed. The correlations between initial scores and difference scores between t0 and t3 of self- and clinician-assessed severity of depression, cognitive and metacognitive maladaptive beliefs with available data are depicted in the appendix (Table 17).

The correlation Tables between the different testing times for each construct are shown in the appendix (Tables 20-25) using available data.

Table 11: *Correlations of initial status and change from t0 to t3 of depression with initial status and change from t0 to t3 of maladaptive cognitive and metacognitive beliefs, LGM data*

	Initial status of DAS	Change of DAS	Initial status of PB	Change of PB	Initial status of NB	Change of NB	Initial status of NFC	Change of NFC
Initial status of HAMD	.36**	.24*	.03	.09	.25*	-.12	.26*	.00
Change of HAMD	-.15	.39**	-.01	-.02	-.15	.34**	-.08	.24*
Initial status of BDI	.49**	.12	.20	-.13	.32**	-.20	.39**	-.19
Change of BDI	-.19	.41**	-.20	.30**	-.20	.47**	-.18	.47**

Note Significant correlations (2-tailed) are flagged: ** = $p < .01$; * = $p < .05$. HAMD = clinician-assessed depression, BDI = self-assessed depression, DAS = cognitive maladaptive beliefs, PB = positive metacognitive beliefs, NB = negative metacognitive beliefs, NFC = metacognitive beliefs about the need for control.

4.5 Hypothesis 3b

To examine the effects of the initial status and the change of cognitive and metacognitive maladaptive beliefs on the change of depression, multiple hierarchical regression analysis was used. The assumptions for regression analysis were satisfied (see 3.4).

The results of the regression models A and B are depicted in Tables 12 and 13, model A using change of clinician-assessed severity of depression as outcome variable (i.e. slope of HAMD), while model B used change of self-assessed severity of depression as outcome variable (i.e. slope of BDI). A preliminary regression analyses revealed that out of the expected risk factors (age, gender, initial status of depression, former history of depression), only initial depressive symptoms had a significant effect on the change of depression and was included in models A and B. The initial depression had a medium effect on the change of depression in model A1 (HAMD) and strong effects in model B1 (BDI). Model A1 explained 18 % of the variance, model B1 could account for 30 % of variance of the outcome.

Table 12: *Model A1-3: Predictors of change of depressive symptoms (outcome: change of HAMD (i.e. slope))*

	b	**β**	***p***	**Partial correlation**
Step 1	**Model A1**			
Constant	1.24		.412	
Initial Status of HAMD (intercept)	-0.39	-0.42	< .001	-.42
Step 2	**Model A2**			
Constant	2.761		.140	
Initial Status of HAMD (intercept)	-0.59	-0.63	< .001	-.60
Initial Status of DAS (intercept)	0.02	0.19	.043	.22
Change of DAS (slope)	0.12	0.58	< .001	.59
Step 3	**Model A3**			
Constant	0.40		.890	
Initial Status of HAMD (intercept)	-0.57	-0.61	< .001	-.60
Initial Status of DAS (intercept)	0.01	0.07	.507	.08
Change of DAS (slope)	0.10	0.50	< .001	.48
Initial Status of PB (intercept)	-0.30	-0.28	.095	-.19
Change of PB (slope)	-0.38	-0.28	.089	-.20
Initial Status of NB (intercept)	0.38	0.31	.057	.22
Change of NB (slope)	0.44	0.40	.015	.28
Initial Status of NFC (intercept)	0.18	0.17	.281	.13
Change of NFC (slope)	0.03	0.03	.870	.02

Note $R^2 = .18$, $F = 17.74$ ($p < .001$) for step 1; $\Delta R^2 = .29$, $F = 23.35$ ($p < .001$) for step 2, $\Delta R^2 = .06$, $F = 9.37$ ($p < .001$) for step 3. HAMD = clinician-assessed depression, DAS = cognitive maladaptive beliefs, PB = positive metacognitive beliefs, NB = negative metacognitive beliefs, NFC = metacognitive beliefs about the need for control, b = unstandardized regression coefficient, β = standardized regression coefficient.

Models A2 and B2 included initial status and change of maladaptive cognitive beliefs (i.e. intercept and slope of DAS) as possible predictors. In models A2 and B2, in addition to initial status of depression, initial status of cognitive maladaptive beliefs predicted a change of depression at small effect size and change of maladaptive cognitive beliefs at strong effect size. The data suggest that high initial scores of DAS or a less beneficial change (i.e. a smaller reduction or a higher increase) of the DAS from t0 to t3 were associated with a less beneficial change of depressive symptoms from t0 to t3. Models A2 and B2 could explain additional 29% of the variance of data in comparison to model A1 and B1.

Table 13: *Model B1-3: Predictors of change of depressive symptoms (outcome: change of BDI (i.e. slope))*

	b	**β**	***p***	**Partial correlation**
Step 1	**Model B1**			
Constant	2.82		.192	
Initial Status of BDI (intercept)	-0.46	-0.55	<.001	-.55
Step 2	**Model B2**			
Constant	0.03		.991	
Initial Status of BDI (intercept)	-0.63	-0.76	< .001	-.71
Initial Status of DAS (intercept)	0.07	0.29	.001	.35
Change of DAS (slope)	0.23	0.56	< .001	.64
Step 3	**Model B3**			
Constant	-13.6		.002	
Initial Status of BDI (intercept)	-0.6	-0.72	< .001	-,75
Initial Status of DAS (intercept)	0.03	0.13	.101	.19
Change of DAS (slope)	0.12	0.30	< .001	.40
Initial Status of PB (intercept)	-0.12	-0.06	.654	-.05
Change of PB (slope)	0.17	0.06	.611	.06
Initial Status of NB (intercept)	0.74	0.31	.011	.29
Change of NB (slope)	0.93	0.42	.001	.38
Initial Status of NFC (intercept)	0.70	0.33	.007	.31
Change of NFC (slope)	0.73	0.31	.013	.28

Note R^2 = .30, F = 35.7 (p < .001) for step 1; ΔR^2 = .29, F = 38.7 (p < .001) for step 2 ΔR^2 = .15, F = 24,01 (p < .001) for step 3. BDI = self-assessed depression, DAS = cognitive maladaptive beliefs, PB = positive metacognitive beliefs, NB = negative metacognitive beliefs, NFC = metacognitive beliefs about the need for control, b = unstandardized regression coefficient, β = standardized regression coefficient.

Models A3 and B3 additionally include the slopes and the intercepts of PB, NB and NFC as possible predictors. In model A3, only initial status of depression, change of DAS and change of NB had a significant effect on the outcome variable. A less beneficial change of DAS and NB were associated with a less beneficial change of depressive symptoms with strong (change of DAS) and medium (change of NB) effect sizes. In model B3, initial status of depression, change of DAS, initial status and change of NB, as well as initial status and change of NFC emerged as predictors of the change of depression. High initial scores of NB and NFC predicted a less beneficial change of depressive symptoms at medium effect size, a less beneficial change in scores of DAS, NB and NFC predicted a less beneficial change of depressive symptoms at medium effect sizes. Model A3 could significantly account for additional 6% of variance of data in comparison to model A2, model B3 could significantly explain additional 15 % as compared to model B2. In total, model A3 could explain 53 % of variance of data and model B3 74 %. An exchange of the order of block 2 and 3 did not

essentially alter results (for models A' and B', see Tables 18 and 19 in the appendix). Hypothesis 3b could be confirmed.

4.6 Hypothesis 3c

Models A4 and B4 additionally include the presence of a critical life event between t2 and t3 as last step in the hierarchical regression analysis (see Tables 14 and 15). In model A4 (HAMD), a critical life event could explain additional 9 % of the variance of data. The presence of a critical life event between t2 and t3 significantly predicted less change in depressive symptoms from t0 to t3 at medium effect size (β > .3). In model B4 (BDI), presence of a critical life event did not emerge as significant predictor of the change of depression. Hypothesis 6 can partially be confirmed.

Table 14: *Model A4: Predictors of change of depressive symptoms (outcome: change of HAMD (i.e. slope))*

	b	**β**	***p***	**Partial correlation**
Step 4	**Model A4**			
Constant	-.25		.942	
Initial Status of HAMD (intercept)	-.59	-.61	< .001	-.58
Initial Status of DAS (intercept)	.00	.02	.874	.02
Change of DAS (slope)	.09	.44	.004	.41
Initial Status of PB (intercept)	-.08	-.07	.722	-.05
Change of PB (slope)	-.06	-.04	.840	-.03
Initial Status of NB (intercept)	.02	.02	.924	.01
Change of NB (slope)	.15	.14	.495	.10
Initial Status of NFC (intercept)	.34	.31	.095	.24
Change of NFC (slope)	-.03	-.02	.900	-.02
Presence of a Critical life event	3.37	.37	.004	.41

Note $\Delta R^2 = .09$, $F = 5.86$ ($p = .004$) for step 4; DAS = cognitive maladaptive beliefs, PB = positive metacognitive beliefs, NB = negative metacognitive beliefs, NFC = metacognitive beliefs about the need for control, b = unstandardized regression coefficient, β = standardized regression coefficient

Table 15: *Model A4 Predictors of change of depressive symptoms (outcome: change of BDI (i.e. slope)*

	b	**β**	***p***	**Partial correlation**
Step 4	**Model B4**			
Constant	-14.38		.003	
Initial Status of BDI (intercept)	-.51	-.68	< .001	-.73
Initial Status of DAS (intercept)	.01	.03	.708	.06
Change of DAS (slope)	.10	.25	.017	.34
Initial Status of PB (intercept)	.12	.05	.704	.06
Change of PB (slope)	.16	.06	.671	.06
Initial Status of NB (intercept)	.72	.32	.036	.30
Change of NB (slope)	.82	.40	.009	.37
Initial Status of NFC (intercept)	.69	.32	.017	.34
Change of NFC (slope)	.86	.34	.013	.35
Presence of a Critical life event	.26	.01	.869	.02

Note ΔR^2 = .00, F = 16.03 ($p <$ .001) for step 4; BDI = self-assessed depression, PB = positive metacognitive beliefs, NB = negative metacognitive beliefs, NFC = metacognitive beliefs about the need for control, b = unstandardized regression coefficient, β = standardized regression coefficient

4.7 Hypothesis 4

Two measures were used to estimate stability: the standardized within-group effect size *d* and the test-retest correlation from t0 to t3 *r* for each construct respectively. The results are presented in Table 1. BDI, DAS, PB, and NFC showed significant positive test-retest correlations at medium (PB) to large (BDI, DAS, NFC) effect sizes. This means that the mean scores of BDI, DAS, PB, and NFC changed in the same way between t0 and t3.

The *d*-scores of HAMD, BDI, and DAS were numerically higher than the *d*-scores of PB, NB and NFC, with the *d*-score of PB being the lowest. As high *d* values suggest higher changes in mean scores than low *d* values, the results suggested that mean change over 3.5 years of HAMD, BDI and DAS were higher than mean change over 3,5 years of PB, NB and NFC. Hence, metacognitive maladaptive beliefs, particularly PB, appeared to be more stable in long-term course than depressive symptoms and cognitive maladaptive beliefs and hypothesis 5 could partially be confirmed.

Table 16*: Relative stability of the different constructs as measured by within-group effect size d and test-retest correlation r*

	HAMD	BDI	DAS	PB	NB	NFC
d	1.16	1.08	1.01	0.31	0.73	0.60
r t0 t3	.20	.40**	.48**	.27*	.13	.37**

Note Significant correlations (2-tailed) are flagged: ** = $p < .01$; * = $p < .05$, HAMD = clinician-assessed depression, BDI = self-assessed depression, DAS = cognitive maladaptive beliefs, PB = positive metacognitive beliefs, NB = negative metacognitive beliefs, NFC = metacognitive beliefs about the need for control, diff t0t3 = Difference score between t0 and t3.

5. Discussion

5.1 Summary of the aims and results of the study

The current study examined the role of cognitive and metacognitive maladaptive beliefs in depression. It aimed at understanding the effect of cognitive versus metacognitive maladaptive beliefs on the long-term course of depressive symptoms using a latent growth model approach. Cognitive maladaptive beliefs concern primarily the content of beliefs whereas metacognitive maladaptive beliefs are roughly defined as thoughts about functions and consequences of one's own thinking. I assumed firstly that results concerning the long-term course of depression regarding recurrence (about 70 - 90 % in literature, see 2.1.3) can be replicated in this study. Furthermore, I hypothesised that initial status and change of metacognitive maladaptive beliefs explain additional variance of the data in the prediction of depressive symptoms above and beyond cognitive maladaptive beliefs. I also supposed that change of depression could be predicted by a stressful life event that had occurred between the 6 months and the 3.5 years follow-up assessment. I finally conjectured that metacognitive maladaptive beliefs are more stable than depressive symptoms and more stable than cognitive maladaptive beliefs and that they constitute a vulnerability factor for depression.

Regarding the long-term course of depression, at 3.5 years follow-up, 78 % of the participants met criteria for a recurrent depressive disorder. Across all testing times, depressive symptoms, cognitive and metacognitive maladaptive beliefs significantly decreased. The change of depressive symptoms from baseline to 3.5 years follow-up assessment was positively correlated with a change of cognitive and metacognitive maladaptive beliefs. Regression analyses showed that out of the alleged risk factors and predictors, initial status of depression, change of cognitive maladaptive beliefs and change of negative metacognitive beliefs had a significant effect on the change of clinician-assessed symptom severity of depression (primary outcome parameter, HAMD) with strong (change of DAS) and medium (change of negative metacognitive beliefs) effect sizes. The final model could account for 53 % of the variance of the data. When repeating the analysis with the change of self-assessed symptom severity of depression (secondary outcome parameter, BDI), the initial scores of negative metacognitive beliefs and metacognitive beliefs about the need for control, as well as the change in cognitive maladaptive beliefs, in negative metacognitive beliefs, and in metacognitive beliefs about the need for control predicted a less beneficial change of depressive symptoms at medium effect sizes. This model could explain 74 % of variance of data. Presence of a critical life event as a predictor of depression could be confirmed when

analysis was conducted using HAMD, but not for BDI. Finally, our data showed that metacognitive maladaptive beliefs, especially positive metacognitive beliefs, followed by negative metacognitive beliefs, and metacognitive beliefs about the need for control, appear to be more stable in long-term course than depressive symptoms and cognitive maladaptive beliefs.

5.2 Discussion of results concerning the long-term course of depression

Concerning the long-term recurrence rates, our results matched findings concerning recurrence in patients with a former history of depression (Eaton et al., 2008; Katon et al., 2001). In our sample, only 46 % of the patients were estimated to be in remission according to the HAMD at 3.5 years follow-up assessment. The high recurrence and low remission rate might be due to the special sample structure examined in this study. The sample was composed of patients in a psychosomatic clinic of whom more than half of the participants were already diagnosed with a recurrent disorder at intake with 3 prior depressive episodes on average and mean illness duration of 8 years. The probability of recurrence increases with each depressive episode, with 50-65 % of the patients suffering a second episode after the first and 70-80 % of the patients who suffered a second episode suffering a third (Eaton et al., 2008; Katon et al., 2001). Risk of recurrence increases by 16% for each successive depressive episode (Solomon et al., 2000). For that reason, our sample was at high risk of recurrence and the recurrence rate corresponds to the rate indicated in literature for individuals who had already suffered two episodes. This could indicate that our findings cannot be transferred to non-clinical samples without precaution, but our results should be valid for clinical samples and individuals with former history of depression. As chronicity and recurrence is one of the major problems of depression, our results can give valuable information about individuals with courses of chronic depression. Also, our findings confirm the known fact that depression is a highly recurrent disorder and highlight the need to recognize and to treat depression early in order to prevent severity and chronicity of depression.

Concerning comorbidities, our data match previous findings with 51 % of the patients suffering a comorbid axis-one disorder at baseline assessment (as compared to 64 % in Kessler et al., 2003), with anxiety disorders being the most frequent (in line with Kessler et al., 2003).

5.3 Predicting the change of depression through cognitive and metacognitive maladaptive beliefs

In the regression analyses, out of the alleged predictors (gender, age, former history of depression), only initial status of depression emerged as significant predictors of the change of self- (HAMD) and clinician-assessed (BDI) symptom severity and was entered in the final models as control variable. Models A1-4 used change of HAMD as primary outcome variable, while models B1-4 employed BDI as secondary outcome variable. In model A2 and B2, initial status and change of maladaptive cognitive beliefs emerged as significant predictors of the change of depression at high (change of cognitive maladaptive beliefs) and low (initial status of cognitive maladaptive beliefs) effect sizes. High initial scores of cognitive maladaptive beliefs or low change of cognitive maladaptive beliefs from baseline to 3.5 years follow-up assessment were associated with a low change of depressive symptoms. Model A2 and B2 could explain additional 29% of variance. Inclusion of initial status and change of positive metacognitive beliefs, negative metacognitive beliefs and metacognitive beliefs about the need for control as possible predictors accounted for additional 6 % (model A3) or 15 % (model B3) of variance of data. In model A3, initial status of depression, change of cognitive maladaptive beliefs, and change of negative metacognitive beliefs had a significant effect on the outcome variable with strong (change of cognitive maladaptive beliefs) and medium (change of negative metacognitive beliefs) effect sizes. In model B3, initial scores of negative metacognitive beliefs and metacognitive beliefs about the need for control as well as small change in cognitive maladaptive beliefs, negative metacognitive beliefs, and metacognitive beliefs about the need for control predicted a less beneficial change (i.e. a smaller reduction or a higher increase) of depressive symptoms at medium effect sizes. The final model A3 could explain 53 % of variance of data and model B3 74 %.

5.3.1 Cognitive maladaptive beliefs

In terms of cognitive maladaptive beliefs in depression, our findings are in line with results from various studies. When only initial status of depression, initial status and change of cognitive maladaptive beliefs was integrated in the regression model (Model A2 and B2), high initial status and less beneficial change in cognitive maladaptive beliefs predicted a less beneficial change of depressive symptoms. Hence, the presence of cognitive maladaptive beliefs appeared to contribute to depressive symptom severity. Prior studies confirmed that the initial score of cognitive maladaptive beliefs is a vulnerability factor for the onset, recurrence, and severity of depression (Alloy et al., 2006; Iacoviello et al., 2006; Otto et al., 2007;

Struijs et al., 2013). The fact that in the final models A3 and B3, only the change of cognitive maladaptive beliefs emerged as predictor of the change of depression, while initial status of DAS did not, is largely in line with findings from Beevers et al. (2003). They found that a low reduction of maladaptive cognitive beliefs predicted shorter periods of time until relapse in depression, while the total score of DAS did not. In combination with our finding, this could mean that change in maladaptive cognitive beliefs is more important in the long-term course of depression than initial scores. Thus, ongoing evaluation of maladaptive cognitive beliefs during therapy as well as monitoring the targeted decrease is vital in the therapy of depression.

5.3.2 Metacognitive maladaptive beliefs

Regarding metacognitive maladaptive beliefs, our results partly confirm prior findings, but indicate that some assumptions may need to be revisited. First, the implementation of negative metacognitive beliefs and metacognitive beliefs about the need for control in the regression model could significantly explain an additional variance of the data ($\Delta R^2 = .06$ in model A3 and $\Delta R^2 = .15$ in model B3) in comparisons to models that only included initial status and change of cognitive maladaptive beliefs. This suggests that metacognitive maladaptive beliefs are not redundant with maladaptive cognitive beliefs and should be considered in the theoretical background of depression. Thus, our findings can confirm the explanatory power of metacognitive maladaptive beliefs in depression assumed by different cross-sectional (Huntley & Fisher, 2016; Papageorgiou & Wells, 2003b; Roelofs et al., 2007; Tajrishi et al., 2011, Yilmaz et al, 2015; Solem et al., 2016;) and one longitudinal study (Ruiz & Odriozola-Gonzalez, 2015). Yilmaz et al. (2015) concluded that metacognitive maladaptive beliefs are a better predictor of depression than cognitive maladaptive beliefs. In contrast, our data do not confirm this conclusion, because in all models, integration of cognitive maladaptive beliefs explained more additional variance than integration of positive metacognitive beliefs, negative metacognitive beliefs and metacognitive beliefs about the need for control, also when swapping the order of the different steps in the regression analyses. For that reason, our data indicates that although initial status and change of negative metacognitive beliefs and metacognitive beliefs about the need for control are predictors of depression, the importance of maladaptive cognitive beliefs in the course of depression could be confirmed.

Results can be discussed in the light of the different categories of metacognitive maladaptive beliefs. Before discussing the results, it has to be remarked that in this study, subscales of MCQ-30 were employed that are not fully congruent with PBRS and NBRS, often

used in the cited studies (for example Papageorgiou & Wells, 2009; Roelofs et al., 2007; Solem et al., 2016; Yilmaz et al., 2015) . This could limit the comparability of the results. Nevertheless, as Roelofs et al. (2010) concluded that NBRS with positive metacognitive beliefs and NBRS with negative metacognitive beliefs and metacognitive beliefs about the need for control show construct validity, sufficient comparability of the different results can be assumed.

Positive metacognitive beliefs have been considered to play an important role in the onset of depression and several investigations support this hypothesis (Weber & Exner, 2013; Halvorsen et al., 2015; Huntley & Fisher, 2016). In our study, however, neither initial status nor change of positive metacognitive beliefs emerged as significant predictors of the change of depression. When analysing and comparing the results with the literature in more detail, this apparent incongruity can be explained. First, whereas Halvorsen et al. (2015) identified positive metacognitive beliefs as a factor discriminating depressed from non-depressed individuals, among the significant factors it was the least correlated. This is in accordance with our findings where change of positive beliefs was significantly correlated with the change of BDI, but it was not significantly correlated with the change of HAMD. Second, while discussing the results, it has to be considered that Weber and Exner (2013) and Huntley and Fisher (2016) conducted their studies with *non-clinical* samples. They both found that positive metacognitive beliefs had an indirect effect on depressive symptoms via rumination. Nevertheless, as the Papageorgiou and Wells (2003b) study illustrates, findings can diverge in clinical and non-clinical samples. For that reason, our results should preferentially be discussed in reference to studies that investigated *clinical* samples. Indeed, different studies are available that investigated the relation between metacognitive beliefs, rumination and depressive symptoms in clinical samples (Papageorgiou & Wells, 2003b; Roelofs et al., 2007). In those clinical studies, instead of positive metacognitive beliefs, *negative* metacognitive beliefs constituted the link between rumination and depressive symptoms. From this observation could follow that in clinical samples, negative metacognitive beliefs are more closely linked to depression than positive metacognitive beliefs. This is consistent with our findings, where neither initial status nor change of positive metacognitive beliefs emerged as predictors of the change of depression. Moreover, in agreement with our results, negative metacognitive beliefs emerged in several studies as predictors of depression. Two longitudinal studies with non-clinical samples found negative metacognitive beliefs to be a predictor of the course of depression (Papageorgiou & Wells, 2009; Yılmaz et al., 2011). Negative met-

acognitive beliefs also emerged in one cross-sectional study with a clinical sample as discriminating depressive and non-depressed individuals and was judged to be a vulnerability factor for depression (Halvorsen et al., 2015). Thus, our data confirm the relevance of negative metacognitive beliefs in depression in a clinical sample with longitudinal study design. Eventually, it has to be noted that our study is the only that investigated a clinical sample in a longitudinal setting so that the interpretation and comparison of the cited studies is faced to some restrictions that I tried to consider.

With regard to metacognitive beliefs about the need for control, in our sample, their initial status and change predicted a change of depression only when analyses were performed with BDI, but not with HAMD. Hence, the results concerning metacognitive beliefs about the need for control are not fully conclusive. It is possible that the results were affected by the modality of the administration of the measures, showing stronger associations when the predictor and the outcome variable are both self-rated instruments (MCQ and BDI). In addition, HAMD has been suggested to measure rather vegetative symptoms of depression and to underrepresent cognitive symptoms, as worthlessness, hopelessness, and helplessness, are not investigated (Zimmerman, Posternak, & Chelminski, 2005). BDI seems better to address cognitive symptoms of depression: in a meta-analysis concerning the factor structure of BDI, it was presented that "negative attitudes" usually emerge as one of three highly interrelated factors (Beck et al., 1988). This could also account for the greater explanation of variance that was found when the regression analyses where computed with BDI. Our results concerning beliefs about the need for control in relation to self-rated depressive symptoms are corroborated by findings from Halvorsen et al. (2015), were metacognitive maladaptive beliefs about the need for control were a constituent factor of a function discriminating never depressed, previously depressed and currently depressed individuals. Hence, despite the discrepancy between our results with HAMD and BDI as outcome variable, the results with BDI should be acknowledged and suggest that metacognitive beliefs about the need for control do play a role in depression.

5.3.3 Further implications

Our results allow drawing some cautious conclusions regarding the aetiology of depression, in particularly regarding the metacognitive model of depression (Wells & Schweiger, 2011), while acknowledging that those conclusions cannot be but preliminary, as not all pathways of the model have explicitly been tested. Positive metacognitive beliefs constitute an integral part of the metacognitive model of depression which presumes that when triggered with sad

mood, an individual is likely to react at first with positive metacognitive beliefs which, in turn, could lead via rumination and negative metacognitive beliefs to depression (Wells & Schweiger, 2011). This model assigns thus a predominant importance to both positive and negative metacognitive beliefs, because it supposes that positive metacognitive beliefs lead to unhelpful coping strategies while negative metacognitive beliefs entail beliefs about the "uncontrollability of rumination and the meaning of depression" (Wells et al., 2012; Yilmaz et al., 2015). The findings as discussed above could challenge the role of positive metacognitive beliefs in the metacognitive model of depression, as neither initial status nor change of positive metacognitive beliefs predicted depressive symptoms. While we could confirm the importance of negative metacognitive beliefs and metacognitive beliefs about the need for control in a longitudinal long-term study design with a clinical sample, our data could not confirm the role of positive metacognitive beliefs in the onset, recurrence and maintenance of depression as supposed by the metacognitive model. To conclude, our data suggest that metacognitions do play an important role in the aetiology of depression, although it seems that negative metacognitive beliefs and metacognitive beliefs about the need for control could be more important in clinical samples than positive metacognitive beliefs.

5.4 The cognitive vulnerability model of depression

This study moreover examined the relative stability of cognitive and metacognitive maladaptive beliefs in the long-term course of depression. Positive metacognitive beliefs seemed to be the most stable, followed by negative metacognitive beliefs and metacognitive beliefs about the need for control. Cognitive maladaptive beliefs seemed to be as stable as depressive symptoms, both also significantly decreasing across all testing times. This suggests that cognitive maladaptive beliefs might partly be concomitants of depression. Zuroff et al. (1999), Beevers and Miller (2004) and Hankin (2008) all concluded that cognitive maladaptive beliefs might be state-dependent, while showing trait-like characteristics at the same time. As I only examined relative stability, which is to say stability in relation to other measured constructs, our findings do not refute the thesis that cognitive maladaptive beliefs might have trait characteristics, though our data can't confirm it. But our data suggest that cognitive maladaptive beliefs are less stable than metacognitive maladaptive beliefs. Until now, to the best of our knowledge, no other studies have investigated the stability of metacognitive maladaptive beliefs in a longitudinal study design. Our data lead to the conclusion that metacognitive maladaptive beliefs might expose trait characteristics and that they might constitute an important underlying vulnerability factor for depression. This hypothesis is in line with

Halvorsen et al. (2015) who found in their cross-sectional comparison of never depressed, previously depressed and currently depressed individuals that negative metacognitive beliefs might have trait characteristics rendering individuals susceptible to depression or, alternatively, that negative metacognitive beliefs might produce scar effects which make individuals who have experienced a depressive episode vulnerable to relapse.

The presence of a critical life event emerged as a predictor of the change of depressive symptoms when introduced as last step in the regression model using HAMD. Our data suggest that critical life events can provoke depression in individuals vulnerable for depression. The vulnerability hypothesis in terms of cognitive vulnerability because of cognitive maladaptive beliefs is supported by findings from Struijs et al. (2013) and Scher et al. (2005). To my knowledge, metacognitive maladaptive beliefs have not yet been examined as vulnerability factors in combination with critical life events. Our findings indicate that not only cognitive maladaptive beliefs, but also metacognitive maladaptive beliefs (most notably negative metacognitive beliefs and metacognitive beliefs about the need for control) contribute to cognitive vulnerability for depression. As limitation, the effect could not be reproduced when using BDI. For that reason, and as the period between the critical life event and assessment of depression could extend up to three years due to our study design, it seems necessary to deepen vulnerability research in this regard. Taken together, the results show that more research should be devoted to the issue of metacognitive maladaptive beliefs as vulnerability factors for depression.

To conclude, our data are compatible with a vulnerability-stress model of depression which includes cognitive as well as metacognitive maladaptive beliefs as vulnerability factors. Our findings are consistent with the idea of stable underlying cognitive assumptions about the own thinking that, when triggered by a negative life event, lead to depressive symptoms via cognitive maladaptive beliefs.

5.5 Limitations and strengths of the study

When interpreting the results, some limitations and strengths of the study should be kept in mind. First, good to satisfying completion rates of the participants should be mentioned. The response rate was very good at post (94 %) and satisfying at both follow-up assessments (71% for t2 and t3). In comparable studies, return quote were of 82 % after three months (Papageorgiou & Wells, 2009), 37% after nine months (Ruiz & Odriozola-Gonzalez, 2015), 77 % after nine years (Halvorsen, Wang, Eisemann, & Waterloo, 2010). Moreover, all parts of the study were registered at trial register data-bases (German Clinical Trials Register for

t0 to t2; ClinicalTrials.gov for t3). Registration contributes to increased transparency and better access to clinical trials. As another strength of the study, the long investigation period of 3,5 years has to be mentioned, since only few studies considering maladaptive metacognitive beliefs in the long-term course of depression have been presented (Papageorgiou & Wells, 2009; Ruiz & Odriozola-Gonzalez, 2015; Weber & Exner, 2013; Yılmaz et al., 2011), of which none examined a clinical sample. However, the long interval of three years without follow-up between 6 months and 3.5 years follow-up also could count as limiting factor, although the course of depression, therapeutic interventions, critical life events and stays in psychiatric departments between the two assessments were retrieved at 3.5 years follow-up via a semi-structural interview. Since remission is defined by the APA (2013) as a period of at least two consecutive months during which complete diagnostic criteria of a major depressive episode have not been met, the choice of the follow-up intervals made it impossible to assess remission at 3.5 years follow-up according to the official guidelines. Consistent with other studies (see for example Zimmerman et al., 2012), HAMD score at 3.5 years follow-up was used to define remission which can be judged as satisfying.

Regarding the employed instruments, the study also has several strengths, though it is not without limitations. The psychopathological status was investigated in detail at baseline, using with M.I.N.I a highly standardized procedure, and examining not only depression, but also other achsis-1 comorbidities. Furthermore, use of HAMD and BDI allowed for self-assessment as well as external assessment of the severity of depressive symptoms. Use of the HAMD as primary outcome measure could restrict findings. Though, the HAMD has been criticized for poor interrater-reliability at individual item level, heterogeneity of item description, overestimation of physical items and incomplete coverage of the symptom criteria used to define major depression by the DSM-IV, such as worthlessness, indecisiveness and concentration problems (Zimmerman et al., 2005). Still, as mainly one examiner conducted the interviews at 3.5 years follow-up, the problem of poor interrater-reliability did not occur at the long-term follow-up. Also, as mentioned, with BDI, a second measure of depression was employed, balancing this limitation. Furthermore, the fact that only three subscales of MCQ-30 were investigated could limit findings, even if the employed subscales were reported to be closely linked to depression (Ruiz & Odriozola-Gonzalez, 2015; see 1.2.3.2). Because of the unclear role and status of metacognitive maladaptive beliefs in depression, assessment of the subscales “cognitive confidence” and “cognitive self-consciousness” could have been adequate for clarification. Furthermore, as rumination was found to mediate the relation between positive metacognitive beliefs and depressive symptoms in

non-clinical samples (Weber & Exner, 2013) and between negative metacognitive beliefs and depressive symptoms in clinical samples (Papageorgiou & Wells, 2003b; Roelofs et al., 2007), it seems to be disadvantageous that no instrument measuring rumination, such as *Ruminative Response Scale* (RRS), was integrated in the study.

Finally, some particularities of the chosen sample have to be taken into account. First, with mean HAMD 10.9 (*SD* = 7.9) and BDI of 17.49 (*SD* = 11.68) at inclusion assessment, the sample seemed to show only mild depression which could imply that generalization to more severely depressed individuals could be problematic. But, on the other hand, at intake, patients had on average already suffered 3 depressive episodes with mean illness duration of 96 months. At long-term follow-up, 78% the collective were diagnosed with a recurrent depression and 22% with a double depression, indicating the prevalence of chronic courses of depression in our sample. Also, the fact that at long-term follow-up, out of 58, 60 % of the patients (*n*=35) had underwent between 6 months and and 3.5 years follow-up or currently followed outpatient psychotherapies and that 41 % of the patients (*n*=34) underwent current antidepressant pharmacotherapy could explain the mean low scores in depression scales. In addition, it has to be kept in mind that out of the 35 patients who underwent a psychotherapy, 54 % (*n*=19) followed a cognitive behavioural therapy (CBT) which is supposed to target cognitive maladaptive beliefs, whereas metacognitive maladaptive beliefs are often not addressed in CBT. This fact could distort our results concerning the stability of cognitive and metacognitive maladaptive beliefs. Nevertheless, it is ethically not justifiable to only assess untreated depressed patients, so that limitations of that kind have to be accepted.

In terms of the data analysis, use of latent growths models (LGMs) can be judged as another strength of the study. LGMs are established as statistical models used to analyse univariate curves of longitudinal data (Muniz-Terrera et al., 2017). The analysis permits flexible examination of change processes over time (Schmiedek & Wolff, 2010). As LGMs are formulated under the structural equation model (SEM) frame, they can be utilized to implement multivariate change processes and to establish relationships between them. Moreover, they are appropriate to test causal hypotheses via path models (Schmiedek & Wolff, 2010). With LGMs, mean as well as subject-specific trajectories can be estimated where time invariant and time variant covariates may be included (Muniz-Terrera et al., 2017). Also, LGMs allow for analyses of incomplete data, as analysis is not restricted to complete cases, but all cases with data from at least one measure time can be included (Schmiedek & Wolff, 2010).

5.6 Conclusions, further research requests and implications for clinical praxis

Cognitive maladaptive beliefs are considered as confirmed vulnerability factors for depression and are integrated in aetiological models of depression. More recent findings suggest that metacognitive maladaptive beliefs might in addition to or instead of cognitive maladaptive beliefs play an important role in the onset, maintenance, and recurrence of depression. This study aimed to investigate the role of cognitive and metacognitive maladaptive beliefs (positive metacognitive beliefs, negative metacognitive beliefs, and metacognitive beliefs about the need for control) in the long-term course of depression. The results suggest that maladaptive cognitive beliefs, negative metacognitive beliefs, and metacognitive beliefs about the need for control, particularly their change, all significantly predict the change of depressive symptom severity. Furthermore, metacognitive maladaptive beliefs seemed to be more stable than cognitive maladaptive beliefs. Our data suggests that they could, besides cognitive maladaptive beliefs, contribute to cognitive vulnerability for depression. As negative metacognitive beliefs and beliefs about the need for control explain additional variance of the change of depressive symptoms, it seems necessary to implement these constructs in the cognitive model of depression.

Nevertheless, the investigations concerning metacognitive maladaptive beliefs is facing serious problems because the term is not consistently used (Semerari et al., 2012). Definitions range from rather broad definitions as “thinking about one’s own thinking” (Moritz et al., 2013, p. 359) to complex constructs subsuming different subtypes of metacognition and implying different cognitive functions and cognitions about those functions (Wells, 2001). As outlined in the introduction (see 3.3.5), the concept of metacognition on which this study is based, draws on the broad definition given by Flavell and Wellman (1977). More precisely, metacognitive maladaptive beliefs assumed to be relevant in depression are defined through the instruments used to assess them (see 2.3.5 *Metacognitions Questionnaire 30*). In this context, it has to be acknowledged that MCQ-30, MCQ65, PBRS and NBRS, mainly employed to investigate metacognitive maladaptive beliefs in depression, are not identical, even if the measured constructs overlap (for evaluation of MCQ-30 in comparison to MCQ65, see Wells & Cartwright-Hatton, 2004; for evaluation of PBRS and NBRS in comparison to MCQ-30, see Roelofs et al., 2010). In line with Roelofs et al. (2010), we may conclude that differences in results concerning the exact interplay of metacognitive maladaptive beliefs and rumination in depression seem to be partly due to the differences of the applied instruments. In addition, it is also possible that subscales of DAS and MCQ-30 partly measure the same constructs. When too many correlated variables are entered in a

regression model, it becomes difficult to compare different predictors (Field, 2016). For that reason, it could be necessary to investigate more profoundly inferences between DAS and MCQ-30 before drawing final conclusions. In order to elucidate the exact role of the different constructs, more research is warranted that illuminates the scope of the different instruments and that elaborates a definition of metacognitive maladaptive beliefs which overlaps the different research approaches. Furthermore, research has suggested that metacognition is also related to other constructs such as mindful attention awareness which has been suggested, besides maladaptive metacognitions, to contribute to cognitive vulnerability (Solem et al., 2015). This highlights the need to conduct more clinical studies that bring together different constructs to create a complete model of the aetiology of depression. One attempt to integrate different concepts has recently been proposed by Pedone et al. (2017) who suggest a model integrating different theories such as theory of mind, metacognition and mentalization. Such a model integrating and precisely defining distinct concepts that seem to be relevant in depression would be very positive. The need for a clear concept of metacognition, metacognitive processes and biases is reinforced by the emergence of a whole field of more experimental research, dedicated for example to the link between physiological and metacognitive processes (for example Capobianco, Morris, & Wells, 2018; Stange, Hamilton, Fresco, & Alloy, 2017).

Depression is one of the most prevalent mental disorder world-wide and contributes significantly to the burden of disease (World Health Organization, 2016). Early and effective treatment of depression is crucial as it is known that longer and more sever episodes often entail early relapses and reduce the likelihood of recovery (Keller et al., 1992). Although different effective psychotherapeutic and pharmacologic therapies have been developed (for an overview concerning the efficacy of different psychotherapies see Cuijpers, 2015), there is still need to further improve established therapies. A study commissioned by the Bertelsmann-Stiftung Germany found that in Germany, 50 % of patients with dysthymia, moderate or severe depression received no or insufficient therapy (Bertelsmann Stiftung, 2014). But even if therapy places were available for every patient, it is assumed that pharmacological and psychotherapeutic therapies only would diminish about 30 % of the disease burden (Andrews, Issakidis, Sanderson, Corry, & Lapsley, 2004) due to high drop-out rates (Swift & Greenberg, 2012) and high recurrence probability (Eaton et al., 2008). This highlights that, while so-called new wave therapies, such as Metacognitive Therapy as conceptualized by Wells or Acceptance-And-Commitment-Therapy, seem, if at all, only slightly different from established therapies such as CB-T in terms of efficacy (Cuijpers, 2015), there is need to

ameliorate existing therapies. Our results support the idea that it could be beneficial for the patients to implement strategies to modify metacognitive maladaptive beliefs in current therapies. One example of this practice is the add-on therapy Metacognitive Training for Depression (D-MCT) which was developed by Jelinek, Hauschildt, and Moritz (2015) as group intervention and which is fully compatible with CBT, while integrating a metacognitive point of view. It yielded promising results in a feasibility study (Jelinek, Otte, Arlt, & Hauschildt, 2013) and in a RCT with 6-months follow-up (Jelinek et al., 2016). Furthermore, D-MCT also encountered high acceptance by patients (Jelinek, Moritz, & Hauschildt, 2017).

Also, depression is a frequent comorbidity that patients develop on the ground of various medical condition (Kang et al., 2015). There is increasing evidence that metacognitive maladaptive beliefs might contribute to distress or depression in Multiple Sklerosis (Heffer-Rahn & Fisher, 2018), in Epilepsy (Fisher & Noble, 2017) and Diabetes (Purewal & Fisher, 2018). Targeting metacognitive maladaptive beliefs in those medical conditions might thus help to prevent comorbid depressive episodes. This is of vital importance, as comorbid depression has been shown to impact the course of the diseases by deteriorating quality of life, leading to increased impairment and a higher mortality (Kang et al., 2015).

To conclude, cognitive and metacognitive maladaptive beliefs play an important role in onset, maintenance, and recurrence of depression. It seems vital to integrate the concept of metacognitive maladaptive beliefs more stringently in therapeutic research and therapeutic interventions.

6. Summary

Cognitive maladaptive beliefs (most commonly assessed using the Dysfunctional Attitudes Scale [DAS]) have been shown to represent an important vulnerability factor in the onset, relapse, and recurrence of depression. Besides, metacognitive maladaptive beliefs (as measured by the Metacognitive Questionnaire-30 [MCQ-30]) are also thought to contribute to depression. However, the course and relationship of cognitive and metacognitive maladaptive beliefs in depressed individuals on the long-term has not been explored. Also, whereas a extensive research concerning stability of cognitive maladaptive beliefs in depression has been conducted, stability of metacognitive maladaptive beliefs has not been examined yet. The aim of the present study was to investigate over a period of 3.5 years the role and stability of cognitive and metacognitive maladaptive beliefs in individuals with depression and to elucidate whether cognitive or metacognitive maladaptive beliefs are a better predictor of the course of depressive symptoms over this time span.

Eighty-four patients with depression were assessed with the DAS, three subscales of the MCQ-30 (positive metacognitive beliefs, negative metacognitive beliefs, beliefs about the need for control), the Hamilton Depression Rating Scale, and the Beck Depression Inventory at baseline and were reassessed after four weeks, 6 months and 3.5 years. Analyses were carried using a longitudinal latent growth model approach.

Our data showed that change on the DAS and on the MCQ-30 subscale ‘negative metacognitive beliefs’ significantly predicted the change in clinician-rated depressive symptom severity over 3.5 years, and that change on the DAS and change and initial scores of the MCQ-30 subscales ‘negative metacognitive beliefs’ and ‘need for control’ predicted change in self-rated depressive symptom severity. Also, the presence of a critical life event significantly predicted the change of clinician-rated depressive symptom severity. In all models, maladaptive cognitive beliefs explained more additional variance than the MCQ-30 subscales. In terms of stability, metacognitive maladaptive beliefs were more stable than cognitive maladaptive beliefs.

Cognitive and metacognitive maladaptive beliefs were both predictors of depression, with the DAS being a better predictor than the MCQ-30 subscales. As maladaptive metacognitive beliefs were more stable than maladaptive cognitive beliefs, they should be considered an important underlying vulnerability factor for depression. In conclusion, both cognitive and metacognitive maladaptive beliefs seem to contribute to depression and should be considered as targets in the therapy of unipolar depression.

6. Zusammenfassung

Es konnte gezeigt werden, dass kognitive maladaptive Einstellungen (durch die Skala dysfunktionaler Einstellungen [DAS] getestet), einen wichtigen Vulnerabilitätsfaktor für Depression darstellen. Daneben scheinen auch metakognitive maladaptive Einstellungen (durch den Metakognitiven Fragebogen-30 [MCQ-30] getestet) zu Depression beizutragen. Bisher wurde jedoch die Beziehung von kognitiven und metakognitiven maladaptiven Einstellungen bei Personen mit Depression im Langzeitverlauf noch nicht untersucht. Während bereits umfangreiche Forschung der Stabilität von kognitiven maladaptiven Einstellungen gewidmet worden ist, wurde die Stabilität von metakognitiven maladaptiven Einstellungen bislang noch nicht untersucht. Das Ziel der vorliegenden Studie war es, über einen Zeitraum von 3.5 Jahren die Rolle und Stabilität von kognitiven und metakognitiven maladaptiven Einstellungen bei depressiven Individuen zu untersuchen und zu klären, ob kognitive oder metakognitive maladaptive Einstellungen einen besseren Prädiktor für den Verlauf depressiver Symptome darstellen.

Vierundachtzig Patient*innen mit Depression wurden bei Einschluss mittels der DAS, drei Skalen des MCQ-30 (positive metakognitive Überzeugungen, negative metakognitive Überzeugungen und Überzeugungen über das Bedürfnis nach Kontrolle), der Hamilton Depression Skala und dem Beck-Depressions-Inventar untersucht. Follow-Up-Untersuchungen erfolgten nach vier Wochen, nach sechs Monaten und nach dreieinhalb Jahren. Die Analysen wurden mithilfe eines latenten Wachstumsmodells ausgewertet.

Die Daten zeigten, dass eine Veränderung im DAS und in negativen metakognitiven Überzeugungen signifikant die Veränderung in den durch eine*n Kliniker*in bewerteten depressiven Symptome vorhersagte, und dass eine Veränderung im DAS und initialer Wert und Veränderung in zwei MCQ-Subskalen den Verlauf der selbst-bewerteten depressiven Symptome vorhersagten. Das Vorhandensein eines kritischen Lebensereignisses sagte depressive Symptome voraus. In allen Modellen erklärten maladaptive kognitive Einstellungen mehr Varianz als die MCQ-30 Subskalen. In Bezug auf die Stabilität zeigten sich metakognitive maladaptive Einstellungen stabiler als kognitive maladaptive Einstellungen.

Sowohl kognitive als auch metakognitive maladaptive Einstellungen sagten den Verlauf depressiver Symptome voraus, die DAS war dabei ein besserer Prädiktor als die MCQ-30 Subskalen. Da metakognitive maladaptive Einstellungen stabiler als kognitive maladaptive Einstellungen waren, sollten sie als ein wichtiger zugrunde liegender Vulnerabilitätsfaktor für Depression gewertet werden, deren Veränderung neben der kognitiver maladaptiver Einstellungen ein Ziel in der Therapie unipolarer Depression darstellen könnte.

7. Publication

Die Publikation ist nicht in der E-Dissertation enthalten. Sie ist verfügbar unter:

Faissner M, Kriston L, Moritz S, Jelinek L. Course and stability of cognitive and metacognitive beliefs in depression. Depress Anxiety. 2018;1–8. https://doi.org/10.1002/da.22834.

8. List of Abbreviations

AAQ-II	Acceptance and Action Questionnaire – II
ACSQ	Adolescent Cognitive Style Questionnaire
APA	American Psychiatric Association
ASQ	Attributional Style Questionnaire
BAI	The Beck Anxiety Inventory
BDI	Beck Depression Inventory, First Edition
BDI-II	Beck Depression Inventory, Second Edition
CAS	Cognitive attentional syndrome
CBT	Cognitive Behavioural Therapy
CBQ	Cognitive Bias Questionnaire
CC	Cognitive Confidence
CDAS	Children's Dysfunctional Attitudes Scale
CDI	Children's Depression Inventory
CIDI	Composite International Diagnostic Interview
CRSQ	Children's Response Styles Questionnaire
CSC	Cognitive self-consciousness
CSQ	Cognitive Style Questionnaire
CQ	Cognitions Questionnaire
DAS	Dysfunctional Attitude Scale
DAS-R	Dysfunctional Attitude Scale – Revised
DASS-21	Depression subscale of the Depression Anxiety and Stress Scales-21
DAS-24	Dysfunctional Attitude Scale-24
D-MCT	Metacognitive Training for Depression
DSS	Depressive Schemas Scale
GHQ-28	General Health Questionnaire
GP	General practitioner
H	Hypothesis
HADS	Hospital Anxiety And Depression Scale

HAMD	Hamilton Depression Rating Scale
HT	Health Training
IAT	Implicit Associations Test
ICSRLE	Inventory of College Students' Recent life Experiences
IDD	Inventory to Diagnose Depression
IDSC	Inventory for Depressive Symptomatology, Clinician Version
IDS-SR	Inventory of Depression Symptomatology – Self Report
ISDA	Implicit self-depressed associations
LEIDS-r	Leiden Index of Depression Sensitivity – revised
LES	Life Experience Survey
LIFE-PSR	Longitudinal Interval Follow-up Evaluation-Psychiatric Status Ratings
LTE	List of Threatening Experiences
M	Mean
MCQ-30	Metacognitions Questionnaire-30
MDE	Major Depressive Episode
MHRSD	Modified Hamilton Rating Scale for Depression
M.I.N.I	Mini International Neuropsychiatric Interview
MSGO	Miskimins Self-Goal Other Discrepancy Scale
NB	Negative metacognitive beliefs concerning uncontrollability, danger and negative consequences of rumination
NBRS1	Negative Beliefs about Rumination Scale: uncontrollability and harm
NBRS2	Negative Beliefs about Rumination Scale: Social and interpersonal consequences of ruminating
NEO-FFI	Neo five-factor inventory
NFC	Metacognitive beliefs about the need for control
PB	Positive metacognitive beliefs about worry and rumination
PBRS	Positive Beliefs about Rumination Scale
PSI	Problem Solving Inventory

PSS	Perceived Stress Scale
PSWQ	Penn State Worry Questionnaire
QIDS	Quick Inventory of Depressive Symptomatology
RCT	Randomized Controlled Study
RRS	Ruminative Response Scale
RSQ	Rumination response scale
SADS–L	Schedule for Affective Disorders and Schizophrenia—Lifetime
SADS-C	Schedule for Affective Disorder and Schizophrenia-Change Version
SBQ-R	Suicide Behaviours Questionnaire-Revised
SCID	Structured Clinical Interview for DSM–IV Axis I Disorders
SD	Standard Deviation
SDS	Self-rating Depression Scale
SEF	Self-Efficacy Scale
SEM	Structural Equation Model
S-REF	Self-Regulatory Executive Function
TCQ	Thought Control Questionnaire
VAS	Visual Analog Scale
VIF	Variance Indicator Factor
WHO	Word Health Organisation
YSQ	Young Schema Questionnaire

9. References

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11. CV

Lebenslauf aus datenschutzrechtlichen Gründen nicht enthalten.

12. Eidesstattliche Erklärung

Ich versichere ausdrücklich, dass ich die Arbeit selbständig und ohne fremde Hilfe verfasst, andere als die von mir angegebenen Quellen und Hilfsmittel nicht benutzt und die aus den benutzten Werken wörtlich oder inhaltlich entnommenen Stellen einzeln nach Ausgabe (Auflage und Jahr des Erscheinens), Band und Seite des benutzten Werkes kenntlich gemacht habe.

Ferner versichere ich, dass ich die Dissertation bisher nicht einem Fachvertreter an einer anderen Hochschule zur Überprüfung vorgelegt oder mich anderweitig um Zulassung zur Promotion beworben habe.

Ich erkläre mich einverstanden, dass meine Dissertation vom Dekanat der Medizinischen Fakultät mit einer gängigen Software zur Erkennung von Plagiaten überprüft werden kann.

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13. Appendix

Table 17: *Correlations of initial status and difference scores between t0 and t3 of depression with initial status and difference scores between t0 and t3 of maladaptive cognitive and metacognitive beliefs, using available data*

	DAS t0	DAS diff t0t3	PB	PB diff t0t3	NB t0	NB diff t0t3	NFC t0	NFC diff t0t3
HDRS t0	.33**	-.09	.06	.08	.25*	-.20	.25*	-.03
HDRS t0t3	-.25	.62**	.00	.05	-.02	.30*	.01	.32*
BDI t0	.50**	-.07	.20	-.10	.31**	-17	.39**	-.17
BDI t0t3	-.44**	.67**	-.07	.21	-.15	.43**	-.14	.52**

Note Significant correlations (2-tailed) are flagged: ** = $p < .01$; * = $p < .05$, HAMD = clinician-assessed depression, BDI = self-assessed depression, DAS = cognitive maladaptive beliefs, PB = positive metacognitive beliefs, NB = negative metacognitive beliefs, NFC = metacognitive beliefs about the need for control, diff t0t3 = Difference score between t0 and t3.

Table 18*: Model A'1-3: Predictors of change of depressive symptoms (outcome: change of HAMD (i.e. slope))*

	b	**β**	***p***	**Partial correlation**
Step 1	**Model A'1**			
Constant	1.24		.412	
Initial Status of HAMD (intercept)	-0.39	-0.42	<.001	-.42
Step 2	**Model A'2**			
Constant	-3.85		0.193	
Initial Status of HAMD (intercept)	-0.47	-0.51	<.001	-.51
Initial Status of PB (intercept)	-0.24	-0.23	.236	-.14
Change of PB (slope)	-0.33	-0.25	.181	-.15
Initial Status of NB (intercept)	0.35	0.29	.107	.18
Change of NB (slope)	0.50	0.45	.013	.28
Initial Status of NFC (intercept)	0.38	0.36	.032	.24
Change of NFC (slope)	0.44	0.38	.026	.25
Step 3	**Model A'3**			
Constant	0.40		.890	
Initial Status of HAMD (intercept)	-0.57	-0.61	<.001	-.60
Initial Status of PB (intercept)	-0.30	-0.28	.095	-.19
Change of PB (slope)	-0.38	-0.28	.089	-.20
Initial Status of NB (intercept)	0.37	0.31	.057	.22
Change of NB(slope)	0.44	0.40	.015	.28
Initial Status of NFC (intercept)	0.18	0.17	.281	.13
Change of NFC (slope)	0.03	0.03	.870	.02
Initial Status of DAS (intercept)	0.01	0.07	.507	.08
Change of DAS (slope)	0.10	0.50	<.001	.48

Note $R^2 = .18$, $F = 17.74$ ($p < .001$) for step 1; $\Delta R^2 = .21$, $F = 6.92$ ($p = .001$) for step 2, $\Delta R^2 = .15$, $F = 9.37$ ($p < .001$) for step 3. HAMD = clinician-assessed depression, DAS = cognitive maladaptive beliefs, PB = positive metacognitive beliefs, NB = negative metacognitive beliefs, NFC = metacognitive beliefs about the need for control, b = unstandardized regression coefficient, β = standardized regression coefficient.

Table 19: *Modell B'1-3: Predictors of change of depressive symptoms (outcome: change of BDI (i.e. slope))*

	b	**β**	***p***	**Partial correlation**
Step 1	**Model B'1**			
Constant	2.82		.192	
Initial Status of BDI (intercept)	-0.46	-.55	<.001	-.55
Step 2				
Constant	-16.41		<.001	
Initial Status of BDI (intercept)	-0.53	-.63	<.001	-.71
Initial Status of PB (intercept)	-0.03	-.01	.918	-.01
Change of PB (slope)	0.25	.09	.485	.08
Initial Status of NB (intercept)	0.73	.30	.020	.26
Change of NB (slope)	1.02	.46	<.001	.39
Initial Status of NFC (intercept)	1.02	.48	<.001	.42
Change of NFC (slope)	1.25	.52	<.001	.46
Step 3				
Constant	-13.60		.002	
Initial Status of BDI (intercept)	-0.60	-.72	<.001	-.75
Initial Status of PB (intercept)	-0.12	-.06	.654	-.05
Change of PB (slope)	0.17	.06	.611	.06
Initial Status of NB (intercept)	0.74	.31	.011	.29
Change of NB(slope)	0.93	.42	<.001	.38
Initial Status of NFC (intercept)	0.70	.33	.007	.31
Change of NFC (slope)	0.73	.31	.013	.28
Initial Status of DAS (intercept)	0.03	.13	.101	.19
Change of DAS (slope)	0.12	.30	<.001	.40

Note $R^2 = .30$. $F = 35.7$ ($p < .001$) for step 1; $\Delta R^2 = .39$. $F = 24.68$ ($p < .001$) for step 2. $\Delta R^2 = .06$. $F = 24.02$ ($p < .001$) for step 3. BDI = self-assessed depression. DAS = cognitive maladaptive beliefs. PB = positive metacognitive beliefs. NB = negative metacognitive beliefs. NFC = metacognitive beliefs about the need for control. b = unstandardized regression coefficient. β = standardized regression coefficient.

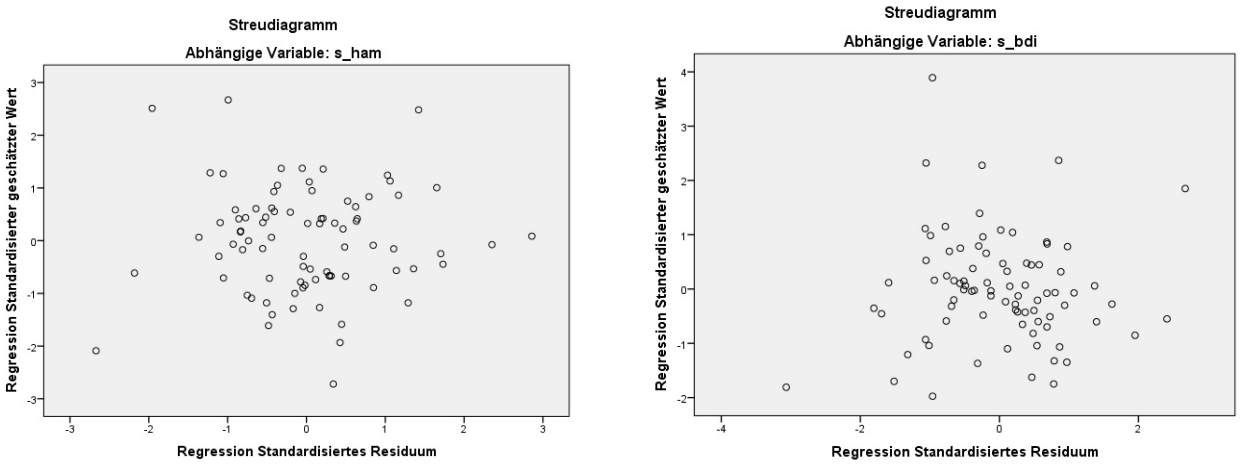

Figure 3 Checking homoscedasticity of model A and B

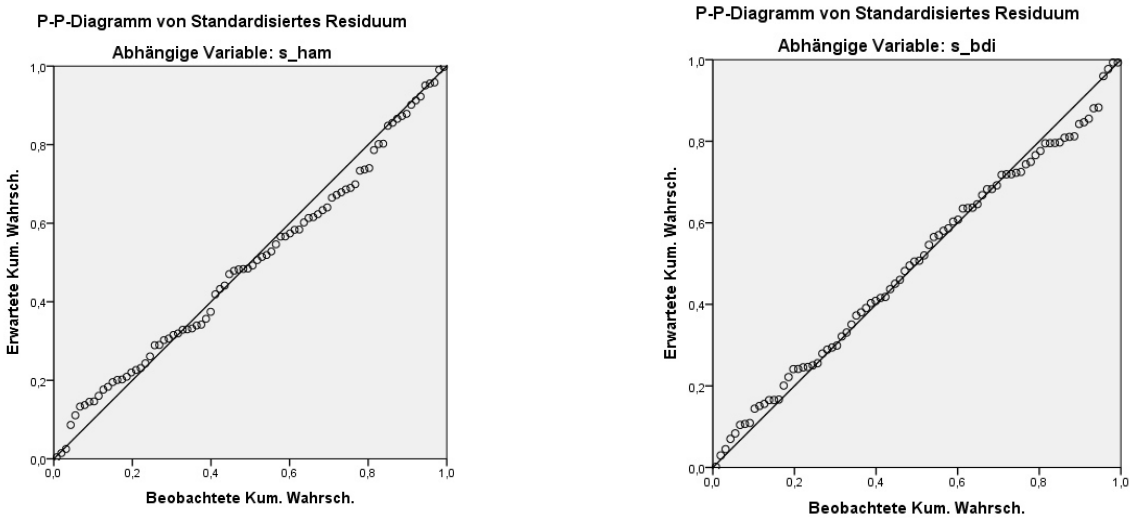

Figure 4 Checking normality distribution of residuals of model A and B

Table 20*: Correlation HAMD t0. t1. t2. t3*

	HAMD t0	HAMD t1	HAMD t2	HAMD t3
HAMD t0	1	.63**	.51**	.20
HAMD t1	.63**	1	.66**	.38**
HAMD t2	.51**	.66**	1	.43**
HAMD t3	.20	.38**	.43**	1

Note Significant correlations (2-tailed) are flagged: **=$p < .01$; *=$p < .05$. HAMD = clinician-assessed depression.

Table 21: *Correlation BDI t0. t1. t2. t3*

	BDI t0	BDI t1	BDI t2	BDI t3
BDI t0	1	.65**	.57**	.40**
BDI t1	.65**	1	.76**	.53**
BDI t2	.57**	.76**	1	.60**
BDI t3	.40**	.53**	.60**	1

Note Significant correlations (2-tailed) are flagged: **=$p < .01$; *=$p < .05$. BDI = self-assessed depression.

Table 22: *Correlation DAS t0. t1. t2. t3*

	DAS t0	DAS t1	DAS t2	DAS t3
DAS t0	1	.80**	.64**	.48**
DAS t1	.80**	1	.81**	.50**
DAS t2	.64**	.81**	1	.55**
DAS t3	.48**	.50**	.55**	1

Note Significant correlations (2-tailed) are flagged: **=$p < .01$; *=$p < .05$., DAS = cognitive mal-adaptive beliefs.

Table 23: *Correlation PB t0. t1. t2. t3*

	PB t0	PB t1	PB t2	PB t3
PB t0	1	.60**	.39**	.27*
PB t1	.60**	1	.53**	.52**
PB t2	.39**	.53**	1	.57**
PB t3	.27*	.52**	.57**	1

Note Significant correlations (2-tailed) are flagged: **=p< .01; *=p < .05. PB = positive metacognitive beliefs.

Table 24: *Correlation NB t0. t1. t2. t3*

	NB t0	NB t1	NB t2	NB t3
NB t0	1	.53**	.32*	.13
NB t1	.53**	1	.41**	.55**
NB t2	.32*	.41**	1	.37**
NB t3	.13	.55**	.37**	1

Note Significant correlations (2-tailed) are flagged: **=p < .01; *=p < .05. NB = negative metacognitive beliefs.

Table 25: *Correlation NFC t0. t1. t2. t3*

	NFC t0	NFC t1	NFC t2	NFC t3
NFC t0	1	.66**	.49**	.37**
NFC t1	.66**	1	.67**	.57**
NFC t2	.49**	.67**	1	.64**
NFC t3	.37**	.57**	.64**	1

Note Significant correlations (2-tailed) are flagged: **=p < .01; *=p < .05. NFC = metacognitive beliefs about the need for control.