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Direktor: Prof. Dr. med. Gerd Schulte-Körne

Transmission and Prevention of Depression in the Offspring of Parents with Depression

Differences and changes in psychopathology, emotion regulation and attributional style

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Johanna Löchner

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Berichterstatter:	Prof. Dr. med. Gerd Schulte-Körne
Mitberichterstatter:	Prof. Dr. Dr. Martin Keck
	Prof. Dr. Joest Martinius
Mitbetreuung durch die promovierte Mitarbeiterin:	Dr. Belinda Platt
Dekan:	Prof. Dr. med. dent. Reinhard Hickel
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Abstract

According to the World Health Organisation, depression is one of the most common psychiatric disorders - affecting around 350 million people across all age groups worldwide. Suffering from major depression not only causes great personal burden for the affected person, but also for their family, society and economy. The later the disease is recognized, the worse is the prognosis, going along with higher treatment costs. Consequently, an early identification of risk factors for depression is necessary to prevent these high personal and economic costs. One of the groups at greatest risk of developing depression is the offspring of parents suffering from depression. Their risk of developing depression is estimated to be three to four times higher during childhood and adolescence alone, and do even persist into adulthood. Since the transmission of depression from parent to child may result from numerous risk and protective factors and their interaction, the high risk for developing a depression is not understood well yet. Furthermore, although evidence-based treatment interventions for depression have been developed and implemented into practice, few prevention programs for the children of depressed parents have been developed, with heterogeneous findings. In the first part of the thesis, I provide a theoretical framework for the trans-generational transmission of depression based on the existing literature. In addition, prevention approaches and their efficiency in reducing the risk for depression are discussed. In the second and empirical part two studies referring to the transmission and prevention of depression in the offspring of depressed parents are reported.

In *study I*, a high-risk group (HR, n = 74) children of parents with depression is compared to a low-risk group (LR, n = 38) consisting of the offspring of parents without depression. The goal of the study was to i) replicate findings of the increased risk in youth that is associated with parental depression and ii) identify most prevalent risk factors in order to explore possible mechanisms of the trans-generational transmission of depression. Therefore, the HR and LR were compared in general psychopathology (self-rated depressive and psychopathology symptoms; parent-rated psychopathology) and the mediators (emotion regulation, attributional style) and moderators (life events). In addition, the role of parental depression and its impact and association on the children's depressive symptoms is investigated. The data supported earlier findings of increased risk for depression for the HR, since the HR showed significantly increased psychopathology and depressive symptoms with a big effect size (d = 1.75). Thereby, the parental depression was associated significantly with children's depression severity. In addition, the data provided strong evidence for group differences in adaptive emotion regulation strategies, positive and negative attributional style and the number of positive life events. Against expectations, groups did not differ in maladaptive emotion regulation strategies and the number of negative life events. Maladaptive emotion regulation strategies, negative life events and parental depression were the strongest predictors of children's depressive symptoms, together accounting for 30.8 % of the variance. These results suggest practical implications for prevention interventions for depression like increasing emotional and cognitive coping strategies and positive life events. Longitudinal highly-powered studies are necessary in future research.

In *study II,* preliminary results of an ongoing randomized controlled trial of one of the most promising prevention programs for the offspring of depressed parents (replicated here for the first time outside of the research group) are presented. Data from n = 61 families who reached post-assessment are provided. It was hypothesized that children in the experimental group (EG) would show decreased symptoms of psychopathology and depression compared to the control group (CG) over time. In addition, mediating factors such as emotion regulation

strategies and attributional style were expected to improve within the EG over time. Rating of treatment fidelity was very high, indicating good reliability of the intervention. The acceptance of families of the program was excellent; children and parents gave a very positive feedback about the intervention and their personal benefit of participating. Results indicate significant reduction of self-reported psychopathological symptoms between groups over time favouring a positive intervention effect. In addition, parent-rated psychopathology symptoms also showed significant decreases from baseline to post-assessment. Against expectations, both groups showed significant lower depression. There was a significant interaction effect of time and group indicating less maladaptive emotion regulation strategies and a more positive internal attributional style in the intervention group compared to the control group over time. Both groups showed improved adaptive emotion regulation strategies but a more negative attributional style over the study period. In contrast to predictions, there was a significant interaction effect of time and group in the negative internal attributional style scale, indicating a more negative attributional style of children in the EG over time. The benefits of the CG are interpreted as general activation for this high risk group for seeking information help. Together these findings are promising, although the results are preliminary and a bigger sample is necessary for more confident interpretations. There is a lack of evidence and number of prevention programs for this high-risk group, especially in Germany. Since effect sizes of prevention interventions were found to be small and diminish over time, further research is needed to identify relevant mediators and moderators in order to increase efficacy.

In sum, this thesis supports previous findings about the increased risk of depression for the offspring of parents suffering from depression and the association of parental and youth depression. In addition, it provides novel information about particular risk factors for children of depressed parents. Moreover, results of the first replication of a promising prevention intervention in Germany suggest that it is possible to modify some of these risk factors (maladaptive emotion regulation strategies and positive internal attributional style) and that doing so has positive effects on reducing self-reported psychopathology in children at risk.

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List of Content

1. Depression in Childhood and Adolescence

In 1980s researchers started to conduct studies focusing mental illness of children and their nature of psychopathology (Achenbach, McConaughy, & Howell, 1987a), treatment and development (Lonigan, Elbert, & Bennett-Johnson, 1998). Hence, a different understanding of psychological disorders in children emerged: children do differ qualitatively in manifest disorder and were no longer been seen as little adults, who basically show the same symptoms of psychiatric disorders. These new perspectives led to a new understanding of child psychopathology, coming along with new research approaches, theories and models and "recognized developmental psychopathology framework" (Huberty, 2012, p. 4). Depression in childhood and adolescence is associated with many negative outcomes like negative educational achievement (Gibb, Fergusson, & Horwood, 2011), negative social outcome and suicidality (Birmaher, Ryan, Williamson, Brent, & Kaufman, 1996; Weissman et al., 2006). Although it is related to depression in adulthood, depression for child and adolescence is facing different challenges in diagnostic and treatment.

1.1. Prevalence of depression in childhood and adolescence

Prevalence rates of major depression in general across the lifetime are nominated with 15 – 20 % (Ihle & Esser, 2002; Wittchen & Uhmann, 2010). In youth, the prevalence of depression varies across childhood and adolescence. Earlier studies found occurrence rates of depression in children from 1- 4 % and for adolescents 5 - 8 % (Birmaher et al., 1996; Jane Costello, Erkanli, & Angold, 2006; Lewinsohn, Rohde, Klein, & Seeley, 1999). A more recent study, the "Great Smoky Mountain Study" (Copeland, Angold, Shanahan, & Costello, 2014; Foley, Goldston, Costello, & Angold, 2006) confirmed these findings and further investigated in three months prevalence rates of depression that was 2.2 %. The most common comorbidity of depression is anxiety disorders with up to 70 % (Axelson & Birmaher, 2001).

Several researchers agree on the fact that depression in children increases markedly during transition from childhood to adolescence (Dietz, Weinberg, Brent, & Mufson, 2015; Roza, Hofstra, van der Ende, & Verhulst, 2003). During this phase of life the disorder rises dramatically and has its' first peak by the age of 15 (Pine, Cohen, Gurley, Brook, & Ma, 2007). Since puberty is a vulnerable period in youth, experiencing depression in this time is associated with significant consequences like diminished social relationships, reduced educational attainment and an elevated risk of suicide (Gibb et al., 2011). Moreover, an onset of depression during adolescence is associated with recurring and chronic trends in adulthood (Lewinsohn et al., 1999).

1.2. Symptoms of depression

Since the main focus of this work is transmission and prevention of depression, more detailed characteristics of the typology of depression are provided in the following section. As depression is manifested in cognitive, behavioural and physical symptoms, different kinds of symptom patterns are displayed in table 1 below. Core symptoms in depression are anhedonia, loss of interest and energy over time, self- confidence and appetite. For children and adolescents, symptoms can be slightly different and their developmental status needs to be taken into account. For example, adolescents with depression can also be rather agitated than sad. Also somatic problems for children (e.g. stomach ache) are more common than for adults. An experimental study characterized youth depression with shorter duration and reduced frequency of positive affects in comparison to a healthy control group (Sheeber et al., 2009).

Table 1 Characteristics of depression, (Huberty 2012, p. 57)

Cognitive	Behavioural	physical
• „all-or-none“-Thinking • catastrophizing • memory problems • concentration problems • attention problems • internal locus of control • negative view of self, world and future • automatic thinking • negative attributional style • negative affect • feelings of helplessness and hopelessness • low self-esteem • difficulty making decisions • feels of loss of control • suicidal thoughts	• depressed mood • social withdrawal • does not participate in usual activities • shows limited effort • decline in self-care or personal appearance • decreased work or school performance • appears detached from others • crying for no apparent reason • inappropriate response to events • irritability • apathy • uncooperative and suicide attempts	• Psychomotor agitation or retardation • somatic complaints • poor appetite or overeating • insomnia or hypersomnia • low energy or fatigue

1.3. Diagnosis “depression”

The challenge for clinicians working with children is to distinguish typical developmental variations of mentally healthy behaviour from those that indicate a manifest mental illness. Typical developmental variations may be interpreted falsely as pathological or significant psychopathological behaviour (Huberty, 2012). A mistake may lead to an inappropriate treatment or no intervention, when abnormal behaviour is not recognised as pathologic. Furthermore, symptoms vary in intensity, frequency and duration, making it essential to observe patterns or clusters of symptoms over a sufficient period of time. Therefore, diagnostic observations should cover different fields (e.g. home, school) and different sources (parents, teachers) (Achenbach, McConaughy, & Howell, 1987b). Lewis (p.3, 1990) defined developmental psychology as “… the study and prediction of maladaptive behaviours and processes over time”. Therefore professional clinicians need to evaluate the on-going dynamic nature of children’s development observing their emotional and behavioural problems over

time. Looking at multiple factors "has had a positive effect on clinical assessment, intervention and prevention research and practice" (Huberty, 2012, p. 5).

Consequently, developmental pathways were established in research and clinical practice. Due to these defined pathways, patterns that evolve and occur over time are more predictable. The primary classification systems for mental disorders are the *Diagnostical and Statistical Manual of Mental Disorders – Fifth Edition* (DSM-V, American Psychiatric Association, 2013) and the *International Statistical Classification of Diseases and Related Health Problems- tenth edition* (World Health Organization, 1993). The DSM is commonly used in the U.S., UK (and other English speaking countries like Australia) and represents the first reference to classify a depressive disorder for children and adults. In Europe the *ICD-10* (World Health Organization, 1993) is used preferably among practitioners, although in the research context it is common practice to use DSM. Both systems are categorical in nature and present a nomenclature to identify clusters of symptoms that lead to a specific diagnosis. With their polythetic, multiaxial approaches these classifications systems are providing a useful descriptive and administrative perspective. Implications for treatments are not established. Furthermore, there are limitations concerning developmental variations, cultural factors and other contributing factors such as the socio-economic status or parenting variables. Especially in the field of child psychopathology, the developmental process must be considered for an accurate diagnosis. The DSM and ICD differ slightly in the handling of diagnosing depression, but cover similar symptoms of depression.

Recently, a new version of the former DSM-IV, which was in practice since 1994, was published (May 18th, 2013). For the DSM-V, except the exclusion of the bereavement criteria, no changes were made concerning major depression. That means that it is now up to the clinician's discretion to differentiate depressive symptoms that follow a bereavement are a major depression episode or a typical grief reaction. The DSM-IV/V criteria suggest that five

of the following symptoms must be displayed for the majority of time for at least two weeks, while a depressive mood during most of the days and loss of interest and joy of activities must be shown. Further symptoms are increased or decreased appetite going along with loss or gain of weight (> 5 % / month), insomnia or hypersomnia, akathisia or deceleration, apathy and loss of energy, low self-esteem and sense of guilt, reduced ability of concentration and decision making, repeated thoughts of death and suicide. Additional criteria also must be fulfilled: There should not be a manic, mixed or hypomanic episode in the past. These symptoms must cause significant suffering and impairment in social, economic and other important areas of functioning.

1.4. Prognosis of depression

The average duration of a depressive episode for adults is around nine months (Birmaher et al., 1996). Even in case of no treatment, the depression is likely to diminish after this time. Nevertheless 70 % of the patients whose depression remits will experience a rezidiv within five years suggesting continuity till adulthood (Birmaher et al., 1996). For children and adolescents showing peculiar risk behaviour, frequency, potential for recurrence or chronicity and the severe morbidity of depressions are alarming factors (Micco, Henin, & Hirshfeld-Becker, 2014). Depressive symptoms in preadolescent youth (age 7-12) were shown to be predictors of adolescent depression. Due to the atypical presentation of symptoms and high frequency of comorbidity, depression often remains undetected, resulting in a more negative prognosis (Angold & Costello, 1993). Depression in this sensitive episode of pubertal, social and neural development may disrupt socio-affective processes and increase preadolescent risk of recurring depression across adolescence and young adulthood (Geller et al., 2001).

Even after an acute depressive episode, longitudinal studies indicate children with depression who recover within a nine month period still carry a significant risk for having repeated and more severe episodes of depression within subsequent two year period (Kovacs et al., 1984). Preadolescents with depression continue to experience more difficulties in interpersonal relationships with parents and peers after their symptoms remit (Puig-Antich et al., 1985). Furthermore, symptom improvement does not always result in improvement of interpersonal functioning. Residual impairment may be the pathway for depression recurrence (Dietz et al., 2015).

There is evidence that many children and adolescents suffering from depression do not seek help, although non-treatment of depression might have catastrophically negative consequences in their further educational, social and emotional development (Jaffee, Moffitt, Caspi, Fombonne, Poulton, & Martin, 2002). Statistics range from 10-30 % of affected children and adolescents receiving psychological treatment (Ezpeleta, Keeler, Erkanli, Costello, & Angold, 2001). Consequently, most of children and adolescents affected by depression don't receive adequate professional help. Besides the problema of correct diagnosis that was discussed earlier, reasons for this phenomenon may be the limited access to treatment due to/and the high costs of professional treatment

1.5. Treatment

The clinical practice guidelines indicate psychotherapy as the first line treatment for mild to moderate depression (National Institute for Clinical Excellence, 2005). In more severe cases and non-response to psychotherapy, pharmaceutics can be augmented (National Institute for Clinical Excellence, 2005). Numerous treatments types for psychotherapy popped out in the last decades. Among those the best evaluated evidence was found for cognitive behavioural

therapy (CBT). There is well-established support for CBT compared to no-treatment control conditions in treating community samples or preadolescents with elevated depressive symptoms. In 2004, Glass compared the efficacy of CBT and e.g. fluoxetine confirming the effectiveness of CBT. Still, 30 % of adolescents with major depression did not improve significantly. Nevertheless, there are very few controlled treatment studies for preadolescent depression (Dietz et al., 2015).

More recently, a meta-analysis showed decreased effect sizes of CBT treatment efficacy (standardized mean differences, ranged from -0.47 to -0.96) (Weisz, McCarty, & Valeri, 2006; Zhou et al., 2015) compared to earlier studies (standardized mean differences, ranged from -1.02 to -0.61) (Reinecke, Ryan, & DuBois, 1998). The reason for this discrepancy might be that earlier meta-analyses were based on small sample sizes in the studies. In addition, treatments were rarely or never directly compared in randomized controlled trials (Zhou et al., 2015). Some meta-analysis reported that CBT is superior to other treatments (David-Ferdon & Kaslow, 2008; Watanabe, Hunot, Omori, Churchill, & Furukawa, 2007). Others argue that non-cognitive therapies like interpersonal therapy (IPT) work just alike (Hetrick, Cox, Witt, Bir, & Merry, 2016; Weisz et al., 2006).

Although CBT seems to be an efficient treatment for depression in child and adolescence, effect sizes are moderate and many children do not respond to treatment. Furthermore, the access to treatment is often limited by numerous reasons, leading to manifestation and chronicity of the disease. Another important approach is therefore to prevent depression in the first hand (see section 4).

1.6. Summary

In summary, depression is one of the most prevalent psychiatric disorders, not only in adults. Prevalence rates vary between children from 1-4 % and for adolescents 5-8 %

(Kovacs, 1996), with a significant increase in adolescence (Pine, Cohen, & Gurley, 1998). Core symptoms of depression are manifested in cognitive, behavioural and physical symptoms as a predominant depressive mood and loss of interest and joy of activities. Depression is diagnosed using the classification systems for disorder DSM-V (DSM-V, American Psychiatric Association, 2013) or ICD-10 (World Health Organization, 1993). For children and adolescent, the developmental stage must be taken into account. There are evidence-based treatments as cognitive behavioural therapy or interpersonal therapy (Zhou et al., 2015). Nevertheless, many cases remain untreated due to limited access to therapy or do not respond to treatment, leading to manifestation and chronicity of the disease.

2. Causes of Depression

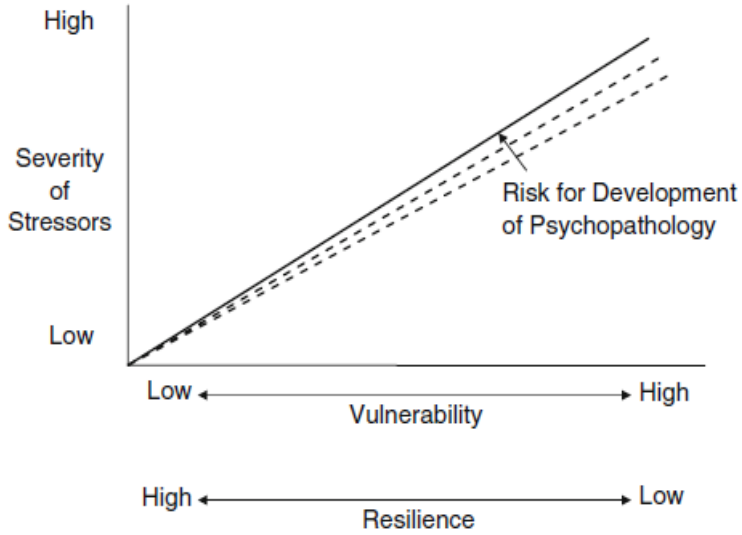
2.1. Diathesis-stress model

The *diathesis–stress* model or *vulnerability-stress* model is a paradigm for understanding how biological, psycho-social and environmental factors interact in the development and maintenance of depression. Vulnerability is defined as the sum of endogenous factors relying on the predisposition of a person to develop a disorder (Hankin & Abela, 2005). Stress is defined as the reaction of an individual to demands that require personal resources (Folkman & Moskowitz, 2004; Lazarus, 1993). For pathways of development in depression, for example, a certain diatheses or vulnerability is required (e.g. genetic predisposition). Thereby the predisposition alone is not sufficient to determine the occurrence of depression: Whether a diathesis is manifested depends greatly on presence and absence of significant stressors. In case an individual is exposed to an external stressor, it is an index of vulnerability or resilience how this person is adapting to it (Huberty, 2012). Individuals with many risk factors are more likely to have a greater diathesis and more difficulties in coping with stress. On the

other hand experience of stress without a vulnerability of mental illness may not cause psychological problems.

Several theories exist about how diathesis and stress interact leading to a psychopathological development (Hankin & Abela, 2005; Monroe & Simons, 1991). Corresponding to the *additive model* (Monroe & Simons, 1991) already a moderate amount of stress may cause psychological disorders, when a person yields a high level of diathesis. An individual with a low vulnerability for mental illness might still develop a psychological disorder in case stress increases above a certain limit. This theory is displayed in the graph 1 below.

Graph 1Vulnerability-resilience-risk-stress-continuum (Huberty 2012, p. 22)



Another variation of the *diathesis-stress model* is *the model of interaction* (Ingram & Luxton, 2005). In this model stress can only lead to a disorder in case there is a certain diathesis. A person without predisposition will not develop psychopathological symptoms, even when the amount of stress is increased. While children are growing up, they are facing

numerous risk factors, but also protective factors that influence their psychological development. This risk and protective factors interact with each other and the vulnerability leading to either a normal and adaptive behaviour or psychiatric disorders (Masten, 2001).

A recent study aimed to replicate these theories by examining genetic vulnerability and stressful life events and their impact on developing major depression on n = 5221 individuals (from 3083 twin families) (Colodro-Conde et al., 2017). Results showed a significant interaction of polygenic risk factors with stressful life events. This interaction accounted for 0.12 % of the variance of depressive symptoms. The authors argue that the amount appears to be small, since heritability of depression was not included as a predictor into the model.

Auerbach, Ho-Ringo Ho and Kim (Auerbach, Ho, & Kim, 2014) emphasize the limitations of this model, since it does not determine how and why stress occurs and what might be the specific individual reaction. Furthermore, they underline the interaction of characteristics of an individual and its reaction to stress. For example “depressotypic” characteristics that are defined as negative inferential style or hopelessness might even shape negative life events in the first hand (Stark, Schmidt, & Joiner, 1996).

Nevertheless, most researchers agree that biological and psychosocial risk factors contribute to the appearance of mental illness. Since risk and protective factor are infinitely numerous and every single one cannot be discussed here, an overview of all risk and resilience factors summarized by Huberty is provided in table 2 (Huberty, 2012).

Table 2 Risk and resilience factors by Huberty (2012)

Context	Risk factors	Vulnerabilities	Protective factors
Genetic	• Genetic disorders or predispositions • Heredity factors	• Problems associated with genetic or hereditary disorders, e.g. language and self-help skill deficits	• Absence of genetic or heredetary disorders • Minimal influence of genetic or hereditary disorders • Lack of stressors that might „trigger“ predispositions
Biological	• Prenatal infections or injury • Neuropsychological deficits/brain damage • Poor maternal care and nutrition • In utero exposure to toxins • Maternal substance abuse	• Difficult temperament • Problems associated with neurological and biological problems, e.g. cortical dysfunction, adaptive skill deficits	• Easy temperament • Absence of or minimal effects of biolodical or neurological problems
Personal/ individual	• Low intelligence • Poor emotional regulation • Low self-efficacy • Low self-esteem • Impulse control problems • Extreme shyness	• Gender • Poor planning ability • Emotional regulation problems • Sociability and social skills deficits • Impulse control • Attention problems • Executive functioning problems	• Gender • Average or above intelligence • Good social acumen and skills • Good emotional regulation skills appropriate for developmental level and situation • Absence of impulse control and attention problems
Family	• Poor parenting practices • Inadequate supervision • Insecure attachment • Parental psychopathology • Parental conflict • Unstable home environment	• Parent-child conflicts • Presence of a developmental, medical, or physical disability • Inadequate coping strategies based on current developmental capacity	• Cohesive family functioning • Good parenting practices • Absence of parental psychopathology • Good coping skills • Able to accept developmentally appropriate personal responsibility
Social	• Antisocial friends • Limited friendships • Limited access to positive social interactions • Poor social models • Socially marginalized	• Social skill deficits • Performance skill deficits • Fluency skill deficits • Social information-processing deficits	• Able to make friends and engage in age-appropriate reciprocal relationships • Absence of or minimal social, performance, and fluency deficits • Good social problem-solving skills • Positive role models

Cultural	• Poverty • Racism • Prejudice • Being a member of a minority cultural or ethnic group within a larger cultural context • Unstable, chaotic, or violent community environment	• Personal characteristics, including disabilities, that are not compatible with the larger social context • Degree of cultural assimilation of child	• Personal characteristics compatible with cultural context • Child is well assimilated into the culture • Positive socioeconomic status • Stable, supportive environment
Educational/ academic	• Poor school environment • Inadequate instruction • Lack of support for mental health and social development in the school setting • "Mismatch" between child´s needs and characteristics and the instructional environment • Disproportional instructional or disciplinary practices • Bullying and relational aggression • Limited family involvement in childs education	• Learning disorders • Difficulties adjusting to demands of school setting • Attention problems • Impulse control problems • Developmental delays	• Positive instructional, mental health, and social school environment • Absence of learning disorders and developmental delays • Individualized instruction adapted to the child`s needs • Cultural, racial, and ethnic equity with regard to instruction and discipline • School recognizes and effectively addresses bullying and relational aggression • Active family involvement in child`s education

2.2. Resilience

In contrast to the *diathesis-stress* model, the approach of resilience focuses on psychological well-being and a healthy development. A healthy development is defined as the children's ability to maintain the balance between *stressors* and *resources* in family, school and peers (Hjemdal, Vogel, Solem, Hagen, & Stiles, 2011). Resources are all protective competencies of an individual at disposal. This balance depends on the individual living conditions and only exists in a dynamic and adapted way (Bauer, 2005). Therefore, *vulnerability, risk factors, resilience* and *protective* factors are intercorrelated concepts, but still distinct from each other. Children with high vulnerability are also seen as having less resilience and are at greater risk to turn to negative psychopathological pathways, with the severity of a disorder being related to one or more stressors. Although vulnerability is a product of genetic, biological and psychological factors, the counterpart resilience can be increased via intervention and

prevention (Hankin & Abela, 2005). For example, a child can have a low vulnerability but simultaneously be able to show resilience to stressful life events. Therefore, children do have different threshold for the development of a disorder, based upon the degree of risk, vulnerability, resilience and stress.

2.3. Cognitive and emotional risk factors

Risk factors are those that have a negative impact on coping with stressors and increase the pathological effect of existing factors and moderate disorders (Jessor, Van Den Bos, Vanderryn, Costa, & Turbin, 1995). Since the appearance of depression is characterized especially by symptoms as negative thinking, hopelessness, depressive mood and loss of motivation, cognitive and emotional factors play an important role in the development and maintenance of depression.

Cognitive risk factors. Cognitive symptoms concern attention, concentration, memory problems as well as the way of thinking and evaluation of the perception. Beck established one of the earliest cognitive models of depression (Beck, Rush, Shaw, & Emery, 1979). He proposed the *cognitive triad* of depression consisting of three aspects: a negative self-evaluation, a pessimistic world view and hopelessness regarding the future. The cognitive triad is highly associated with depressive symptoms with a magnitude of r = .65 (Beck & Perkins, 2001). Negative cognitive patterns are also present in psychopathology of children (Laurent & Stark, 1998). Goodman and Gotlib (1999) stated that several studies confirmed the relation between a negative view of the self and depressive symptoms having a negative self-image endorsing excessive rates of negative self-talk (Lodge, Harte, & Tripp, 1998) and a more negative assessment of their environment (Jacobs & Joseph, 1997). In addition, negative self-evaluations, perception of rejection and self-blame (defined as negative self-talk) were

associated with depressive symptoms (Calvete & Cardeñoso, 2005). Negative expectation of the future like being certain about occurrences of negative events and lack of positive outcomes were found to predict depressive symptoms (Miranda, Fontes, & Marroquín, 2008; Miranda & Mennin, 2007) . Muris and van der Heiden (2006) also reported findings of positive correlations of symptoms of major depression and a more negative view of personal future events rated by children (Muris & Van Der Heiden, 2006). In contrast, positive self-statements were correlated negatively with depressive psychopathology (Cho & Telch, 2005).

This negative thinking style is often displayed and therefore captured by the attributional style. The attributional style is defined as an individual approach in the way to explain causes of events. Thereby, events are commonly classified by internality, stability and globalization of attribution (Stiensmeier-Pelster, Schürmann, Eckert, & Pelster, 1994). Abramson, Seligman and Teasdale argue that individuals differ in the attribution of positive and negatives event in these three dimensions (Abramson, Seligman, & Teasdale, 1978). Depressive patients are usually characterized by a negative attributional style in all three entities and those are often precursors of a depressive episode and endure the acute phase.

Horowitz and colleagues (Horowitz, Garber, Ciesla, Young, & Mufson, 2007) explored the attributional style in adolescents that were taking part in a randomized controlled trial in which the authors compared two prevention interventions for depression with a non-intervention control group. They found attributional style to be associated with the depressive symptoms in adolescents. Beyond that, the attributional style mediated the effect of the intervention on depressive symptoms. Braet and colleagues (2013) underline the importance of focusing on cognitive aspects in prevention of depression for children and adolescents with subclinical symptoms (Braet, Vlierberghe, Vandevivere, Theuwis, & Bosmans, 2013).

Emotional risk factors. Another relevant resilience factor of depression are emotion regulation strategies. Grob and Smolenksi (2005) refer to Thompson's definition of emotion regulation where those are defined as extrinsic and intrinsic processes which are responsible for monitoring, evaluating, and modifying emotional reactions (Thompson, 1994). It is a developmental task that involves initiating, inhibiting and modulating one's emotional state. Since individuals are exposed continually to a vast variety of potentially arousing stimuli in society that evoke emotions, emotion regulation is a highly significant skill in human life. In order to function as a healthy individual in the social context, it is obligatory to learn to manage the emotional state (Koole, 2009). In addition, affective symptoms are not solely present but linked to cognitive functions (Somerville, Jones, & Casey, 2010). Emotion regulation was found to be influenced by executive functions (e.g. inhibition, decision making) and also by social process (e.g. social model learning) (Somerville et al., 2010).

In case an adaptive emotion regulation style is conducted, negative emotions can be reduced (Grob, & Smolenski, 2005). In contrast, when maladaptive strategies are more frequently used, the emotional state is unbalanced, what might lead to psychopathological development and maladaptive behaviour (Garber & Dodge, 1991). In depression, maladaptive emotion regulation strategies as avoidance, suppression and rumination are overrepresented, while adaptive strategies as cognitive reappraisal, problem solving or acceptance are less frequent. Furthermore, children and adolescents that show more adaptive coping strategies when they experience negative life events were observed to show higher rates of psychological well-being in general (Kraaij et al., 2003). This is crucial especially in the developmental period of adolescence, when a more intense and frequent experience of emotions is substantial (de Veld, Riksen-Walraven, & de Weerth, 2012).

In a recent meta-analysis (Schäfer, Naumann, Holmes, Tuschen-Caffier, & Samson, 2016) 35 studies and 68 effect sizes on the difference of the relationship between adaptive

emotion regulation strategies (defined as cognitive reappraisal, problem solving, and acceptance) and maladaptive emotion regulation strategies (defined as avoidance, suppression, and rumination) with depressive and anxiety symptoms in adolescence were analysed. Thereby, adaptive emotion regulation was negatively associated with depressive and anxiety symptoms, while maladaptive regulation strategies showed positive associations. Furthermore, the authors revealed that the habitual use of all emotion regulation strategies was correlated significantly to depressive symptoms. The frequency of usage of adaptive or maladaptive emotion regulation strategies also made a difference in the association with psychopathology: the more adaptive coping strategies were used, the less depressive or anxiety symptoms were present. One major point of criticism is that in this study a non-clinical sample was used to assess self-reported emotion regulation strategies only. Therefore, the data is restricted to make concise conclusion about the association of maladaptive emotion regulation strategies and major depression. Furthermore, since the data is rather cross-sectional than longitudinal, the effect of maladaptive emotion regulation strategies as a predictor for major depression remains unclear.

Van Beveren and colleagues (2016) investigated in the association between temperamental reactivity, emotion regulation and depression in youth (n = 176, 9-18 years) (Van Beveren et al., 2016). The authors not only confirmed the named results of Schäfer and colleagues (2016), but also found significant correlations between higher levels of negative emotionality as a trait, depressive symptoms and the use of maladaptive emotion regulation strategies. Whether emotion regulation strategies or emotionality traits can be interpreted as causal factors leading to psychopathological symptoms remains unclear. Van Beveren and colleagues (2016) underline the need of identifying resilience factors for depression in youth.

In summary, cognitive and emotional factors are central in the development and maintenance of depression. Especially adaptive emotion regulation strategies as well as

positive attributional style were discussed as important resilience factors for major depression. For better understanding the role of emotion regulation and the attributional style in the development of youth depression must be further explored.

2.4. Summary

The *diathesis–stress* model or *vulnerability-stress* model is a paradigm for understanding how biological, psycho-social and environmental factors interact in the development and maintenance of depression. Different theories exist on the accumulation or interaction of different risk and protective factors accounting for the development of a disease like depression. In contrast, the concept of resilience on psychological well-being and a healthy development, defined as the ability to maintain the balance between *stressors* and *resources* (Hjemdal et al., 2011). Resilience factors are those that have a positive impact on coping with stressors and decrease the pathological effect of existing risk-factors and moderate disorders (Jessor et al., 1995). Most relevant resilience factors for depression are emotional and cognitive resilience factors, since depression is characterized especially by these factors.

3. Transmission of Depression

3.1. Risk factor: Parental Depression

One of the most prevalent risk factors of developing a depression is having a parent with depression (Beardslee et al., 1998; Weissman et al., 2006; Weissman et al., 1997). Although there is a consensus in research about that heightened risk for depression in this group, estimations of the specific risk vary. One of the most reliable sources is a longitudinal study with follow-up measures ten and 20 years after baseline (Weissman et al., 2006). Here, the offspring of depressed parents developed the disorder three (20 years post-baseline) to four (10 years post-baseline) times more often compared to the offspring of psychiatrically healthy parents. Other rates vary between three (Garber et al., 2009) to six times (Downey & Coyne,

1990) of increased risk. Mattejat and Remschmidt estimated that 50 % of children of depressed parents have experienced a depressive episode at the age of 20 (Beardslee et al., 1998; Mattejat & Remschmidt, 2008). In case both parents suffer from a depressive disorder, the probability of getting a depression raises to 70 % (Downey & Coyne, 1990).

Beside the risk of incidence of depression, numerous studies focused in psychopathology symptoms and risk of developing mental illnesses in the offspring of depressed parents (Heitmann & Bauer, 2007; Ihle & Esser, 2002; Weissman et al., 2006; Weissman et al., 1997). Here, children and adolescents were found to show increased psychopathological symptoms, e.g. on internalizing, externalizing or abnormal social behaviour (England, & Sim, 2009). In addition, parental depression was found to be associated with the children's psychopathology concerning early onset of mental illness, longer duration, high likelihood of recurrence and symptom severity (England, & Sim, 2009). In a meta-analysis of 193 studies on associations of maternal depression and child maladaptation, correlations of children's internalizing and externalizing symptoms ranged between r = .21 -.23(Goodman et al., 2011). In addition, children's negative effect and behaviour (e.g. sadness, fear) and less positive behaviour (e.g. less smiling, approaching) were also associated significantly with the maternal depression (r's = .10-.15). Several vulnerability factors in the child as a "difficult" temperament" (Green et al., 2010), a more insecure infant attachment style, dysfunctional emotional regulation, anhedonia and cognitive vulnerability to depression (e.g. negative attributional style, self-blame, low self-esteem) were correlated with the mental illness of their parents (England, & Sim, 2009).

The increased risk for depression in children and adolescents growing up with parents suffering from depression can be easily imagined, by thinking of depression characteristics like anhedonia, loss of motivation, interest and energy and the possible environmental stressors that may accompany a depressive episode (e.g. loss of job, marital issues) that

interfere with parenting tasks. Depression was found to be associated significantly a harsher and more negative parenting style and/or emotional unavailable, inconsistency with moderate effect size (England, & Sim, 2009). These impairments even may outlast an acute depressive episode (Rutter & Quinton, 1984). Aggravating this, children and adolescents are usually not informed about the parental disease, leading to unpredictable situations and feelings of confusion and insecurity (Lenz, 2005). For many children a diagnosis of mental illness is initially discovered when the disease is deteriorated and parents leave homes for in-patient stay and treatment. This event and the accompanied separation can be traumatic, especially for little children that have not been enlightened about the parental disease at an earlier point of (Lenz, 2005).

Nevertheless, some children seem to be more resilient and not for all of them parental depression necessary leads to a psychiatric disorder. It is still debated how children manage their developmental tasks and how mental disease might be transmitted. Although some researchers argue that it might be more likely for children to develop the exact same disorders as their parents (Hosman, van Doesum, & van Santvoort, 2009), it remains unclear, what kind of diagnosis children of parents with mental illness might evolve (McLaughlin, 2011). So far there is a consensus of a rather unspecific transmission of psychiatric disorders, except for bipolar disorders that have a greater heritability factor (Birmaher et al., 2009). This means that a particular parental disorder as e.g. social phobia does not necessarily lead to the exact same kind of disorder in the child (but e.g. depression). This phenomenon is called *multi-finality* (a specific risk factor leads to different outcomes) whereas *equi-finality* is referring to a specific disorder as a result of multiple causes (Hosman et al., 2009). In contrast, maternal depression was also found to be linked to earlier onset and more severe course of depression in the offspring (Lieb, Isensee, Höfler, Pfister, & Wittchen, 2002). Two important approaches in order to understand trans-generational pathways of depression and mental illness in general

are *model of transition* for depression of Goodman and Gotlib (Goodman & Gotlib, 1999) as well as the *model of transition* of Hosman and colleagues (2009) presented.

3.2. Models of Transition

In the following section two models of transition are presented and discussed. These *models of transition* of aim to include evidence on vulnerabilities, risk factors mechanisms and moderators in order to understand the transgenerational transition of i) depression (Goodman & Gotlib, 1999) or ii) mental illnesses in general (Hosman, 2009). Firstly, the *model of transition* of depression (Goodman & Gotlib, 1999) is explained in detail and updated with current findings in research supporting the model. Secondly, the model of Goodman and Gotlib is complemented by the more recent model of transition by Hosman and colleagues (Hosman, 2009).

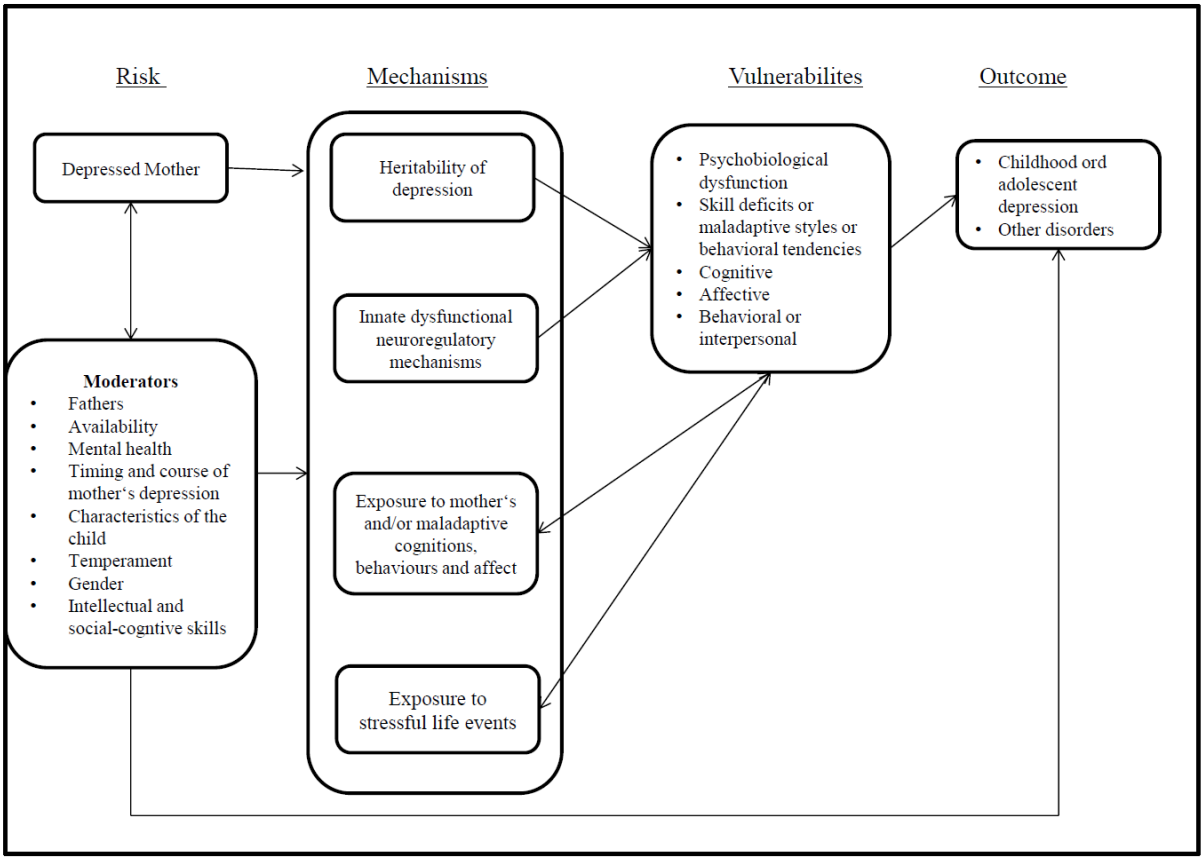
3.2.1. Overview of model of transition of depression

The *model of transition* of depression of Goodman and Gotlib (1999) integrates biological and psychosocial aspects within a transactional perspective in order to uncover the mediation and moderation roles of important factors between the effects of mother's depression on their children (Goodman & Gotlib, 1999). This integrative model (graph 2) displays a maternal depression first with four main variables that are likely to happen due to the mental illness: a) heritability of depression, b) innate dysfunctional neuro-regulatory mechanisms, c) negative maternal cognitions, behaviours and affect and d) stressful context of the children's live. All of these factors display a potential mechanism for the transmission of risk for developing a mental illness; still, any depressed mother-child dyad may be characterized by one, more than one or none of the four mechanisms. Furthermore, the model assumes a number of interactions of the different factors that may affect the transmission of risk. For example, the

genetic factors interact most likely with all the other mechanisms and moderators, as well as biological and psychosocial factors. As indicated in graph 2, the occurrence of none or more of the proposed mechanisms for the transmissions of risk is associated with the emergence of vulnerabilities in any of several domains of functioning: cognitive (e.g. dysfunctional cognitions, low self-esteem, helplessness or hopelessness beliefs, biased attention and interpretation or memory functioning), emotional (e.g. low stress resilience, difficulties in emotional regulation) and behavioural or interpersonal (e.g. inadequate social and social-cognitive skills, dysfunctional impulse control, problems in concentration, low mastery motivation) and psychobiological (the central nervous systems, especially the hypothalamic-pituitary-adrenocortical (HPA) axis). These vulnerability factors are also very likely to affect each other and interact. For example children characterized by dysregulation of the HPA axis may be predisposed both - to act in a lethargic manner and to exhibit hyper-responsiveness to the challenges of novel environments (Coplan et al., 1996). These tendencies would be expected to lead to a low rate of rewarding experience that is also a vulnerability to depression. Furthermore, this behaviour might lead to an increased maternal stress, lower maternal perceived parenting efficacy and poorer quality of mother-child interactions.

Finally, the model includes three moderators the vulnerability factors interact with: the father's health and involvement in parenting tasks, the course and timing of the mother's depression and characteristics of the child such as gender and temperament. In the following, important mechanisms, moderators and the children's vulnerability factors are constituted. Goodman and Gotlib reported scientific evidence for the validation of their model the data they referred to was published before 1999 (Goodman & Gotlib, 1999). Furthermore, additional recent findings are stated and complemented in order to update the past findings.

Graph 2 Model of Transition (Goodman & Gotlib, 1999)



3.2.2. Mechanisms of Transition

Firstly, the four mechanisms of the *model of transition* are constituted in the following sections.

3.2.2.1. Mechanism 1: Genetic factor (heritability and vulnerability)

There is a consistent body of literature demonstrating pattern of genetic transmission of depressive disorders in adults (Grillon et al., 2005). Family members in general have a heightened risk of developing a mental illness in case there is a genetic predisposition. Goodman and Gotlib (1999) referred to studies of twins, adoption and family study designs that were reporting the risk for an affective disorder in adult first-degree relatives of a patient with unipolar affective disorder to be around 20-25 %, compared with general risk of 7 % (Tsuang & Faraone, 1990). Earlier studies found that early onset of depression is the result of increased frequency of depression within families (Wissman et al., 1992), This fact is no longer supported by more recent research on the base of twin-studies (Cohen-Woods, Craig, & McGuffin, 2013; Sullivan, Neale, & Kendler, 2000). For example Sullivan and colleagues conducted a meta-analysis including five twin studies (Sullivan et al., 2000). The authors found that genetic factors explained 37 % of the variance, with unique environment accounting for 63 % and non-shared environmental effects.

Another current approach is the investigation in underlying epigenetic changes of depression. Epigenetic changes cover only the chemical change in a genom by leaving the base sequence unaltered and is therefore different to a genetic mutation (Januar, Saffery, & Ryan, 2015). Epigenetic modification occurs for example by a process called methylation that can be understood as “wrapping” of the deoxyribonucleic acid (DNA). Consequently, decoding the methylated DNA that is necessary for cellular processes is much harder or not possible at all. Interestingly, epigenetic modification can be caused and influenced by environmental factors. For example the stress reactivity can be affected by epigenetic changes

of a glucocorticoid-receptor that is responsible for the negative glucocorticoid feedback of the HPA-axis leading to a higher cortisol level (Smart, Strathdee, Watson, Murgatroyd, & McAllister-Williams, 2015). Due to these processes certain vulnerability for depression evolves. There is subsequent evidence that children, who experienced maltreatment show epigenetical and neuro-endocrinological changes (Romens, Mcdonald, Svaren, & Pollak, 2015; Smart et al., 2015).

3.2.2.2. Mechanism 2: Innate neuro-regulation

Another hypothesis of Goodman and Gotlib (1999) is that infants of depressed mothers are born with dysfunctional neuro-regulatory mechanisms that interfere with emotional regulation processes and consequently, increase vulnerability to depression. Specific neurological structures as the amygdala, specific cortical areas as the prefrontal cortex and the hypothalamic-pituitary-adrenal axis play an important role in the psychopathological development. Those structures are involved in emotions-, cognitive and stress regulating mechanisms that are central in the clinical picture of mental diseases (Meyer, Chrousos, & Gold, 2001). These dysfunctional neuro-regulation mechanisms are either caused by genetic factors or adverse prenatal experience (foetus' exposure to neuroendocrine alterations, constricted blood flow to foetus, poor health behaviours and use of antidepressant medicine) (when pregnant or in past, but neuroendocrine dysfunction of the mother nor recovered after episode). Goodman and Gotlib (1999) reported findings concerning higher levels of beta-endorphin and corticotrophin realising hormone (CRH) (Handley, Dunn, Waldron, & Baker, 1980) as well as higher urinary cortisol and norepinephrine (Field, 1998) among depressed mothers(Goodman & Gotlib, 1999). Furthermore, Goodman and Gotlib (1999) referred to findings on acute stress that is a characteristic of depressive episodes, and effects the neuro-endocrine functioning and the cortisol level in the placenta resulting in

abnormal stress reactivity, abnormal behaviour and affective functioning and abnormal EEG patterns in the child (Emory, Hatch, Blackmore, & Strock, 1993). Several other studies replicated findings concerning the negative consequences of depressive episodes during pregnancy on the child due to high levels of cortisol and the negative impact on brain development, emotion regulation in the HPA-axis and increased stress-reactivity and behavioural problems during childhood and adolescence (Huizink, Robles de Medina, Mulder, Visser, & Buitelaar, 2003; Ronsaville et al., 2006).

3.2.2.3. Mechanism 3: exposure to maladaptive cognitions, behaviors and affects

Beside the biological risk factors, Goodman and Gotlib (1999) included several psychological factors, which place the children at elevated risk for developing depression. In the following paragraph three components are discussed: 1) parental depression and its association with negative emotions cognitions, and behaviour, 2) social and model learning, 3) acquisition of depressotypic cognitions and behaviour.

3.2.2.3.1. Parental depression and negative cognitions, behaviour and affects

Goodman and Gotlib (1999) argue that it is not the depression itself that displays the risk factor for their offspring but the psycho-social impairments of the patient that are associated with depression. They emphasize the inadequate parenting, changed daily routine and social behaviour of parents that were often observed in families with a depressive parent. Parents often seem to be unable to meet the children's needs, resulting in deficits and delays in the children's development.

Gröhe and colleagues (2003) for example found that mothers suffering from depression were less empathetic and insecure in interpreting the children's signals resulting in even more stress and negative consequences on their recovery (Gröhe, 2003). They doubt

their own parenting competencies and report feelings of guilt and insufficiency. Parental psychopathology was associated with insensitive responsiveness as well as with low involvement with the offspring, low monitoring and child maltreatment (Elgar, Mills, McGrath, Waschbusch, & Brownridge, 2007). Parental depression is associated with less frequent positive interactions with children as well as parent child conflicts, poorer family communication and problem solving in other relationships (Beardslee, Gladstone, & O'Connor, 2011; Dietz et al., 2015).

Goodman and Gotlib (1999) differentiate the consequences of inadequate parenting and interaction with the offspring concerning the children's age: for infants, mostly attachment might be affected as well as the early acquisition of emotion regulation strategies. Goodman and Gotlib (1999) stated that insensitive or unresponsive parenting has been found to be among the strongest predictor for both, insecure attachment (Sroufe, Carlson, Levy, & Egeland, 1999) and infants difficulties in establishing effective self-regulation skills (Tronick, Als, Adamson, Wise, & Brazelton, 1978a). More recent studies found similar associations between a secure attachment style and mental health, focusing on the interaction of children and their depressed parents (Lenz, 2005; Mattejat & Remschmidt, 2008). Reck and colleagues (Reck, 2007) observed interaction styles of post-partum depressed mothers and their children by doing the Still-Face Paradigm (Tronick, Als, Adamson, Wise, & Brazelton, 1978b)[1]. The researchers found that mothers are often intrusive or unresponsive in their interaction, while

[1] *Still- Face Paradigm (Tronick, Als, Adamson, Wise, & Brazelton, 1978).* In this experimental setting, the direct effects of observed mother-child interactions by interpreting synchronies, contingencies or patterns of behaviours are analysed. This paradigm consists of three sections: In the first section, the baseline assessment, the mothers are told to interact just as usual with their infant that is seated right in front of her. In the second section, the mother is asked to face the child with a blank expression to her three to four months old infant for two minutes. In this phase of the experiment, the infants usually experience a high level of distress and react with high expressed emotions. The last section is the reunion, in which mothers are allowed to respond to their child again and calm them. Giniano and Tronick (1985, 1986) were investigating the effects of depression: Predominantly negative affect in facial expression and gesture were displayed, when mothers were simulating depression (e.g. being unresponsive to infants). Consequently, infants began to engage in self-directed regulatory behaviours, when external regulation from mother wasn't present.

children seem disturbed and irritated. In the second phase, when mothers are told to be non-responsive, children of depressed mothers are less disturbed and disengaged, which could be interpreted as being used to the mother's unresponsiveness. This negative interactive style was found to affect the attachment style negatively and has been observed to be carried out in the further childhood (Stringaris, Maughan, Copeland, Costello, & Angold, 2013). In sum, these findings of difficulties in parent-child interaction due to depression indicate an impaired attachment with negative consequences for a healthy development of the child.

Social network, peers and role of parenting are also discussed in the model of transition as specific risk and resilience factors for children of depressed parents: Goodman and Gotlib argue, that children face a lot of stressors in school and with peers and need parental support in these vulnerable phase (Goodman & Gotlib, 1999). Therefore, important positive parenting is helping the children to maintain their focus on cognitive –intellectual and social tasks (Hops et al., 1987). In case parents are not able to achieve their parenting duties, school failures, emotional and behavioural problems might be the consequences. Peer stressors were shown to be consistent predictors of depressive symptoms from middle childhood to early adolescence (Copeland, Wolke, Angold, & Costello, 2013) (Manuscript, Depression, & Predicts, 2013) whereas positive parent-child relationships may buffer peer stress and decrease the risk of depressive symptoms (Young et al., 2005).

3.2.2.3.2. Social learning

By social learning or modelling, children acquire cognitions, behaviour and affects that resemble those exhibited by their depressed mothers (Goodman & Gotlib, 1999). They state that children of depressed parents show behaviour like being less active and less content, have poorer peer relations, have lower self-esteem and negative cognitive styles (Weissman, Wickramaratne, et al., 2006). Goodman and Gotlib (1999) argued that parents show similar behaviour when they are depressed and that the behaviour of child and parent is related. The

social cognitive theory (Bandura, 1971) suggests that children acquire knowledge via cognitive processes in social contexts. Learning occurs through observation or direct instruction. Consequently, this matching behaviour might be the result of social model learning. For example Breznitz and Sherman (1987) showed that children match low rates of speech of their mothers in conversations with them (Breznitz & Sherman, 1987).

In more recent findings these effects are supported: Sidebotham and Heorn (Sidebotham & Heron, 2006) report how parents who experienced maltreatment in their childhood themselves were showing violent and neglectful behaviour to their children. On the other hand there were also positive consequences of social learning observed: Schneider and colleagues (Schneider, In-Albon, Nuendel, & Margraf, 2013) investigated in the effects of psychotherapy of parents on their children's well-being. They found less psychopathological symptoms not only in the patient, but also their offspring, although children were not at all engaged in psychotherapy. Schneider and colleagues (2013) discussed positive reciprocal processes of the new skills that were acquired in the psychotherapy course (like self-efficacy, positive thinking, and coping with stress). Parents "performing" these skills might function as role models and children automatically adapt to it by social learning. Schneider and colleagues did neither find gender differences, nor differences in the diagnosis of the parent.

3.2.2.3.3. Acquisition of depressotypic style

Goodman and Gotlib (1999) argue that children of depressed parents are facing the risk of developing a so-called "depressotypic" style of cognitions, affect skills and the resulting behaviour. This again might pave the way to develop a major depression since a negative attributional style or negative coping strategies are linked directly to psychopathological symptoms as described earlier (see section 2.3.). Goodman and Gotlib (1999) cited a study of Hammen and colleagues (Hammen, 1988), who found that children of depressed mothers showed more negative cognitions in their self-concept and negative self-schemata that

predicted adjustment problems at six- month follow-up assessment. Furthermore, the authors provided meta- analytical findings of Joiner and Wagner (1995) reporting moderate support for overall negative attributional style as prospective predictor of increases in depressive symptoms in children (Joiner & Wagner, 1995).

3.2.2.4. Mechanism 4: The Context of the lives of children in families with depression, particularly the stressors, contributes significantly to the development of psychopathology in the children

Goodman and Gotlib (1999) discussed that children of depressed parents are not only exposed to parental psychopathology, but also the psycho-social stress that might have caused the parental disease in the first place. Here, they differentiate between chronic stressors as financial and health problems and other stressful life events (e.g. loss of job, death of related party). For both cases, the authors reported findings how those stressors are accompanied in general with depression (Monroe & Hadjiyannakis, 2002) and how children are affected by those events. For example findings on the effects of poverty (Pound, Puckering, And, & Mills, 1988), chronic stress (Constance Hammen et al., 1987) and maternal depression as significant predictors of adjustment problems in children are discussed (Billings & Moos, 1982). Hammen and colleagues (1991) underline these findings: in this study children of depressed mothers report significantly more episodic and chronic stressors than children with mothers that did not suffer from depression (Hammen, Burge, & Adrian, 1991). Since depressed patients perceive stressors and life events more negative, due to the negative thinking style, research might be impaired by biased self-reports (Beck, 1967). In more recent reviews new methods were implemented in order to avoid false causal interpretation of stressful events (Monroe & Harkness, 2005). On the other hand, a negative cognitive style might “shape” negative life events.

3.2.3. Moderators of Transition

Despite the discussed mediators Goodman and Gotlib (1999) define several moderators that might play an important role in the transition of depression.

3.2.3.1. Partner of depressed parent

Firstly, the role of the father is discussed in Goodman and Gotlib's *model of transition* (1999) and report findings that show the impact of a coexisting parental depression on the development of children. Thereby, a significantly greater risk for disorder for children was shown in case of two depressed parents than in case only one parent suffers from depression (Downey & Coyne, 1990; Weissman, 1997). Healthy fathers – or mothers - may compensate the difficulties in parenting of the affected parent and offer special support to their children (Belsky, 1984). More recent studies confirmed the increased risk of developing a depression in case of having two depressed parents (Mclaughlin et al., n.d.). In addition it was confirmed that a second parent without mental illness may buffer the negative impact of depression in the family by caring and supportive behaviour (Chang, Halpern, & Kaufman, 2007). Nevertheless, the second parent also tends to be unable to cope with the daily hassles and situation, and might not be able to compensate negative effects of the mental illness for children either (Lenz, 2005).

3.2.3.2. Timing and chronicity of mothers depression

The authors further stated that first exposure to maternal depression has a stronger effect on the psychological development for children at a younger age than when they crossed specific sensitive periods. This might be the case, due to the fact that in the first year the neurophysiological development is quite immature, like the regulation of the HPA system or cortical inhibitory controls over arousal (Dawson, 1994; Porges, Doussard-Roosevelt, & Maiti, 1994). Therefore, mother's external regulation is significant in the first year of life, but may be constrained by postnatal depression. Another aspect is the chronicity of the parental

depression. The exposure to a single depressive episode has a less severe impact than a chronic course of depression (Rao, 2006). In a recent study the effects of maternal chronicity and severity of depression on their children's internalizing and externalizing behaviour was explored (Tompson, O Connor, Kemp, Langer, & Asarnow, 2015). The authors found that a prior severity and chronicity of maternal depression predicted internalizing and externalizing symptoms in their children, when the current status of maternal depression was controlled. Furthermore, chronicity of depression was a predictor for rate of change in the children's externalizing behaviour over time.

3.2.3.3. Children's variables

In the model of transition Goodman and Gotlib (1999) child-related factors are included as moderators and vulnerability factors (Goodman & Gotlib, 1999). Thereby, children's variables such as temper, gender, intellectual and social-cognitive skills are discussed as moderators of maternal depression and the risk of developing psychopathological symptoms (Goodman & Gotlib, 1999). The authors argued that these variables interact differently with the exposure to a depressed parent and that there is evidence that children vary in stress resistance, coping styles and being a stressor to their depressed parents. At that time, there were not studies that confirmed the association of temperament or gender with maternal depression and child dysfunction. Nevertheless, studies were published which discussed the role of temperament as vulnerability factor for the development of depression (Clark, Watson, & Mineka, 1994). It was further examined, whether depression influences personality traits and therefore be the result, not the cause of depression. A more recent study of 2011, Hankin and colleagues found evidence for moderate to substantial percentage of association between temperament and depressive symptoms in a sample of 131 pairs of twins and siblings at early adulthood and 326 pairs of twins in middle adulthood (Hankin et al., 2011). The others emphasized the role of genetic influences. One big limitation of this study is that only females

were included. The authors argue that the investigated factors would not differ by gender for depression (Lyons et al., 1998). Nevertheless, they also state that it is not clear, whether the results can be generalized to males.

Another risk factor discussed by Goodman and Gotlib (1999) is intelligence. The authors quoted one study indicating that a higher intelligence in children of depressed mothers might function as protective factor (Radke-Yarrow & Sherman, 1990). This finding was supported by Rost and colleagues (2009), who conducted a 20-year longitudinal study and observed and compared high-minded children to children with average intelligence (Rost, 2009). Since highly intelligent children were found to show slightly better stress coping strategies, the auhtor concluded that intelligence is a protective factor. Another more recent study confirmed that a cheerful temperament, high intelligence and good educational achievement were correlated to psychological well-being (Masten, 2001).

Furthermore, Goodman and Gotlib (1999) underlined that cognitive, affective and interpersonal skill deficits or maladaptive styles in the child increase the risk of developing a major depression. Only two studies were reported that indicate “limited support” for social-cognitive functioning as mediator between the maternal depression and children’s dysfunction (Beardslee, Schultz, & Selman, 1987). Another study reported interpersonal problem-solving competence, attributional and response bias that were found to reduce the risk of aggression and peer rejection, but only in a sample of children who were maltreated by their depressed mothers (Downey & Walker, 1989). Since 1999, more evidence supporting skill deficits in children as risk factors was reported: Jaser and colleagues emphasized the importance of the children’s coping skills particularly for the offspring of parents with depression (Jaser et al., 2005). Researchers investigated thereby in abnormalities in this high-risk group. For example, Lenz observed a more passive- avoiding coping strategy in children of parents with mental illness (Lenz, 2005). In addition, Garber and Flynn (2001) demonstrated that maternal

depression history is positively associated with depressive cognitions in adolescent (Garber & Flynn, 2001). These findings were significant for the dimensions hopelessness, self-worth and the attributional style. In case of chronically depressed mothers their 12 year old children had even stronger negative cognitions (Garber Robinson, Garber, & Robinson, 1997). They were found to be more likely to withdraw and hide their emotions, ruminate about problems or try to distract themselves to avoid anxiety and worries. Most of the time children develop feelings of guilt and have conflicts of loyalty. These tendencies might be reinforced by the family, avoiding the open discussion and information about the parental disorder, making it even harder to cope with the situation. In summary, children and adolescent appear to show less adaptive coping strategies concerning relevant emotional and cognitive resilience factors that are associated with the development of depression.

3.2.4. Criticism on the model of G&G

Although being published in 1999, the *model of transmission* of Goodman and Gotlib still displays the most prominent theoretical framework for the transmission of depression (Goodman & Gotlib, 1999). Similar as the *diathesis-stress model* (Hankin & Abela, 2005) the *model of transition* thereby integrates biological and psychosocial aspects within a transactional perspective. The model aims to uncover the mediation and moderation roles of important factors between the effects of parent's depression on their children. The big advantage of the model is the connection and interactions between biological aspects of functioning with psychological aspects that are inextricably linked in order to understand the transmission of risk of developing a depression. In 1999, there was little evidence for most of the named risk and resilience factors for the high-risk group of the offspring of depressed parents. Here it was shown that most hypotheses that were stated in the model are still up-to-date. Nevertheless, there is some criticism. The authors claim to constitute a comprehensive model of the transgenerational transmission of depression and provide an overview of the

interplay of the declared moderators, mediators, vulnerability factors and outcome variables. Unfortunately, this model was never validated as a whole but relies on evidence that investigated in mostly one or two factors only that were related to negative outcomes in children of depressed mothers. This results in an accumulation of possible risk factors that might play a role in the transmission of depression with an unclear concept behind it. For example, the children's social-cognitive skills are stated as vulnerability factors, but are simultaneously depicted as shaped by the parental depression (through model learning) and could therefore be interpreted as mediator or mechanism of transmission of depression. Goodman and Gotlib (1999) declared it as moderator, by providing evidence about a mediating role (Beardslee, 1987). In addition, the e.g. social influence to social-cognitive skills is not targeted. Therefore, the role and the interaction of those factors remain unclear and contradictory in the model.

Furthermore, some relevant factors are missing as the influence of culture, social network, environment, parental social skills and personality, professional help-system, parentification, epigenetics, family context and treatment experience of parental depression. Another big topic that was left out in the model is the so-called parentification many children display. Parentification is the process of role reversal whereby a child is obliged to act as parent to their own parent (Boszormenyi-Nagy & Spark, 1981). Two ways of parentification are known: the adaptive and destructive style of parentification. The adaptive parentification signifies no impairment of the development of the child. The child is being accredited for its behaviour and reinforced resulted in an increased self-esteem, belief in self-efficacy and empathy (Mattejat, Lenz, & Wiegand-Grefe, 2012). Conversely the destructive parentification has a negative impact on the child psychological development. The children's needs are neglected and the requirements the child has to cope with are inadequate concerning its developmental stage. Destructive parentification results negative long-term consequences as a

low self-esteem, feelings of insufficiency, problems of identification and autonomy, depression and suicidality. Especially adolescents try to confine to protect themselves from negative feelings what may even lead to aggressive avoidance and "internal escape". This coping behaviour on the other hand may increase feelings of guilt. Many adolescents also carry a lot of responsibility and due to their developmental stage easily take the role of the partner, take care of the medicine and household e.g. inevitably the process of identification at this stage is made much more difficult, also because of the missing figure of identification. This mechanism is reinforced by the parent's need of support. Other highly relevant cognitive and emotional factors that were shown earlier to correlate with depressive symptoms (see section 2.3.) (Braet et al., 2015; Horowitz et al., 2007; Schäfer et al., 2016) were not precisely targeted. Instead of focusing on attributional style, the cognitive triad or emotion regulation strategies the authors report findings about "depressogenic cognitive style" and "social-cognitive skill deficits (Goodman & Gotlib, 1999a). In addition, the model only focused on maternal depression. Although women are more often affected by depression than men are, there is nevertheless a substantial number of fathers suffering from depression (Wittchen, Jacobi, Klose, & Ryl, 2010). The update that was done in this literature review implicates that processes may be equivalent when a father is affected. This hypothesis is supported by results of a longitudinal study that explored differences in the offspring of fathers and mothers with depression (Lieb et al., 2002). Lieb and colleagues (2002) found no differences in the risk of depression whether mother or father was affected. Nevertheless, the specific gender aspects that might have an impact on child-parent interaction, relationship and role modelling are not discussed in the model of transition.

One can argue that in 1999 the state of research was less developed than nowadays. Nevertheless, the model of transition of depression (Goodman & Gotlib, 1999) should be interpreted as a theoretical conglomerate of findings of risk factors with unclear

conceptualization. More research on risk factors with experimental and longitudinal data is needed in order to provide a sufficient foundation for the identification of the specific role and interaction of relevant risk factors.

3.2.5. Model of transition of psychopathology of Hosman et al., (2009)

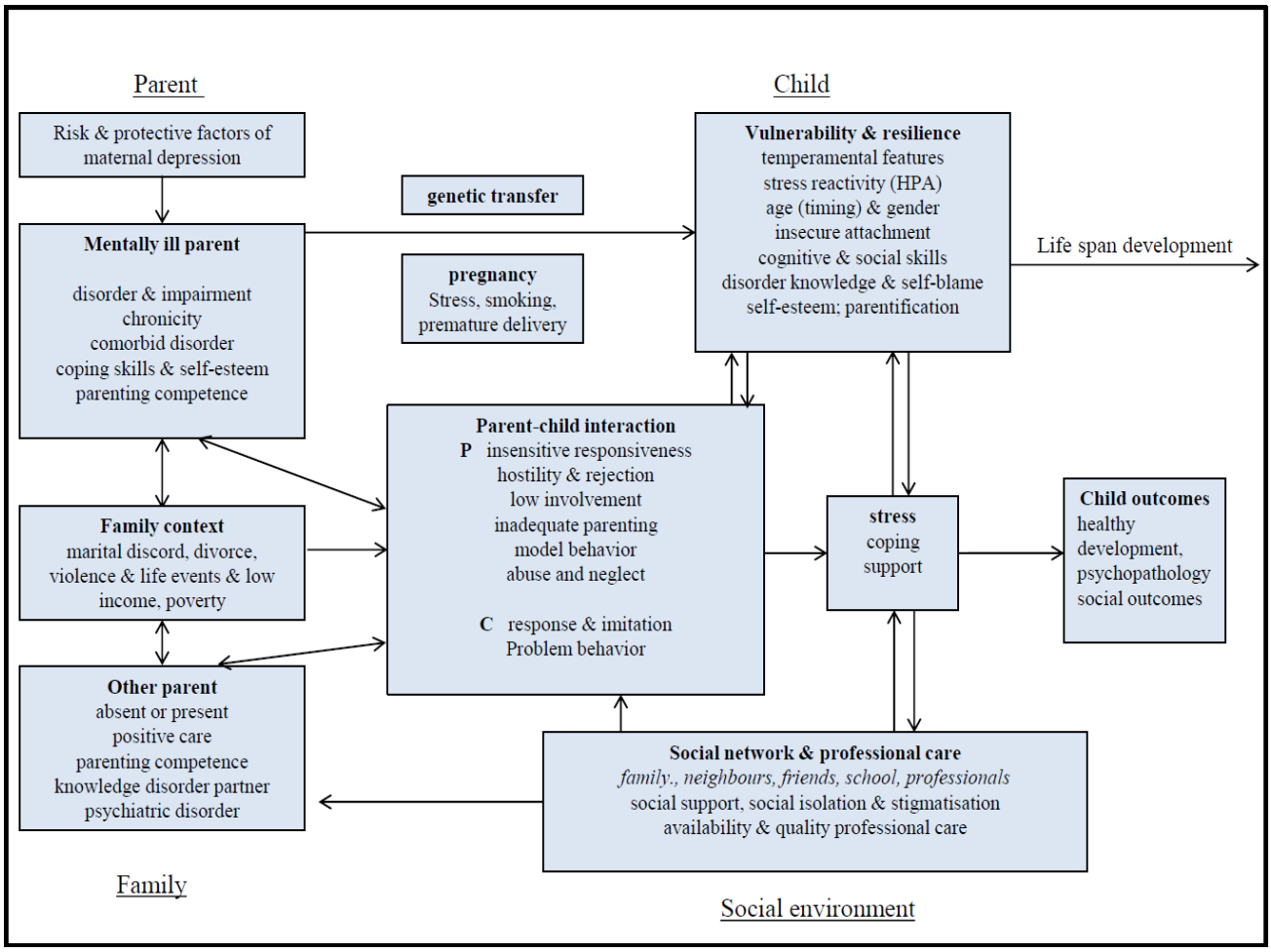
Another model of transition, aiming to explain transgenerational transition of mental illness in general, was developed by Hosman and colleagues (see graph 3) (Hosman et al., 2009). Similar as Goodman and Gotlib (Goodman & Gotlib, 1999), the research group integrated findings of numerous studies that appeared in the past 20 years (before 2009) on the trans-generational development of psychopathology in children of parents (mothers and fathers) with different kinds of mental diseases, in order to identify and study opportunities for preventive interventions. They also included the various mechanism of trans-generational risk transmission referring to Goodman and Gotlib (1999): 1) genetic risk transmission, 2) prenatal influences, 3) parent-child interactions, 4) family processes and conditions, and 5) social influences from outside the family. Furthermore, multiple interacting domains and systems of influence enter the model: 1) parents, 2) children, 3) family, 4) social network, 5) professionals and the wider community. Other additional components like the different developmental stages of children and adolescents as well as the principles of equi- and multi-finality (see section 2.4.) were taken into account. The authors postulated that early impairment has greater effects on the psychopathological development of the child due to attachment and emotional regulation problems (Silk, Shaw, Forbes, Lane, & Kovacs, 2006a).

In contrast to Goodman and Gotlib's model, Hosman and colleagues (2009) underlined that they were not only focusing on the development of psychiatric and related problems but also on factors of resilience and social-emotional development. Hosman and colleagues point out the impact of the parental mental illness as mediator of the marital

relationships, the family life and the children's psychopathology as well as the extra-familial environment, community and care system (Avenevoli & Merikangas, 2006). As all other factors, these can be either protective or risk-increasing for the offspring of depressed parents. For example school could provide a place for children where they can escape the stress of harsh family environment and find opportunities for diversion and positive experiences (Avenevoli & Merikangas, 2006). On the other hand peers can show bullying behaviour and children might be afraid of talking openly about their parent's depression, fearing further exclusion (Hosman et al., 2009). For example a large study in the U.S. investigated the social support by caregivers, who were others than their mothers (Lee, Halpern, Irva, & Martin, 2006). It turned out that the onset of internalizing problems in children of depressed mothers was lower when the family received social support. Hosman and colleagues (2009) criticized the wide-spread lack of child-targeted skills among professionals treating adults and the link to child care.

In sum, Hosman and colleagues (2009) suggest to assess carefully the accumulation of potential risk and protective factors within and across domains in their multi-causal model: The more risk factors accumulate, the higher is the probability of developing psychopathological problems (Rutter & Quinton, 1984). Although the authors carefully constitute conclusions, their model also faces the same problems as the model of transition of depression (Goodman & Gotlib, 1999). Likewise the model is an accumulation of single findings rather than a comprehensive model that was validated as a whole. In addition, cultural factors and the influence of gender are not reported.

Graph 3 A developmental model of trans-generational transmission of psychopathology (Hosman et al., 2009)



3.3.5. Conclusion

Although the *model of transition* of Goodman and Gotlib was published in 1999 and its references are even older, it is still the most prominent framework of transition of depression. The comparison with recent findings showed that most hypotheses are still up-to-date. The expansion of this model by Hosman and colleagues (2009) contributes with additional factors, making it accessible and useful also for other populations than just mothers with depression. Furthermore, resilience factors, the social network and professional care system are taken into account. The great advantage of these models making it prevailing in the field of risk and resilience research is the integration of numerous relevant variables.

Major criticism refers to the accumulation of single and often insufficient findings and the lack of experimental evidence as base of a complex multi-factorial and –causal model. In addition, the model only focuses on maternal depression and does not include various important risk factors. Hosman and colleagues (2009) include both sexes in their model, nevertheless, the discussion of gender relevant aspects is missing in this model (Hosman, 2009). The impact of single risk factors in the interplay of vulnerability and resilience, especially in the light of different developmental stages, of an individual is indefinite. Further research is needed that combines several of these risk and protective factors for children of depressed parents in order to explore the interplay and consequences in sensitive periods.

Current research approaches underlined the findings of these models of transition from a different perspective: Schneider and colleagues (2013) found positive trans-generational effects of parents with mental illnesses doing psychotherapy (Schneider et al., 2013). Children of parents in treatment were found to have less psychopathological symptoms, especially when the parental treatment was successful. Even when parents did not benefit vastly from psychotherapy, their children were still better off. Interestingly, parents of children with mental illness doing psychotherapy also benefit from their children's treatment showing less depressive and stress symptoms. Schneider and colleagues (2013) discuss a positive reciprocal process of new skills (like self-efficacy, positive thinking, coping with stress) learned in the treatment that might be transmitted into the family by e.g. social learning. These findings were not specific for particular diagnosis and indicate an interruption in the trans-generational transmission of mental illnesses.

3.3. Summary

In this paragraph the risk factor of parental depression for developing a depression was discussed. Although researchers agree on a heightened risk of developing a depression for this

risk group, risk estimations are heterogeneous and vary between three (Garber et al., 2009; Weissman et al., 2006) to six times (Downey & Coyne, 1990) of increased risk.

The *model of transition* by Goodman and Gotlib (1999) which was further presented aiming to include evidence on vulnerabilities, risk factors mechanisms and moderators in order to understand the transgenerational transition of depression. Additionally, an update with findings of the current research was provided, indicating the validity of the theoretical framework of Goodman and Gotlib (1999). Furthermore, the trans-generational transmission of psychiatric disorders in general by Hosman and colleagues (2009) was presented. Thereby, the importance of resilience factors, critical time periods social network and professional health care are additional important factors.

Although there is major criticism on these two *models of transition* (Goodman & Gotlib, 1999; Hosman et al., 2009) they represented substantial theoretical frameworks leading to practical implications for children of depressed parents. Although there is a vast number of risk factors that cannot be changed like biological factors (e.g. genetics, child's temper) or certain circumstances (e.g. critical life events, parental depression), the models of transition also display numerous factors (e.g. cognitive, emotional coping skills) that are well known to be modifiable in psychotherapy (Zhou et al., 2015). Consequently, in the recent decades a growing number of preventive approaches aroused focusing on those modifiable risk factors in order to prevent depression in general and in the offspring of depressed parents.

4. Prevention of depression in the offspring of depressed parents

Concerning the high risk of children with depressed parents, the negative prognosis of early incidence and the limited access and high costs of treatment, preventive approaches are clearly necessary. As shown in the models of transition, there are numerous risk factors that are modifiable and are targeted in psychotherapeutical interventions for depression (e.g. negative thinking style) (Zhou et al., 2015). Surprisingly, research of prevention of depression has a short research history. In the following paragraph, the concept of prevention, a description of basic ingredients and selected prevention programs that were evaluated in a randomized controlled trial (RCT) are presented. In addition, the efficiency of prevention programs for the offspring of depressed parents is discussed. At the end of this section existing programs for the offspring of depressed parents as well as results of a recent meta-analysis of those are presented.

4.1. Definition

Prevention (lat. *praevenire,* „to forestall“) contains actions that aim to avert unwanted occasion or disease that could occur with a certain probability, in case those actions are not implemented (Hurrelmann, Klotz, & Haisch, 2009). Prevention assumes the existence of treatments that are suitable and do have an impact on the unwanted disease – in this case the onset of depression. In general preventive interventions have the aim to promote a healthy future for children, by reducing the number of risk factors in its environment and extending protective factors instead. There are two different kinds of nomenclatures in order to distinguish prevention interventions in the field of depression. Programs are either clustered 1) concerning the symptomology in primary, secondary and tertiary prevention or 2)

concerning the targeted group into selective and/or indicated prevention or universal prevention programs (Mrazek & Haggerty, 1994).

Primary prevention. Primary prevention takes place before a disease occurred and in order to prevent it (Hurrelmann et al., 2009). Target groups are populations with specific risks (e.g. parental depression), but also any healthy individual without any kind of symptoms. A popular example is vaccination in the general population to prevent the occurrence of e.g. infantile cerebral palsy, or nutrition training in schools to prevent obesity.

Secondary prevention. Secondary prevention occurs at an early stage of a disease. Its main goal is the early detection of illness to prevent its progression or chronicity (Hurrelmann et al., 2009). Programs for adolescents that had already abused illegal substances or alcohol in order to prevent addiction are an example. In the field of depression children who have already shown elevated depressive symptoms would be the target group for secondary prevention interventions.

Tertiary prevention. When a disease is manifest or was treated acutely, tertiary prevention is implemented for relapse prevention or reduces secondary damages (Hurrelmann et al., 2009). Target groups are mostly patients with chronic illnesses, like diabetes or major depressive episodes.

Selective/targeted/indicated Prevention. Selective prevention approaches target a specific group of children, adolescents or families facing a specific risk of developing a depression, like parental depression or a children's enduring anxiety (Petermann & Petermann, 2011). In case of elevated but subclinical depressive symptoms of children, prevention interventions are "indicated" (Dolle, Schulte-Körne, von Hofacker, Izat, & Allgaier, 2012). These types of prevention have many similarities to treatment contents of

depressive disorders, as e.g. psycho-education, cognitive reappraisal, coping with stress, communication and social skill training and problem-solving.

Universal prevention. In contrast, universal prevention programs include children and adolescents of the general population without a specific selection (Hurrelmann et al., 2009). It is quite common for this type of prevention intervention to take place in schools – sometimes with an additional parent session - in order to reduce general risk factors. Therefore children learn contents as problem solving, coping with stress and relaxation techniques.

4.2. Basic ingredients of prevention interventions for the offspring of depressed parents

There is a growing number of upcoming prevention interventions for depression that have a vast variety in content, number of session, setting, target group and level of scientific evidence. In the following, basic ingredients that are mostly used in these different kinds of depression prevention programs for the offspring of depressed parents are presented.

4.2.1. Psycho-education

Psycho-education of the parental illness is one of the basic ingredients that is included commonly in most of the prevention programs in order to prevent negative outcomes in the children's psychopathology (Beardslee et al., 2011; Clarke, Hornbrook, Lynch, et al., et al., 2001; Compas et al., 2011; Sanford et al., 2003a). In a qualitative investigation children of mentally ill parents Lenz (2005) reported the need of information about course, symptoms and side effects (Lenz, 2005). Consequently most interventions aim to empower children to reach a good understanding of their parents' disease in order to increase their feeling of security and control. Knowing about facts reduces worrying, hopelessness and anxiety that come along

with an unpredictable behaviour due to depressive symptoms (Lenz, 2005). Lenz emphasizes in his work, that the age-adequate psycho-education works as an important protective factor, increasing the children's resilience. Information about the mental illness might have an impact on a person's perception and cognitive appraisal leading to a change of the experience of stress. Feelings of guilt, anger and anxiety, that many children report can be reduced due to the information of causes and symptoms of depression (Scherrmann, Seizer, Rutow, & Vieten, 1992). There is a variety of information provided for adults in order to make them the "expert of their own disease"

but little literature about parental depression or living with a depressed parent for children (Lenz, 2005). Lenz argues that an open dialogue with children and adolescent might be advantageous anyway facing the possibility to respond to children's feelings concerning the parental depression. Psycho-education for children should furthermore be a standard in the clinical practice, especially in cases of forced hospitalization that could be a traumatic event for children (Lenz, 2005).

4.2.2. Coping with stress

As showed earlier, children that are growing up with a parent suffering from major depression are exposed to a high level of stress (as psychosocial stressors, conflicts, depression). Since stress contributes to the development of depression, many prevention programs focus on stress coping skills (Compas et al., 2015; Garber et al., 2009). The aim is to develop positive coping strategies, in order to increase the children's resilience. Positive coping skills or problem-solving skills are based on cognitive behavioural therapy and are used to enable children to adapt a more adaptive style and more flexible possibilities in order to cope with their daily stressors (Lenz & Kuhn, 2011). Relying on Goodman & Gotlib's *model of transition* children of depressed parents often might adapt a "depressotypic" thinking style (e.g. learned helplessness). Because of that children are reinforced to observe their reaction to

stress: cognitive, emotional, physical and behavioural. In a second step, children can evaluate how helpful their way of coping is and whether there may be more adjuvant ways of thinking and acting in a situation. Furthermore, the association between positive thinking and well-being is displayed. Children are trained to take over an active role in their mental experience and learn self-efficacy while they are overcoming feelings of helplessness. These abilities again have a high impact on perception of stress and their general well-being. In their daily lives children ought to improve their problem solving skills when conflicts in the family occur.

4.2.3. Parenting training

Although there is evidence of poor parenting skills of parents suffering from depression(England & Sim, 2009), few prevention programs focus on teaching parenting skills (Compas et al., 2015; Sanford et al., 2003a). Concerning the vast problems of parenting for a depressed parent as described earlier, this is quite surprising. Although other programs don't focus on parenting trainings they still may involve the parent as in family talks (FTI, Beardslee et al., 1997) or have psycho-educative sessions for parents accompanying the children group sessions(CWD, Clarke, Hornbrook, Lynch, et al., et al., 2001). Targeting the challenges of being a parent suffering from depressive episodes, some prevention programs try to establish a positive parenting style. Positive parenting includes a warm and accepting base, enhancement of family cohesion, praise, positive reinforcement, social support and open communication. Furthermore, a certain structure and family rules are often implemented.

4.2.4. Settings

Most prevention programs take place in a group setting. A group can be a context, which enables individuals to share experience in a protected environment (Gundelfinger, 1997). Feeling understood by the group members, that might find themselves in a similar situation, can be a great relief. Children – as well as parents - might realize that they are not alone in

their situation and other children or families gain the exact same experiences (Yalom, 1989). Usually children and adolescent have constraints talking about specific strained experiences and feelings concerning the life with a sick parent (Gundelfinger, 1997). They look out for peers in a comparable situation to solidarize and feel as part of one group. Still, they might prefer to talk about heavily loaded themes in single settings or with their family (Gundelfinger, 1997). Some prevention programs therefore focus on peer group and exclude parents in the active sessions (Clarke, Hornbrook, Lynch, et al., et al., 2001). Like this it might be easier for most of the children to open up about their fears, sorrows, as well as feelings of helplessness, shame, anger and guilt. Family settings can open the dialogue on both sides: parent and children, in order to prevent fears and distrust and liberate transparency (Lenz, 2005). Families might learn to express their feelings more openly and clearly, children can learn to dare to ask questions (e.g. about the depression) and express their needs. Furthermore, a family is usually constraint with many dysfunctional patterns of interaction that can be resolved the best when all members of the family are joining the intervention (Gundelfinger, 1997). Wiedermann and Buckremer (1996) came to the conclusion, that a family setting is efficient especially for communication problems and problem-solving strategies(Wunderlich, Wiedemann, & Buchkremer, 1996). Therefore, some prevention interventions are based mainly on family communication (Beardslee et al., 1997; Mason, Haggerty, Fleming, & Casey-Goldtein, 2012).

4.2.5. Communication

Communication can be interpreted as the base of social functioning (Lenz, 2005). Dysfunctional communication patterns are a significant factor for a tense and conflictual climate (Henggeler & Borduin, 1990). Therefore many family therapeutic interventions focus on communicational aspects (Beardslee et al., 1997; Compas et al., 2009; Mason, Haggerty, Fleming, & Casey-Goldtein, 2012). The aim is to replace dysfunctional and problematic style

of communication with more helpful ways as speaker as well as listener. The basic attitude requires honest interest, acceptance, esteem and honesty. The focus of the communication lies on relevant contents as feelings, needs, wishes and perceptions (Henggeler & Borduin, 1990). These trainings usually make use of role-plays to establish this positive way of communication. The commonly existing taboo about depression in families challenges the open dialogue about the disease but therefore is even more significant. Exchanging thoughts and talking about feelings may benefit to a stable structure of communication (Stieglitz, 2002). Most of the prevention programs that were implemented so far rely on the enhancement of communication within the families (Beardslee et al., 1997; Compas et al., 2011) .

4.2.6. Summary

Five basic ingredients that are performed differently in existing prevention programs were constituted. Psycho-education of the parental illness is an indisputable important and the most commonly used content in the field of prevention of depression in the offspring of depressed parents. Since positive coping strategies of children and adolescents are often impaired and linked to the development of depression (see section 2.3.), many prevention programs target therapeutic techniques in order to facilitate more adaptive coping strategies for children and adolescents. Due to negative consequences of poor parenting skills and parent-child interactions, an important ingredient of depression prevention is improving the parenting style and the communication skills. Most prevention programs rely on the beneficial aspects of group setting, whereas they differ whether they include children and parents simultaneously. Furthermore, many interventions focus on communicational aspects in order to replace dysfunctional style of communication with more helpful ways as speaker as well as listener.

4.3. Selected prevention programs

Most of the intervention programs focusing on children of depressed parents were developed and evaluated in the U.S..

4.3.1. International prevention programs

Five interventions have been developed to prevent depression in the offspring of depressed parents and have been evaluated through randomized controlled trials (RCTs): i) *Family Talk intervention* (FTI, Beardslee et al., 1997), ii) *Project Hope* (PH, Mason et al., 2012) iii) *Coping with Depression* (CWD, Clarke, Hornbrook, Lynch, Polen, et al., 2001), iv) *Raising Healthy Children* (RHC, Compas et al., 2009) and v) *Parenting Training* (PT, Sanford et al., 2003b). These interventions aim to reduce depression risk by improving knowledge of depression within the family and building resilience to stress in parents and/or children. All programs have been developed in the U.S. and Canada but were evaluated also in other countries as i.e. the FTI in Finland (Punamäki, Paavonen, Toikka, & Solantaus, 2013; Tytti Solantaus, Paavonen, Toikka, & Punamäki, 2010) or Germany (Christiansen, Anding, Schrott, & Röhrle, 2015). They all take place across multiple sessions in a face-to-face, group-based setting.

However, they differ in the extent to which they involve psycho-education versus cognitive-behavioural therapy (CBT). Furthermore, they differ with regard to the family members who are involved (parents and/or children), the age range of children included, and the length of the intervention. Some studies also included children with a history of depression so that the presented programs mix in primary prevention and tertiary prevention trials (Beardslee et al., 1997; Beardslee, Gladstone, Wright, & Cooper, 2003; Clarke et al., 2001; Compas et al., 2010, 2011). In the following paragraph, the five programs and their effects on preventing depression are reported.

4.4.1.1. Family Talk Intervention (Beardslee et al., 1997)

The program is based on family systems therapy and has its main focus on psycho-education and family communication, rather than on CBT. The clinician-facilitated intervention contains sessions for the entire family as well as individual parent and child sessions. The program is designed for children aged 8-15 years and consists of 6-11 sessions with refresher meetings or telephone contacts 6-9 months after the final intervention session. Clinicians discuss common experiences of depression as well as concerns about and functioning of the offspring. Parents are encouraged to initiate a dialogue about depression within the family in order to discuss how the family could cope better with depression.

In the original trial, 52 children aged eight to fifteen years were randomised to either the experimental group (FTI) or a lecture control group (Beardslee et al., 1997; Beardslee et al., 2003; Beardslee et al., 2007). The control group consisted of two sessions for parents, providing general (non-personalised) information about parental depression and how to support children. Here, both groups showed improvements in communication skills and understanding their parents' depression at the post-assessment and 18-month follow-up. Nevertheless, these improvements were greater in the experimental than the control group ($F_{1,49} = 3.91$, $p < .05$ and $F_{1,48} = 11.62$, $p < .001$ respectively) (Beardslee et al., 1997). The latest publication of this trial reported the 4.5 year follow-up from baseline in which a sample of n = 122 children remained (Beardslee et al., 2007). Here, children's change of understanding of parental illness over time was significant ($\chi^2(3) = 9.0$, $p < .05$), as were children's internalizing symptoms ($\chi^2(1) = 9.0$, $p < .001$), but did not differ between groups. In a replication of this study in Finland with 149 children, there was a significantly greater reduction in emotional symptoms in the intervention (versus control) group at four-month follow-up ($p = .040$) when the parent's depressiveness at baseline and its change over time was controlled. There was no evidence of group differences in internalizing or depressive

symptoms at 10- and 18-month follow-up (Punamäki et al., 2013; Solantaus et al., 2010; Solantaus, Toikka, Alasuutari, Beardslee, & Paavonen, 2009).

4.4.1.2. Project Hope (Mason et al., 2012)

Another intervention that focuses on psycho-education and communication aspects is Project Hope (PH) (Mason et al., 2012). PH is based not only on depression prevention (inspired by the FTI intervention) (Beardslee et al., 2003) but includes aspects related to the prevention of substance abuse in addition (from the "Family Matters" program) (Bauman, Foshee, Ennett, Hicks, & Pemberton, 2001). The main aims are to strengthen parenting and family relationships and increase youth resilience. The ten weekly sessions for parents and their children aged 12-15 years provide information about depression and substance abuse, as well as changing problematic attitudes towards these issues and enhancing family communication. Furthermore, the family's self-efficacy is promoted. In order to deal with influences from peers and media, family rules and norms surrounding substance use, refusal skills and anti-substance attitudes are taught.

Mason and colleagues (2012) evaluated their program in a randomized controlled trial including N = 30 families, consisting of a parent suffering from depression and one child. Families were randomized to either the intervention group or a waiting list control group. Assessment was conducted at baseline, four and nine months after baseline measuring several parent and child relevant outcome variables (parents: depressive symptoms, parenting skills; youth: depressive symptoms, substance use beliefs, substance use count, coping). Across time, adolescents in the intervention group showed less consumption of alcohol than those in the control group ($F_{1, 21} = 6.5$, $p = .019$). Although there was some evidence from parent reports of improved communication about depression in the intervention vs. control group, this pattern was not evident across all related variables. There was no evidence of a beneficial effect of the intervention on child-reported depressive symptoms ($F_{2,40} = 0.63$, $p = 0.539$).

4.4.1.3. Coping with Depression (Clarke, Hornbrook, Lynch, Polen, et al., 2001; Lewinsohn & Clarke, 1999)

In contrast to the FTI and PH, the CWD is a modified version of a CBT treatment manual for adolescents and therefore focuses on CBT techniques such as cognitive restructuring, interpersonal problem-solving and communication. Children built the focus group of the intervention (rather than their parents). Up to ten adolescents aged 13 to 18 years participate in 15 sessions over four months. Separate psycho-educational sessions for parents are conducted at three time points (baseline, middle and end of the intervention). In these sessions parents receive information about the skills that the offspring have learned and themes that have been discussed during adolescent sessions. Parenting strategies and personal concerns are not discussed in these sessions.

Similarly to the FTI, the CWD has been investigated in more than one trial (both conducted in the U.S.A.). Clarke, Hornbrook, and Lynch (2001) randomised 104 13-18 year old adolescents with sub-clinical depressive symptoms to the intervention or a usual care control group. The authors report significant positive effects of the intervention (versus usual care) on child-reported depressive symptoms, but not parent-reported depressive symptoms (CBCL). In the experimental (versus control) group significantly less children were depressed at 12-month follow-up (9.3 % vs. 28.8 %; $p = 0.003$). At 18- and 24-month follow-up, these effects remained but had diminished. The time to onset of depression was significantly longer in the experimental group compared to the control group ($t_{19} = 2.90$; $p = .009$).

The effects of the CWD intervention were then tested in another larger trial over a six year period (Beardslee et al., 2013; Brent et al., 2015; Garber et al., 2009) . In this study, 316 adolescents aged 13-17 who i) had a history of depression or ii) showed elevated depressive symptoms were randomized to either the intervention group or usual care. The hazard ratio

(HR) and rate for onset of depression was significantly lower in the intervention group than the control group at the 9-month follow-up [21.4% vs. 32.7%; hazard ratio = 0.63, 95%; CI (0.40;0.98), p = .03] (Garber et al., 2009). In addition, this was reflected in a significant interaction of time and condition for change in depressive symptoms (coefficient, -1.10: z = -2.22: p = .03). At the 33- month follow-up, participants in the intervention group also developed less frequently a depressive episode than those in the control group [36.8 % vs. 47.7 %; NNTB = 10; 95% CI (5;2624)] (Beardslee et al., 2013). This difference was only significant for children whose parents did not have an acute episode of depression at baseline. Change scores of depressive symptoms were not significant at the 33-month follow-up. The CWD is the only intervention to have been evaluated for effectiveness at six-year follow-up (Brent et al., 2015). Based on the 278 (of the initial 316) participants assessed at this time point, there were significant positive effects of the intervention on the reduction of onset of depression, again only when they controlled for paternal depression at baseline [hazard ratio = 0.71, 95% CI (0.53;0.96)].

4.4.1.4. Raising Healthy Children (Compas et al., 2009)

This intervention combines elements of the previously described programs. RHC is based on psycho-education and CBT elements such as coping strategies for children (similarly to CWD), but also actively involves both children and their parents (similarly to FTI). In the eight weekly and four monthly booster sessions families with children and adolescents aged 9-15 years of age learn theoretical contents and are encouraged to practice those individually and in the family setting. In the first three sessions all participating family members cooperate as one group all together, whereas children and parents are separated into different rooms in the following sessions.

The intervention was evaluated with n = 188 families with children aged 9 to 15 that were randomised to receive either the intervention or a written-information control group

(Compas et al., 2009, 2011, 2015) and followed them up over 24 months. The incidence of depression at the combined 6- and 12-month follow-up was lower in the experimental group (8.9 %) versus the control group (20.8 %), although this difference was not statistically significant ($\chi^2(3) = 3.04$, $p = .070$) (Compas et al., 2009). Major group effects of the intervention were further displayed in self-reported anxiety/depression and internalizing symptoms scores with increasing effects from post-intervention to 12-month follow-up (YSR; d = 0.31-0.57). Surprisingly, no significant group differences over time were found on the parent-rated measure of children's psychopathology (CBCL). In the most recent publication of this trial, Compas and colleagues (2015) clustered all 242 participating children (i.e. including siblings) into one statistical analysis and reported data across the study period. The positive effect of the intervention (versus control) on incidence of depression was significant at 24-month follow-up (13.1 % vs. 26.3 %, $\chi^2(1) = 4.46$, $p = .035$) (Compas et al., 2015). Interestingly, in this publication most self-reported outcome variables did not differ significantly at the 2-month follow-up assessment but emerged at the 12-month follow-up and remained stable at the 18- month follow-up. Some effects diminished at the 24-month follow-up (internalizing symptoms YSR), but not all of them (depressive symptoms: CES-D, YSR). Again, the parent-rated child psychopathology (CBCL) did not appear to change over time between the groups. For externalizing symptoms, the authors did not find significant effects for conditions. Besides, Compas and colleagues observed significant positive effects of the intervention on parental depressive symptoms at all assessment time points (d = 0.49 - d = 0.26).

4.4.1.5. Parenting Training (Sanford et al., 2003b)

In contrast to former programs that involve children, this program only actively includes the depressed parent of children aged six to thirteen years, taking part in eight weekly sessions. The aim of the program is to deliver information and strategies to parents, which are then

indirectly transferred to the offspring by changes of parental attitude and behaviour (Sanford et al., 2003b). The program is based on psycho-education about family topics and parenting training. Information is provided to foster communication as well as family problem-solving and coping skills. The parenting training was originally designed for parents with children with behavioural disorders (rather than the non-depressed children of depressed parents). It contains concepts and methods derived from social-learning theory (coping-modelling procedures), parent-education theory (cognitive strategies, contingency-management) and family-system theory (family-problem solving, supportive communication).

In the only RCT of the intervention, 44 parents were randomised to the intervention or a waiting-list control group (Sanford et al., 2003b). The authors report significant effects favouring the intervention on family functioning (F_{31} = 7.6, p = 0.01) and non-significant trends on the family conflict scale (F_{31} =3.5, p = 0.07) parenting sense of competence (F_{30} =3.7, p = 0.06) with medium-size effects (d = .40 - .60). The children's depressive symptoms did not differ between conditions. The PT is yet to be evaluated outside of the initial research group.

4.3.2. Prevention interventions in Germany

In Germany there is little research done on selective or indicative prevention and no program has yet been evaluated in a randomized controlled trial that focused on the offspring of depressed parents. Nevertheless, also in Germany research groups start to focus on the high risk of children of mentally ill parents. For example, the *Children of Mentally Ill Parents* program (CHIMPS, Wiegand-Grefe, Werkmeister, Bullinger, Plass, & Petermann, 2012) does include parents with all kind of psychological illness and their children aged two to eighteen years. The program rather focuses on social support and disease coping and consists of twelve to sixteen family or single sessions. 67 children were randomised to the intervention or a

waiting control group. Quality of life was increased in the experimental group (d = 0.46) and social support increased (d = 0.30). Criticism on the study relate to limited reported methodology. In addition, the scale of "social support" only consisted of three items.

Another quasi experimental trial was conducted in a mother-child ward in the south of Germany. The Program EFFEKT-E (Bühler, Kötter, Stemmler, Jaursch, & Lösel, 2013) was offered to 406 mothers suffering from depression during their clinic stay. The intervention is a six-session mother-child-oriented program targeting positive parenting as well as the children's social competence. The authors report decreased perceived parental stress and parental competence (d = .72) as well as less emotional disturbance of the child (d = .52), whereas no differences in social competences of the child were to be seen. Still, these findings need to be interpreted with caution due to the non-existence of randomisation. Furthermore, 60 % of the patients the program was offered to, did refuse to take part. As mentioned before, another approach in Germany was done by Christiansen and colleagues (2015) replicating the FTI in a modified version, in a controlled trial indicating high effects in the decreases of psychopathological symptoms (d = 1.45) (Christiansen et al., 2015).

4.4. Efficiency of prevention programs of depression

In the last 20 years, an increasing number of prevention programs for depression emerged, going along with more studies evaluation their efficacy. Due to that fact, reviews and meta-analysis on both, universal and selective prevention programs for depression, were conducted. Existing reviews and meta-analyses suggest that to a certain degree youth depression can be prevented (Hetrick et al., 2016; Stockings et al., 2016). For example, in a recent Cochrane meta-analysis of 83 prevention interventions for children and adolescents, small but significant effects of interventions were found on depressive symptoms up to, but not beyond, 12 months (Hetrick et al., 2016). The estimated numbers needed to treat to benefit (NNTB) was 11, which is comparable with other public health interventions. The meta-analysis also

indicates that some approaches to the prevention of youth depression may be more effective than others.

Most authors found discriminant effects concerning type of prevention: universal or indicative and selective programs. Concerning universal programs, effects are rather heterogeneous: Calear and Christensen (Calear & Christensen, 2010) report data about mainly school based, universal programs and found effect sizes in a range of *d'* = .21 and *d'* = 1.4. Reasons for these mixed findings might be the varying quality of the included studies as well as duration, intensity and study design. Furthermore, individuals that don't face any risk or need for prevention might benefit less than high-risk population.

Schulte-Körne and Schiller (2012) focused on the efficacy of universal and selective or indicated prevention programs of depression (Schulte-Körne & Schiller, 2012). The authors report in their review an overall significant effect of prevention programs of depression for the reduction of depressive symptoms in short and long term for selective prevention interventions. The long-term (beyond a 24- month follow-up assessment) effects were no longer significant in both types of prevention. Selective and indicated approaches (together known as 'targeted' approaches) were found to be more efficient than universal approaches. These effects were confirmed by the recent Cochrance Review (Hetrick et al., 2016). This effect might be caused due to the fact that effect sizes in targeted (versus universal) interventions may in part be the result of including a non-active control group (Hetrick et al., 2016). Stocking and colleagues (2015) also reviewed multiple selective programs for preventing depression (and anxiety) in young people and found positive effect sizes of 0.29 and 0.34 at immediate and 6-9 month follow-up respectively (compared to 123 no-intervention control groups, 23 active control groups). At the follow-up assessments effect sizes thereby show a greater variability but still are significant with small effects (d = .18-.29) for the nine-month follow-up assessment and small effects at the twelve-month follow-up

assessment (Calear & Christensen, 2010; Horowitz & Garber, 2006). Effects further diminish at the 24-month follow-up.

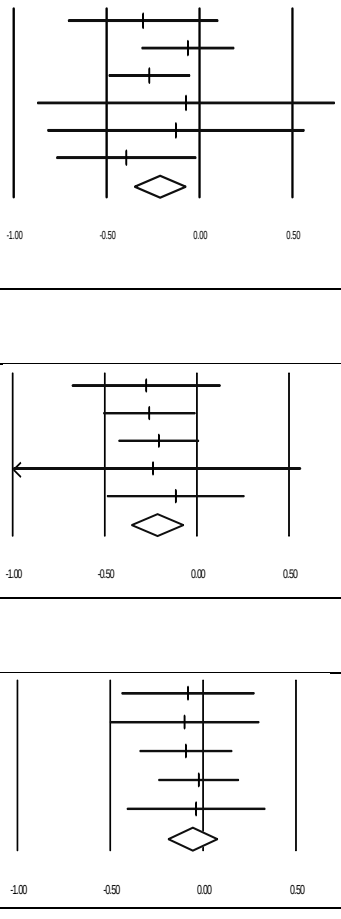
4.4.1. Meta-analytical findings of prevention trials for the offspring of depressed parents

Since there was no review on prevention programs for the high-risk group of the offspring of depressed parents, we recently performed the first systematic review and meta-analysis of prevention programs for children of depressed parents (Loechner et al., n.d.). Here, the main outcomes of the meta-analysis are summarised. Treatment efficiency on depressive and internalizing symptoms as well as incidence of depression of the child was determined at post-assessment, intermediate follow-up (up to 12 months post-intervention), and long-term (15-72 months post-intervention). A systematic literature research resulted in 14 publications from seven independent RCTs (n = 935 children, aged 6-18) that were based on five different types of intervention (see section 4.3.1. for a detailed description of the single programs). All interventions aimed to reduce depression risk by improving knowledge of depression within the family and building resilience to stress in parents and/or children. They all took place across multiple sessions in a face-to-face group-based setting. However, they differed in the extent to which they involved psycho-education versus CBT. Furthermore, they varied with regard to the family members who are involved (parents and/or children), the age range of participating children, and the length of the intervention. The included studies were conducted with high methodological quality and we only found a small overall risk of bias. There was evidence that the interventions had a positive effect on depressive symptoms immediately after the intervention [*d*' = -0.22; 95% CI (-0.36;-0.08) *p* = .002], an effect which remained significant at short-term (up to 12 months) follow-up effect [*d*' = -0.22, range -0.11 to -0.28; 95% CI (-0.36;-0.08) *p* =.002]. There was no evidence of long-term effects of the intervention beyond 12 months. Nevertheless, some studies that investigated moderator variables found

significant intervention effects at long-term, when parents were not currently depressed at baseline (e.g. Brent et al., 2015). Other moderators as for example children's ages, gender, parental education or symptoms of anxiety in the child were less persuasive, but also less investigated. Table 3 provides effects sizes at post-assessment, short- and long-term follow-up.

Table 3 Effect sizes based on depressive (and internalizing) symptoms at post-intervention, short-term and long-term follow-up

Study or Subgroup			95 % CI			Residual (random)		Std. diff. in means and 95% CI
Post assessment	intervention	*d*	Lower	Upper	Var.	Std. residual	Relative weight	
Clarke 2001	CWD	-0.30	-0.71	0.10	0.04	-0.44	11.45	
Compas 2009/2011/2015	RHC	-0.06	-0. 31	0.19	0.02	1.43	29.85	
Garber 2009/Beardslee 2013/Brent2015	CWD	-0.27	-0.49	-0.05	0.01	-0.60	38.65	
Mason 2012	PH	-0.07	-0.89	0.73	0.17	0.36	2.94	
Sanford 2003	PT	-0.13	-0.82	0.57	0.13	0.26	3.93	
Solantaus 2010/Punamäki 2013	FTI	-0.40	-0.77	-0.01	0.04	-0.98	13.19	
overall		-0.22	-0.36	-0.08	0.01			
Short-term follow -up								
Clarke 2001	CWD	-0.28	-0.68	0.13	0.04	-0.29	11.92	
Compas 2009/2011/2015	RHC	-0.26	-0.51	-0.01	0.02	-0.36	30.78	
Garber2009/Beardslee2013/Brent2015	CWD	-0.21	-0.43	0.01	0.01	0.14	40.31	
Mason 2012	PT	-0.24	-1.04	0.57	0.17	-0.05	3.04	
Solantaus 2010/Punamäki 2013	FTI	-0.11	-0.49	0.26	0.04	0.59	13.95	
overall		-0.22	-0.36	-0.08	0.05			
Long-term follow- up								
Beardslee 1997/2003/2007	FTI	-0.08	-0.44	0.28	0.03	-0.12	13.38	
Clarke 2001	CWD	-0.10	-0.50	0.30	0.04	-0.20	10.65	
Compas 2009/2011/2015	RHC	-0.93	-0.34	0.16	0.02	-0.30	27.47	
Garber2009/Beardslee2013/Brent2015	CWD	-0.02	-0.24	0.20	0.01	0.41	36.13	
Solantaus 2010/Punamäki 2013	FTI	-0.04	-0.41	0.34	0.04	0.13	12.37	
overall		-0.06	-0.19	0.07	0.05			



Note: d - cohen's d; CI - confidence interval; lower - lower limit; upper - upper limit; var - variance, std. residual - standardized residual, std. diff. - standard differences; depr. symp. - depressive symptoms, int. symp. - internalizing symptoms. CWD – Coping with Depression, RHC – Rasing Healthy Children, PT – Parenting Training, PH – Project Hope, FTI – Family Talk Intervention.

In addition, we calculated the effects of the interventions on incidence of depression and found small to medium effects incidence [Risk Ratio = 0.56; 95 % CI (0.41;0.77); *d*' = -.42, NNTB = 4.28]. Unfortunately, this clinical highly relevant measure was only reported by four studies. Nevertheless, this measure indicates that depression can be indeed prevented for some children in this high-risk group. In this work we pointed out that research is still limited in number and cultural contribution. Further research is needed that focuses on moderators and mediators in order to replicate these findings and increases preventive effects. No significant differences in other subgroup analysis like effects of intervention (or control group) type or type of control group were found, although effects sizes differed. In other studies, effect sizes were found to be smaller or non-significant in study designs with an active control group (Merry et al., 2011). Another important factor might be the qualification of the group leader: Clinically trained group leaders might increase intervention efficacy (Calear & Christensen, 2010; Stice, Shaw, Bohon, & Marti, 2010).

In summary, there is evidence of efficiency of prevention interventions favouring indicated and selective interventions. Effect sizes range from moderate to small and diminish over time. Similar effects were found for the prevention of depression in the offspring of depressed parents. Since this work is about the offspring of depressed parents, the following sections focus on this specific high-risk group.

4.4.2. Conclusion

There are five prevention programs for the high-risk group of the offspring of depressed parents that were evaluated by a randomized controlled trial: i) *Family Talk intervention* (FTI, Beardslee et al., 1997), ii) *Project Hope* (PH, Mason et al., 2012) iii) *Coping with Depression* (CWD, Clarke, Hornbrook, Lynch, Polen, et al., 2001), iv) *Raising Healthy Children* (RHC, Compas et al., 2009) and v) *Parenting Training* (PT, Sanford et al.,

2003b). All of them were developed and evaluated in America (U.S. and Canada). Although some programs show promising effects, especially on the incidence of depression (Compas et al., 2015; Garber et al., 2009) only the FTI was replicated outside the research group in Europe, (Punamäki et al., 2013; Solantaus et al., 2010).

The presented prevention programs differ in numerous aspects as targeted group (parents, children or family) or focus on CBT (Garber et al., 2009) vs. psycho-education and family communication (Beardslee et al., 1997). Nevertheless, especially the RHC (Compas et al., 2009) manages to include many ingredients that were discussed to be helpful: The program contains i) psycho-education, ii) CBT-techniques for improving emotional and cognitive coping strategies, iii) parenting training in a iv) family-, parents-, child- and group setting. In addition, results on the reduction of internalizing, externalizing and depressive symptoms are very promising (d = -.42 at short-term follow up on depressive symptoms). Especially the rates of onset of depression at the 24- month follow-up were impressive with 14 % incidence of depression in the experimental group versus 33 % onset of depression in the control group. Since long-term effects on this clinically highly relevant outcome measure are rare, this program appears to be most promising. Surprisingly, this intervention has never been replicated by an independent research group. In Germany, there are only few attempts in the field of prevention research. Consequently, more research in Germany on prevention of depression in the offspring of depressed parents is needed.

4.5. Summary

Prevention interventions can be distinguished concerning an individual's symptomology (primary, secondary and tertiary prevention) or the targeted group (selective and/or indicated prevention or universal prevention) (Mrazek & Haggerty, 1994). Prevention programs for depression in general are often focused and were found to be efficient (Hetrick et al., 2015; Stockings et al., 2016), few research groups focused on the high-risk group of

children of depressed parents. Although the number of conducted trials is limited, those interventions differ greatly on the included ingredients (e.g. psycho-education, parenting training, setting). In a recent systematic review and meta-analysis on RCTs in the field of depression prevention for this high-risk group we identified five different interventions showing small to moderate effects on the children's depressive and internalizing symptoms and onset of depression (Loechner & Starman et al., n.d.). Most of the studies were never replicated outside the research groups and were mostly conducted in the U.S.. The RHC (Compas et al., 2009) appeared to be especially promising, but was never replicated. In Germany, there is little research in the field of depression prevention, especially on RCTs.

5. Summary of Literature Review

As shown in the first part of this work, depression is one of the most common psychiatric disorders (WHO, 2004) causing great personal and economic burden (Mathers, Fat, & Boerma, 2008). In adolescence, prevalence rates rise dramatically, but are prevailing in children already (Dietz et al., 2015). Especially children of depressed parents face an increased risk to develop a depression themselves (Weissman et al., 2006). In general, the onset of a depressive disorder underlies multifactorial processes (Nickel, et al., 2009) and is therefore influenced by a wide range of malleable risk and protective factors – which include biological, familiar, psychological, societal and social conditions (WHO, 2004). The *diathesis–stress* model or *vulnerability-stress* model is a paradigm for understanding how these factors interact in the development and maintenance of depression(B L Hankin et al., 1998). Since core symptoms in child and adolescent depression are anhedonia, loss of interest and low self- confidence, especially emotional and cognitive factors were detected to play a key role in the development and maintenance of depression (Braet et al., 2015; J L Horowitz et al., 2007; Schäfer et al., 2016). Although there is evidence-based treatment – psychotherapy and antidepressants (WHO, 2015; Zhou et al., 2015) – the prognosis for early onset is poor, access to treatment is limited and expensive.

One of the biggest risk factors for developing a depression is having a parent with depression (Weissman, et al., 2006). In Germany approximately 3.8 million children and adolescents grow up with a parent who currently suffers or has suffered from a depressive disorder (Plass & Wiegand-Grefe, 2012, Statistisches Bundesamt, 2006). For the the offspring of these parents, the risk to develop a depressive disorder is estimated to be three to four times higher than for the offspring of non-depressed parents (Weissman et al., 2006; Weissman, 1997). Furthermore, for early onset of depression, the prognosis is often more chronic and severe than for later incidence of the disease. In order to understand the heightened risk in this

group, two models of transition of depression (Goodman & Gotlib, 1999) and mental illness in general (Hosman, 2009) were presented and updated with findings of the current research. Those models integrate biological and psychosocial aspects within a transactional perspective in order to uncover the mediation and moderation roles of important factors between the effects of parent's depression on their children. A major criticism of both models is that they aim to explain the trans-generational transmission of depression but only rely on single findings that are included in one model. Furthermore, evidence on experimental studies is neglected. Although there is a consistent body of literature explaining the specific risk of depression in the offspring of depressed parents, no study included several prevalent risk factors simultaneously.

The World Health Organisation (WHO) stated, that prevention of youth depression is a public health priority (WHO, 2004). Consequently, numerous depression prevention programs emerged in the last decades. Meta-analytical findings suggest that prevention interventions that target high-risk groups are more effective than those universally administered to all youth (Hetrick et al., 2015). As shown earlier, one of those high-risk groups are children of parents with depression (Weissman et al., 2006). A systematic review and meta-analysis of RCTs to prevent depression in the children of parents with depression, conducted by colleagues and myself, showed small to moderate but significant effects in the reduction of the incidence of depression (*d'* = 0.42) (Loechner & Starman, et al., underreview). Research in this field has been dominated by interventions developed and evaluated in the U.S.. Five different prevention programs that focus on the offspring of depressed parents were evaluated in RCTs. One of the most promising interventions, which delivers CBT in a family- and group-based setting, is yet to be replicated outside of the original research group - the "Raising Healthy Children (RHC)" program (Compas et al.,

2015). In Germany, there is little research done in the field of depression prevention for the offspring of depression.

Consequently, the following two studies investigate the transmission and prevention of depression. *Study I* intends to replicate earlier findings on the increased risk for depression in the offspring of depressed parents. In addition, numerous risk factors and their impact on children's depressive symptoms are explored (emotional and cognitive factors, stressful life events). Furthermore, the significance of the parental depression for developing a major depression is investigated.

In *study II*, preliminary results of the first replication of the translated and culturally adapted prevention program "Raising Healthy Children" (Compas et al., 2009) for the offspring of parents with depression in Germany are presented.

Study I

Transmission of depression in the offspring of depressed parents

6. Introduction study I

6.1. Theoretical Background

As shown earlier, the offspring of parents with depression represent a specific high-risk group (Weissman, et al., 2006). Children and adolescents that are growing up with a parent suffering from major depression were found to be three to four times more likely to develop a major depression than children of non-depressed parents (Weissman et al., 2006). Numerous studies were depicting abnormalities in their psychopathological development and an increased risk of increased psychopathological symptoms and mental illnesses (Heitmann & Bauer, 2007; Ihle & Esser, 2002; Weissman et al., 2006; Weissman et al., 1997). The *model of transition, Goodman and Gotlib* (1999) summarized numerous risk factors, aiming to explain the heightened risk of depression and the trans-generational transfer of depression (Goodman & Gotlib, 1999). It was shown that the reported findings of risk factors in the model are mostly still up-to-date and were extended by the current research. For example the evidence about biological predisposition was confirmed in many studies (e.g. Meyer, Chrousos, & Gold, 2001; Smart, Strathdee, Watson, Murgatroyd, & McAllister-Williams, 2015). Nevertheless, some important risk factors that were shown to be related to depression, have poorly been addressed in the model. Although Goodman and Gotlib (1999) state, that children inherit or might adapt through model learning a "depressogenic style" of their mothers (concerning cognitive, emotional and behavioural factors), no empirical evidence has underlined this hypothesis yet. More recently, findings of association of cognitive and emotional factors of depression in general were investigated in order to explore the specific role in development and maintenance of depression (see section 2.3.) (Abela & Hankin, 2008; Auerbach et al., 2014; Braet et al., 2015; Mathews & Macleod, 2005; Schäfer et al., 2016). This is surprising since those factors are modifiable and constitute the base in evidence-based treatment and prevention of depression (Zhou et al., 2015; Clarke, Hornbrook, Lynch, et al., et al., 2001).

For example Braet and colleagues (Braet et al., 2015) found that the cognitive triad (negative self-evaluation, a pessimistic world view and hopelessness regarding the future) significantly predicted depressive symptoms and accounted for 43.5 % of the variance in depressive symptoms of n = 171 children and adolescents. The authors interpreted this finding as a marker of depressive symptoms, since participants were not suffering from major depression. Nevertheless, the data is cross-sectional and predictions about future diagnosis cannot be made. Confirming this, Joiner and Wagner (Joiner & Wagner, 1995) reported moderate support for overall negative attributional style as prospective predictor in their meta-analysis on depressive symptoms in children. Unfortunately, the authors did not investigate the offspring of depressed parents. In contrast, one promising study interrogated long-term effects of cognitive vulnerability to depression in n = 205 seven year old children of parents with major depression (Hayden et al., 2014). In one to two one-year intervals the authors measured the maternal affective style and the children's cognitive vulnerability in an experimental task as well as a self-rating questionnaire in order to test their attributional style. They found that a negative cognitive style was prospectively and concurrently associated with depressive symptoms of the children with modest stability. In addition, the parental affect was correlated to this cognitive style. Hayden and colleagues (2014) discussed whether higher rates of maternal criticism caused this cognitive vulnerability or whether children with this predisposition elicit more paternal criticism. Furthermore, the effect of paternal depression on the children's cognitive style might be a mediator of the risk of depression. In the sample only 33 % of mothers and 17 % of fathers had a lifetime history of major depression. Therefore, results cannot be generalized for the population of the offspring of depressed parents. Unfortunately, the sample was not divided into two groups (children with parents with depression and without) in order to explore differences in the outcome variables. In another study, Horowitz and colleagues (2007) explored the attributional style in adolescents that were taking part in a randomized controlled trial where the authors compared two prevention

interventions for depression with a no-intervention control group (Horowitz, Garber, Ciesla, Young, & Mufson, 2007). They found attributional style to be associated with the depressive symptoms of the adolescents. Beyond that, the attributional style mediated the effect of the intervention on depressive symptoms.

Another important factor that is related to the development and maintenance of depression is referring to emotion regulation strategies (Schäfer et al., 2016). Ehring and colleagues (2010) found that dysfunctional use of emotion regulation strategies (e.g. suppression of emotion) are linked to depression vulnerability (Ehring, Tuschen-Caffier, Schnülle, Fischer, & Gross, 2010). Again, this study is cross- sectional and therefore limited to causal attributions. Nevertheless, those findings were confirmed in a longitudinal study investigating the predictive value of maladaptive and adaptive emotion regulation strategies for psychopathological symptoms in a relatively big sample of n = 1.317 (Aldao & Nolen-Hoeksema, 2012). Here, adaptive strategies only had a negative association with psychopathology symptoms in case of high levers of maladaptive strategies. Both samples were community samples without a predefined risk of depression. Although there are many studies (e.g. Corinna Reck, Nonnenmacher, & Zietlow, 2016; Zietlow, Schlüter, Nonnenmacher, Müller, & Reck, 2014) on mothers suffering from post-natal depression and emotion-related factors in children, those mostly refer to the resulting attachment style, but not to emotion regulation strategies. One study focused on n = 45 children aged four to seven of mothers suffering from depression and n = 33 children of never depressed mothers and identified emotion regulation strategies as moderating factor of maternal depression and children's internalizing symptoms and discuss positive emotion regulation strategies as protective factor (Silk, Shaw, Forbes, Lane, & Kovacs, 2006b). In this study, emotion regulation strategies only were conceptualized as 1) negative focus on delay, 2) positive reward anticipation, and 3) behavioural distraction and therefore don't cover the earlier

described range of possible strategies. In addition, emotion regulation strategies are often discussed as mechanisms or mediators, but not moderators (Compas et al., 2010; Schäfer et al., 2016).

Stressful life events in an individual's life are constituting another important factor in the development of mental illness. As shown earlier in the *diathesis-stress model* (section 2.1.) stressful life events might trigger a certain vulnerability and provoke the incidence of depression (Colodro-Conde et al., 2017). The offspring of depressed parents are more likely to experience negative life events, due to environmental circumstances that might have caused the parental depression in the first place (Monroe, Slavich, Torres, & Gotlib, 2007; Pound et al., 1988). A recent study investigated the effects of child-experienced parenting and peer stressors on the development of depression in adolescents (n = 275) (Oppenheimer, Hankin, & Young, 2017). In this longitudinal study a negative impact of low levels of observed positive parenting was associated with an increased likelihood of the occurrence of an episode of major depression, but only for adolescents who simultaneously experienced a high amount of peer stressors. A cross-sectional study confirmed this finding in a sample of the offspring of depressed parents (Jaser et al., 2005). Here, children's symptoms of depression and anxiety were linked to peer and family stressors, but partially mediated by dysfunctional coping strategies. The occurrence of stressful life events and its impact on an individual's well-being raises the question of coping strategies. Coping strategies are defined as "conscious volitional efforts to regulate emotion, cognition, behavior, physiology, and the environment in response to stressful events or circumstances" (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001, p. 89). Numerous studies investigated the mediating effect of coping strategies between stressful life events and psychopathological symptoms (e.g. Aldao & Nolen-Hoeksema, 2012; Compas et al., 2001). Again, the number of researchers that focused on the high-risk group of children of parents with depression is limited. One longitudinal

study aimed to examine whether coping strategies mediate the effect of stressful life events on depressive symptoms among children (7-17 years) with parents with (n = 129, high-risk group) and without depression (n = 98, low-risk group) (Evans et al., 2015). Here, stressful life events, symptoms of depression and coping strategies were measured at four time points over 22 months. The authors tested structural equation models, indicating that stressful life events significantly predicted children's depressive symptoms over time. In addition, there was a mediating effect of some coping strategies (primary control coping and disengagement coping) linking the effect between life events and depressive symptoms. There were small but significant correlations between secondary coping strategies (e.g. emotion regulation strategies) and stressful life events with the depressive symptoms in the child among all time points. Furthermore, reciprocal effects of negative life events and coping styles are discussed. The study shows several strengths by providing longitudinal data and including outcome measures of the offspring of depressed parents. Surprisingly, the authors don't report group-based differences (high-risk vs. low-risk group) in outcome variables in the model. Compas and colleagues observed coping strategies in a sample that consisted only of families with parental depression who took part in a prevention program (Compas et al., 2010). Here, children's secondary control coping strategies mediated the effect of the intervention on children's psychopathology by accounting for approximately 50 % of the significant intervention effect.

In sum, there is evidence of how emotion regulation, cognitive factors and stressful life events are associated with depressive and psychopathology symptoms. In addition, it was shown, that the offspring of depressed parents are showing higher psychopathological symptoms compared to children of non-depressed parents. Moreover, they are exposed to more stressful life events and face an increased risk for developing a depression. Although Goodman and Gotlib (1999) strived to explain trans-generational pathways in their model of

transition, there is little evidence on relevant mediators as emotion regulation strategies, attributional style and moderators as stressful life events for the offspring of depressed parents compared to the offspring of non-depressed parents. This gap in research is surprising, since the offspring of depressed parents face a heightened risk of developing a major depression and findings about modifiable risk factors (as cognitive and emotional factors) are substantial for clinical implications.

The current study adds to the literature among transmission of depression by addressing potential risk factors for the transmission of depression in the offspring of depressed parents using an opportunistic sample of children and their parents recruited to a preventive intervention. Firstly, a moderate sample size of N = 112 parent-child dyads is collected. Secondly, findings of increased psychopathological symptoms in the offspring of parents with depression (high-risk group, HR) compared to children of non-depressed parents (low-risk group, LR) are aimed to be replicated. In addition, correlation of children's and parental depression characteristics are explored. Thirdly, most prevalent emotional and cognitive factors are compared between groups, as well as negative and positive life events to investigate whether children of parents with depression show more risk factors than children with non-depressed parents. Fourthly, those mediating and moderating risk factors are explored concerning their association with parental and children's depression characteristics. Finally, the impact of those moderating, mediating factors and the parental depression on the children's subclinical depressive symptoms is explored.

6.2. Hypotheses Study I

The following hypotheses are tested:

Group differences in children's psychopathology and association of parental depression. The first aim was to replicate the finding of increased psychopathology in children of depressed parents. In addition, the association of the children's depressive symptoms and parental depression characteristics is explored.

H1.1a: Children of depressed parents show more depressive and psychopathology symptoms than children of parents without depression.

H1.1b. Children's depressive symptoms are associated with the parental depression variables: i) current status of depression and the ii) parental depressive symptoms.

Group differences in moderators and mediators of major depression. In the next step, potentially key factors for the transmission, development and maintenance of depression are analysed for group differences between the high- and the low-risk group.

H1.2: Children of depressed parents show more i) maladaptive and less adaptive emotional regulation strategies, ii) a more negative and less positive attributional style and iii) report more negative and less positive life events than children of parents without mental health problems.

Exploring risk factors for major depression. The last research questions addresses the influence of the relevant risk factors for depression in the offspring of depressed parents (parental depression, mediators as emotion regulation, attributional style and moderators as life events). Firstly, the association of these factors with the children's depressive status are explored. Secondly, it is investigated what the most prevalent risk factors in predicting the children's depressive symptoms are.

H1.3a: The risk factors maladaptive emotion regulation, negative attributional style and negative life events are correlated positively with the children's depressive symptoms, whereas adaptive emotion regulation strategies, positive attributional style and positive life events are correlated negatively with the children's depressive symptoms.

H1.3b The factors emotional regulation strategies, attributional style, negative life events and the parental depressive status account for the variance in the child's depressive symptoms.

7. Method study I

7.1. Study design

In a between-groups design, psychopathology, emotion regulation strategies, attributional style and stressful life events were compared between N = 112 children of depressed (high-risk group = HR, n = 74) versus non-depressed parents (low-risk group = LR, n = 38). In addition, the extend of influence of those factors and the parental depression on depressive symptoms in children was tested among groups. Because the data were taken from a study of a family intervention (Study 2), data from more than one child per family were available. For these analyses the oldest child was chosen for inclusion in the high-risk group. Data from children in the low-risk group acquired from a study where only one child per family was recruited.

7.2. Participants

High-risk group (HR). Parents were eligible in case they fulfilled the diagnostic criteria of a depressive disorder according to the DSM-IV, occurring during the children's lifetime. Children and adolescent were included in the study if they did not meet the DSM-IV diagnostic criteria for a psychiatric disorder (in the present or past). They had to be aged eight to seventeen and have at least an IQ of 85. Parents were excluded if they suffered from alcohol or substance abuse, bipolar disorder, reported psychotic symptoms, had a personality disorder or a suicidal crisis.

The high-risk group consisted of n = 74 families, originating from Munich and suburban parts. 80.5 % of the families had German background; others had a migration background of Turkey or Bulgaria. Families were recruited in different kinds of institutions (e.g. clinics, newspaper articles, pediatricians, see section 11.2. for detailed information). The

biggest group was invited due to direct contacts in clinics (26.0 %) and to newspaper articles (24.7 %).

Low-risk (LR) group. Parents, children and adolescents were included if they did not meet the diagnostic criteria of any psychiatric disorder according to the DSM-IV (in the present or past). Children and adolescents had to be aged nine to fifteen with an IQ of at least 85. The families were recruited to an ongoing study conducted by colleagues in the department (Anca Sfärlea, Belinda Platt), hence the slight difference in age range (9-15 rather than 8-17). Nevertheless, the mean age was the same for both groups (see Table 6).

The low-risk group sample consisted of n = 38 families from Munich and surrounding suburbs. Since the expected variance of this group is expected to be lower than in the high-risk group, the sample sizes were smaller but still sufficiently big for the analysis. Most of the families were contacted because they were registered in the study databank of the research team (40.0 %); another part was recruited with the help of the local administration office (36.0 %) or public advertisement (24.0 %). 92 % of the families were German, 8.0 % had Austrian, Bulgarian or Turkish background. The majority of children (92.0 %) were living together with their mother and father, 8.0 % were single-parents.

Each family received 25 € as reward for participating. All participants were informed about the study procedure and possible risks and gave their written consent for study participation. The ethic approval was positive, confirming that the collected data is in line with the Helsinki guidelines.

7.3. Procedure

When parents contacted the research team in response to study advertisement, the initial exclusionary criteria were addressed and the participating parents were screened regarding their general psychopathology and that of their participating children. Additionally, they

received more details about the study protocol. In case the family was suitable and interested in taking part in the study, a date for the assessment session was made. Hence, participants were again informed about the study procedure and a written informed consent was given to the parent and the child. After that the child was screened for intelligence and a structured, standardized diagnostically interview for psychological disorders was conducted. The parent (at least one) was also interviewed about their psychopathological symptoms as well as about their children's symptoms. Questionnaires were handed out to be filled in at home and asked to send back. After the first assessment, a decision was made about the family's eligibility for the study on the base of the information which the research team had gathered at the assessment.

7.4. Measures

Table 4 gives an overview of the instruments used to determine eligibility for the study and measure outcomes.

Table 4 Eligibility and outcome variables

	Measure	Instrument
Eligibility criteria	Diagnostic status (child)	K-DIPS
	Intelligence test (child)	CFT 20-R
	Diagnostic status (parent)	DIPS
	Personality disorder (parent)	SKID II
	Psychopathology (2nd parent)	SCL-90-R
Outcome measures	Depressive symptoms (child)	DIKJ
	Psychopathological symptoms (child)	YSR, CBCL
	Emotion regulation strategies (child)	FEEL-KJ
	Attributional style (child)	ASF
	Stressful life events (child)	CASE (C/P)
	Depressive symptoms (parent)	BDI-II
	Status and history of depression (parent)	DIPS

Note. K-DIPS = Diagnostisches Interview für psychische Störungen, Child Version; CFT 20-R = Culture Fair Test. DIPS = Diagnostisches Interview für psychische Störungen, Parent Version; SKID II = Strukturiertes Klinisches Interview für DSM-I; SCL-90-R = Symptomcheckliste. DIKJ = Depressions-Inventar für Kinder und Jugendliche; YSR = Youth Self-Report; CBCL = Child Behaviour Checklist; FEEL-KJ = Fragebogen zur Erhebung der Emotionsregulation bei Kindern und Jugendlichen; ASF = Attributionsstil-Fragebogen; CASE = Child and Adolescent Survey of Experiences; BDI-II Beck's Depression Inventory;

7.4.1. Demographic variables

Participants were asked to provide information on a number of important demographic variables that are displayed in table 5.

Table 5 Demographic questionnaire

Parent version	
	Age
	gender
	Marital status
Cultural background	Country of birth
	Nationality
	Mother tongue
Socio economic status	Educational level
	Employment (full time vs. part time, type of job)
	Family income
Therapeutical experience	Experience with psychotherapeutical treatment
	medication[1]
	In-patient stays[1]
Child version	
	Age
	Gender
Cultural background	Country of birth
	Nationality
	Mother tongue
School	Grade
	Type of school
	Friends
Social network	Social support (e.g. by grandparents)

Note. [1] provided only for parents with depression.

7.4.2. Eligibility measures

7.4.2.1. Parental diagnostic status

To assess whether parents met the diagnostic criteria for inclusion in the study (see 5.2. participants), the *Diagnostisches Interview für Psychische Störungen* (Schneider, Margraf, Spörkel, & Franzen, 1992) was administered. It is a semi-structured, clinical interview that serves as a checklist for the diagnosis of psychiatric disorders on the basis of the DSM-IV. The standardized manual enables an objective implementation and evaluation, when it is done by a psychologically trained person. Exact formulation of criteria increase reliability and validity additionally. With selective screening questions at the beginning of each section, the interviewer is being led step by step through the diagnostic. Firstly, general demographic questions and potential stressors are explored. In the next step the interviewer asks the participant about their symptoms concerning panic attacks and disorder, phobia, general anxiety disorder, obsessive compulsive disorder, post-traumatic stress disorder or potential traumas in their biography, affective disorders, alcohol- and substance abuse, somatic disorders, non-organics psychotic symptoms and medication.

Test objectivity is more vulnerable than other questionnaires due to its semi-standardized structure. Authors warn that the instrument must only be used by a trained clinician. In that case objectivity can be seen giving concerning standardised instructions and standard values for implementation, evaluation and interpretation.

Suppiger and colleagues (Suppiger et al., 2008) tested the reliability through interrater accordance and report kappa scores between k =.72 and k =.92 for general factors. The retest-reliability is likewise satisfying with scores between k = .62 and k = .94. An exception is reported for the scale sleeping disorders, where the kappa is only k = .35. Schneider and colleagues (1992) found the retest-reliability scores to be substantial across different scales – the concord rate varies from k = .42 (somatoform disorders), k = .73 (anxiety disorders), k =

.66 (depression) and k = .87 (eating disorders). Concerning the interrater-reliability Schneider and colleagues (1992) report percentage congruence of at least 92 % and kappa´s between k = .82 and k = 1.0, rating higher than the retest-reliability. Margraf et al. (1991) report retest-reliability scores of Yule´s Y between Y = .67 (somatoform disorder) and Y = 1.0 (psychoses), while kappa varies between k = .68 (depression) and k = .78 (no disorder). In-Albon et al. (2008) analysed the validity of the DIPS, which was tested through other disorder questionnaires and found predominantly good to very good validity scores for most scales. Solely the results for sleeping disorders and generalized anxiety disorder form an exception – validity was inadequate, as the authors report.

Trained and experienced staff of the research team conducted all clinical interviews. In this work, 20 % of interviews were checked for interrater reliability. Therefore, 20 interviews were selected randomly and re-rated by an independent researcher (Laura Thomsen). The pre-defined criterion was the accordance of diagnosis concerning the current and previous status of depression. The accordance rate was excellent with 100 % (kappa = 1.00), especially compared to other publications. This index indicates a high interrater reliability (Schneider et al., 1992).

7.4.2.2. Child diagnostic status

To ensure that children had no current or past psychiatric diagnosis, the child version (K-DIPS) was administered (Schneider et al., 1992). This contains both a child self-report and a parent-report. Sections are similar as in the adult version with additional sections concerning attention deficit hyperactivity disorder, oppositional behaviour, conduct disorder, tic disorder, sleeping problems, separation anxiety, selective mutism, enuresis/encopresis, and pica. In addition, parents are asked about their children's symptomology. Similar as in the parent version, the test objectivity depends on the implementation by trained clinicians.

The reliability was tested mainly by interrater accordance and was found to be sufficiently high (In-Albon et al., 2008). In the child version kappas are between k = .39 (sleeping disorder), and k = .95 (depression), or Yule's Y = .86 (sleeping disorder) to Y = .99 (dysthymia). In the part where parents are interviewed about their children, kappa ranges from k = .42 (pica) to k = .96 (depression), Yule's Y again showed better accordance Y = .98 (pica) to k = .99 (depression). In the section "depression" In-Albon and colleagues found high interrater reliability scores as well as high retest-reliability scores after one week (98-100 % accordance) (In-Albon et al., 2008). The parent-child accordance of 6-17 year olds (mean = 10.5 years) was lower (k = .31). The validity is claimed to be good or very good for the subscales anxiety disorder, affective disorder, eating disorder, somatic disorder, alcohol and substance abuse as well as for single diagnosis as social phobia, obsessive compulsive disorder, panic disorder with/without agora phobia. The validity was tested with extern questionnaires. Individuals that had no psychiatric disorder concerning the K-DIPS rating did have very low rates in other questionnaires (In-Albon et al., 2008). Another validity measure was the rating of a clinician with low to moderate accordance rates for depression k = .25 (Dolle et al., 2012).

Like in the parent version, all clinical interviews were conducted by trained and experienced staff of the research team. In this work, 20 % of conducted interviews were checked for interrater reliability. Therefore 20 interviews were selected randomly and re-rated by an independent researcher (Laura Thomsen). Again, the pre-defined criterion was the accordance of diagnosis concerning the current and previous status of depression. The accordance rate was excellent with 100 % (kappa = 1.00), especially compared to other publications. This index indicates a high interrater-reliability, especially compared to other publications (Schneider et al., 1992).

7.4.2.3. Intelligence screening (child)

In order to estimate the children's intelligence, the *Culture Fair Test* (CFT 20-R, Weiß, 2006) was administered. The CFT 20-R is a basic intelligence assessment, testing the general mental ability *g* or the fluid intelligence. The test claims to be untouched by social and cultural influences. The CFT-20-R is split into four sub tests: 1) serial continuation series, 2) object classification 3) matrix and 4) topologies. The total of 101 items is exclusively figural with a multiple choice answer format. The duration is 60 minutes, in the short form 35-40 minutes and is constructed for eight to nineteen year old children and adolescents. Single or group sessions are possible. The re-test reliability for the first part is r = .92, for the second part r = .91 and r = .96 for both parts. Correlation of the first and the second part is r = .82. The test validity was confirmed with correlations of external measures as grades in math r = .45-.53 what can be interpreted as sufficiently high, concerning the language free test construction and other inferring factors with grades. Standard values were calculated using a sample of 4.400 students in Germany with IQ-, T- and standard values for class and age groups. In this work only part one has been used with the short time version in order to screen the children's intelligence (IQ > 85).

7.4.2.4. Screening for personality disorder (parents)

For screening for parental personality disorders, the *Strukturiertes Klinisches Interview für DSM-IV* (SKID II, Wittchen, Zaudig, & Fydrich, 1997) was conducted. The SKID II is a psychometrical instrument in order to evaluate and diagnose personality disorders as defined in DSM-IV axis II. It is a two-step instrument, consisting of a screening questionnaire and a following interview. In the interview, items are directed to the patient, in case a dimension crossed a specific cut off of „yes"-responds. The SKID-II is directed to adults only and can be applied in clinic as well as out-patient settings. The duration of the questionnaire is estimated to be 30 minutes; the interview differs depending on the number of „yes" responds but

around 30 minutes for in-clinic patients. The interview must be performed by a trained clinician in order to give a correct diagnose.

7.4.2.5. Psychopathology (second parent)

Partners of the parent suffering from depression, were also screened for their psychopathological symptoms using the *Symptomcheckliste* (SCL-90-R, Franke, 2002). The SCl-90-R is a screening instrument in order to evaluate the impact and perception of psychological and physical symptoms in the last week. This assessment was used in order to screen the healthy parent for psychopathological problems. The 90 items self-rating scale can be applied from 12-years on. There are nine subscales including somatization, obsession, social insecurity, depression, anxiety, phobia, aggression, paranoiac thinking, psychotic symptoms. The test duration lies between ten to fifteen minutes. Test objectivity is given due to standardized instructions, detailed analyzing material, and interpretation guidance. Cronbach's Alpha in all subscales was sufficiently high ($r_{min} \geq .76$), especially the global score reached very high values of internal consistency ($\alpha = .97 - .98$). Re-rest reliability was measured in an interval of one week and was moderate to high. There are standard values for age and gender (T-values) for 12- 70 years olds (n = 2.025).

7.4.3. Outcome measures

7.4.3.1. Symptoms of depression(child)

To assess self-reported symptoms of depression in children, the *Depressions Inventar für Kinder und Jugendliche* (DIKJ, Stiensmeier-Pelster, Schürmann, & Duda, 2000) was implemented. It's the translation of the well-established English Children's Depression Inventory (CDI, Kovacs, 1992). The DIKJ was constructed on the base of the diagnostic criteria of the DSM-IV and includes all significant symptoms of a depressive disorder in a

child-friendly version. There are 26 items with three equal response options. The duration is about ten to fifteen minutes. Due to standardized instructions and standard values for implementation, evaluation and interpretation test objectivity are guaranteed. Standard values are relying on a sample of n = 3.395 students in the age of eight to sixteen divided in age, gender and school type. There are T-values as well as percentile ranks. There is numerous evidence of high reliability: the internal consistency (Cronbach's alpha) was α = .92 in a small clinical sample (n = 139) and α = .87 in an unselected sample of students (n = 3.403). Construct validity can be regarded as high, since the items are directly based on the DSM-criteria for depression.

7.4.3.2. Children's psychopathology (parent report)

The German version of the *Child Behavior Checklist* (CBCL, Döpfner, Schmeck, & Berner, 1994) was used to assess the parental judgement of the children's personal, social and academic competences, internal and external psychopathological symptoms. The questionnaire is constructed for parents of children aged four to eighteen years; the duration is fifteen to twenty minutes. The CBCL is divided in two subscales: the competence scale measuring activities, social competences and school achievement, where parents report in 13 questions in an open format about their children's engagement in sports, hobbies, extracurricular activities, friends and school; and the syndrome scale measuring internalizing, externalizing and other general symptoms in 113 items with three response options ("0 = not applicable; 1 = sometime/ a bit applicable; 2 = applicable"). Internalizing symptoms are covering social withdrawal, physical impairment, anxiety and depression. External symptoms are defined as delinquent and aggressive behavior. Social, obsessive compulsive and attentional symptoms are reported in the general symptoms scale. Test objectivity can be accepted concerning standardized instructions and standard values for implementation, evaluation and interpretation. Reliability of subscale and global scale was confirmed in a

German clinical sample (n = 1.653) and in a non-clinical sample (n = 1.622). Internal consistency of the internalizing and externalizing subscales was r > .85. Factor validity was confirmed in a clinical sample for all scales except the "social problem" and "social withdrawal" scales. Using confirmatory analysis the factorial structure was confirmed not only in the German sample (n = 2.900), but also in 28 other cultures. There are standard values for age (4-11 years and 12-18 years) and gender reporting T- and %-values.

7.4.3.3. Children's psychopathology (child report)

For the assessment of the children's psychopathology, the German version of the *Youth Self-Report* (YSR, Döpfner, Berner, & Lehmkuhl, 1994), was administered. The YSR is the equivalent of the CBCL (Döpfner et al., 1994) but for the children's response. The questionnaire is constructed for children aged eleven to eighteen years; the duration is fifteen to twenty minutes. Like the CBCL, the YSR is divided in two subscales: the competence scale and the symptoms scale. The competence scale measuring activities, social competences and school achievement children report in eleven questions in an open format about their engagement in sports, hobbies, extracurricular activities, friends and school. The syndrome scale covers 113 items about internalizing, externalizing and other general symptoms offering the response three options ("0 = not applicable; 1 = sometime/ a bit applicable; 2 = applicable"). Interpretation of scales is equivalent to the CBCL scales. Test objectivity can be seen given concerning standardized instructions and standard values for implementation, evaluation and interpretation. The reliability of subscales was tested in a clinical sample (n = 292) and confirmed. High internal consistencies are reported for the internal and external symptoms scale (r ≥ .86), sufficient internal consistencies were found for subscale "aggressive behaviour", "anxiety/depression", "physical impairment", "antisocial behaviour" and "attention problems" (r > .70). Standard values were investigated in a nationwide German sample of n = 1.800 children and adolescents. Factorial validity was proven using main

component analysis with a following varimax rotation. The subscale construction could be confirmed, except the scale “social withdraw”. There are standard values reported for gender and age in T-values and percentile ranks.

7.4.3.4. Children’s emotion regulation strategies

The *Fragebogen zur Erhebung der Emotionsregulation bei Kindern und Jugendlichen* (FEEL-KJ; Grob & Smolenski, 2005) was administered in order to evaluate the children’s emotion regulation strategies. The questionnaire evaluates in two dimensions (adaptive and maladaptive) how children and adolescents cope with the emotions anxiety, sadness and anger. The self-rating questionnaire was constructed for children and adolescent aged ten to nineteen and can be applied in a group or single setting. The duration is estimated between 10 to 30 minutes, depending on the children’s age and consists of 30 items with a five-point Likert scale (“1 = never, 2 = rare, 3 = sometimes, 4 = often, 5 = almost always”). Adaptive (problem focused action, distraction, increased happiness, acceptance, cognitive reappraisal, problem solving) and maladaptive coping strategies (giving up, aggressive behaviour, withdrawal, negative self-evaluation, perseveration) are estimated. Furthermore, the FEEL-KJ obtains secondary subscales that are independent from expression, social support and control of emotion. Moreover, the questionnaire is a screening instrument for the risk of developing psychopathological symptoms. Items are not clustered to specific disorders but to take psychosocial competences into account. Like this it provides useful information about the children’s resources as well. The internal consistency of the fifteen scales lie between $\alpha = .69$ (giving up) und $\alpha = .91$ (social support), for the subscale adaptive strategy Cronbach’s alpha was $\alpha = .93$, for maladaptive strategies $\alpha = .82$. The six-weeks re-test- reliability of the single scales was $r_{tt} = .62$ -.81 for the fifteen subscales, for the two secondary scales $r_{tt} = .81$ (adaptive strategies) and $r_{tt} = .73$ (maladaptive strategies). Construct validity, factorial

structure, differential and internal validity was confirmed in a sample of n= 1.446 children and adolescents.

7.4.3.5. Child attributional style

The *Attributionsstil-Fragebogen* (ASF-KJ; Stiensmeier-Pelster et al., 1994) was conducted in order to rate the children's attributional style. It is a self-rating questionnaire for children and adolescents aged eight to sixteen. Children and adolescents are asked to evaluate and name eight positive and negative situations concerning their cause referring to internality, globalism and stability. The questionnaire is interpreted by the negative of positive ratings of these three dimensions. A negative internal, global and stable attributional style is linked to e.g. depressive symptoms. The duration is 20 to 40 minutes and consists of 16 items. Each item refers to a specific situation that is first described briefly (e.g. "Imagine a classmate is celebrating her birthday but you are not invited"). Children are asked to respond first how they evaluate the situation in an open format. In the second step, three questions with four response options are offered, in order to further explore the attributional style (e.g. negative or positive). Due to standardized instructions and standard values for implementation, evaluation and interpretation test objectivity can be seen given. Depending on the specific study, coefficients of consistency (Cronbach's alpha) of the global and stability dimension lie between $\alpha = .72$ and $\alpha = .81$, the internality dimension between $\alpha = .52$ and $\alpha = .57$. Retest-Reliability (four weeks) was observed to vary between $r_{tt} = .49$ and $r_{tt} = .65$. The construct validity can be seen as given, since the questionnaire is strictly theory-led. Furthermore significant correlations of depressions score, self-esteem and evaluation of own abilities were shown. Standard values are reported in T-values and percentile ranks (n = 1500).

7.4.3.6. Child's life events

The *Child and Adolescent Survey of Experiences* (CASE; Allen, Rapee, & Sandberg, 2012) was administered for capturing the child's negative life events. The CASE is a checklist

including about 38 life events that might have happened in someone's life in the past twelve months rated by parents (CASE-P) or children (CASE-C). Individuals are asked to rate firstly whether this life event happened to them and secondly how severe the impact of this event was on their life on a six-step scale. Life events range from e.g. a holiday experience to diseases, accidents or experiences in school in order to capture threatening as well as positive experiences. Test objectivity can be seen given concerning standardized instructions and standard values for implementation, evaluation and interpretation. There are moderate retest-reliability (one week) for mothers and children r_{tt} = .75, the accordance rate of mother and child was 60 %. There were accordance rates found between as similar instrument PACE (Psychological Assessment of Childhood Experiences(Sandberg et al., 1993) of k = .13 (leisure activities) and k = .73 (experiences with pets) but not satisfying in the scales "leisure" and "conflicts in family" (Allen et al., 2012). The external validity of the CASE is given with a significant correlation with the PACE of r = .47 for negative and r = .28 for positive life events. It was also observed that children with anxiety disorder show different score than children without mental illnesses (Allen et al., 2012).

7.4.3.7. Parent's depressive symptoms

The German version of *Beck's Depression Inventory* (BDI-II, Hautzinger, Bailer, Worall, & Keller, 1994) was conducted for measuring the parent`s depressive symptoms. In 21 items covering different depressive symptoms with four response options which mirror the intensity of each symptom, the severity of depressive symptoms is evaluated. The duration is around five to ten minutes. Test objectivity can be seen given concerning standardized instructions and standard values for implementation, evaluation and interpretation. The German version was applied in multiple studies with clinical patients (depression and other disorders; n = 1079) as well as in the general population testing for re-test reliability. In a period of five months a retest reliability of r = .78 was identified. There were high correlations found

between the BDI-II and other questionnaires concerning depressive symptoms as the FDD-DSM-IV (Fragebogen zur Depressionsdiagnostik nach DSM IV, Kühner, 1997) (r = .72-.89) and the MADRS (Montgomery Asberg Depression Scale, Montgomery & Asperg, 1979) (r = .68 -.70).There are standard values for depressed patients (n = 266) as well as for healthy population (n = 582) reported in the manual.

7.5. Data preparation

7.5.1. Outlier

In order to detect outliers, all variables were z-transformed and screened for values above +/- 3.29. There were just few outliers. In three cases they could be corrected, since it turned out to be IQ-scores that were invalid. Those IQ-values were suspiciously low (IQ = 67 -72). The IQ-test was followed by a two-hour clinical interview, in which the validity of the IQ-values and the children's motivation could be observed in a personal setting. Since those three children appeared did not show any indication of intelligence below the average but were less motivated to do the intelligence test, those values could be classified as invalid. Therefore, the outliers were adjusted to two standard deviations below the mean. Two increased values were found in CBCL and YSR scores in the high-risk group that were reasonable for the analysis and therefore were not corrected.

7.5.2. Missing values

In empirical researches, incomplete data is well known (Lüdtke, Robitzsch, Rautwein, & Köller, 2007). The causes of missing data are numerous, even when a thorough and standardized method was implemented (Lüdtke et al., 2007). Here, the range of missing outcome values was 0.9 – 21.4 % ($x_{missing}$ = 13.1 %), consequently above the critical values of 5 %, suggesting non-coincidence (Rost, 2007, p. 177). Families who attended the assessment

session of the intervention but dropped out of the program later, are responsible for most missing data. More than 80 % of the outcome variables were missing from fourteen participants (families of the high-risk group that dropped out of the program). More than 50.00 % of the outcome variables were missing from four other participants. Nevertheless, the high and the low-risk group did not differ significantly in the amount of missing values ($t_{1,59}$ = .23; p = .470). Table 6 displays percentage of missing variables of the outcome variables. Since data was missing completely at random (MCAR) missing values were imputed based on the expectation-maximization method (Stephens, Smith, & Donnelly, 2001). This method enables imputation without changes of group means, standard deviations and covariance.

Table 6 Missing data study I

Outcome variable	DIKJ	YSR	CBCL	FEEL-KJ	ASF	CASE	BDI-II parent
n complete data	90	91	94	98	88	111	109
n missing	22	21	18	14	24	1	3
% missings	19.6	18.7	16.1	12.5	21.4	0.9	2.7

Note. DIKJ = Depressions-Inventar für Kinder und Jugendliche; YSR = Youth Self-Report; CBCL = Child Behaviour Checklist; FEEL-KJ = Fragebogen zur Erhebung der Emotionsregulation bei Kindern und Jugendlichen; ASF = Attributionsstil-Fragebogen; CASE = Child and Adolescent Survey of Experiences; BDI-II Beck's Depression Inventory; HR = high-risk group.

7.5.3. Testing assumptions

Data is assumed to be interval scaled due to the implemented assessment instruments. The independence of samples can be regarded as given. In this study there is data of N = 112 independent families. Before each analysis was run, relevant assumptions were tested as e.g. the normal distribution and equality of variances using the equivalent test statistics. Corrections were applied when necessary.

7.6. Analyzing strategy

The data was analyzed using the statistic program SPSS Version 19 (SPSS Inc., 1989-2006) for Windows and JASP Version 0.8.1.1 for Mac Os x for calculating additional Bayesian statistics. Since the age range was quite big (8-17 years), T-values were used for the analysis for all outcome measures that provided standard tables (YSR, CBCL, DIKJ, ASF, FEEL-KJ) in order to control age and gender. In addition to the following analysis, the Bayes factor (BF_{10}) was calculated. In contrast to *p*-values, the Bayes factor allows the researcher statements about the alternative hypothesis, and evidence in order to reject null hypothesis. Consequently, an additional and more precise estimation of the amount of evidence present in the data is provided (Jarosz & Wiley, 2014).

1. For testing hypothesis H1.1 one-factorial multiple variance analysis (MANOVA) was run to estimate the group differences between depressive and psychopathological symptoms of children and adolescents.
2. For Hypothesis H1.1b, Spearman's correlations coefficients were calculated to evaluate the association between the variables i) current status of parental depression (no history of depression, remitted or currently depressed), ii) parent's depressive symptoms and iii) the children's depressive symptoms with an alpha level of $\alpha = .05$, two-tailed.
3. Another MANOVA was conducted for testing hypothesis H1.2 and group differences on 1) emotional regulation strategies, 2) attributional style and 3) life events of children with parents with depression (high-risk group) and children with healthy parents (low-risk group).
4. In order to test hypothesis H1.3a Spearman's correlation coefficients were calculated on several risk factors and the children's depressive symptoms: i) emotion regulation strategies, ii) attributional style, iii) life events and iv) parental depression characteristics.

5. For predicting the children's depressive symptoms, two regression models were conducted (H3.1b). Firstly, four clusters of possibly relevant predictors were entered stepwise in the hierarchical regression model (1) background variables of child, 2) parental depression characteristics, 3) moderators and 4) mediators). In the next step, a regression model with forward inclusion was calculated in order to explore the most significant predictors for the depressive symptoms in children.

8. Results study I

8.1. Sample description

A total of 112 families was recruited for this study, 74 in the high-risk group and 38 low-risk group. In general, families had a high economical background and parents were mostly well educated. Families did not differ significantly in the demographic variables, except in the parent's marital status and, as expected, the parental depressive symptoms (BDI-II). More parents in the high-risk group were married than in the low-risk group. Demographic characteristics are displayed in table 7 (children) and 8 (parents).

Table 7 Demographic characteristics, children

variable	High-risk group (n = 74)	Low-risk group (n = 38)	Total sample (N = 112)	*p*-value
Age				
Mean (SD)	12.0 (2.97)	11.77 (1.65)	11.92 (2.59)	
Range (min.-max.)	2.97		8-17	.639
Gender (%)				
female	52.7	36.8	56.3	.450
IQ				
Mean (SD)	106.6 (14.76)	111.66 (11.03)	108.27 (13.77)	
Range (min.-max.)	85-141	91 - 133	82-141	.099
Siblings (%)				
yes	66.2	76.0	77.2	
no	33.8	24.0	20.2	.652
School type (%)				
Elementary school	28.6	31.5	37.5	
Secondary school	16.6	7.9	12.5	
High school	36.4	55.3	47.1	.385

Table 8 Demographic characteristics, parents

	High-risk group (n = 74)	Low-risk group (n = 38)	Total sample (N = 112)	*p*-value
Age, parent				
Mean (SD)	46.61 (6.33)	45.08 (4.70)	46.04 (5.86)	
Range (min.-max.)	34-60	34-54	34-60	.107
Education parent (%)				
Basic education	20.6	21	20.6	
A-levels	27.1	15.8	22.7	
University	42.4	57.9	48.5	
Doctoral degree	10.2	4.5.3	8.2	.724
Marital status (%)				
Single parent	5.4	52,6	24.0	
Married	72.6	42.1	64.0	
separated	12.9	45.2	10.0	.000
Employment(%)				
Full time	41.6	39.5	55.2	
Part time	22.1	60.5	40.6	
Unemployed	3.4	0	2.1	
Retired	7.8	0	6.3	.246
Family income (%)				
– 2000 € /months	13.2	10.6	12.1	
2000 – 3000 € /months	22.6	5.3	15.4	
3000 – 4000 € /months	17.0	18.4	17.6	
4000 – 5000 € /months	22.6	18.4	20.9	
> 5000 € /months	24.5	47.4	34.1	.073
Parent depressive symptoms (BDI-II)				
Mean (SD)	17.59 (10.98)	1.79 (3.47)	12.14 (11.82)	
Range (min.-max.)	0-53	0-14	0-53	.000

Psychopathology. To qualify for the study parents were required to either meet criteria for at least one episode of major depressive according to the DSM-IV criteria (high-risk group) or have no lifetime diagnosis of any DSM-IV disorder (low-risk group). Therefore, the sample differed in the parental psychopathology. In the high-risk sample, most parents were diagnosed with a recurrent depressive disorder that was remitted (23.0 %) moderate (12.5 %)

or light (64.5 %). 84 % of all children were not having severe symptoms in the clinical interview; 9.1 % showed light subclinical symptoms. Four children of the high-risk group showed elevated symptoms of major depression and anxiety and were therefore excluded from the study[2]. The screening of the non-affected second parent (SCL-20R) did not reveal any increased values (mean global score GS = 0.02).

[2] Children and parents were supported to seek professional help in order to receive adequate treatment by providing contact information of therapists for children and adolescents and giving advice.

8.2. Testing hypotheses

8.2.1. Testing Hypothesis 1.1

8.2.1.1. Assumptions H 1.1

The Kolmogorov-Smirnoff test revealed evidence that the collected data were distributed normally. However, the self-reported depressive symptoms of the child indicated non-normality (DIKJ: K-S statistic = .14, df = 61, p = .003). According to West and colleagues (1995) normally distributed data can be assumed, when the values of skewness and kurtosis divided by its standard error are s < 1.96 and k < 1.96, which was the case in the sample (West, Finch, & Curran, 1995)[3]. Although this analysis indicated left-skewness, the visual check indicated normality of the data. In addition, the MANOVA is quite robust against the non-normality of the data. Box-M-test revealed non-significance, implicating homogeneity of covariance matrices ($F_{1, 59}$ = .56, p = .943).

8.2.1.2. Results H1.1

Table 9 describes the psychopathology of children in the high-risk and low-risk group. In order to evaluate whether children of depressed parents differ in their psychopathological outcome variables, a one-way MANOVA was calculated.

[3] *Standardized skewness and kurtosis*: DIKJ: s = 1.14/0.26 = 4.38 > 1.96, k = 1.29/0.52 = 0.02

Table 9 Psychopathology of children in the high risk and low-risk group

	Descriptives		Univariate effects		
	High-risk group M (SD)	Low-risk group M (SD)	F	*p*	η^2
Self-report depressive symptoms (DIKJ)	46.79 (7.43)	40.89 (6.84)	11.21	.001*	.129
Youth-self report (YSR)					
Internalizing symptoms	52.05 (10.35)	47.00 (8.31)	4.18	.044*	.052
Externalizing symptoms	50.86 (7.2)	46.88 (8.27)	4.35	.040*	.054
General psychopathology	53.19 (8.72)	48.70 (7.98)	7.58	.007*	.091
Child behaviour checklist, parent report (CBCL)					
Internalizing symptoms	58.31 (9.53)	47.48 (6.47)	28.08	.000*	.276
Externalizing symptoms	51.42(7.60)	48.28 (8.01)	5.21	.025*	.063
General psychopathology	55.46 (7.73)	47.10 (7.01)	27.39	.000*	.262

Note. * p< .05; ** p < .001

The MANOVA revealed in a significant multivariate main effect for condition concerning children's depressive symptoms and psychopathology (Wilks' $\lambda = .565$, $F_{1,79} = 3.78$, $p = .000$; $\eta^2 = .435$, d = 1.75). Given the significance of the overall test, the univariate main effects were examined. In all variables, depressive and psychopathological symptoms rated by parents and children, the high-risk group showed significantly higher values than the low-risk group with high effect sizes. Supporting these findings, the Bayes factor indicated indicating anecdotal (BF_{10} YSR = 2.97) to decisive effects (BF_{10} DIKJ = 50.01; BF_{10} CBCL= 27709.01)[4] evidence in favour of rejecting the null-hypotheses

[4] Interpretations of Bayes Factor (Jarosz &Wiley 2014)

BF_{10}		BF_{10}	
< 1/100	desicive support for H_0	1 – 3	anectodal support for H_1
<1/10	strong support for H_0	3-10	moderate support for H_1
1/10-1/3	moderate support for H_0	10-30	strong support for H_1
1/3 – 1	anectodal support for H_0	30-100	very strong support for H_1
1	H_0 is as likely as H_1	>100	desicive support for H_1

8.2.1.3. Summary H.1.1a

Thus hypothesis H1.1a was confirmed. The high-risk group did differ significantly from the low-risk group concerning the children's psychopathological and depressive symptoms: children of parents with depression showed significantly higher values in depressive symptoms, self-reported externalizing symptoms, internalizing, externalizing symptoms and general psychopathology reported by their parents and self-report.

8.2.2. Testing Hypothesis 1.1b

8.2.2.1. Assumptions H1.1b

Since parents with and without depression were included in the sample, it is not surprising that the Kolmogorov-Smirnoff-statistic revealed in significance indicating non-normality of the distribution (BDI-II; K-S statistic = 0.15, df = 110, p = .000). Since the sample consists of depressed and non-depressed parents (with many 0-values in the distribution), the left skewed distribution is not surprising. By visual check the data appeared normal distributed, but showed numerous 0-scores, deriving from non-depressed parents. Consequently, a two-tailed Spearman's correlation was conducted for H1.1b and H1.3a that is assumed to be more robust against violations of the normal distribution (Field, 2005).

8.2.2.2. Results H1.1b

Table 10 displays Spearman's correlations between the variables i) current status of parental depression (1) no history of depression, 2) remitted or 3) currently depressed) and the continuous variable parental and children's depressive symptoms.

Table 10 Correlation matrix of parent and child outcome variables

	Current status of depression	BDI-II
Self-report depressive symptoms, child (DIKJ)	.376**	.233**
Current status of depression, parent[1]		.695**
Self- rating depressive symptoms, parent (BDI-II)		1

*Note. N = 77-99. *Correlation is significant for α = .05 (two tailed). ** Correlation is significant for α = .001 (two tailed). 1 = never depressed, 2 = remitted, 3 = currently depressed; [2] 1 = currently depressed, 2 = remitted or not depressed; [3] = Spearmans correlation coefficient. DIKJ = Depressions-Inventar für Kinder und Jugendliche, BDI-II Beck's Depression Inventor.*

All correlations were significant, indicating positive associations between the children's depressive symptoms, the current status of depression and the parental depressive symptoms. The magnitude between child and parent outcome variables were small to moderate (r = .233 - .376). The Bayes factors for the associations of self-reported depressive symptoms of children and the parental depression indicator variables were ranging from anecdotal evidence (BF_{10} BDI-II x DIKJ = 1.27) to strong evidence (BF_{10} current status of depression of parent x DIKJ = 60.01).

8.2.2.3. Summary H1.1b

Hypothesis H1.1b was confirmed. Parental depression variables like current status of depression as well as depressive symptoms correlated significantly with small to moderate magnitude with the children's depressive symptoms.

8.2.3. Testing Hypothesis 1.2

8.2.3.1. Assumptions H1.2

All assumptions required for calculating the MANOVA were met, except two subscales of negative and positive life events rating (CASE) the data were normally distributed[5]. In case of non-normality due to the Kolmogorov Smirnoff-test, the data was further analysed by visual checks and examinations of standardized skewness and kurtosis[6]. The extend of the violation of the assumption of normality was rated to be low.

Box-M-test revealed non-significance, implicating homogeneity of covariance matrices for emotion regulation strategies (FEEL-KJ: $F_{1, 110} = .845$, $p = .665$) and attributional style (ASF: $F_{1, 81} = 1.19$, $p = .241$). The covariance matrices of negative and positive life events, rated by the children did not fulfil this assumption (CASE: $F_{1, 66} = 1.95$, $p = .034$). Since Levene-test[7] statistic revealed, that variance are equal between groups, no further corrections were made.

8.2.3.2. Results H1.2

8.2.3.2.1. Emotion regulation strategies

Table 11 shows emotion regulation strategies, of children in the high and low-risk group, as well as an overview of descriptive and results of univariate tests of subscales of FEEL-KJ. In

[5] *Kolmogorov-Smirnoff statistics*: CASE (positive life events); K-S statistic = 0.15, df = 112; $p = .002$; CASE (negative life events); K-S statistic=0.17, df = 112, p = .000).

[6] *Analysis of standardized skewness and kurstosis:* CASE (positive life events); K = 0.45/0.48 = 0.93 < 2.58, S = -0.42/0.25 = 1.68 < 2.58; CASE (negative life events); K = -0.10/0.49= 0.20 < 2.58, S = 0.75/0.26 = 2.88 >2.58;

[7] *Levene-statistic for homogeneity of variances*: CASE (positive life events, child rating): $F_{1,69} = 4.37$, $p = .040$; CASE (positive life events): $F_{1,69} = 1.75$, $p = .190$; CASE (negative life events, child rating): $F_{1,69} = 0.71$, $p = .403$; CASE (positive life events, child rating): $F_{1,69} = 0.33$, $p = .568$.

order to evaluate whether children of depressed parents differ in their emotion regulation strategies a MANOVA was calculated.

Table 11 Results of MANOVA, children's emotional regulation

	Descriptives		Univariate effects		
FEEL-KJ subscale	High-risk group M (SD)	Low-risk group M (SD)	F	*p*	η^2
Adaptiv strategies					
Anger	44.91 (12.31)	50.31 (12.29)	4.58	.035*	.045
Anxiety	46.18 (12.01)	51.07 (12.86)	4.38	.039*	.035
Sadness	48.62 (10.17)	50.13 (11.68)	0.46	.410	.005
Maladaptiv strategies					
Anger	47.95 (10.39)	43.00 (10.51)	3.87	.052	.035
Anxiety	46.47 (10.64)	44.34 (10.10)	0.71	.341	.009
Sadness	45.47 (9.97)	43.65 (10.19)	1.12	.292	.011

Note. * p< .05; ** p < .001.

The overall group differences in emotion regulation were significant (Wilks' $\lambda = .872$, $F_{1,91} = 2.6$, $p = .039$, $\eta^2 = .13$, d = 0.77). Across all sub-scales, children of depressed parents showed less adaptive emotion regulation strategies and tend to have more negative emotion regulation strategies (Table 11). Nevertheless, only the subscales *adaptive regulation strategies anger* and *anxiety* reached statistical significance between groups. The adaptive strategies *anger* and *anxiety* were further tested for evidence with the Bayes factor revealing in an anecdotal effect (BF_{10} adaptive strategy anger = 0.93; BF_{10} adaptive strategy anxiety = 0.52).

8.2.3.2.2. Attributional style

Table 12 displays means and standard deviations of the attributional style of children in the high risk and low-risk group. In order to evaluate whether children of depressed parents differ in their attributional style, a MANOVA was calculated.

Table 12 Children's attributional style

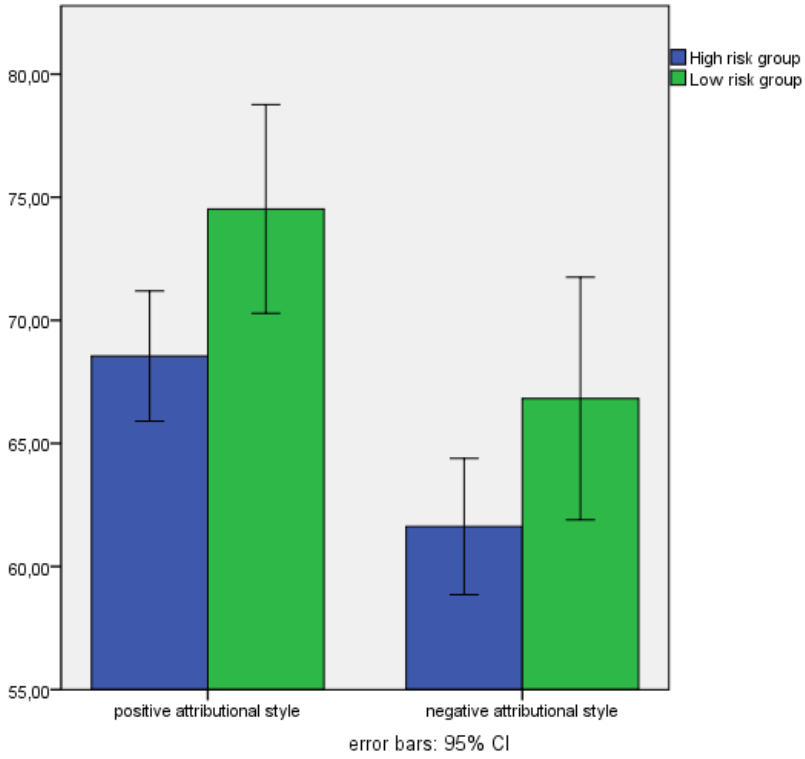
	Descriptives	
Dimensions of attributional style (ASF)	High-risk group M (SD)	Low-risk group M (SD)
Positive		
internal	45.15 (9.04)	50.08 (10.26)
stable	49.96 (10.83)	56.52 (11.71)
global	47.92 (11.89)	53.17 (13.39)
Negative		
internal	44.15 (9.84)	46.73 (9.28)
stable	50.86 (9.88)	57.13 (10.63)
global	48.79 (9.84)	52.30 (13.37)

The one-way MANOVA revealed in non-significant multivariate main effect for condition concerning children's attributional style (Wilks' $\lambda = .920$, $F_{1,81} = 1.17$, $p = .329$; $\eta^2 = .080$).

Since the six subscales are built on 16 items only, the power might be not enough to reject the H_0. Therefore, post-hoc sum scores of the positive and negative attributional scales were built and two univariate ANOVA were calculated. For positive attributional style, the groups differed significantly ($F_{1,74} = 6.12$, $p = .015$, $\eta^2 = 0.077$, $d = 0.58$) as well as marginally for the negative attributional style score ($F_{1,74} = 3.96$, $p = .050$, $\eta^2 = 0.051$, $d = 0.46$). Children of the high-risk group showed less positive and less negative attributional style as displayed on graph 4. This effect was supported by the Bayesian statistic indicating a

moderate effect on the group differences in the positive attributional style (BF_{10} = 3.29) and an anecdotal effect on the negative attributional style (BF_{10} = 1.34).

Graph 4 Means of positive and negative attributional style



8.2.3.2.3. Life events

Table 13 describes the self-rating of negative life events of children in the high risk and low-risk group. In order to evaluate whether children of depressed parents differ in their rating of negative life events, a MANOVA was calculated.

Table 13 Results of MANOVA, children's life events

Rating of life events	Descriptives High-risk group M (SD)	Low-risk group M (SD)	Univariate effects F	*p*	η^2
Number of positive life events	5.38 (1.90)	6.30 (1.41)	5.59	.020*	.065
Number of negative life events	3.80 (2.32)	3.20 (1.93)	1.39	.242	.017
Impact of positive life events	13.87 (5.37)	15.12 (5.11)	1.34	.250	.017
Impact of negative life events	7.80 (5.15)	6.90 (4.55)	0.68	.411	.008

*Note. * p < .05; ** p < .001.*

The one-way MANOVA revealed a significant multivariate main effect for condition concerning children's report of positive and negative life events and their rating of its impact (Wilks' $\lambda = .873$, $F_{1,77} = 2.8$, $p = .031$; eta² = .127, d = 0.78). In the post hoc univariate test children of depressed parents showed significantly lower values in the number of positive life events, but not in its impact. There was no difference between negative life events and their impact on children of the low and high-risk group. The Baysian statistic confirmed these findings revealing in a strong effect in the report of positive life events ($BF_{10} = 14.30$), but no evidence for the group differences in all other comparisons (BF_{10} negative life events = 0.27; BF_{10} impact of positive life events = 0.70; BF_{10} impact of negative life events = 0.58).

8.2.3.3. Summary H 1.2

Thus hypothesis H1.2 was partly confirmed. Although the main analysis of differences in emotion regulation strategies and positive and negative life events revealed significant effects, not all post-hoc univariate comparisons remained stable: The high risk sample did differ significantly from the low-risk group concerning, adaptive emotion regulation strategies (anger and anxiety) as well as the number of positive life events. Only when the global scores positive and negative attributional style were compared, group differences were significant. Children showed significantly less positive attributional style in the high-risk group, and also

less negative attributions than children in the low-risk group. Nevertheless, the groups did not differ in the subscales that distinguish further the internal, stable and global attributional style of the negative and positive scales.

8.2.4. Testing Hypothesis 1.3a

8.2.4.1. Results H1.3b

Assumptions for H1.3b were tested earlier and confirmed (see 8.2.1. and 8.2.2.). Table 14 provides the correlation matrix of the risk factors for depression (i) emotion regulation strategies, ii) attributional style, iii) life events and iv) parental depression characteristics) for the children's depressive symptoms.

Table 14 Correlation matrix (Pearsons's r) of parent and child outcome variables

	Adaptive Str.	Maladaptive Str.	Pos. life events	Neg. life events	Pos. attribution	Neg. attribution	Status of depr. (P)	BDI-II
Depressive symptoms, child (DIKJ)	-.14	.35**	-.28*	.10	-.18	-.11	.46**	.31*
Adaptive strategies (FEEL-KJ)		-.05	-.02	-.11	.18	.06	-.17*	-.12
Maladaptive strategies (FEEL-KJ)			-.03	.22*	-.06	-.01	.13	.02
Positive life events (CASE)				.43**	.28**	.28*	-.37**	-.21*
Negative life events (CASE)					.12	.23*	-.11	-.03
Positive attributional style (ASF)						-.73*	-.37*	-.31**
Negative attributional style (ASF)							-.27*	-.24*
Current status of depression, parent[1]								.71**
Depressive symptoms, parent (BDI-II)								

*Note. N = 65-112. *Correlation is significant for α = .05 (two tailed). ** Correlation is significant for α = .001 (two tailed). Spearman's correlation coefficients: [1]current status of depression, parent: 1 = no lifetime depression, 2 = currently remitted, 3 = currently depressed. DIKJ = Depressions-Inventar für Kinder und Jugendliche; FEEL-KJ = Fragebogen zur Erhebung der Emotionsregulation bei Kindern und Jugendlichen; ASF = Attributionsstil-Fragebogen; CASE = Child and Adolescent Survey of Experiences; BDI-II Beck's Depression Inventory.*

Maladaptive strategies, history of parental depression, current status of parental depression and the parental depression score were significant positive correlations of the children's depressive symptoms with small to moderate size. The factor positive life events (child rating) was associated significantly negative.

Parental depression variables also showed various significant correlations with the risk factors of children: for example the current status of depression showed significant negative

associations with adaptive emotion regulation strategies, positive life events, positive and negative attributional style of children. Similarly, the depressive symptoms of parents were correlated negatively and significantly to these variables, but not to the adaptive emotion regulation strategies.

The risk factors among each other were also showing intercorrelations: Maladaptive emotion regulation strategies were positively associated with negative life events. The maladaptive and adaptive emotion regulation strategies were not associated. In contrast, the positive and negative attributional style scale showed significant and high negative correlation. The positive attributional style was also correlated with small to moderate magnitude but significant to positive life events. The negative attributional style was correlated positively with positive and negative life events.

8.2.4.2. Summary H1.3b

There was evidence for significant and positive correlations of risk factors as maladaptive strategies, current status of parental depression and the parental depression with the children's depressive symptoms. Correlations were of small to moderate magnitude. In contrast, adaptive emotion regulation strategies, positive attributional style and negative life events were not associated.

In addition, the parental depression characteristics were linked negatively to the children's variables adaptive emotion regulation strategies, positive life events, positive and negative attributional.

8.2.5. Testing Hypothesis H1.3b

8.2.5.1. Assumptions of multiple regression

The data was explored for the numerous assumptions for multiple regression analysis and corrected if necessary[8]. There was no indication for multi-collinearity (all reported bivariate correlations < r = .80) that was additionally confirmed by the Variance-Inflation factor (VIF < 1 for all variables)

8.2.5.2. Results Hypothesis H1.3b

Table 15 displays the results of the multiple regression analysis that was performed on the basis of theoretical background. Therefore, four blocks of variables entered the regression model in order to account for the variance in the children's depression symptoms.

Table 15 Regression model summary

Step	R	R^2	Change in R^2	Change in F	Sig. change in F	*p*-value
1	.321	.103	.103	1.24	.302	.302
2	.499	.249	.146	5.05	.010	.029
3	.534	.285	.036	1.25	.291	.036
4	.606	.368	.082	1.50	.218	.037

Note. Dependent variable: children's depressive symptoms (DIKJ)

The first block was background variables of children variables (age, gender, IQ-score, type of school, socio-economic status) accounting significantly for 10.3 % of the variance. In the next step the parental depression characteristics were included (parental depression score (BDI-II); parental status of depression). Changes in R^2 were significant and the model accounted significantly for 24.9 % of the variance. In the third step, potential moderator variables (positive and negative life events) entered the regression model with resulting significant changes in R^2 and further 3.6 % (total: 28.5 %) of accounted variance. In the last step,

[8] Assumptions of multiple regression analysis: quantitative or categorical variables, the criterion quantitative, continuous and independent, non-zero-variance of predictors, homoscedasticity of residuals, confirmed by P-Plots exploration and Durban-Watson Test confirming non-correlation of residuals (all values < 2)

potential mediator variables as adaptive and maladaptive emotion regulation strategies, positive and negative attributional style) were included resulting in R^2 = 36.8 % of accounted variance and further significant changes in R^2. Table 16 provides information of beta-weights standard errors and *p*-values of all predictor variables.

Table 16 Coefficients of regression model

Step		Beta	SE	Standardized Beta	*p*-value
1	Constant	6.70	6.36		0.30
	Age	0.46	0.30	0.26	0.13
	Gender	0.48	1.13	0.06	0.68
	IQ-score	-0.07	0.05	-0.21	0.15
	School-type	-0.06	0.53	-0.02	0.92
	SES[1]	0.40	1.03	0.05	0.70
2	Constant	-1.49	6.50		0.82
	Age	0.48	0.28	0.27	0.10
	Gender	0.95	1.07	0.11	0.38
	IQ-score	-0.03	0.04	-0.10	0.47
	School-type	0.08	0.50	0.03	0.87
	SES	0.03	0.97	0.00	0.98
	Depressive status parent	2.34	0.76	0.51	0.00
	Depressive symptoms parent (BDI-II)	-0.07	0.06	-0.19	0.23
3	Constant	-2.82	6.53		0.67
	Age	0.50	0.28	0.28	0.09
	Gender	1.12	1.07	0.13	0.30
	IQ-score	-0.04	0.05	-0.12	0.41
	School-type	0.03	0.50	0.01	0.96
	SES	-0.28	0.99	-0.04	0.78
	Depressive status parent	2.42	0.78	0.52	0.00
	Depressive symptoms parent (BDI-II)	-0.07	0.06	-0.18	0.26
	Positive life events	0.30	0.30	0.14	0.32
	Negative life events	0.25	0.27	0.12	0.37
4	Constant	0.04	9.37		1.00
	Age	0.56	0.28	0.31	0.05
	Gender	0.45	1.10	0.05	0.69
	IQ-score	-0.03	0.05	-0.08	0.57
	School-type	-0.15	0.51	-0.05	0.76
	SES	-0.27	0.99	-0.04	0.79
	Depressive status parents	1.63	0.86	0.35	0.06
	Depressive symptoms parent (BDI-II)	-0.05	0.06	-0.13	0.42
	Positive life events	0.30	0.30	0.13	0.34
	Negative life events	0.26	0.27	0.12	0.35
	Positive attributional style	0.02	0.09	0.05	0.80
	Negative attributional style	-0.10	0.08	-0.27	0.20
	Adaptive emotion regulation strategies	-0.02	0.05	-0.06	0.66
	Maladaptive emotion regulation strategies	0.09	0.05	0.22	0.09

Note. dependent variable: child depression score (DIKJ); all variables are child variables except depressive status of parent and depressive symptoms parent (BDI-II).[1]SES- socio-economic status. Step 1: background variables, step 2; characteristics of parental depression, step 3: moderators, step 4: mediators.

In the next exploratory analysis, a regression model with forward selection of predictors was conducted. Table 17 displays the summary of the model, Table 18 coefficients of the resulting significant predictors.

Table 17 Regression model summary, forward selection

Step	R	R^2	Change in R^2	Change in F	Sig. change in F	*p*-value
1	.406[a]	.165	.165	16.37	.000	.000
2	.513[b]	.263	.098	10.91	.001	.000
3	.555[c]	.308	.045	5.29	.024	.000

Note. dependent variable: child depression score (DIKJ); all variables are child variables except depressive status of parent and depressive symptoms parent (BDI-II)

The factors maladaptive regulation strategy, negative life events and the parental depression status significantly predicted the children's depressive symptoms and accounted for 30.8 % in the variance[9]. Thereby, the predictor status of parental depression had the highest beta-weight.

Table 18 Coefficients of regression model, forward inclusion of predictors

		B	SE	Stanardized Beta	
1	Constant	-1.73	2.18		.429
	Maladaptive emotion regulation strategies	0.19	0.04	0.40	.000
2	Constant	-4.26	2.20		.056
	Maladaptive emotion regulation strategies	0.17	0.04	0.37	.000
	Depressive status parents	1.64	0.49	0.31	.001
3	Constant	-4.48	2.14		.040
	Maladaptive emotion regulation strategies	0.15	0.04	0.32	.001
	Depressive status parents	1.64	0.48	0.31	.001
	Negative life events	0.47	0.20	0.21	.024

Note. dependent variable: child depression score (DIKJ); all variables are child variables except depressive status of parent and depressive symptoms parent (BDI-II)

The Bayse factor revealed in a decisive effect for the whole model with BF_{10} = 68 034.71. Furthermore, there was strong to decisive evidence for the predictor variables *maladaptive emotion regulation strategies* (BF_{10} = 502.63) and *current status of parental depression* (BF_{10} = 66.81) and *negative life events* (BF_{10} = 15.59).

[9] The same model was tested with the child's general psychopathological (parent rating, CBCL; child rating, YSR) symptoms as independent variables. For the YSR, only the maladaptive emotion regulation strategies and negative life events contributed significantly to the model: R^2 = .230; Only current status of parental depression was a significant factor, when the CBCL score was predicted: R^2 = .212.

8.2.5.3. Summary Hypothesis 1.3b

Hypothesis H13.b was partly confirmed. The first stepwise hierarchical regression model including all background variables, parental depression characteristics, moderators and mediators revealed in 36.8 % of accounted variance in the children's depressive symptoms. The last exploratory analysis of relevant risk factors for depressive symptoms resulted in three prevalent predictors accounting for 30.8 % of the variance: current status of parental depression, maladaptive emotional regulation strategies and negative life events predicted the children's depressive symptoms. The attributional style did not account for the variance in dependent variables. Calculations on the Bayse factor supported the model indicating a decisive effect, especially for the predictors maladaptive regulation strategies and current status of parental depression.

9. Discussion study I

Capitalising on data collected from families being recruited to an intervention study (see study 2), the present study sought to investigate the factors that predicted vulnerability for depression in the children of depressed (n = 74) vs. non-depressed (n = 38) parents. This study aimed to replicate the findings about the increased risk for depression in children of depressed parents compared to children of non-depressed parent (H1.1a). On top of that, the association of parental depression with subclinical depressive symptoms in children was estimated (H1.1b). In the second hypothesis (H1.2), the most prevalent emotional (adaptive and maladaptive emotion regulation strategies), cognitive (attributional style) factors and life events were compared between the low and high-risk group. At last (H1.3a), the association of modifiable risk factors with youth depression and their impact on the children's depressive symptoms (H1.3b) were explored.

9.1. Summary findings

The present study supports the primary hypothesis (H1.1a) that children of depressed parents show significantly increased depressive and psychopathology symptoms compared to children of non-depressed parents with a decisive effect size (d = 1.75). These differences were shown by values in all measures of self- and parent-reported depressive and psychopathological symptoms. Correlations between children's depressive symptoms and parental history of depression or depressive symptoms were small to moderate and significant (H1.1b, r = .23 – 38). The Bayesian statistics supported the rejection of the null hypothesis by indicating a strong effect for both analyses.

There was further evidence for the second hypothesis (H1.2), indicating statistically significant group differences in emotion regulation strategies (d = 0.77), positive and negative attributional style (d = 0.46) and positive and negative life events (d = 0.78) of children with

and without parents with depression. More specifically, children of the high-risk group tended to show less adaptive emotion regulation strategies (e.g. acceptance, cognitive reappraisal) when they were confronted with anger and anxiety. Non-statistically significant trends suggested that they tend to conduct more maladaptive regulations strategies (e.g. withdrawal, deny). In line with the expectations, children of the low-risk group showed a more positive attributional style than children of parents with depression, what was supported by the Bayes statistic with moderate evidence. In contrast to the hypothesis, children of the high-risk group did show less negative attributional strategies than children of the low-risk group. The Bayes factor indicated only anecdotal support against the null-hypothesis. There was little evidence that positive and negative life events had an impact on children's psychopathology (most univariate *p*s > 0.05), although the high-risk group did report less positive life events than the low-risk group. The impact of positive and negative life events as well as the number of positive life events did not differ significantly.

In addition, there was evidence that risk factors as maladaptive strategies, current status of parental depression and the parental depression score were associated significantly with the children's depressive symptoms (H1.3a). Furthermore, positive life events were correlated negatively with the variables. Correlations were of small to moderate magnitude. In addition, the parental depression characteristics were linked negatively to the children's variables adaptive emotion regulation strategies, positive life events, positive and negative attributional.

In the last hypothesis (H.3b), three prevalent risk factors for depression were identified: maladaptive emotion regulation strategies, negative life events and the status of parental depression accounting significantly for the variance in the children's depressive symptoms with 30.8 %. Again, the Bayesian statistic confirmed decisive evidence for this, but only for the predictors maladaptive emotion regulation strategies, status of parental depression

and negative life events. Thereby, maladaptive emotion regulation strategies and parental depression had the same and more impact on the children's depressive symptoms than negative life events.

9.2. Interpretation of findings

By demonstrating significantly more psychopathology and depressive symptoms among the high-risk group with a decisive effect size, the data replicates earlier findings that children of depressed parents (Goodman & Garber, 2017; Goodman & Gotlib, 1999a; Weissman, et al., 2006). Moreover, it was shown how the parental depression as well as the children's vulnerabilities is associated with the children's depressive symptoms.

The data confirms more recent studies that found that children of depressed parents tend to conduct less positive coping strategies (Compas et al., 2010). Furthermore, the link of less adaptive coping strategies and a less positive attributional style to the children's depressive symptoms was approved (Braet et al., 2015; Dearing & Gotlib, 2009; Horowitz et al., 2007; Huberty, 2012; Schäfer et al., 2016). Although there is a consensus on the association of emotion regulation (Ehring et al., 2010; Schäfer et al., 2016) and cognitive factors (Abela & Hankin, 2008; Auerbach et al., 2014; Braet et al., 2015; Mathews & Macleod, 2005) and negative life events (Colodro-Conde et al., 2017; Oppenheimer et al., 2017) with the development of depression, current researches mostly refer to community samples or children of depressed parents only (Compas et al., 2010; Hayden et al., 2014; Horowitz et al., 2007) or did not compare groups in case both groups were represented in the sample (Evans et al., 2015).

In contrast to the expectations, the high-risk group showed significantly less adaptive emotion regulation strategies but did not differ from the low-risk group in maladaptive regulation strategies. Earlier findings and the present data showed that maladaptive emotion

regulation strategies are linked to depressive symptoms (Ehring et al., 2010; Schäfer et al., 2016). Since the high-risk group showed significantly more depressive symptoms, it is surprising that groups did not differ in this outcome variable. One explanation is that children of the high-risk group try to avoid using maladaptive strategies. A reason therefore might be that they avoid to act like their parents or parents might try to instruct their children actively how not to behave in a "depressive way". The sample consists mostly of well-educated parents that stated to be well informed about the diagnosis, symptoms and causes. Most parents gave the feedback that they know about the risk for depression in their children and that they worry about them. Due to their major depression, parents in this sample are more likely to show skill deficits in adaptive coping strategies than parents without mental health problems. Consequently, they might not be a role-model for adaptive emotion regulation strategies. Since most of participating parents were well experienced in psychotherapy, they might be well aware of their negative coping strategies and try to encourage their children in not behaving this way. Nevertheless, Goodman and Gotlib (1999) stated the opposite explanation (about how children adapt the "depressotypic" style of their parents) and this explanation is rather hypothetical. Further research and a bigger sample is needed to verify this effect.

Another finding that was partly unexpected is the differences in attributional style: Children of the high-risk group showed less positive, but also less negative attributional style than the comparison group. For the negative attributional style, the effect was only marginal, but is nevertheless contradictory to earlier findings about the association of negative thinking style and depressive symptoms (Braet et al., 2015). In addition, neither attributional style was correlated with depressive symptoms in the children. A reason therefore might be that the questionnaire (ASF) does not capture broadly enough the negative cognitive style children might adapt from their parents, since it only consists of 16 situations that have to be rated by

the child. Useful additional measures would include more components of the cognitive triad and self-esteem or more objective measures (e.g. experimental tasks, assessment of cognitive biases) for more reliable assessment of the general cognitive style. In addition, children responded that they cannot identify well with the given situations of the questionnaire and that they have difficulties in responding adequately. These reasons decrease reliability of the instrument and consequently the data.

Since parents with depression experience more stress (e.g. financial problems, unemployment), I expected to see more negative life events (e.g. divorce of parents) reported by children (Monroe et al., 2007; Pound et al., 1988). However, this was not the case. One reason therefore might be that children are not well aware of negative life events like financial problems, marital problems of parents, healthy issues of parents since parents might try to shield negative life events from their children in order to protect them. Nevertheless, children of depressed parents did differ significantly from their report of positive life events. Fewer positive life events might indirectly mirror the environment that goes along with parental depression (e.g. less family activities, holidays) and confirm the earlier findings about differences in the environment of the high-risk group. For example, in case of financial problems and conflicts in marriage or work, children might not experience directly those problems, but there might be lack of money, time and energy for positive activities what was mirrored in less positive activities (e.g. holidays).

Although in this study the most relevant factors that are associated with the development of depression were analysed (background variables: e.g. age, gender; mediators: emotion regulation, attributional style, moderators: life events; characteristics of parental depression), not all factors contributed significantly in the regression model. Only maladaptive strategies, the current status of parental depression and negative life events significantly predicted the children's depressive symptoms. In addition, the parental

depression characteristics were linked negatively to the children's variables adaptive emotion regulation strategies, positive life events, positive and negative attributional. Consequently, the factor parental depression status might represent more latent variables (genetic factors, the so called "depressotypic style") that is not "uncovered here" and therefore accounts for the variance in the children's depressive symptoms. It is possible that there are numerous conceptual overlaps of risk factors that cannot be further explored with the present data. The sample is limited in size to calculate structural equation models that would be necessary to explore the relations between latent data better. Furthermore, the data is cross-sectional and not longitudinal. Therefore findings must be interpreted with caution, since the data is correlational rather than causal.

Furthermore, the sample consisted of children without a diagnosis of a major depression that only showed subclinical symptoms of depression. Therefore, some risk factors might be less prevalent as in depressive samples.

9.3. Strengths

This is the first study exploring and investigating differences of numerous mediating and moderating risk factors in the offspring of depressed and non-depressed parents, in order to achieve a better understanding of the heightened risk for depression this group. In addition, those risk factors were explored concerning their association and predictive power of the children's depressive symptoms what is novel in the field. The data replicates findings from single studies about elevated symptoms and individual vulnerability factors in the offspring of depressed parents. Furthermore, these important risk and resilience factors are integrated. Differences in relevant risk factors for depression were observed between groups, contributing to the explanation of transmission of depression. Most importantly, it could be shown that although vulnerabilities in the child, like a maladaptive emotion regulation

strategy and negative life events are significant predictor for the children's depressive symptoms, the parental depression also accounted to the variability the children's depressive symptoms. This finding underline the theory of the model of transition (Goodman & Gotlib, 1999), but shows additionally how severe the impact of the parental depression on the children's well-being is. Moreover, the data enables a quantification of the impact of the most relevant risk factors. Due to the better understanding of these risk factors, clinical implications can be drawn. For example, prevention interventions can be tailored more specifically to the particular needs and skill deficits. Since children of depressed parents showed significantly less adaptive emotion regulation strategies and a less positive attributional style, clinical interventions should focus on these specific coping strategies. This is a highly relevant topic, since prevention interventions show only small to moderate effect sizes and tend to diminish over time (Hetrick et al., 2016). Based on the better understanding of problems and needs in this group, this knowledge can be used in order to increase efficiency and sustainability of prevention interventions.

Another strength is the sample size, since the recruitment of families, suffering from depression is challenging as these families face numerous daily stressors. In addition, due to depressive characteristics as loss of energy, motivation and interest, normally, these families are difficult to motivate for participating in studies, especially with their children. The sample size was big enough to detect group differences in the outcome variables what is important for generalizing effects. Other studies failed to recruit the sample for a prevention trial describing numerous reasons why these families are less motivated to take part in running trials (Phikala & Johansson, 2008).

9.4. Limitations

On the other hand, the mentioned sample might constitute a limitation of the study. The data of the high-risk group were collected from an opportunistic sample of families who were

recruited for an intervention (see *study II*). Taking part in a time consuming intervention with the whole family demands a high motivation to do so, especially for depressed parents, so the sample might be less representative. Furthermore, the socio-economic background of all families was high, indicating a less problematic financial and economic situation. In addition, there were hardly any parents suffering from severe depressive episodes in the sample and most of the participating families were German. The reason for this selective sample might be that families from low socio-economic background may be less interested in participating in on-going studies, due to their everyday stress. Families with e.g. a migration background or with a low socio-economic background often don't benefit from offers and initiatives. One reason therefore might be that they often face numerous other stress factors in their daily life, that there is less energy to join an additional program, especially an intervention program. Furthermore, information about causes of depression and the genetic contribution to the transmission of depression alarmed parents and motivated to take part in the study. A lack of this information might result in the opposite effect and non-activation for participating in a prevention program, since the children were not suffering from any mental illness yet. Consequently, high motivation, less severe forms of depression and a more comforting background are characteristics of the sample leading to less representativeness and challenge generalization of effects. Following this argumentation, the effects of the findings might be even stronger, since the families with more risk factors due to their socio-economic background are underrepresented. Nevertheless, this sample might be quite informative for future interventions for children of depressed parents, since this group would be the one possible to recruit.

Although missing values could be imputed, a limitation of this study is the amount of missing data. Since this study is about families with parental depression, it is not surprising that impairments of those families are mirrored in the response rates of questionnaires.

Families were encouraged and supported to fill in the questionnaires and reminded to send them back, nevertheless, many parents felt stressed by just another "to do" in their daily routine. Furthermore, the number of questionnaires was probably too high for this group. This hypothesis is supported by the observation of the missing data of the low-risk groups, the families with non-depressed parents. Here, the amount of missing data was low (n = 2) and families did not report to have problems with the questionnaires.

Moreover, the sample sizes consisted of unequal groups in the high (n = 74) and low (n = 38) risk group. Since the risk sample, in which more variability was expected than in the low risk sample, was sufficiently big that limitations should not have a vast impact on the results.

Another criticism is that the data is cross-sectional rather than longitudinal and therefore not allowing causal interpretations. Although the risk for depression in children with parents with depression appears evident, longitudinal data is necessary to secure the effect. In addition, a bigger sample would allow structural equation modelling and therefore enable the exploration of all several factors – including latent factors – in one model, in order to better understand the transmission of depression. The inclusion of other relevant depression related factors (e.g. children's self-esteem and temperament) would be beneficial therefore.

9.5. Future research

Given the infancy developmental pathways of depression for the offspring of depressed parents, there are numerous avenues for future research. Although this study combines several relevant risk factors in order to understand the transmission of depression, the data is rather exploratory. Longitudinal studies that base on representative and big samples are needed to explore the role of risk and protective factors that were found to be prevalent as maladaptive

emotion regulation strategies, parental depression and negative life events. Data that allows structural equation modelling in order to achieve a better understanding of impact, overlap and interaction of risk factors would be beneficial. Moreover, more reliable instruments for the assessment of the attributional style and other cognitive variables are needed. Further experimental data for verifying the effects of risk factors on the children's well-being might also be helpful for a better understanding of transmission of depression in this high-risk group.

Furthermore, future clinical research should focus on prevention of depression in this high-risk group, due to the high risk of the offspring of depressed parents and the lack of preventive offers for this group. In addition, those prevention programs only show small effects that diminish over time. One major finding was that children and adolescents of the high-risk group have less adaptive emotion regulation strategies and positive attributional style. Research on prevention programs in which the children's specific skill deficits are taken into account, need to be developed and evaluated for efficiency. By addressing skill deficits and increasing the children's resilience against depression, prevention interventions might boost their efficacy. This data provides evidence that vulnerability factors are particular relevant for this high-risk group and therefore provides a beneficial foundation for higher intervention effects. In *study II*, a promising prevention program that focuses on coping strategies is evaluated for its efficiency in the reduction of depressive symptoms and psychopathology in the offspring of depressed parents. Here, those factors are assessed at pre- and post-assessment in order to estimate their beneficial contribution.

9.6. Summary

In summary, data collected from 112 children and adolescents of parents with (n = 74) and without depression (n = 38) showed group differences in depressive symptoms and general psychopathology that is associated with an increased risk of incidence of depression.

The present data provides novel evidence on numerous vulnerability factors that play an important role in the development for this high risk: the offspring of depressed and non-depressed parents did differ in overall and adaptive emotion regulation strategies, overall, positive and negative attributional style and the amount of positive life events. Against expectations, groups did not differ in maladaptive emotion regulation strategies and the amount of negative life events. Furthermore, the association of children's and parents' depression characteristics was significant and of moderate size. The present study further provides novel evidence about most prevalent risk factors predicting the depressive symptoms in the offspring of depressed parents. In this sample, it was shown that maladaptive emotion regulation strategies, negative life events and the parental depression are the most important predictors among numerous other environmental, moderating and mediating factors in order to explain the children's depressive symptoms. Thereby, maladaptive emotion regulation strategies and parental depression had the same and more impact on the children's depressive symptoms than negative life events. In addition, the data provided evidence on how parental depression characteristics were associated negatively to the children's outcome variables as adaptive emotion regulation strategies, positive life events, positive and negative attributional that may are associated with the development of depression.

Although the data is rather exploratory, the theoretical framework of transmission of depression of Goodman and Gotlib (1999) is supported. Nevertheless, more longitudinal studies are necessary including more families, especially with a low socio economical background. Clinical implications are prevention programs that target skill deficits that were uncovered in *study I* (emotion regulation strategies, attributional style) in order to reduce the risk of the offspring of depressed parents.

Study II

Evaluation of the prevention intervention

„Gug auf –Gesund und glücklich aufwachsen!“

10. Introduction Study II

10.1. Theoretical background

As discussed in the first part of this work and shown in *study I*, the offspring of parents with depression face an elevated risk for depression (e.g. Weissman et al., 2006). Children and adolescents of depressed parents were shown to be three to four times more likely to develop a mental illness, compared to a community sample (Weissman, Wickramaratne, et al., 2006). In general, the rising number of depression prevention programs, which were developed in the recent decades, indicate that depression is to some extend preventable. This evidence is supported by reviews and meta-analysis, favoring targeted interventions over universal prevention programs (Hetrick et al., 2016; Stockings et al., 2016). Mostly small to moderate effect sizes on the reduction of internalizing or depressive symptoms are reported, that diminish over time. Surprisingly, among these prevention trials there is a limited number of studies, especially of randomised controlled trials that investigated the effects of prevention programs for the offspring of depressed children. The effect sizes were shown to vary from small to moderate, and diminish over time (Loechner et al., n.d.). Five interventions were detected that focus on this high-risk group and have been evaluated by randomized controlled trials (RCTs): i) *Family Talk intervention* (FTI, Beardslee et al., 1997), ii) *Project Hope* (PH, Mason et al., 2012) iii) *Coping with Depression* (CWD, Clarke, Hornbrook, Lynch, Polen, et al., 2001), iv) *Raising Healthy Children* (RHC, Compas et al., 2009) and v) *Parenting Training* (PT, Sanford et al., 2003b) (see section 4.3.1. for further details). Although the programs differ in a variety of characteristics (e.g. participants included, content, number of sessions), no differences in their efficacy were detected between groups in a recent meta-analysis (Loechner et al., n.d.). This might be the consequence of limited number of studies with great homogeneity that were included in the subgroup-analysis. Although the meta-analysis included too few studies to systematically investigate the factors that contribute to

most effective interventions, findings from the individual studies revealed some ingredients that seemed to characterise more effective interventions. For example, most researchers agree on the importance of psycho-education about the parental disease (Beardslee et al., 1997; Clarke et al., 2001; Compas et al., 2010; Garber, 2006). Another important ingredient of prevention interventions is the teaching of positive coping strategies in order to increase the children's resilience (Compas et al., 2015; Garber et al., 2009). As shown in the first part of this work, coping skills might buffer the negative effect of stress and decrease the children's vulnerabilities. Compas and colleagues reported the mediating role of children's coping strategies between the effects of the prevention program (RHC) and children's depressive symptoms, accounting for approximately half of the intervention effect (Compas et al., 2010). In addition, these basic CBT-techniques focusing on the improvement of coping skills in the therapy of depression are well examined and evidence based elsewhere (Zhou et al., 2015). Surprisingly, only few prevention programs included these contents. Another ingredient is the parenting training. Although it is well known that depressed parents display great skill deficits in positive parenting and interaction with their children (Hart, Newell, & Olsen, 2003), parenting skills are rarely included in existing interventions (Compas et al., 2010; Sanford et al., 2003a). Some prevention programs do not even include parents at all in their sessions (CWD, Garber et al., 2009), although there is evidence for the positive effect of open and positive communication in families. They are normally characterized by dysfunctional way of communicating (Stieglitz, 2002).

Compas and colleagues (2009, 2011) managed to include all these significant ingredients in their program. The program contains i) psycho-education, ii) CBT-techniques for improving emotional and cognitive coping strategies, iii) parenting training in a iv) family- and group setting. In addition, results of the reduction of internalizing, externalizing and depressive symptoms are very promising (d = -.42 at short-term follow up on depressive

symptoms). Especially the rates of the onset of depression at the 24- months' follow-up were impressive with 14 % incidence of depression in the experimental group versus 33 % onset of depression in the control group. Since long-term effects on this clinical highly relevant outcome measure are rare, this program appears to be the most promising. The more surprising it is that this intervention has never been replicated by an independent research group. Moreover, it remains unclear, whether the program works in a different cultural background. Cultural backgrounds vary between nations and were found to influence how people deal with mental illness (Glaesmer, Brähler, & Lersner, 2012). Consequently, it is necessary to replicate these findings outside the U.S.. In addition, it was shown that there exists a limited number of randomised controlled trials of prevention interventions in Germany (see section 4.3.2). It was showing *study I* that the offspring of depressed parents show skill deficits in important depression associated risk factors as adaptive emotion regulation strategies and positive and negative attributional style. Only few studies focused on the mediating role of these factors in prevention trials, reporting beneficial effects of teaching positive coping strategies (Compas et al., 2010) and a positive attributional style (Horowitz & Garber, 2006).

Therefore, *study II* focuses on the efficiency evaluation of the translated and culturally adapted program of *Raising Healthy Children* (Compas et al., 2010) in a randomized controlled trial. The adopted German version of the program *GuG auf – gesund und glücklich aufwachsen!* is evaluated concerning its effectiveness in reducing depressive symptoms and generally psychopathology of children of depressed parents. It is further investigated how underlying mechanisms like emotional regulation and attributional style that were examined in *study I* and are associated with the development of depressive symptoms, change within and between groups. Data was further collected on incidence of depression. Since the study is

ongoing, data on this outcome variable is not sufficient yet to warrant an analysis (< 20 % complete data at the last assessment time point).

10.2. Hypotheses study II

The following hypotheses are tested:

Differences in psychopathology. The first aim was to replicate the findings of Compas and colleagues (2009, 2011) on efficiency of the intervention in the reduction of depressive symptoms and general psychopathology in the offspring of depressed parents.

H2.1: Compared to the waiting control group, children of the experimental group show reduced psychopathological symptoms from baseline to post-assessment.

Differences in mediators. Secondly, underlying mechanisms, like i) emotional regulation and ii) attributional style that are associated with the development of depressive symptoms, are explored for changes between and within groups over time.

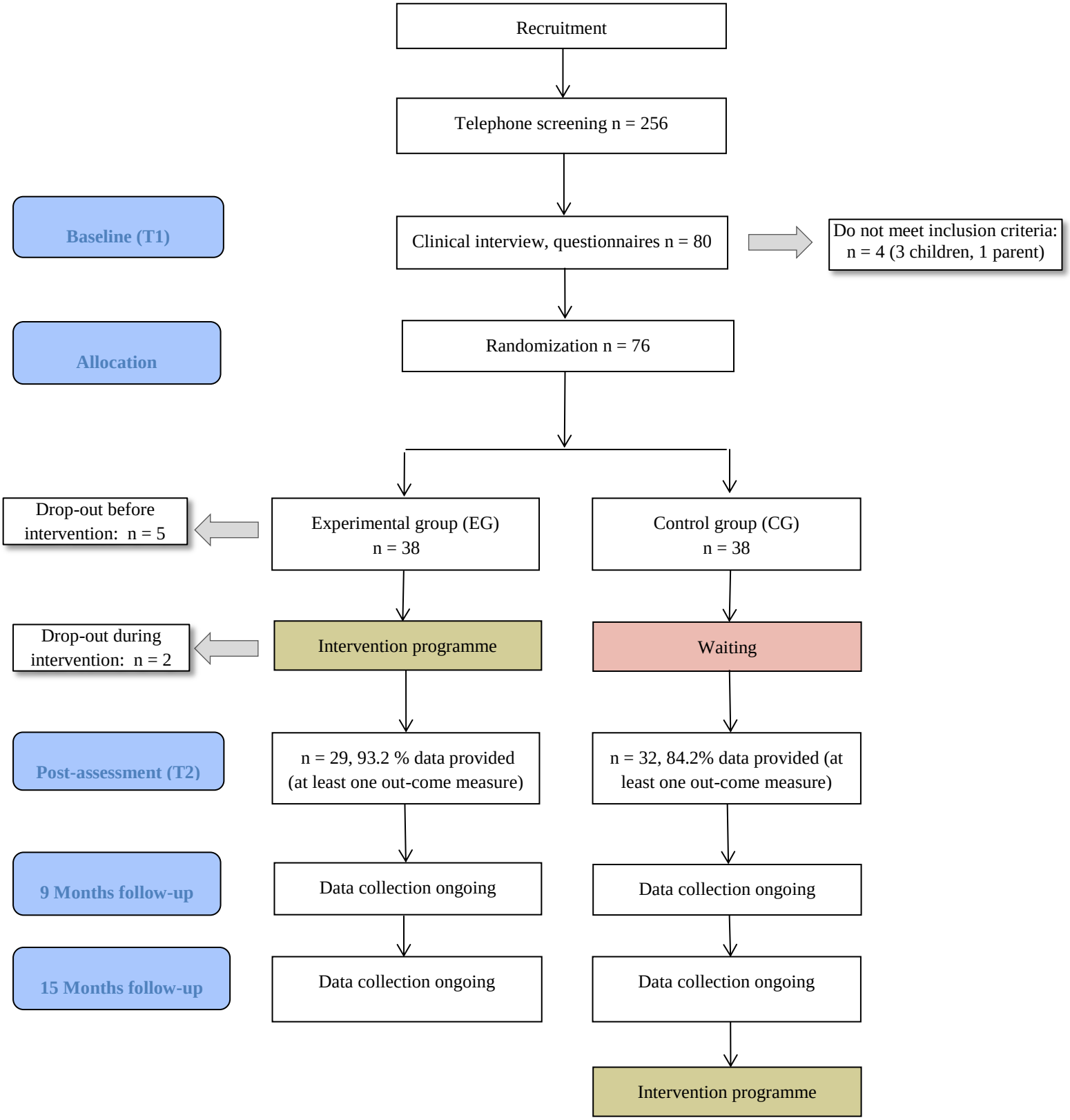
H2.2: Compared to the waiting control group, children of the experimental group show improved i) emotional and ii) cognitive coping strategies from baseline to post-assessment.

11. Method Study II

11.1. Study design

In a randomized controlled trial (RCT) conducted in line with the CONSORT statement, the efficacy of the prevention intervention *GuG auf – Gesund und glücklich aufwachsen* was evaluated. Psychiatrically healthy children and their parent with depression were allocated to either the group-based and cognitive-behavioural intervention (experimental group, EG) or to a waiting control group (control group, CG). In addition to the baseline assessment (T1) both groups were assessed immediately after the intervention at six months (T2), as well as after nine months (T3) and fifteen months (T4) after baseline. In the single blind design participants were aware of the allocated group, outcome assessors were not. Results of T3 and T4 are not reported, since the data collection is still ongoing. Figure 1 displays an overview of the study design. Since the study is still ongoing, only n = 61 of 76 recruited families reached T2 and were included in the analysis. In order to detect significant small effects with an alpha level of 5 % and power of 80 % a one-sided Fisher's exact test based on earlier findings of Compas and colleagues (2015) revealed in sufficient sample size (n = 43). In addition, the Bayse statistics will be run which also inform about the extent to which there is sufficient evidence for the null hypothesis.

Figure 1 Study design



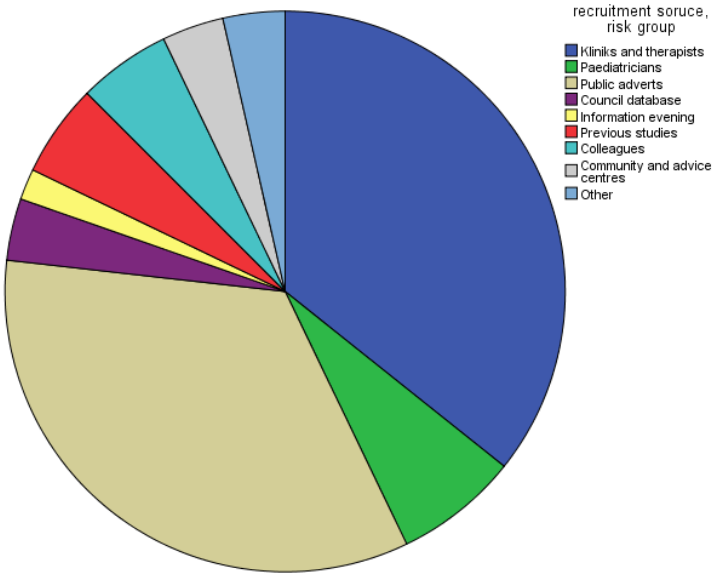
11.2. Participants

The n = 76 families were included in the study if a parent fulfilled the DSM-IV diagnostic criteria of a depressive disorder during the children's lifetime and a child (aged 8-17, IQ > 85) did not meet the diagnostic criteria for a psychiatric disorder in the present or past. Participants had to be fluent in German in order to be able to participate in the group setting. Parents were excluded if they suffered from alcohol or substance abuse, bipolar disorder, reported psychotic symptoms, had a personality disorder or a suicidal crisis. In case both parents were suffering from depression, both parents were entitled to receive the intervention as well as siblings, unless they were in a crisis or had severe psychological symptoms. Families, taking part in a similar family therapy training that might interfere with the intervention effects, were excluded.

Each family received 25 € at the beginning and the end of the study period as a reward for participating. All participants were informed about the study procedure and possible risks and gave their written consent for study participation. The ethic approval was positive, confirming that the collected data is in line with the Helsinki guidelines.

In the ongoing study, families were recruited at multiple sites in Munich, as psychiatry clinics, information centres, self-help groups, paediatricians, psychiatrists and psychotherapists. Another source was advertisement placed in newspaper and local radio. The local town administration supported the research group by providing contact information of families with children at the eligible age to offer the program directly. Parents or children who have been involved in previous research projects of the research group and were interested in being informed about new studies were also invited to take part. The largest group was invited due to direct contacts in clinics (26.0 %) and to newspaper articles (24.7 %). In graph 5 the distribution of recruitment sources are displayed.

Graph 5 Recruitment source



11.3. Procedure

Figure 1 illustrates the study procedure. The initial procedure of the study (recruitment, screening, assessment session T1) was identical to *study I*, described in 7.5. after the first assessment, a decision was made about the family's eligibility in the study. In case of severe psychological problems of the child, the family was excluded from the study and further information about potential sources of support was provided. When ten families were found to be suitable, randomization took place. Randomization was performed by a statistician in blocks of ten families (five per group) and stratified concerning the current status of parental depression (currently depressed or remitted) and the children's age. At six (T2) and nine (T3) months after baseline, families received outcome measure questionnaires by mail and were asked to send it back. At the fifteen month follow-up (T4), all participants were invited again for the final assessment, where a clinical interview was performed. Here, the 76 families were randomized to either the experimental (n = 38) or the control group (n = 38). Seven families (9.2 %) that were randomized to the intervention group dropped out before the intervention

started (n = 5) or during the six-month intervention period (n = 2), leaving 69 families (90.7 %) who reached T2. Seven families in the EG and one family in the CG had reached T2 but did not provide data on at least one outcome measure. There were complete data sets of at least one measure at post-assessment of 29 families (76.3 % of those randomised) of the experimental group (84.2 % of those randomised) and 32 families of the control group.

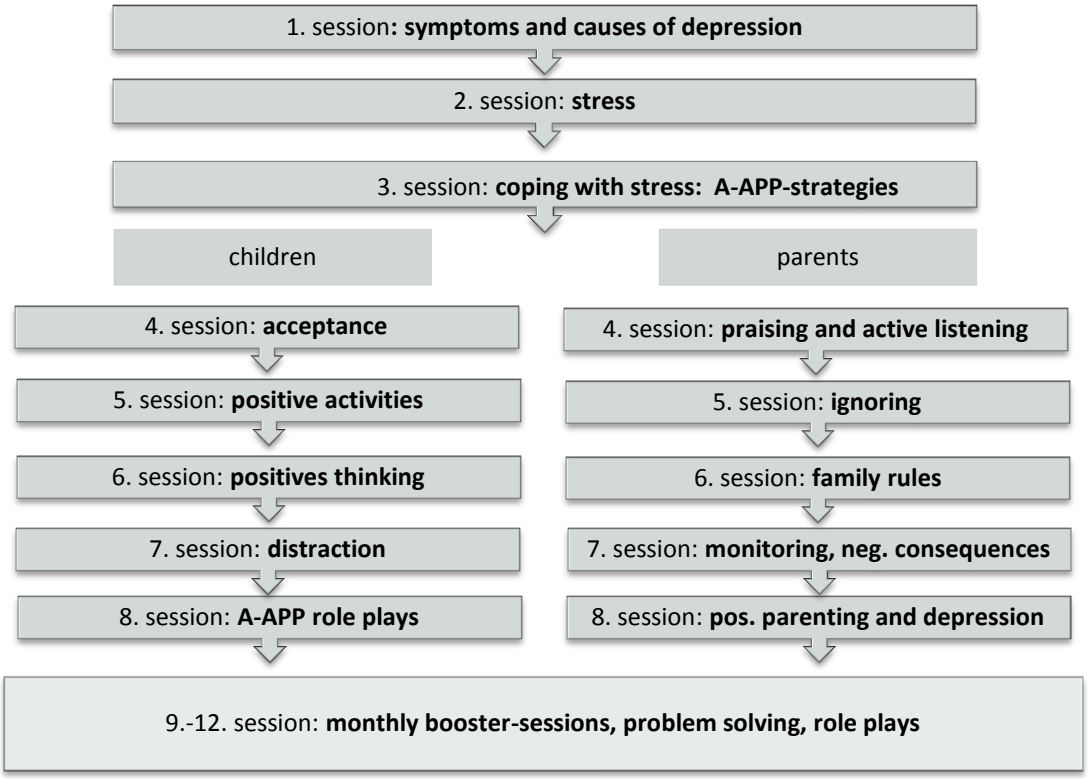
11.4. Intervention

The program *GuG auf – Gesund und glücklich aufwachsen* is the German replication of the original program *Raising Healthy Children (RHC)* by Compas and colleagues (2009). The manualized program (available upon request) is a group- and family- based cognitive-behavioural intervention targeting parents with depression and their psychiatrically healthy children. Figure 2 depicts the structure of the program. In eight weekly and four monthly booster sessions the basic ingredients are psycho-education, stress coping strategies and parenting training. In session 1-3, parents and children in a group of three to four families discuss depressive symptoms, causes of depression and the impact on the family. Additionally, they talk about stress and the individual family member's response to specific stressors. Four specific stress coping strategies are presented to the whole group in order to enable the parents to support their children when they practice these. In the following sessions, children are separated from parents after a starting ritual (talking about family activities) in order to practice the so called "A-APP" coping strategies that is an acronym for acceptance, distraction, positive activities and positive thinking (in German: Akzeptanz, Ablenkung, positives Denken, positive Aktivitäten). On the other hand, parents learn about positive parenting skills. Those skills consists of displaying a caring and warm behaviour, being consistent and structured in parenting and maintaining this positive parenting style also in acute depressive episodes. It is further discussed, how they can activate a supporting network in case of depressive days and to respond to personal early warnings of depressive

episodes to increase family bonding and positive time. Families are encouraged to spend qualitative time together and do fun activities. In the booster sessions individual stress situations are discussed and practiced in role-plays applying the A-APP coping skills, parent and children learned earlier in the program. In the last session the whole group is doing a quiz about contents of the past twelve sessions in family teams.

All group leaders were post-graduate psychologists or medical doctors that were trained in conducting the sessions. In addition, regular supervision was performed to further ensure treatment fidelity.

Figure 2 Overview of sessions "GuG auf- Gesund und Glücklich aufwachsen!"



The sessions took place in the conference rooms of the department of children and adolescents psychiatry, psychosomatic and psychotherapy.

11.5. Control condition

The control group was a waiting control group, in which participants could receive the program after the study period. Families were still allowed to take advantage of the usual care system. Like this, the program can be compared to the natural conditions and development of psychopathology of this high-risk group. However, intervention mechanisms and placebo effects cannot be tested in this design. To address this limitation, numerous hypothesized mechanisms of action are measured (children's coping skills, attributional style) in both groups.

11.6. Measures

Table 19 provides an overview of the assessment instruments that are described in detail in *study I*, section 7.4..

Table 19 Eligibility criteria and outcome variables

	Measure	Instrument
Eligibility criteria	Diagnostic status (child)	K-DIPS
	Intelligence test (child)	CFT 20-R
	Diagnostic status (parent)	DIPS, BDI-II
	Personality disorder (parent)	SKID II
	Psychopathology (2nd parent)	SCL-90-R
Main outcome variables	Depressive symptoms (child)	DIKJ
	Psychopathological symptoms (child)	YSR, CBCL
Secondary outcome variables	Emotion regulation strategies (child)	FEEL-KJ
	Attributional style (child)	ASF
Fidelity of intervention	Content of session	Self-generated checklist & video recording
	Presence of participants	Self-generated checklist
	Homework compliance	Self-generated checklist
Acceptance of participants	Feedback of participants	Self-generated questionnaire

Note. K-DIPS = Diagnostisches Interview für psychische Störungen, Child Version; CFT 20-R = Culture Fair Test. DIPS = Diagnostisches Interview für psychische Störungen, Parent Version; BDI-II Beck's Depression Inventory; SKID II = Strukturiertes Klinisches Interview für DSM-I; SCL-90-R = Symptomcheckliste. DIKJ = Depressions-Inventar für Kinder und Jugendliche; YSR = Youth Self-Report; CBCL = Child Behaviour Checklist;, FEEL-KJ = Fragebogen zur Erhebung der Emotionsregulation bei Kindern und Jugendlichen; ASF = Attributionsstil-Fragebogen.

Beside the measures that were implemented in *study I*, the fidelity of intervention and the acceptance of participants were assessed. For both measures, the research team developed checklists and questionnaires that mirrored the intervention content.

11.6.1. Treatment fidelity

Fidelity "(…) refers to the level in which the treatment as implemented matches the treatment as intended" (Summerfelt, 2003). It is crucial to test fidelity in order to directly attribute outcomes to the intervention but not to confounding variables enabling a more confidential interpretation of the results (Spillane et al., 2007). For maximizing the pure treatment effect and increase the level of fidelity, the influence of confounding variables must be minimized, e.g. by using a manualized intervention a priori. By testing for fidelity, other influence variables can be detected and taken into consideration at the post-intervention analysis. Objectivity, reliability and validity can be estimated.

The intervention is based on a detailed manual and consequently simplifies the standardized implementation in general (Compas et al., 2010, 2015). To further ensure treatment fidelity, all group leaders were well experienced in clinical psychology and had at least a master degree of either psychology or medicine. Furthermore, regular supervisions by the principle investigator (Belinda Platt) were performed in order to discuss problematic situations and possible deviations from the manual (e.g. how to handle acute crisis of parents). Moreover, an adherence checklist that included all relevant topics of the group sessions was provided by the developer of the program (see Appendix A). After each single session, the group leaders checked the fulfilment of the items (see Appendix A). For the sake of later examination, all sessions were videotaped. An independent researcher (Andrea Hauslbauer) who was not involved in conducting sessions, checked the completeness rates of 25 % randomly selected video tapes, following the adherence checklist.

11.6.2. Acceptance of participants

Participants were asked for feedback at the end of each session by an anonymous questionnaire (see Appendix B). The questionnaire was created by the research team and consisted of items that mirrored the intervention. Parents and children were asked to rate on a 5-point Likert scale whether they understood the content of the session (1 = "not at all"; 5 = "very well"), whether they participated actively (1 = "not at all"; 5 = "a lot"), whether they felt comfortable (1 = not at all"; 5 = "very much"), how they felt supported and understood by the group leader (1 = "not at all"; 5 = "very much"), how well they understood the homework assignment (1 = "not at all"; 5 = "very much") and how helpful they experienced the session (1 = "not at all"; 5 = "very much"). At the end of the questionnaire there was space for qualitative comments.

11.7. Analyzing Strategy

The data was analyzed using the statistic program SPSS Version 19 (SPSS Inc., 1989-2006) for Windows and JASP Version 0.8.1.1. for Mac OX s for calculating additional Bayesian statistics. T-values were used for the analysis for all outcome measures that provided standard tables (YSR, CBCL, DIKJ, ASF, FEEL-KJ) in order to control for age and gender. The oldest child from each family was chosen for the analysis in the experimental group.

Characteristics of the Intervention

- *Fidelity of intervention.* 25 % of the videotaped sessions across groups (n = 40, 20 videos of parent sessions, 20 videos of child sessions) were randomly selected, re-watched, and rated for adherence on the pre-defined adherence-checklist by an independent researcher (AH). An ANOVA, based on the percentage of items completed per session (DV), was calculated to examine fidelity differences between groups one to eight (IV). Any significant effects were followed by post-hoc tests and effect size calculations.

- *Acceptance of intervention*. Means and standard deviations of parent and child feedback questionnaire for all sessions were analyzed in order to estimate the participant's evaluation on the intervention.

Testing hypotheses

- For hypotheses H2.1 and H2.2 a one-factorial repeated measures univariate analysis of variance (ANOVA) was calculated with group as a between subjects factor (EG; CG) and time as a within-subjects variable (T1-T2). Significant effects were followed up with post-hoc tests. Due to baseline differences in the parent's rating of psychopathology of the child (ASF internal positive and negative scale), an ANCOVA with the ASF scores as covariate at baseline was calculated in order to evaluate differences between groups at post-assessment. In addition the Bayes factor (BF_{10}) was calculated in order to estimate the validity of the effect (see section 7.6. for further explanation).

11.8. Data preparation

11.8.1. Outlier

In order to detect outliers all variables were z-transformed and screened for values above +/- 3.29. There were just few outliers: In three cases they could be corrected, by adjusting the values to two standard deviations below the mean (see 7.5.1. for further explanation). Two increased values were found in CBCL and YSR scores that were reasonable for the analysis and therefore were not corrected.

11.8.2. Missing values

Table 20 displays an overview of missing outcome variables. The extend of missing values due to missing questionnaires or incomplete responses was ranging from 7- 42.6 % ($x_{missing}$ = 26.8 %), consequently above the critical values of 5 % suggesting non-coincidence (Rost, 2007, p. 177). Missing data was higher at post-assessment (31.4 %) than at baseline (5.6 %). Most missing values were found in variable YSR global score with 42.6 % missing values at post-assessment. Nine cases had more than 80.0 % missing values and were detected as drop-outs. Further four cases had more than 50.0 % missing values. The experimental and control group did not differ significantly in missing values ($t_{1,59}$ = .86; p = .419). Consequently, missing values were imputed based on expectation-maximization procedure (Stephens et al., 2001). This method enables imputation without changes in group means, standard deviations and covariance.

Table 20 Missing data, study II

Outcome variable	DIKJ		YSR		CBCL		FEEL-KJ		ASF	
	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2
n complete data	54	37	48	35	48	34	51	36	46	37
n missing	7	24	13	26	13	27	10	25	15	24
% missings	11.5	39.3	21.3	42.6	21.3	11.5	16.4	40.9	24.6	39.3

Note. DIKJ = Depressions-Inventar für Kinder und Jugendliche; YSR = Youth Self-Report; CBCL = Child Behaviour Checklist; FEEL-KJ = Fragebogen zur Erhebung der Emotionsregulation bei Kindern und Jugendlichen; ASF = Attributionsstil-Fragebogen; CASE = Child and Adolescent Survey of Experiences.

12. Results Study II

12.1. Sample description

A total of n = 61 families were analyzed for this study. Most of the participating families originated from Munich and surrounding areas 91.5 % of the sample was German, 8.5 % reported to have background from Turkey, Bulgaria and Austria. In general, families had a high economical background and parents were mostly well educated. Tables 21 and 22 display an overview of demographic and clinical variables of children and parent. Groups were comparable in all outcome variables and did not reveal significant differences, except in two variables (ASF positive internal score: $t_{1,44} = -3.35$, $p = .002$; ASF negative internal score: $t_{1,44} = -3.82$, $p = .000$). These differences were taken into consideration for the interpretation of the results.

Table 21 Demographic and clinical characteristics, children

	Experimental group n = 29	Control group n = 32	Total sample N = 61	*p*-value
Age				
Mean (SD)	12.20 (3.03)	12.30 (3.18)	12.25 (3.09)	
Range (min.-max.)	8-17	8-17	8-17	.909
Gender (%)				
female	58.6	53.1	52.7	.367
IQ				
Mean (SD)	103.3 (15.94)	109.7 (13.69)	106.55	
Range (min.-max.)	85-141	85-133	85-141	.086
Siblings (%)				
yes	85.2	73.3	78.9	
School type (%)				
Elementary school	38.6	41.3	40.0	
Secondary school	19.2	10.3	14.5	
High school	42.3	44.8	43.6	.986

Characteristics of psychopathology, children				
	Experimental group n = 29	Control group n = 32	Total sample N = 61	*p*-value
Self-report depressive symptoms (DIKJ)				
Mean (SD)	46.52 (8.75)	46.90 (7.05)	46.70 (7.87)	
Range (min.-max.)	36.00-69.00	38.00-63.00	36.00-69.00	.645
Self-report psychopathological symptoms (YSR)				
Mean (SD)	55.20(8.71)	50.43 (8.67)	52.91 (8.75)	
Range (min.-max.)	41.00–80.00	35.00-69.00	35.00-80.00	.154
Parent-report psychopathological symptoms (CBCL)				
Mean (SD)	57.75 (6.88)	53.54 (7.45)	55.65 (7.40)	
Range (min.-max.)	43.00-71.00	40.00–68.00	40.00-69.00	.137
Adaptive emotion regulation strategies (FEEL-KJ)				
Mean (SD)	45.11 (9.17)	46.40 (13.80)	45.74 (11.57)	
Range (min.-max.)	30.00-67	23.00-73.00	23.00-73.00	.707
Maladaptive emotion regulation strategies (FEEL-KJ)				
Mean (SD)	47.00 (9.8)	45.24 (12.23)	46.14 (10.99)	
Range (min.-max.)	25.00-67.00	20.00-71.00	20.00-71.00	.839
Attributional style				
Positive internal				
Mean (SD)	40.86 (8.32)	49.17 (8.37)	45.02 (9.29)	
Range (min.-max.)	31.00-62.00	36.00-69.00	31.00-69.00	.002
Positive stable				
Mean (SD)	49.69 (12.48)	50.69 (10.14)	50.19 (11.25)	
Range (min.-max.)	28.00-80.00	32.00-80.00	28.00-80.00	.525
Positive global				
Mean (SD)	48.00 (12.98)	48.60(12.43)	48.39 (12.58)	
Range (min.-max.)	32.00-80.00	24.00-84.00	24.00-80.00	.556
Negative internal				
Mean (SD)	39.04 (6.75)	48.40 (9.58)	43.85 (9.67)	
Range (min.-max.)	28.00-55.00	31.00-69.00	28.00-69.00	.000
Negative stable				
Mean (SD)	50.52 (9.84)	52.75 (14.07)	51.65 (12.41)	
Range (min.-max.)	36.00-71.00	29.00-99.00	29.00-99.00	.735
Negative global				
Mean (SD)	48.13 (11.06)	65.04 (12.34)	48.93 (10.27)	
Range (min.-max.)	21.00-72.00	21.00-72.00	21.00-72.00	.442

Note. SD = standard deviations, Min = minimum, Max = maximum.

Psychopathology of children. Of the resulting sample, 77.0 % of the children did not show any severe symptoms according to the DSM-IV criteria. Nevertheless, 23.0 % showed light

subclinical symptoms as sleeping problems (3.2 %), ADHD (5.4 %), specific phobia (3.2 %) depression (2.7 %) and tic-disorder (2.7 %), eating disorder (2.6 %) nightmare (1.6 %) obsessive compulsive disorder (1.6 %).

Table 22 Demographic and clinical characteristics, parents

	Experimental group n = 29	Control group n = 32	Total sample N = 61	*p*-value
Age				
Mean (SD)	46.63 (6.26)	47.78 (6.49)	47.17 (6.33)	.526
Range (min.-max.)	34-56	36-58	34.58	
Gender (%)				
female	58.6	68.8	63.9	.419
Education (%)				
Basic education	15.4	17.4	16.4	
A-levels	23.1	30.4	26.5	
University	42.3	47.8	44.9	
Doctoral degree	19.2	4.3	12.2	.384
Marital status (%)				
Single	3.7	8.3	4.9	
Married	81.5	83.4	72.6	
separated	14.8	8.3	11.8	.365
Single parent	14.8	20.8	17.6	.583
Employment (%)				
Employed	84.5	100	85.8	
Full time	60.0	32.0	64.6	
Part time	40.0	68.0	31.3	
Unemployed	3.8	0	2.0	
Retired	11.5	13.0	12.2	.805
Family income (%)				
– 2000 € /months	13.0	14.3	13.2	
2000 – 3000 € /months	21.7	28.6	25.0	
3000 – 4000 € /months	13.0	14.3	13.6	
4000 – 5000 € /months	31.7	14.3	18.2	
> 5000 € /months	30.4	28.6	29.5	.648

Characteristics of psychopathology, parents

	Experimental group n = 29	Control group n = 32	Total sample N = 61	*p*-value
Depressive Symptoms (BDI-II)				
Mean (SD)	17.5 (9.8)	18.15 (12.50)	17.86 (11.28)	.837
Range (min.-max.)	0-40	0-53	0-53	
Depressive Episodes				
Mean (SD)	7.27 (5.29)	4.9 (5.3)	6.11 (5.3)	
Range (min.-max.)	1-20	1-20	1-20	.156
Subjective impairment1				
Mean (SD)	5.00 (1.52)	5.3 (1.62)	5.16 (1.55)	
Range (min.-max.)	2-7	1-7	0-7	.477
Comorbid disorder (%)				
Anxiety	100	87.4	93.4	
other	0	12.6	6.6	.857
Currents status of depression				
Currently depressed	75.9	78.1	77.0	
Remitted	24.1	21.9	23.0	.945
Treatment experience (%)				
Psychotherapy	91.3	92.0	91.7	.933
Psychopharmaceuticals	87.0	69.6	78.3	.160
Clinic stays	69.6	69.6	69.6	.845

Note. SD = standard deviations, Min = minimum, Max = maximum. [1]Subjective general impairment (0 = none - 8 = very strong).

Psychopathology parents. Most parents were diagnosed with a recurrent depressive disorder that was remitted (23.0 %) moderate (12.5 %) or light (64.5 %). 10 % fulfilled the criteria for a double depression. The majority of the parents suffering from depression were female (56.8 %). Only 14.8 % experienced a single depressive episode in their lifetime. 11.5 % of the families consisted of two parents suffering from depression. The partner of parents with depression that reported not to be affected by a mental illness was also screened for psychopathological impairment and was showing no critical clinical scores (BDI-II x_{mean} = 5.6, SD = 6.03, range 0-15; SCL-20-R x_{mean} = 28.41, SD = 9.24, range 3-60). 15 % had slightly increased values on the personality disorder screening questionnaire (SKID II), but

only in one case the SKID II screening was clinically relevant and the family therefore excluded.

12.2. Characteristics of the intervention

12.2.1. Fidelity of intervention

Table 23 displays an overview of the percentages of completeness and number of sessions that were included in the analysis.

Table 23 Percentages of completeness of sessions

Group	1	2	3	4	5	6	7	8	total
# sessions included	5	5	5	4	5	5	5	5	39
% completed	98	100	100	100	97	100	96	100	98.9

Although 25 % of the videotaped sessions were randomly selected for analysis, only 22.5 % were analyzed, due to incomplete recordings. The average rate of completeness of intervention characteristics was high with 98.9 % of completed contents, with a range of 96.0 % to 100 %. No significant differences between groups were found ($F_{7, 39} = 1.16$, $p = .351$). Consequently, treatment fidelity does not differ between groups.

These findings support the thesis that the program has been delivered thoroughly concerning the intended intervention and that results can be interpreted with high fidelity.

12.2.2. Acceptance of participants

In Table 24 (parents) and 25 (children) the evaluation of the intervention (in means and standard deviations) rated by participants of all single sessions is displayed.

Table 24 Parent's evaluation of intervention program

	M	SD	Min	Max
Understanding the content	4.64	0.62	1	5
Active participation	4.02	0.75	2	5
Feeling comfortable	4.27	0.78	1	5
Feeling understood / supported	4.59	0.73	2	5
Understanding the exercises	4.44	0.65	1	5
Usefulness of exercises	4.14	0.77	1	5

Note. N = 25. M = Mean SD = standard deviations, Min = minimum, Max = maximum. (1 = lowest rating; 5 = highest rating).

Parents gave a lot of open feedback at the end of the single sessions. Most of them liked to exchange ideas with other families ("I learned a lot from the other parents", "I like the atmosphere") and that they liked the program in general ("it was great- as always!"; " I'm afraid it's over soon", "I feel understood and the group leaders are patient – thank you!"). Some parents also gave negative feedback ("sessions are too long with too much content!" "I had concentration problems with all the input!", "I don't like the negative consequences."- referring to the parenting section).

Table 25 Children's evaluation of intervention program

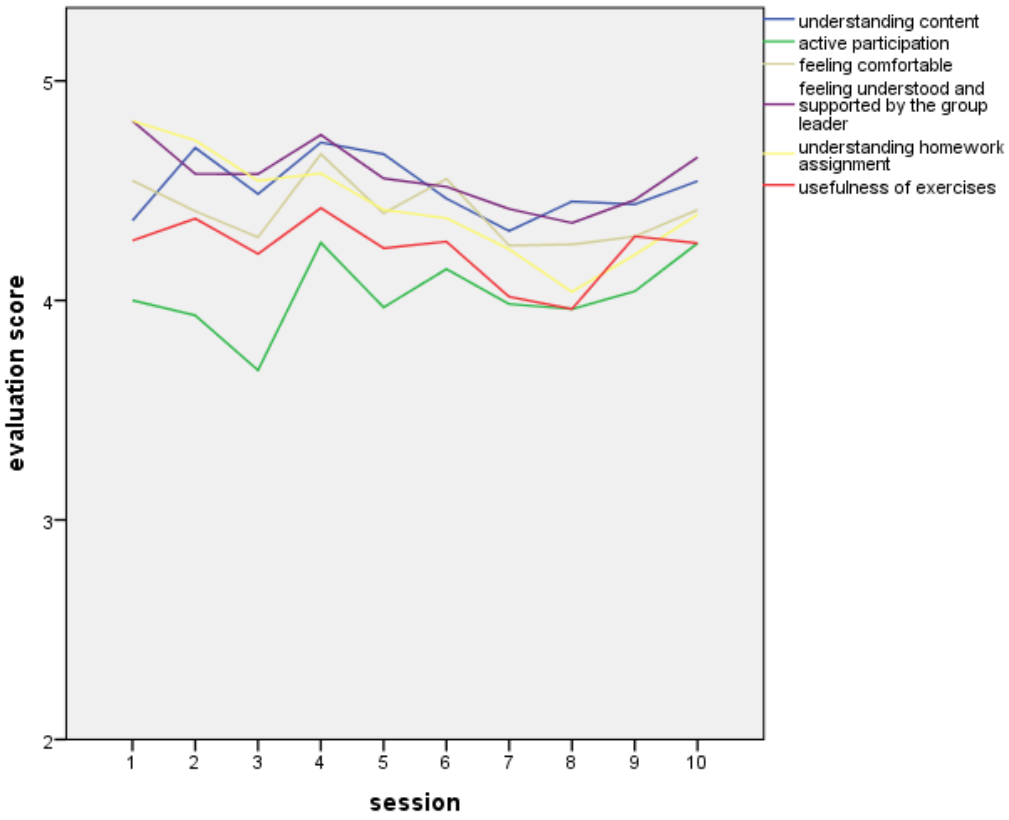
	M	SD	Min	Max
Understanding the content	4.50	0.71	1	5
Active participation	4.01	1.07	1	5
Feeling comfortable	4.48	0.81	1	5
Feeling understood / supported	4.52	0.71	1	5
Understanding the exercises	4.38	0.89	1	5
Usefulness of exercises	4.33	0.82	1	5

Note. N = 26. M = Mean SD = standard deviations, Min = minimum, Max = maximum. (1 = lowest rating; 5 = highest rating).

Children mostly commented positively in the open feedback, that they had fun ("That was fun! Everybody was laughing!", "Everything was great!") and liked the program ("grade = excellent!" "I wish, the program had more than only twelve sessions.", "I liked the role plays") and that the program was informative ("it was very revealing"). Children and adolescent also reported to feel comfortable in the group ("I felt very good"). Two critical

voices complained about too much input and that they preferred more games to play ("It was boring! I need more breaks!"). In graph 6, the means of the evaluation of sessions one to twelve of the six variables is displayed.

Graph 6 Evaluation of families, session 1-12



12.3. Testing hypotheses

In order to test the intervention's efficacy, repeated measures ANOVA was calculated for different outcome measures over time (baseline, post-assessment) and groups (experimental group; control group) as well as an ANCOVA for two subscales (positive and negative internalizing attributional style, ASF). In the following paragraph, change in groups over time is shown for psychopathological symptoms, emotion regulation and cognitive coping style.

12.3.1. Testing Hypothesis 2.1

12.3.1.1. Assumptions H2.1

The Kolmogorov-Smirnoff test revealed evidence that the majority of collected data were distributed normally. However, two variables revealed significance at T2 (DIKJ, YSR)[10]. For the evaluation of the extend of the violation, skewness and kurtosis were explored[11] as well as a visual check of the distributions and rated to be low. Due to the robustness of ANOVA of infringing premises of normal distribution and since all other data were normally distributed, an ANOVA was performed. The Box-M-test revealed mostly non-significance, implicating homogeneity of covariance matrices[12]. The Levene-test of homogeneity of variances was non-significant in all cases, indicating homogeneity of variances.

12.3.1.2. Results H2.1

Effects of the intervention on child self-reported depressive symptoms (DIKJ). Firstly, the depression score of groups were explored. Table 26 provides an overview of means, standard

[10] *Kolmogorov-Smirnoff statistic*: $DIKJ_{T2}$: K-S statistic = .13, df = 61, p = .013; YSR_{T2}; K-S statistic = 1.58, df = 61, p= .001

[11] *Analysis of skewness and kurtosis*: $DIKJ_{T2}$ s = 0.84/0.31 = 2.71 k = 3.64/0.61 = 5.68; YSR_{T2} s = -1.25/0.31 = 4.03, k 4.79/0.61=7.48;

[12] *Box-M-Test*: DIKJ: $F_{3,59}$ = 5.02, p = .002; YSR: $F_{3,59}$ = 4.24, p = .005, CBCL: $F_{3,\ 59}$ = 1.90, p = .126. *Levene-Test*: $DIKJ_{T1}$: $F_{1,59}$ = .004, p = .095; $DIKJ_{T1}$: $F_{1,59}$ = 3.31, p = .074; YSR_{T1}: $F_{3,59}$ = 1.02, p = .317; YSR_{T2}: $F_{3,59}$ = 2.10, p = .153,$CBCL_{T1}$: $F_{3,\ 59}$ = 0.01, p = .906, $CBCL_{T2}$: $F_{3,\ 59}$ = 0.54, p = .466

deviation and sample size per condition at baseline and post-assessment of the children's depressive symptoms (DIKJ).

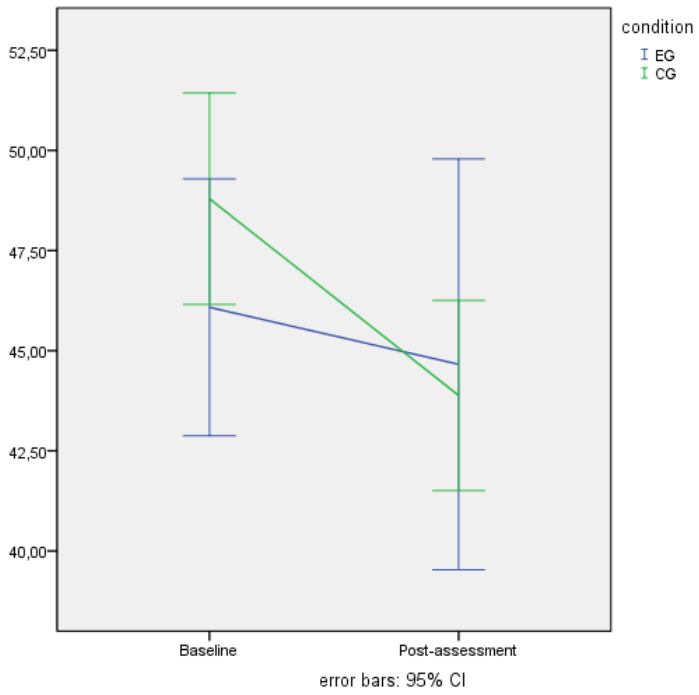
Table 26 Means and standard deviations per group, child-rated depressive symptoms (DIKJ)

	DIKJ	
	EG	CG
Baseline	46.68 (8.75)	48.04 (7.40)
Post-assessment	44.38 (12.14)	43.10 (7.10)

Note. Mean (SD).

Differences in means of the DIKJ scores were not significant concerning interaction ($F_{1, 59}$ = 1.45, p = .232, η2 = .03, d = 0.34, BF_{10} = 1.05). Both groups showed decreased values over time ($F_{1, 59}$ = 4.79, p = .032, η2 = .07, d = 0.54). Confirming this result, the Bayes factor revealed in a moderate effect favouring the null-hypothesis (BF_{10} = 4.79). Graph 7 provides an overview of means of depressive symptoms of both groups over time.

Graph 7 Means of the children's depression score (self-rated, DIKJ) of groups over time



Effects of the intervention on child self-reported psychopathological symptoms (YSR). In the next step, differences in means over time of the children's psychopathology were calculated. Table 27 provides an overview of means, standard deviation and sample size per condition at baseline and post-assessment of the child-reported psychopathology.

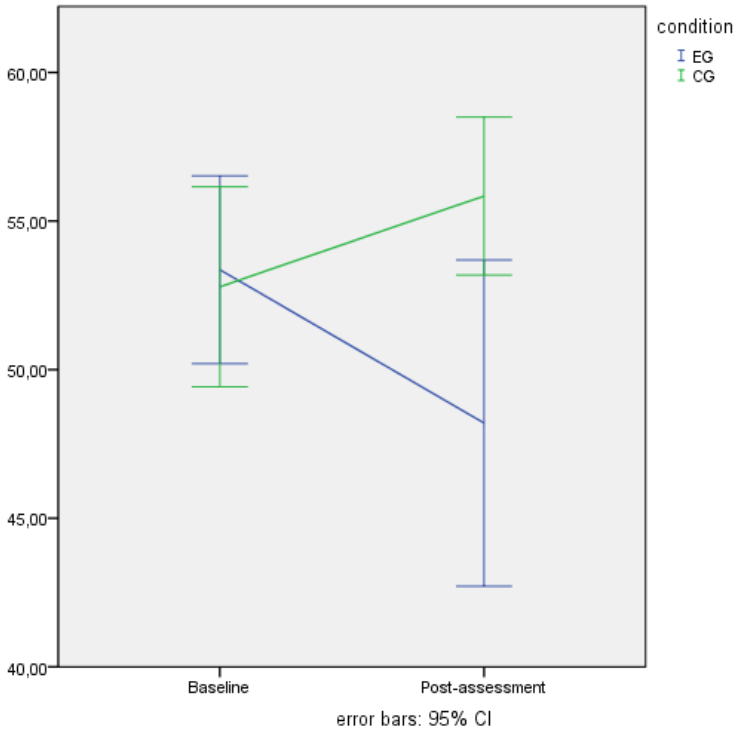
Table 27 Means and standard deviations per group, child-rated psychopathology (YSR)

	Group statistics	
	EG Mean (SD)	CG Mean (SD)
YSR global score		
baseline	53.31 (7.31)	52.78 (9.07)
post-assessment	48.82 (12.34)	55.72 (9.93)
YSR internalizing symptoms		
baseline	53.01 (9.9)	52.03 (12.1)
post-assessment	48.40 (9.5)	53.26 (10.5)
YSR externalizing symptoms		
baseline	50.56 (6.9)	51.24 (6.8)
post-assessment	49.94 (8.8)	53.17 (9.8)

A significant interaction of time and group ($F_{1, 59} = 7.79$; $p = .007$) of an effect size of $\eta^2 = .11$ (d = 0.81) was found for the global score of the YSR. The experimental group showed decreased ($t_{28} = 1.83$, $p = .079$), whereas the control group increased values over time ($t_{59} = -2.01$, $p = .052$). At post-assessment, the groups differed significantly from each other ($t_{59} = -2.85$, $p = .006$). A similar picture was observed for the subscales internalizing symptoms ($F_{1,59} = 6.63$, $p = .013$, $\eta2 = .11$, d = 0.68), and externalizing symptoms ($F_{1,59} = 8.24$, $p = .006$, $\eta2 = .12$, d = .73). Post-hoc t-tests showed that the control group showed increasing values in their YSR subscale scores (internalizing: $t_{31} = -1.65$, $p = .109$, externalizing: $t_{31} = -2.98$, $p = .005$) while the experimental group decreased over time (internalizing: $t_{28} = 2.18$, $p = .010$,

externalizing: $t_{28} = 1.27$, $p = .217$), leading to significant differences at post-assessment (YSR, internalizing: $t_{59} = -2.45$, $p = .028$; YSR, externalizing: $t_{59} = -2.46$, $p = .017$). Moreover, the Bayes factor revealed in a moderate effect favouring an interaction effect of time and group for the overall psychopathological symptoms ($BF_{10} = .5.00$), internalizing symptoms ($BF_{10} = 6.63$) and externalizing symptoms ($BF_{10} = 8.24$). In graph 8 the YSR global score of the intervention group (EG) and the control group (CG) is displayed over time at baseline assessment and post assessment.

Graph 8 Means of the children's psychopathology (self-rated, YSR) of groups over time



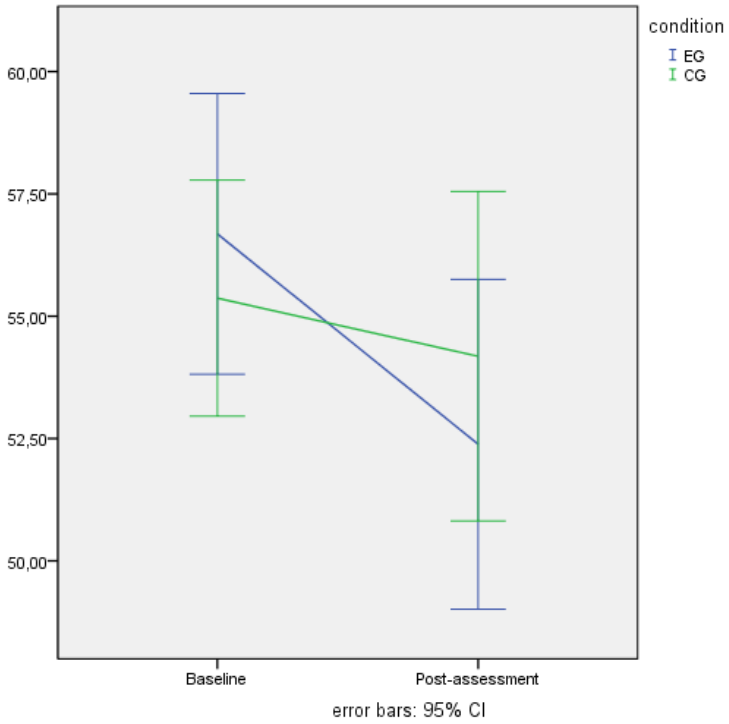
Effects of the intervention on parent-reported psychopathological symptoms of the child (CBCL). Table 28 provides an overview of means, standard deviation and sample size per condition at baseline and post-assessment of the parent-reported psychopathological symptoms (CBCL).

Table 28 Means and standard deviations per group, parent rated psychopathology (CBCL)

	Group statistics	
	EG Mean (SD)	CG Mean (SD)
CBCL global score		
baseline	56.5 (6.8)	55.3 (6.7)
post-assessment	52.1(8.06)	54.3 (7.34)
CBCL internalizing symptoms		
baseline	57.4 (9.7)	59.4 (6.9)
post-assessment	53.5 (8.8)	57.6 (9.1)
CBCL externalizing symptoms		
baseline	53.1 (6.5)	50.7 (7.1)
post-assessment	50.1 (7.5)	49.4 (7.6)

Both groups showed significant changes over time in their parent-rated general psychopathological outcomes ($F_{1,59}$ = 5.31, p = .025, η2 = .08, d = 0.58), but did not differ from each other ($F_{1,59}$ = 1.71, p = .196, η2 = .03, d = 0.34). This picture was also observed in the internalizing subscale of the CBCL: both groups showed reduced internalizing ($F_{1,59}$ = 6.70, p = .012, η2 = .10, d = 0.66) over time, but did not differ from each other (internalizing subscale: $F_{1,59}$ = .083, p = .774, η2 = .01, d = 0.20). For the subscale externalizing symptoms, there was no effect either of time ($F_{1,59}$ = 2.48, p = .121, η2 = .04, d = 0. 14) or interaction ($F_{1,59}$ = 0.30, p = .585, η2 = .01, d = 0.20). The Bayesian statistics depicted anecdotal to moderate effects for changes of both groups over time in the parent-rated children's psychopathology (CBCL: global score: BF_{10} = 5.30, internalizing subscale: BF_{10} = 6.61; externalizing subscale: BF_{10} = 2.48). Graph 9 displays changes in means of groups over time of the CBCL global score.

Graph 9 Means of the children's psychopathology (parent-rating, CBCL) of groups over time



12.3.1.3. Summary H.2.1

Hypothesis H2.1 was partly confirmed. As predicted, there was a significant interaction effect of time and condition with decreasing self-rated psychopathology symptoms in the experimental group and increasing values in the control group over time. These developments revealed in significant group differences at post-assessment. The Bayse statistics indicated group this interaction of time and condition with a moderate effect favoring the rejection of the H_0. Although both groups showed decreased depression scores and parent-rated psychopathology (global score and internalizing symptoms) over time, there were no group differences in these outcome variables. The Bayes statistics revealed in moderate support of these findings.

12.3.2. Testing Hypothesis 2.2

12.3.2.1. Assumptions H2.2.

The data was normally distributed except in two subscales at T2.(FEEL-KJ maladaptive strategies, ASF negative)[13]. By visual check of the distributions and examining the skewness and kurtosis of each scale, the extend of the violation turned out to be low[14]. Since the ANOVA is quite robust against the violation of the normality no further corrections were made. Box-M-test was not significant for all analysis, implicating homogeneity of covariance matrices[15].

12.3.2.2. Results H2.2

Effects of intervention on emotion regulation strategies (FEEL-KJ). Table 29 provides an overview of means, standard deviations and sample size of all subscales of the FEEL-KJ.

Table 29 Means and standard deviations, Emotion regulation strategies (FEEL-KJ)

	Group statistics	
	EG Mean (SD)	CG Mean (SD)
Adaptive emotion regulation strategies		
Baseline	45.23 (9.41)	45.72 (10.56)
Post-assessment	50.11 (12.81)	49.24 (8.75)
Maladaptive emotion regulation strategies		
Baseline	45.03 (8.18)	47.65 (11.67)
Post-assessment	43.99 (10.22)	52.31 (6.74)

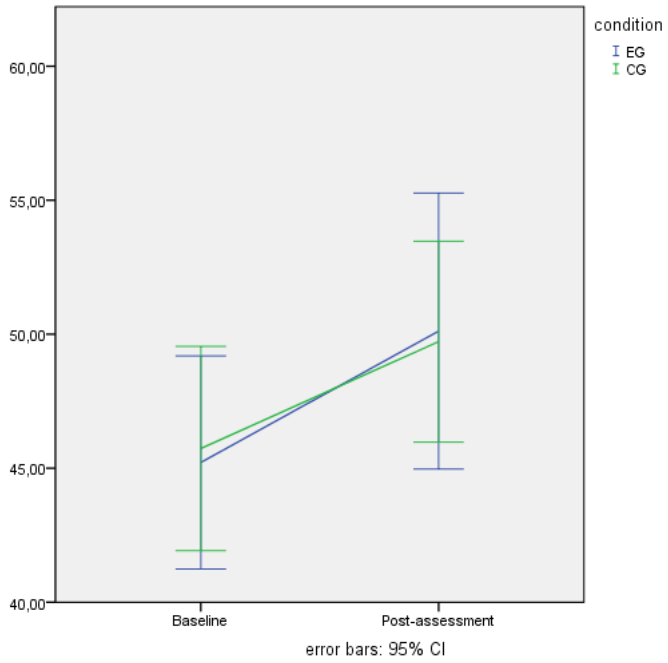
[13] *Kolmogorov-Smirnoff statistic*: FEEL-KJ maladaptive strategies$_{T2}$; K-S statistic = 0.13, df = 61, p = .010, ASF neg.$_{T2}$ K-S statistic = 0.15, df = 61, p = .001

[14] *Analysis of standardized skewness and kurtosis: Analysis of skewness and kurtosis*: DIKJ$_{T2}$ S = 2.80, k = 5.95; YSR$_{T2}$ s = -3.44, k = -7.48; FEEL-KJ maladaptive Straegies$_{T2}$ S = 0.04, K = 0.61, ASF neg.$_{T2}$ S = 0.68, K = 2.43

[15] *Box-M-Test*: adaptive strategies (FEEL-KJ): $F_{3,61} = 1.67$, p = .657; maladaptive strategies (FEEL-KJ): $F_{1,32} = 1.36$, p = .252; positive internal attributional style (ASF): $F_{1,31} = 0.256$, p = .857; positive stable attributional style (ASF): $F_{1,31} = 0.88$, p = .451; positive global attributional style (ASF): $F_{1,31} = 0.10$, p = .959; negative internal attributional style (ASF): $F_{1,31} = 1.94$, p = .121; negative stable attributional style (ASF): $F_{1,31} = 0.382$, p = .766; negative stable attributional style (ASF): $F_{1,31} = 1.25$, p = .290

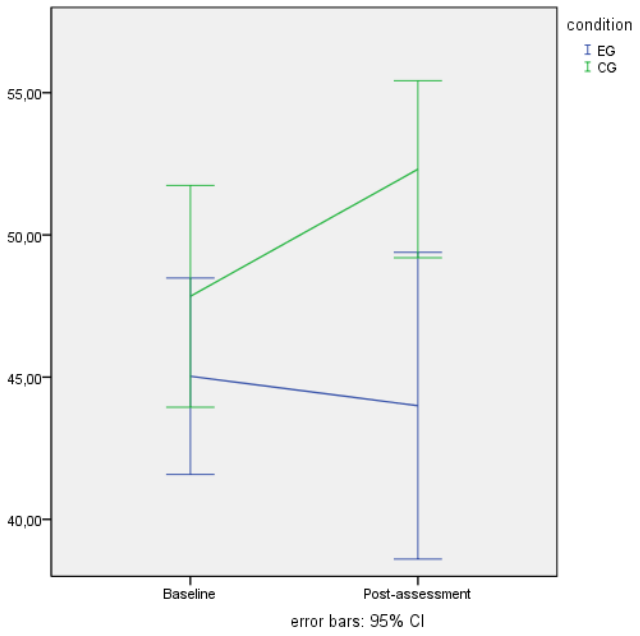
Both groups showed significant increased adaptive emotion regulation over time ($F_{1,59}$ = 8.56, p = .005, η2 = .13, d = .77), but did not differ from each other ($F_{1,59}$ = 0.09, p = .764, η2 = .00, d = .00). The Bayse statistics indicated a moderate effect of group differences over time (BF_{10} = 8.56). Graph 10 displays changes in means and error bars over time.

Graph 10 Adaptive emotion regulation strategies over time



In contrast, there was a significant interaction effect of time and condition in maladaptive strategies ($F_{1, 59}$ = 4.63, p = .035, η^2 = .07, d = 0.54). Here, the control group showed increased values (t_{37} = -3.32, p = .06) while the experimental showed non-significant decreased values over time (t_{28} = 0.44, p = .664), leading to significant group differences at post-assessment (t_{59} = -2.93, p = .005). This interaction effect was confirmed by the Bayse factor indicating a moderate support for the rejection of the H_0 (BF_{10} = 4.63).

Graph 11 Maladaptive emotion regulation strategies over time



Effects of intervention on attributional style (ASF). In table 30 means, standards deviations and sample size of all six subscales at baseline and post-assessment are provided.

Table 30 Means and standard deviations per group, attributional style (ASF)

	Group statistics	
	EG	CG
Attributional style	Mean (SD)	Mean (SD)
Positive internal		
Baseline	40.48 (8.33)	49.96 (7.92
Post-assessment	43.10 (8.70)	46.00 (8.28)
Positive stable		
Baseline	50.07 (11.99)	52.66 (10.47)
Post-assessment	54.91 (11.58)	52.04 (10.71)
Positive global		
Baseline	48.31 (12.61)	50.88 (11.26)
Post-assessment	48.60 (11.37)	54.44 (10.46)
Negative internal		
Baseline	37.72 (6.01)	48.82 (8.26)
Post-assessment	42.21 (8.28)	47.81 (5.81)
Negative stable		
Baseline	50.57 (9.81)	53.83 (12.31)
Post-assessment	52.61 (10.30)	56.86 (14.90)
Negative global		
Baseline	48.39 (10.84)	51.81 (9.63)
Post-assessment	50.84 (7.00)	54.81 (12.90)

Since the internal positive and negative attributional scales differed at baseline between groups, an ANCOVA was calculated for these subscales. Among the six subscales of the measure of the attributional style, there were significant effects on the positive and negative internal attributional style scale. There was a significant interaction effect in the internal positive attributional style ($F_{1, 59}$ = 3.5, p = .019, η^2 = .09, d = 0.62) indicating increased values for the experimental (t_{28} = -1.53, p = .139) and decreased values for the control group (t_{31} = -1.85, p = .079) over time. This effect relied mostly on the differences of groups at baseline (t_{59} = -3.85, p = .000), since differences at post-assessment were not significant (t_{31} = -1.06, p = .294). Confirming this, the Bayse factor of BF_{10} = 1.39 revealed only in an anecdotal effect. In addition, groups differed over time in the internal negative attributional style ($F_{1, 59}$ = 5.03, p = .029, η^2 = .08, d = 0.61) with significantly increased values of the experimental group (t_{28} = -2.94, p = .007) and non-significant decreased values in the control group (t_{31} = 0.48, p = .629) over time. In post-hoc t-test significant differences of groups were observed at post-assessment (t_{59} = -2.68, p = .010). Again, the Bayse statistics showed weak evidence for the rejection of the null-hypothesis (BF_{10} = 1.51). All results of the attributional strategies are shown in table 31.

Table 31 Results of ANOVA with repeated measure, attributional style (ASF)

Attributional style (ASF): positive subscales					
scale	effect	$F_{1, 59}$	p	η2	BF_{10}
stable	time	3.64	.061	0.06	1.00
	interaction	1.54	.219	0.03	0.64
global	time	0.90	.347	0.01	1.00
	interaction	1.46	.232	0.03	0.34
Attributional style (ASF): negative subscales					
scale	effect	$F_{1, 59}$	p	η2	
stable	time	2.09	.153	0.03	1.00
	interaction	0.33	.565	0.06	0.64
global	time	2.77	.101	0.04	1.00
	interaction	0.02	.875	0.00	0.74

Note. BF_{10} = Bayse Factor.

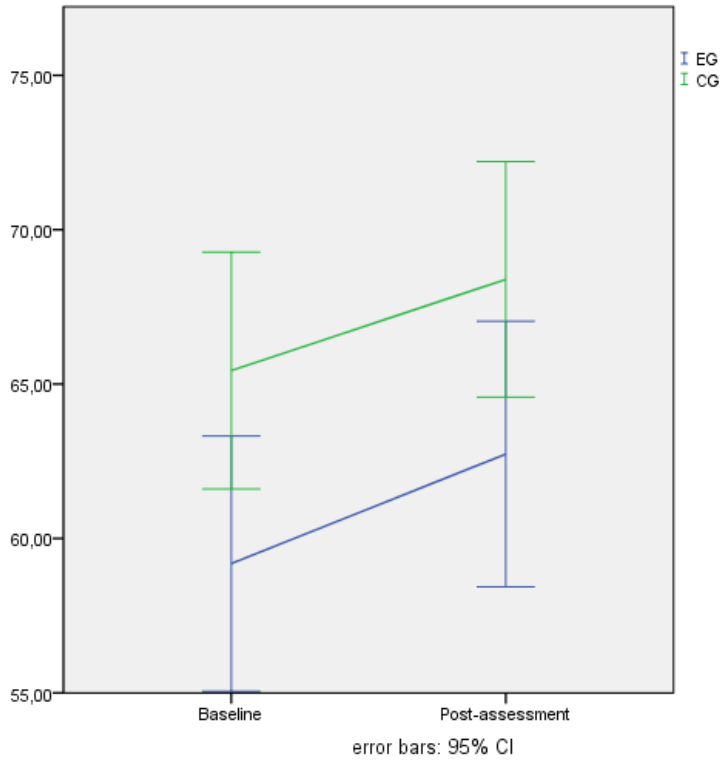
Since the power might be too low to discover effects in all six subscales of the 16-item questionnaire, post-hoc sum scores of positive and negative attribution style scales were built (s. table 32).

Table 32 Means and standard deviations per group, attributional style – (ASF) sum scores

	Group statistics	
	EG	CG
Sum scores of attributional style	Mean (SD)	Mean (SD)
Positive Attributional style		
Baseline	67.40 (10.58)	72.12 (10.32)
Post-assessment	69.82 (10.82)	72.85 (10.34)
Negative Attributional style		
Baseline	59.18 (9.78)	65.44 (11.51)
Post-assessment	62.73 (10.18)	68.39 (11.44)

Here, the groups did not differ over time in their positive attributional style ($F_{1, 59} = 0.79$, $p = .376$, $\eta^2 = .01$, d = 0.20; $BF_{10} = 0.87$) or change scores ($F_{1, 59} = 2.77$, $p = .101$, $\eta^2 = .04$, d = 0.40; $BF_{10} = 0.46$). In contrast data provided evidence for a significant main effect of time of the negative attributional style ($F_{1, 59} = 5.57$, $p = .022$, $\eta^2 = .086$ d = .61). This finding was supported moderately by the Bayse statistics ($BF_{10} = 6.39$). Groups did not differ from each other over time ($F_{1, 59} = 0.04$, $p = .830$, $\eta^2 = .00$, d = 0.00; $BF_{10} = 0.23$). Graph 12 displays the development means of the negative attributional style of groups over time.

Graph 12 Sumscore negative attributional style over time



12.3.2.3. Summary H2.2

Hypothesis H2.2 was partly confirmed. Both groups showed increased values of adaptive emotion regulation strategies over time, but did not differ from each other. The Bayse statistics indicated a moderate effect of group differences over time. As predicted, children of the intervention group showed significantly less maladaptive emotion regulation strategies over time compared to the control group. This finding relied mostly on significant increased maladaptive emotion regulation strategies of the control group. Again, this effect was confirmed by the Bayse factor with moderate support. In addition, there were significant interaction effects of time and group for the internal positive and negative attributional style: the experimental group showed a non-significant more positive, the control group a non-significant less positive attributional style over time. The difference in the internal positive attributional style mostly relied on differences at baseline, since groups did not differ from

each other at post-assessment. Confirming this, there was only anecdotal support for rejecting the null-hypothesis by the Bayse statistics. In contrast to expectations, the experimental group showed a significantly more negative internal attributional style over time as the control group and differed significantly from the control group at post-assessment. On the global scales of positive attributional scale, there were no differences of time and group. In contrast, both groups showed a significantly more negative more negative attributional style over time. The evidence was supported moderately by the Bayse statistics.

13. Discussion study II

This study aimed to evaluate the effectiveness of the translated and culturally adapted program of *Raising Healthy Children* (Compas et al., 2009) in a randomized controlled trial. The adopted German version of the program *GuG auf – gesund und glücklich aufwachsen!* was evaluated concerning its effectiveness in reducing depressive symptoms and generally psychopathology from baseline to post-assessment in children of depressed parents. It was further investigated how underlying mechanisms like emotional regulation and attributional style that are associated with the development of depressive symptoms and mediate the intervention effect, change within and between groups.

13.1. Summary of findings

The rating of treatment fidelity was high, indicating a very good accordance rate. In addition, participating families evaluated the intervention program very positive and gave positive qualitative feedback on the intervention concerning content, group leaders, atmosphere and general benefit. Along with hypothesis H1.1, there was a significant effect of interaction of time and group in the child-rated psychopathology. Against expectations, both groups showed decreased values in depressive symptoms and parents-rated psychopathology. Nevertheless, the experimental group showed significant reduced internalizing symptoms (parent and self-rating) from baseline to post-assessment. As predicted in hypothesis H2.2, groups differed in their emotion regulation strategies over time: the experimental group showed less maladaptive emotion regulation strategies compared to the control group over time. This finding relied mostly on significant increased maladaptive emotion regulation strategies of the control group resulting in post-assessment group differences. This effect was confirmed by the Bayse factor with moderate support. Surprisingly, both groups showed significantly increased adaptive emotion regulation strategies from baseline to post-assessment, but did not differ in this variable from each other. Furthermore, there were significant interaction effects of time and

condition in the internal positive and negative attributional style. The interaction effect of time and condition in the positive internal attributional style mostly relied on differences at baseline and were only supported anecdotally by the Bayse statistics. Against predictions, the experimental group also showed a significantly more negative internal attributional style over time and differed significantly from the control group at post-assessment. In addition, there was a significant main effect of time in the post-hoc built sum score of all negative attributional style scales indicating a more negative attributional style in both groups at post-assessment. The evidence was supported anecdotally by the Bayse statistics.

13.2. Interpretation

The high treatment fidelity rating increases reliability of current findings. Like this, group leader effects of other confounding variables were tried to diminish as much as possible enabling a valid interpretation of quantitative results. In addition, the positive rating of participants is a very important piece of information. Children and adolescents without subjective suffer pressure are asked to participate in a time-consuming intervention that targets the parental depression. One can easily imagine that especially adolescents prefer other activities to a two-hour weekly intervention with their parents, talking about difficult topics as depression. Nevertheless, especially children and adolescents gave a very positive feedback favouring the intervention. Rasing and colleagues (2016) found no significant group differences in a RCT on depression and anxiety prevention in schools and reported that the students did not like the intervention at all (Rasing, Creemers, Stikkelbroek, Kuijpers, & Engels, 2016). The authors interpreted this feedback as possible cause of non-significance.

As expected, the experimental group showed significant changes in most of the hypothesized variables. Against predictions, the comparison group also did. Although

children of the experimental group showed decreased values in depressive symptoms, parent- and child-rated psychopathological symptoms over time, the groups only differed in the child-rated psychopathology. Treatment effects were observed in a trend on the children's self-reported psychopathology and findings on two mediators (maladaptive emotion regulation and positive internal attributional style). Children in the experimental group showed significantly less maladaptive emotion regulation strategies and a more positive attributional styles. Except for the results in the attributional style, those trends were supported by the Baysian statistics indication moderate support for those effects. These findings are in line with Compas and colleagues (2010), who reported that the effect of a prevention intervention was mainly transmitted by coping strategies (Compas et al., 2010). Since the prevention program includes skill trainings for coping with stress and negative emotions, it can be assumed that the data reflects an improvement in these skills.

Similar findings were observed in prevention programs of Punamäki and colleagues (Punamäki et al., 2013; Solantaus et al., 2010). Although only a short personal contact was offered to the participants of the control group, both groups showed significantly decreased child-rated psychopathology and improved understanding of the parental depression. The authors argue that an extensive interview with the children about their fears and sorrows might have a beneficial effect, since these children normally do not get a lot of attention. Another reason might be that parents who sign in in this time-consuming program start to open up and talk to their children about their disease. Consequently, the offspring of depressed parents get information about the parental illness which might lead to more control and security. This was found to be a protective factor for this high-risk group (Lenz, 2009). In addition, the fact of signing in into a prevention program alone might activate families for seeking additional help, especially when they were not allocated to the experimental group. Those families might be motivated and ready to do an extensive program and be frustrated

when they hear about being randomized to the control group. Due to this activation and frustration, it is understandable, when families strive for information and support elsewhere rather than wait 15 months till the end of the study period.

In addition, the discrepancy between the parent- vs. child-rated psychopathology is noteworthy. That parents and children perceive the children's psychopathological symptoms differently, is well known (Choudhoury, Pimentel, & Kendall, 2003). Here, there was interaction effect in the child's rated instrument while parent rated decreased psychopathology symptoms in both groups. One reason therefore might be that the participating parents were suffering from depression and therefore have difficulties in observing symptoms in someone else, since depression goes along with loss of empathy. On the other hand, children might spare their parents with their psychological difficulties, since they avoid being a burden to them. Another explanation is the expectation of parents who sign in in to a time consuming program. Even if they are in the control group, they spent a lot of time by organizing and engaging in the program. As a consequence, they expect some improvement and might therefore be biased by filling in the questionnaires.

Although the data supported most of the hypothesis, the findings are mixed and not all predictions were met. The experimental group showed significantly increased negative internal attributions over time and both groups showed a more negative attributional style in general over time. In addition, the control group showed similar developments in reduced depressive symptoms, parent-rated psychopathology and increased adaptive emotion regulations. Firstly, it is possible that the program was superior in the reduction of the expected outcome variables, especially in depressive symptoms compared to the non-active control group. Effect sizes of prevention trials for this risk group were shown to be small in general (Loechner & Starman, et al., n.d.) and in some trials there were no effects at all on depressive and internalizing symptoms of children (Beardslee et al., 1997). In those studies,

outcome variables as “family communication”, “global functioning” or “understanding of the parental depression” were included and significant changes observed (Beardslee et al., 1997). Those measures were not included and assessed. Moreover, due reduced power possible effects might not be detected. As the study is on-going, the sample size is yet small, resulting in power deficits. Furthermore, the sample refers to families suffering from depression. On top of that, a big amount of missing values further reduced the power in the data, although imputation of missing data was conducted. Sample characteristics of families with depression might be the reason for this. Although the research team supported the participants continuously, many parents reported to feel “stressed” about the questionnaires. The reason therefore might be that parents with depression face numerous daily hassles (e.g. homework, managing the household and work). In the conducted sessions, many parents were complaining about their daily routines and that they felt overstrained. Parents gave the feedback that mentoring the children not only doing their homework but also encouraging them to fill in the questionnaires was sometimes too much. Consequently, the number of missing data was increased in the sample reducing the power. Taking this impairment into account and consequently including a bigger sample is necessary.

Most important, the data provides results of the first post assessment time point of an on-going study at six months after baseline, achieving to find change scores between groups in psychopathology, attributional style and emotion regulation strategies. Naturally, changes in these factors might not occur rapidly and a longer assessment period might be necessary to actually see changes in complex psychological patterns as thinking style, emotion regulation and consequently psychopathology. Although some trials found differences in depressive symptoms right at post-intervention (Garber et al., 2009; Punamäki et al., 2013) many researchers in the field of prevention of depression did not find significant changes in depression scores, right after the intervention (Clarke, Hornbrook, Lynch, et al., et al., 2001;

Compas et al., 2011; Sanford et al., 2003a). In the replicated study here (Compas et al., 2009) group differences that were not significant at the post-assessment (depressive symptoms, internalizing symptoms) reached significance at the 10- months and 12-month follow-up. A possible interpretation for this finding might be that children and adolescent need more time to integrate new-learned coping strategies, before the new skills are mirrored in their symptoms. Similarly, coping strategies as emotion regulation strategies and the attributional style might not be changed in a time period of sixth month only. Since there is little literature in the field of prevention of depression that took these mediating factors into account, further explorative studies are necessary. Although, there were some changes in the attributional style, not all scales showed changes over time. In addition, the Bayse factor supported most of the effects on the attributional style with anecdotal evidence only. Consequently, results on the attributional style are less trustworthy. Since cognitive factors are known to be more stable over time (Beck et al., 1979), children and adolescent might need a longer or more intense time period.

Another reason of undetected intervention effects in depressive symptoms might be due the depression measure (DIKJ). Recently, there is a lot of criticism in using those questionnaires in order to identify depressive symptoms that are rather heterogeneous and differ greatly between individuals who suffer from depression (Fried & Nesse, 2015). The major criticism is that those questionnaires lump all symptoms of depression together and neglect single characteristics. Those questionnaires do not provide information about single symptoms (e.g. hopelessness) that might have been affected by the intervention. Nevertheless, differences in changes score might get visible when children and adolescents finished the study and depressive symptoms are measured over a longer time period than only six months. At the 15- month follow-up, the onset of depression will be assessed by a clinical interview

that represent for this specific discussed case a more valid source for uncovering depressive symptoms for youth that tries to avoid trouble for their parents.

Still, the observed effects in different outcome variables are very promising for the efficiency of the program when the sample is complete, even though the statistical power might seem reduced. Furthermore, the acceptance of participants was extremely high. Regarding the high-risk group that is characterized by symptoms of depression (loss of motivation, energy and joy) and is therefore normally hard to reach, this finding is particularly interesting. In case participants enjoy sessions, the chance that they benefit from the intervention is much higher. In addition, it reflects that the content was adequate and that families felt understood and appreciated at the right point. Moreover, children of depressed parents did not suffer from depression and might not have felt the need for treatment. However, they rated the program to be very helpful and that they enjoyed the sessions.

13.3. Strengths

The study focuses on a research field that is neglected in Germany, although the risk and consequences of depression for the offspring of depressed parents is evident. This is the first randomized controlled trial evaluating one of the most promising prevention interventions that are currently existing (Compas et al., 2009). Besides the importance of the topic, this study attracts attention in its methodological quality. Treatment fidelity of the manualized intervention was excellent, ensuring reliability of the intervention. Furthermore, the majority of participants gave positive feedback about the sessions. They felt understood by the group leaders, liked the content of the sessions and responded that they benefit they take home was massive. This is an essential result, since it is not natural that children and especially adolescents enjoy time-consuming interventions with their parents about a difficult topic as

depression. Moreover, since the offspring of the participating families were not suffering from depression, they might not feel the need to receive help. This finding is mirrored in the low drop-out rate (n = 2) during the intervention enabling more positive intervention effects.

In addition, participants were randomly allocated to the intervention or control group, enabling visible effects of the intervention effects compared to the course of this high-risk group. An active control group might have the benefit of a more valid assessment of the prevention program, but has the disadvantage of hiding a "more natural" development of children and adolescent of this high-risk group. Although there is much research about characteristics of the offspring of depressed parents, there are only few longitudinal studies (Weissman, et al., 2006). Hence, we are able to show how surprisingly rapid some outcome variables have changed within only six months. As argued earlier, these processes are normally slow (Beck et al., 1979). Observing these quick changes in psychopathology, these developments are not only in line with other preventive interventions that had a non-active control group (Beardslee et al., 2013; Garber et al., 2009), but are alarming concerning the already known risk for depression in this subgroup. These findings further underline the importance of support and preventive interventions for this high-risk group.

Another advantage of the study design is the inclusion of numerous moderators and mediator variables that were proved in *study I* to be important in the development of depression (like attributional style and emotion regulation strategies). Only few studies on prevention trials reported underlying mechanisms as attributional style or emotion regulation strategies (Compas et al., 2010; Horowitz and Garber, 2006). This is very surprising, since effect sizes for prevention programs are small to moderate and information about mediating effects are essential for improving the effects (Hetrick et al., 2016). Based on the better understanding of problems and needs in this group, this knowledge can be used in order to increase efficiency and sustainability of prevention interventions.

Another strength is the sample size, since the recruitment of families, suffering from depression is challenging due to characteristics of depression. For example loss of energy, motivation and interest that go along with depression interfere with signing in to a time consuming program, especially with the whole family. On top of that, many parents told the research group not to have talked to their children about their disease yet. Although some might deny participation for this reason, many parents initiated open family talks and participated in the study.

13.4. Limitation

A major limitation is the high number of missing values, especially at the post-assessment. Although data could be imputed, the imputed values were created based on the present information of the sample. This fact might reduce the power to detect effects. A reason for the high percentage of the missing data might be the general impairment of these high-risk families. Although participants were supported at all times by the research team, many of the participating families felt stressed about the questionnaires due to their daily hassles. This fact mirrors how parents with depression might be easily stressed with tasks on top of their daily routines. Another reason of the high number of missing values might be the amount of questionnaires, children ha to fill in. Nevertheless, children of non-depressed parents, as reported in *study I*, did not report to have any problems with the amount of questionnaires. The amount of missing data in the low-risk group was low (n = 2). Still, these families only participated for one assessment time point and not for a study period of 15 months. Future research in this field could avoid difficulties like this by simplifying data collection by using electronical support. In recent years, apps were developed that are more attractive to fill in for children and adolescents and are less expensive to monitor for parents. Furthermore, these

apps enable completeness of items avoiding missing data due to not properly answered questionnaires. For this study, there was no such technical device available.

Another uncontrollable limitation is the sample representation. The average socio-economical background of the sample is not representing families with low socio-economical background that are often affected by depression and may face more stressors (e.g. financial problems, unemployment). In addition, most of the participating families were German. Families that participated were mostly high income families with well-educated parents and children. Perrino, Beardslee, & Bernal (2015) discussed the lack of "scientific equity". Certain minorities (e.g. racial/ethnic minorities, socioeconomically disadvantaged) are often neglected regarding psychosocial support and have limited access to quality mental healthcare, although they might be at high risk for developing depression. Therefore, the current study can be seen as rarely representative for ethnic minorities. The reason for this selective sample might be that families from low socio-economical background may be less interested in participating in a time-consuming intervention program that requires a lot of energy due to economical restrains. The present sample provided feedback that filling in the questionnaire is "stressful", families with a more problematic financial background might be having even more constraints. Another reason may be that many parents suffering from depression with a problematic economical background try to not to quit the job and do not seek professional help therefore. Due to financial restrictions in the study, there was hardly money for public advertisement especially in various newspaper that target minority groups. In addition, many of the parents the program was offered to did not want to participate due to numerous reasons: many had fears and felt ashamed to open up about their diagnosis or to overburden their children with difficult topics. Furthermore parents often may try to hide their illness due to possible loss of custody (Hearle et al., 1999). Although family support is fundamental in this case, joining a prevention program is challenging for the whole concerned

family. Another side effect of this selective sample is that the high motivation of participants may be reflected in the data. These well-educated families might be well informed about possible support in society, the depressive disorder itself and parenting strategies compared to families with a lower socio-economic status. In case there are skill deficits, highly motivated individuals also are more likely to soak up information to get help. Consequently, intervention effects might be either increased, according to the participant's commitment or decreased, due to high baseline competences. In addition, families that were allocated to the control group had the skills to achieve information and alternative support. Further research is needed with more representative samples to answer this question. To enable this, more funding for recruitment is necessary. In general, families without contacts to clinical doctors are more difficult to reach and inform about the studies. A higher budget for recruitment is inevitable for advertisement and information of the study for families that are not in contact with health institutions, for example in public radio and several newspapers that target minority groups. Moreover, families could be supported financially in their expanses to come to the sessions or rewarded with more than 50 € for participation.

Since the onset of depression was not assessed by a clinical interview at post-assessment yet the preventive effect of incidence of major depression was not captured. This will happen at the 15-month follow-up that is much more reasonable, since depressive symptoms are not to be expected to evolve among a period of only six months. Nevertheless, the outcome variables at the post-assessment are only proxy for depression prevention.

Moreover, there was no active control group included in the study. Although there are many advantages on a comparison with a non-active control group, like observing the natural development of this high-risk group, there are also some disadvantages. A comparable, active control intervention would enable a better estimation of the treatment effect, since confounding variables are reduced (such as e.g. attention by group leader, exchange with

families). The latest Cochrane review (Hetrick et al., 2015) indicated that effect sizes are higher in case no active control group was included in randomized controlled trials in the field of prevention of depression. Nevertheless, observing an untreated comparison group might enable to estimate the "natural course" of psychopathological development of this high-risk group. The actual risk can be evaluated as well as the benefit of a prevention program compared to the support that these at-risk families normally experience – that is marginal.

13.5. Future research

Generally, there is little research in the field of depression prevention for children of depressed parents. Besides this research group, two in Finland and one in Canada, most studies are conducted in the U.S. Only one more controlled, but not randomized trial was identified in Germany (Christiansen et al., 2015). Although many prevention programs seem to be efficient, findings of efficiency evaluation of prevention interventions are still heterogeneous, (Hetrick et al. 2016, Schulte-Körne & Schiller, 2012; Loechner, Starman et al., under rev.). More research and replications of existing trials is needed to achieve more homogenous findings.

More specifically, future research must focus on bigger samples including families with a lower socio-economic status for estimating the efficacy of prevention programs for children of depressed parents in a more representative way.

In addition, studies should take important moderators and mediators into account. For example, research groups (Beardslee 2013; Brent, 2015) found, that the efficiency of prevention programs depends greatly on the current status of parental depression. When parents were currently depressed, the former significant intervention effect disappeared. Although this was not the case in the present study, only few studies investigated in this

moderator. Moreover, from other fields like anxiety prevention for example, we know how substantial the question of the involvement of parents in interventions is (Siddaway, Wood., & Cartwright-Hatton, 2014; Warwick. et al., 2017). There are numerous important mediating and moderating variables like time period of program, size of intervention groups, family history of depression, negative live events of families, socio-economic background of families and age group of participating children. Future research must achieve a better understanding of the underlying mechanisms in order to improve the existing prevention programs.

The present study investigated in a CBT-based intervention and a non-active control group. There are other approaches in the treatment of youth depression as the interpersonal psychotherapy, that are efficient (Zhou et al., 2015). Surprisingly, few prevention programs focus on this approach, although one study found significant effects in a prevention trial for children and adolescents (Horowitz & Garber, 2006). Future research should investigate in different treatment approaches and compare those to each other and active and non-active control conditions.

It is further essential to explore cost effectiveness of interventions that is an important issue to focus on in future for enabling the dissemination of prevention programs to a broader population that is not only represented in study trials but communities.

13.6. Summary

The aim of the present study was to evaluate a prevention program for depression for the offspring of depressed parents in a randomized controlled trial. The main hypothesis was, whether children and adolescents of depressed parents show fewer depressive and general psychopathological symptoms from baseline to post-assessment than the waiting control group. In most of the scales children in the experimental group showed significantly lower psychopathology symptoms (CBCL, YSR) from baseline to post-assessment and differed

from the control group (YSR). In contrast to expectations, both groups showed significant decreases in depressive symptoms. Differences in changes scores of emotional regulation strategies were observed in maladaptive regulation strategies favouring the experimental group and positive internal attributional style. Against predictions, both groups showed increased adaptive emotion regulation strategies over time. In contrast to expectations, the experimental group showed a significant more negative internal attributional style than the control group, while a more negative attributional style in general was found for both groups over time. Treatment fidelity was rated excellent, as well as the acceptance of participating families. Most participants evaluated the program very positive.

This is one of the biggest studies about prevention programs for at-risk children in Germany so far. Future research must focus on bigger samples and include important moderators and mediators and calculate cost-effectiveness.

14. Conclusion

Depression is one of the most common psychiatric illness and with high prevalence rates already in childhood and adolescence, causing great personal and economic burden for individuals, families and society (Wittchen 2010; Costello, Mustillo, Erkanil, Keeler, & Angold 2003). In global comparison, the depressive disorder currently ranks the third place, but is projected to rise up to the top by 2030 (Mathers, Boerma, & Ma Fat, 2008). Consequently, preventing depression is a public health priority (WHO, 2004).

There are several theories about the interaction of risk and vulnerability factors increasing the risk of developing and maintaining depression, like e.g. the accumulation of risk factors (Lewinsohn et al., 1994, Sameroff 1998) or the interaction of those increasing the risk of depression for individuals with a certain diathesis (Abramson, 1898; Beck, 1967, Huberty, 2012; Masten, 2001; Rutter, 2001). Since children of depressed parents are exposed naturally to numerous of risk factors (Goodman & Gotlib 1999, Hosman et al., 2009), one of the biggest risk factors for developing depression is having a parent who has depression (Hosman, 2009; Weissman, 1997). The offspring of depressed parents were found to be three to four times more likely to develop a depressive disorder than children of non-depressed parents (Weissman et al. (2006). In addition, these children are more likely to experience more severe and continuous courses of depression.

The trans-generational continuation of depression is explained by the *model of transition* (Goodman & Gotlib, 1999) including multiple biological and psycho-social factors and pathways for the development of depression in the offspring of depressed parents. Goodman & Gotlib (1999) accumulated findings about mechanisms and process explaining the high risk for depression in this group. Numerous studies were conducted on individual pathways as the genetic contribution of association of cognitive vulnerabilities with depression. It was shown that many of the single findings that were reported by Goodman and

Gotlib in 1999 appeared to be up-to-date, when they were compared to current researches. Nevertheless, there is a lot of criticism on the constituted model. One major criticism is that the conceptualism of those risk factors is indistinct in their role and impact on the children's psychopathology outcomes. In addition, hypothesis often lack evidence and experimental studies and are rather a conglomerate of single findings than a global model integrating and explaining the transmission of depression. The model only focuses on maternal depression and does not obtain other relevant risk factors (e.g. cultural aspects, support by significant others).

Undoubtedly, prevention interventions for children and adolescents facing the high risk of mental illness are needed. Although there is an existing number of various supporting offers, most of them are not evidence based (Christiansen, Anding, & Donath, 2014). Previous meta-analyses suggest that it is possible to prevent depression in children and adolescents in general, indicating small to moderate effects (Hetrick et al., 2016; Mendelson & Tandon, 2016; Stockings et al., 2016). Nevertheless, there are only few prevention trials focussing on the offspring of depressed parents, mostly conducted in the U.S. Findings are heterogeneous and the role of significant moderators and mediators remains unclear (Loechner & Starman, et al., n.d.).

Study I. The first study sought to replicate findings of the increased risk for depression in youth that is associated with parental depression and identify most prevalent risk factors in order to explore possible mechanisms of the trans-generational transmission of depression. Data collected from 112 children and adolescents of parents with and without depression showed big group differences in depressive and psychopathology symptoms. Therefore, the offspring of depressed (HR, n = 74) and non-depressed parents (LR, n = 38) were compared in general psychopathology (self-rated depressive and psychopathology symptoms; parent-rated psychopathology), the mediators (emotion regulation, attributional style), and

moderators (life events). In addition, the role of parental depression and its impact and association on the children's depressive symptoms was investigated. The data supported earlier findings of increased risk for depression for the HR, since the HR showed significantly increased psychopathology and depressive symptoms with a big effect size (d = 1.75). Thereby, the parental depression was associated significantly with children's depression severity. In addition, the data provided strong evidence for group differences in adaptive emotion regulation strategies, positive and negative attributional style and the number of positive life events. Against expectations, groups did not differ in maladaptive emotion regulation strategies and the number of negative life events. Nevertheless, maladaptive emotion regulation strategies, negative life events and parental depression were the strongest predictors of children's depressive symptoms, together accounting for 30.8 % of the variance. The study provides novel evidence about the impact of specific risk factors on the children's depressive symptoms and therefore the increased risk for depression. Nevertheless, more longitudinal studies are necessary including more families with a low socio economical background. The findings constitute information for the improvement of existing prevention programs by giving information about skill deficits and potential mediating factors. For example, the development of future intervention programs might benefit from the finding that children of depressed parents have skill deficits in maladaptive emotion regulation strategies and the attributional style. Contents of interventions should focus on improvement of coping skills and stress regulation. Additionally, the high-risk group was found to report less positive life-event. Positive activities for this group may represent a useful ingredient of clinical interventions.

Study II. In *study II*, preliminary results of an ongoing randomized controlled trial of one of the most promising prevention programs for the offspring of depressed parents (replicated here for the first time outside of the research group) are presented. Data from n =

61 families who reached post-assessment are provided. It was hypothesized that children in the experimental group (EG, n = 29) would show decreased symptoms of psychopathology and depression compared to the control group (CG, n = 32) over time. In addition, mediating factors such as emotion regulation strategies and attributional style were expected to improve within the EG over time. Rating of treatment fidelity was very high, indicating good reliability of the intervention. The acceptance of families of the program was excellent; children and parents gave a very positive feedback about the intervention and their personal benefit of participating. Results indicate significant reduction of self-reported psychopathological symptoms between groups over time favouring a positive intervention effect. Against expectations, both groups showed lower depression and parent-rated psychopathology symptoms from baseline to post-assessment. As predicted, there was a significant interaction effect of time and group indicating less maladaptive emotion regulation strategies and a more positive internal attributional style in the intervention group compared to the control group over time. Again, both groups showed improved adaptive emotion regulation strategies over the study period. The attributional style was found to be more negative in both groups from baseline to post-assessment. In addition, children in the experimental group showed a more negative internal attributional style over time than children in the control group. The benefits of the CG are interpreted as general activation for this high risk group for seeking information help. Overall, these findings are promising, although the results are preliminary and a bigger sample is necessary for more confident interpretations. There is a lack of evidence and number of prevention programs for this high-risk group, especially in Germany. Since effect sizes of prevention interventions were found to be small and diminish over time, further research is needed to identify relevant mediators and moderators in order to increase efficacy.

In sum, this thesis supports previous findings about the increased risk of depression for the offspring of parents suffering from depression and the association of parental and youth depression. In addition, it provides novel information about particular risk factors for children of depressed parents by outlining group differences in depressive symptoms, general psychopathology, adaptive emotion regulation strategies, positive and negative attributional style and positive life events between children of parents with and without depression.

The data provides evidence that most prevalent risk factors for youth depression in this sample are maladaptive emotion regulation strategies, negative life events and the parental depression. Moreover, results of the first replication of a promising prevention intervention in Germany suggest that it is possible to modify some of these risk factors (maladaptive emotion regulation strategies and positive internal attributional style) and that doing so has positive effects on reducing self-reported psychopathology in children at risk. Consequently, these findings enable treatment and prevention implications in order to increase the children's resilience. Further research conducting longitudinal studies with representative and big samples, including important mediator and moderator variables are needed in order to further investigate in these factors and increase the efficacy of prevention interventions. Future approaches on prevention should target integration of preventive offers in primary care and make prevention accessible for this high-risk group. In addition, existing prevention programs should be replicated in more geographically distributed samples, including different approaches and comparison conditions. On top of this, cost-effectiveness calculations are necessary for optimizing the care provided.

15. Appendices

Appendix A: Fidelity Checklist

Checkliste Gruppenleiter für PRODO Sitzungen

Sitzung 1 - Psychoedukation Depression

Gruppenleiter		**Datum**	
Gruppe		Uhrzeit	

Bitte abhaken, wenn erledigt:

Gliederung	Ja	Nein
Teil 1 (20 Minuten)		
Vorstellungsrunde/Kennenlernübung, Überblick über Kursziele, Regeln und Erwartungen		
Teil 2 (10 Minuten)		
Gruppendiskussion: Symptome von Depression		
Teil 3 (10 Minuten)		
Interaktive Psychoedukation: Depression definieren		
Teil 4 (20 Minuten)		
Interaktive Psychoedukation: Ursachen der Depression		
Pause (10 Minuten) nach 55 Minuten		
Teil 5 (15 Minuten)		
Gruppenaktivität: Der Umgang mit Depression innerhalb der Familie. Video – Diskussion mit den Familien		
Teil 6 (15 Minuten)		
Gruppendiskussion: Depression in der Familie		
Teil 7 (5 Minuten)		
Grundprinzip und Durchführung der Trainingsblätter		
Teil 8 (15 Minuten)		
Die Trainingsblätter für diese Woche		
Teil 9 (5 Minuten)		
Abschluss und Evaluation der Sitzung		

Sitzung 2 - Stressreaktionen und A-APP-Bewältigung

Gruppenleiter		**Datum**	
Gruppe		Uhrzeit	

Bitte abhaken, wenn erledigt:

Gliederung	Ja	Nein
Teil 1 (15 Minuten)		
Trainingsblätter einsammeln und besprechen		
Teil 2 (25 Minuten)		
Psychoedukation: Reaktionen auf Stress		
Teil 3 (15 Minuten)		
Gruppenaktivität: Stress-Ballons		
Pause (10 Minuten) nach 55 Minuten		
Teil 4 (20 Minuten)		
Psychoedukation: A-APP-Bewältigung und positive Aktivität		
Teil 5 (15 Minuten)		
Gruppenaktivität: positive Aktivität		
Teil 6 (5 Minuten)		
Gruppenaktivität: öffentliche Verpflichtung zu positiver Aktivität		
Teil 7 (15 Minuten)		
Trainingsblätter für diese Woche		
Teil 8 (5 Minuten)		
Abschluss und Evaluation der Sitzung		

Sitzung 3 - A-APP-Bewältigung

Gruppenleiter		**Datum**	
Gruppe		Uhrzeit	

Bitte abhaken, wenn erledigt:

Gliederung	Ja	Nein
Teil 1 (20 Minuten)		
Trainingsblätter einsammeln und besprechen		
Teil 2 (15 Minuten)		
Psychoedukation: Akzeptanz		
Teil 3 (20 Minuten)		
Psychoedukation: Positives Denken		
Pause (10 Minuten) nach 55 Minuten		
Teil 4 (20 Minuten)		
Gruppenaktivität: Negatives und Positives Denken		
Teil 5 (20 Minuten)		
Psychoedukation: Ablenkung		
Teil 6 (1 Minute)		
Gruppendiskussion: Kurze Zusammenfassung der A-APP-Fertigkeiten		
Teil 7 (14 Minuten)		
Trainingsblätter für diese Woche		
Teil 8 (5 Minuten)		
Abschluss und Evaluation der Sitzung		

Sitzung 4 - Erziehungskompetenzen I und A-APP

Gruppenleiter		**Datum**	
Gruppe		Uhrzeit	

Bitte abhaken, wenn erledigt:

Gliederung	Ja	Nein
Teil 1 (10 Minuten)		
Trainingsblätter einsammeln und besprechen		
Eltern und Kinder gehen in getrennte Räume		
ELTERN		
Teil 2 (1 Minute)		
Einführung in die getrennten Sitzungen		
Teil 3 (14 Minuten)		
Trainingsblätter einsammeln und besprechen		
Teil 4 (15 Minuten)		
Psychoedukation: Positive Erziehung und Erziehungsstile		
Teil 5 (15 Minuten)		
Psychoedukation: Positive Zeit und Lob		
Pause (5 Minuten)		
Teil 6 (10 Minute)		
Gruppenaktivität: Rollenspiel positive Zeit		
Teil 7 (10 Minuten)		
Gruppenaktivität: Positive Zeit Üben		
Teil 8 (5 Minuten)		
Gruppenaktivität: Positive Zeit Planen		
Teil 9 (8 Minuten)		
Trainingsblätter für diese Woche		
Teil 10 (2 Minuten)		
Vorbereitung für das Zusammenkommen mit der Familie		
KINDER		
Teil 2 (1 Minute)		
Einführung in die getrennten Sitzungen		
Teil 3 (14 Minuten)		
Trainingsblätter einsammeln und besprechen		
Teil 4 (15 Minuten)		

Psychoedukation: Kontrollierbare vs. Unkontrollierbare Stressoren		
Teil 5 (15 Minuten)		
Psychoedukation: Unkontrollierbare Familienstressoren		
Pause (10 Minuten)		
Teil 6 (5 Minuten)		
Gruppendiskussion: A-APP-Fertigkeiten wiederholen		
Teil 7 (5 Minuten)		
Psychoedukation: Einführung in Akzeptanz		
Teil 8 (10 Minuten)		
Psychoedukation: Akzeptanz Definieren		
Teil 9 (15 Minuten)		
Psychoedukation: Akzeptanz anwenden		
Teil 10 (15 Minuten)		
Trainingsblätter für diese Woche		
Eltern und Kinder kommen wieder zusammen		
Teil 11 (15 Minute)		
Wöchentliche Familienzeit in der Sitzung		
Teil 12 (5 Minuten)		
Familientraining FUN		
Teil 13 (5 Minuten)		
Abschluss und Evaluation der Sitzung		

Sitzung 5 - Erziehungskompetenzen II und A-APP

Gruppenleiter		**Datum**	
Gruppe		Uhrzeit	

Bitte abhaken, wenn erledigt:

Gliederung	Ja	Nein
Teil 1 (10 Minuten)		
FUN Positive Familienunternehmung besprechen		
Eltern und Kinder gehen in getrennte Räume		
ELTERN		
Teil 2 (15 Minuten)		
Trainingsblätter einsammeln und besprechen		
Teil 3 (10 Minuten)		
Psychoedukation: Einführung in Ignorieren		
Teil 4 (10 Minuten)		
Gruppenaktivität: Rollenspiel Ignorieren und Lob		
Pause (5 Minuten)		
Teil 5 (15 Minuten)		
Psychoedukation: Lob und Ignorieren anwenden		
Teil 6 (5 Minuten)		
Psychoedukation: Wenn Ignorieren schwierig ist		
Teil 7 (10 Minuten)		
Gruppenaktivität: Unterstützung aktivieren		
Teil 8 (13 Minuten)		
Trainingsblätter für diese Woche		
Teil 9 (2 Minuten)		
Vorbereitung für das Zusammenkommen mit der Familie		
KINDER		
Teil 2 (15 Minuten)		
Trainingsblätter einsammeln und besprechen		
Teil 3 (10 Minuten)		
Psychoedukation: Einführung in positive Aktivitäten		
Teil 4 (5 Minuten)		
Psychoedukation: Tägliche positive Aktivitäten		
Teil 5 (15 Minuten)		
Psychoedukation: Kategorien von positiver Aktivitäten		
Teil 6 (5 Minuten)		

Gruppenaktivität: positive Aktivitäten einüben		
Pause (10 Minuten)		
Teil 7 (10 Minuten)		
Gruppenaktivität: positive Aktivitäten sammeln		
Teil 8 (15 Minuten)		
Trainingsblätter für diese Woche		
Eltern und Kinder kommen wieder zusammen		
Teil 9 (15 Minuten)		
Wöchentliche Familienzeit in der Sitzung		
Teil 10 (5 Minuten)		
Familientraining FUN		
Teil 11 (5 Minuten)		
Abschluss und Evaluation der Sitzung		

Sitzung 6 - Erziehungskompetenzen III und A-APP

Gruppenleiter		**Datum**	
Gruppe		Uhrzeit	

Bitte abhaken, wenn erledigt:

Gliederung	Ja	Nein
Teil 1 (10 Minuten)		
FUN Positive Familienunternehmung besprechen		
Eltern und Kinder gehen in getrennte Räume		
ELTERN		
Teil 2 (15 Minuten)		
Trainingsblätter einsammeln und besprechen		
Teil 3 (10 Minuten)		
Psychoedukation: Anweisungen geben		
Teil 4 (10 Minuten)		
Gruppenaktivität: Hausregeln ausmachen		
Teil 5 (15 Minuten)		

Gruppenaktivität: Hausregeln Rollenspiel *Optional		
Pause (5 Minuten)		
Teil 6 (5 Minuten)		
Psychoedukation: Belohnungen		
Teil 7 (10 Minuten)		
Psychoedukation: Zieltabelle		
Teil 8 (20 Minuten)		
Psychoedukation: Die Zieltabelle aufbauen		
Teil 9 (8 Minuten)		
Trainingsblätter für diese Woche		
Teil 10 (2 Minuten)		
Vorbereitung für das Zusammenkommen mit der Familie		
KINDER		
Teil 2 (15 Minuten)		
Trainingsblätter einsammeln und besprechen		
Teil 3 (10 Minuten)		
Psychoedukation: Was ist negatives Denken?		
Teil 4 (10 Minuten)		
Gruppenaktivität: Warum positives und negatives Denken anwenden?		
Pause (10 Minuten)		
Teil 5 (10 Minuten)		
Psychoedukation: Positives Denken		
Teil 6 (15 Minute)		
Gruppenaktivität: Negative Gedanken in Positive umwandeln		
Teil 7 (15 Minuten)		
Trainingsblätter für diese Woche		
Eltern und Kinder kommen wieder zusammen		
Teil 9 (15 Minuten)		
Wöchentliche Familienzeit in der Sitzung		
Teil 10 (5 Minuten)		
Familientraining FUN		
Teil 11 (5 Minuten)		
Abschluss und Evaluation der Sitzung		

Sitzung 7 - Erziehungskompetenzen IV und A-APP

Gruppenleiter		**Datum**	
Gruppe		Uhrzeit	

Bitte abhaken, wenn erledigt:

Gliederung	Ja	Nein
Teil 1 (10 Minuten)		
FUN Positive Familienunternehmung besprechen		
Eltern und Kinder gehen in getrennte Räume		
ELTERN		
Teil 2 (15 Minuten)		
Trainingsblätter einsammeln und besprechen		
Teil 3 (10 Minuten)		
Psychoedukation "Bescheid Wissen"		
Teil 4 (5 Minuten)		
Gruppenaktivität: "Bescheid Wissen" üben		
Pause (5 Minuten)		
Teil 5 (10 Minuten)		
Psychoedukation: Negative Konsequenzen		
Teil 6 (10 Minuten)		
Gruppenaktivität: Eine Zieltabelle mit negativen Konsequenzen vorbereiten		
Teil 7 (5 Minuten)		
Psychoedukation: Konsequenzen kommunizieren		
Teil 8 (10 Minuten)		
Trainingsblätter für diese Woche		
KINDER		
Teil 3 (10 Minuten)		
Psychoedukation: Wann Ablenkung angewandt wird		
Teil 4 (10 Minuten)		
Psychoedukation: Was ist Ablenkung?		
Pause (10 Minuten)		
Teil 5 (10 Minuten)		
Psychoedukation: Akzeptanz und Ablenkung		
Teil 6 (10 Minuten)		

Gruppenaktivität: Akzeptanz und Ablenkung anwenden		
Teil 7 (5 Minuten)		
Psychoedukation: Zusammenfassung Ablenkung		
Teil 8 (15 Minuten)		
Trainingsblätter für diese Woche		
Eltern und Kinder kommen wieder zusammen		
Teil 9 (15 Minuten)		
Wöchentliche Familienzeit in der Sitzung		
Teil 10 (5 Minuten)		
Familientraining FUN		
Teil 11 (5 Minuten)		
Abschluss und Evaluation der Sitzung		

Sitzung 8 - Planen mit der Familie und Ihre gelernten Fähigkeiten

Gruppenleiter		**Datum**	
Gruppe		Uhrzeit	

Bitte abhaken, wenn erledigt:

Gliederung	Ja	Nein
Teil 1 (10 Minuten)		
Besprechung der positiven Familienunternehmung FUN		
Eltern und Kinder gehen in getrennte Räume		
ELTERN		
Teil 2 (15 Minuten)		
Einsammeln und Besprechung der Trainingsblätter		
Teil 3 (15 Minuten)		
Psychoedukation: Positive Erziehung bei Depression		
Teil 4 (10 Minuten)		
Psychoedukation: Depressive Symptome erkennen		
Teil 5 (15 Minuten)		
Psychoedukation: Die Kinder bei A-APP unterstützen		

Teil 6 (15 Minuten)		
Trainingsblätter für diese Woche		
Pause (10 Minuten)		
KINDER		
Teil 2 (15 Minuten)		
Einsammeln und Besprechung der Trainingsblätter		
Teil 3 (40 Minuten)		
Gruppenaktivität: A-APP Rollenspiele		
Teil 4 (15 Minuten)		
Trainingsblätter für diese Woche		
Pause (10 Minuten)		
Eltern und Kinder kommen wieder zusammen		
Teil 5 (20 Minuten)		
Wöchentliche Familienzeit in der Sitzung		
Teil 6 (5 Minute)		
Familientraining FUN		
Teil 7 (5 Minuten)		
Abschluss und Evaluation der Sitzung		

Sitzung 9 - Wiederholung und Übung

Gruppenleiter		**Datum**	
Gruppe		Uhrzeit	

Bitte abhaken, wenn erledigt:

Gliederung	**Ja**	**Nein**
Teil 1 (10 Minuten)		
FUN Positive Familienunternehmung besprechen		
Eltern und Kinder gehen in getrennte Räume		
ELTERN		
Teil 2 (15 Minuten)		
Einsammeln und Besprechung der Trainingsblätter		

Teil 3 (5 Minuten)		
Gruppendiskussion: Wiederholung der Vorteile positiver Erziehung		
Teil 4 (5 Minuten)		
Gruppenaktivität: Erzieherische Situationen vorhersehen		
Teil 5 (25 Minuten)		
Gruppenaktivität: Eltern Rollenspiele		
Teil 6 (5 Minuten)		
Vorbereitung für Rollenspiele mit der Familie		
Teil 7 (5 Minuten)		
Trainingsblätter für diese Woche		
Pause (10 Minuten)		
KINDER		
Teil 2 (15 Minuten)		
Einsammeln und Besprechung der Trainingsblätter		
Teil 3 (5 Minuten)		
Gruppenaktivität: Stressige Situationen vorhersehen		
Teil 4 (30 Minuten)		
Gruppenaktivität: A-APP Rollenspiele		
Teil 5 (5 Minuten)		
Vorbereitung für Rollenspiele mit der Familie		
Teil 6 [OPTIONAL]		
Gruppenaktivität: Rollenspiel, zusätzliche stressige Situationen		
Teil 7 (5 Minuten)		
Trainingsblätter für diese Woche		
Pause (10 Minuten)		
Eltern und Kinder kommen wieder zusammen		
Teil 8 (30 Minuten)		
Gruppenaktivität: Rollenspiele mit der Familie		
Teil 9 (5 Minute)		
Familientraining FUN		
Teil 10 (5 Minuten)		
Abschluss und Evaluation der Sitzung		

Sitzung 10 - Wiederholung und Übung

Gruppenleiter		**Datum**	
Gruppe		Uhrzeit	

Bitte abhaken, wenn erledigt:

Gliederung	Ja	Nein
Teil 1 (10 Minuten)		
FUN Positive Familienunternehmung besprechen		
Eltern und Kinder gehen in getrennte Räume		
ELTERN		
Teil 2 (15 Minuten)		
Einsammeln und Besprechung der Trainingsblätter		
Teil 3 (30 Minuten)		
Gruppenaktivität: Eltern Rollenspiele		
Teil 4 (5 Minuten)		
Gruppenaktivität: Erzieherische Situationen vorhersehen		
Teil 5 (5 Minuten)		
Vorbereitung für die Rollenspiele mit der Familie		
Teil 6 (5 Minuten)		
Trainingsblätter für diese Woche		
Pause (10 Minuten)		
KINDER		
Teil 2 (15 Minuten)		
Einsammeln und Besprechung der Trainingsblätter		
Teil 3 (5 Minuten)		
Gruppenaktivität: Stressige Situationen vorhersehen		
Teil 4 (30 Minuten)		
Gruppenaktivität: A-APP Rollenspiele		
Teil 5 (5 Minuten)		
Vorbereitung für Rollenspiele mit der Familie		
Teil 6 [OPTIONAL]		
Gruppenaktivität: Rollenspiel, zusätzliche stressige Situationen		
Teil 7 (5 Minuten)		
Trainingsblätter für diese Woche		

Pause (10 Minuten)		
Eltern und Kinder kommen wieder zusammen		
Teil 8 (30 Minuten)		
Gruppenaktivität: Rollenspiele mit der Familie		
Teil 9 (5 Minute)		
Familientraining FUN		
Teil 10 (5 Minuten)		
Abschluss und Evaluation der Sitzung		

Sitzung 11 - Wiederholung und Übung

Gruppenleiter		**Datum**	
Gruppe		Uhrzeit	

Bitte abhaken, wenn erledigt:

Gliederung	Ja	Nein
Teil 1 (10 Minuten)		
FUN Positive Familienunternehmung besprechen		
Eltern und Kinder gehen in getrennte Räume		
ELTERN		
Teil 2 (15 Minuten)		
Einsammeln und Besprechung der Trainingsblätter		
Teil 3 (30 Minuten)		
Gruppenaktivität: Eltern Rollenspiele		
Teil 4 (5 Minuten)		
Gruppenaktivität: Erzieherische Situationen vorhersehen		
Teil 5 (5 Minuten)		
Vorbereitung für die Rollenspiele mit der Familie		
Teil 6 (5 Minuten)		
Trainingsblätter für diese Woche		
Pause (10 Minuten)		
KINDER		

Teil 2 (15 Minuten)		
Einsammeln und Besprechung der Trainingsblätter		
Teil 3 (5 Minuten)		
Gruppenaktivität: Stressige Situationen vorhersehen		
Teil 4 (30 Minuten)		
Gruppenaktivität: A-APP Rollenspiele		
Teil 5 (5 Minuten)		
Vorbereitung für Rollenspiele mit der Familie		
Teil 6 [OPTIONAL]		
Gruppenaktivität: Rollenspiel, zusätzliche stressige Situationen		
Teil 7 (5 Minuten)		
Trainingsblätter für diese Woche		
Pause (10 Minuten)		
Eltern und Kinder kommen wieder zusammen		
Teil 8 (30 Minuten)		
Gruppenaktivität: Rollenspiele mit der Familie		
Teil 9 (5 Minuten)		
Familientraining FUN		
Teil 10 (5 Minuten)		
Abschluss und Evaluation der Sitzung		

Sitzung 12 – Wiederholung und Übung

Gruppenleiter		**Datum**	
Gruppe		Uhrzeit	

Bitte abhaken, wenn erledigt:

Gliederung	Ja	Nein
Teil 1 (30 Minuten)		
Trainingsblätter einsammeln und besprechen		
Teil 2 (15 Minuten)		
Gruppendiskussion: Fortschritt besprechen und Probleme lösen		

Teil 3 (15 Minuten)		
Gruppenaktivität: Familienziele		
Pause (10 Minuten)		
Teil 4 (30 Minuten)		
Gruppenaktivität „Wer wird Millionär“ Spiel		
Teil 5 (20 Minuten)		
Abschluss, Zertifikate und Evaluation der Sitzung		

Gesund und Glücklich aufwachsen!

Bewertungsbogen

Sitzung: ____

Datum: _____

Bitte beantworte/beantworten Sie die folgenden Fragen über die heutige Sitzung, um uns zu helfen das Programm zu verbessern!

□ **Elternteil** □ **Kind**

Bitte kreise/kreisen Sie hierfür die Zahl ein, die am ehesten deine/Ihre Meinung wiederspiegelt! **Danke!**

1. **Wie gut hast du/haben Sie den Inhalt der heutigen Stunde verstanden?**

1	2	3	4	5
Überhaupt nicht		ein bisschen		sehr gut

2. **Wie aktiv hast du/haben Sie während der heutigen Sitzung mitgearbeitet? (Z.B. in Diskussionen oder Rollenspielen mitgemacht)**

1	2	3	4	5
gar nicht		ein bisschen		sehr viel

3. **Hast du dich/Haben Sie sich während der heutigen Stunde wohl gefühlt?**

1	2	3	4	5

überhaupt nicht	ein bisschen	sehr

4. Wie sehr hast du dich/Wie sehr haben Sie sich von den Gruppenleitern verstanden und unterstützt gefühlt?

1	2	3	4	5
überhaupt nicht		ein bisschen		sehr

5. Wie gut hast du/haben Sie die Trainingsblätter der vergangenen Woche verstanden, nachdem wir sie heute besprochen haben?

1	2	3	4	5
gar nicht		ein bisschen		sehr viel

6. Wie hilfreich fandest du/fanden Sie die Übungen in der heutigen Stunde?

1	2	3	4	5
gar nicht		ein bisschen		sehr hilfreich

Bitte schreibe/schreiben Sie in die folgenden Zeilen, was du/Sie uns noch zusätzlich gerne über die heutige Stunde mitteilen willst/wollen.

Danke!

16. List of Tables

17. List of Figures

18. List of Graphs

References

Abela, J. R. Z., & Hankin, B. L. (2008). Cognitive vulnerability to depression in children and adolescents: A developmental psychopathology perspective. *Handbook of Depression in Children and Adolescents*, 35–78.

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

Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987a). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin*, *101*(2), 213–232. https://doi.org/10.1037/0033-2909.101.2.213

Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987b). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin*, *101*(2), 213–232. https://doi.org/10.1037/0033-2909.101.2.213

Aldao, A., & Nolen-Hoeksema, S. (2012). When are adaptive strategies most predictive of psychopathology? *Journal of Abnormal Psychology*, *121*(1), 276–281. https://doi.org/10.1037/a0023598

Allen, J. L., Rapee, R. M., & Sandberg, S. (2012). Assessment of maternally reported life events in children and adolescents: A comparison of interview and checklist methods. *Journal of Psychopathology and Behavioral Assessment*, *34*(2), 204–215. https://doi.org/10.1007/s10862-011-9270-5

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of*. *Mental Disorders* (Vol. 4ªed). Washington DC.

Angold, A., & Costello, E. J. (1993). Depressive comorbidity in children and adolescents: Empirical, theoretical, and methodological issues. *The American Journal of Psychiatry*, *150*(December), 1779–1791. https://doi.org/10.1176/ajp.150.12.1779

Auerbach, R. P., Ho, M. H. R., & Kim, J. C. (2014). Identifying cognitive and interpersonal predictors of adolescent depression. *Journal of Abnormal Child Psychology*, *42*(6), 913–924. https://doi.org/10.1007/s10802-013-9845-6

Avenevoli, S., & Merikangas, K. R. (2006). Implications of High-Risk Family Studies for Prevention of Depression. *American Journal of Preventive Medicine*, *31*(6 SUPPL. 1), 126–135. https://doi.org/10.1016/j.amepre.2006.07.003

Axelson, D. A., & Birmaher, B. (2001). Relation between anxiety and depressive disorders in childhood and adolescence. *Depression & Anxiety (1091-4269)*, *14*(2), 67–78. https://doi.org/http://dx.doi.org/10.1002/da.1048

Bandura, A. (1971). Social learning theory. *Social Learning Theory*. https://doi.org/10.1111/j.1460-2466.1978.tb01621.x

Bauer, U. (2005). *Das Präventionsdilemna\rPotenziale schulischer Kompetenzförderung im Spiegel sozialer Polarisierung*. Wiesbaden.

Bauman, K. E., Foshee, V. A., Ennett, S. T., Hicks, K., & Pemberton, M. (2001). Family

matters: a family-directed program designed to prevent adolescent tobacco and alcohol use. *Health Promotion Practice*, *2*(1), 81–96. https://doi.org/10.1177/152483990100200112

Beardslee, W. R., Brent, D. A., Weersing, V. R., Clarke, G. N., Porta, G., Hollon, S. D., … Garber, J. (2013). Prevention of Depression in At-Risk Adolescents. *JAMA Psychiatry*, *70*(11), 1161. https://doi.org/10.1001/jamapsychiatry.2013.295

Beardslee, W. R., Brent, D. A., Weersing, V. R., Clarke, G. N., Porta, G., Hollon, S. D., … Garber, J. (2013). Prevention of depression in at-risk adolescents: Longer-term effects. *JAMA Psychiatry*, *70*(11), 1161–1170. https://doi.org/10.1001/jamapsychiatry.2013.295

Beardslee, W. R., Gladstone, T. R. G., & O'Connor, E. E. (2011). Transmission and prevention of mood disorders among children of affectively ill parents: A review. *Journal of the American Academy of Child and Adolescent Psychiatry*, *50*(11), 1098–1109. https://doi.org/10.1016/j.jaac.2011.07.020

Beardslee, W. R., Gladstone, T. R. G., Wright, E. J., & Cooper, A. B. (2003). A Family-Based Approach to the Prevention of Depressive Symptoms in Children at Risk : Evidence of Parental and Child Change The online version of this article , along with updated information and services , is located on the World Wide Web at : A Family-.

Beardslee, W. R., Gladstone, T. R., Wright, E. J., & Cooper, A. B. (2003). A family-based approach to the prevention of depressive symptoms in children at risk: evidence of parental and child change. *Pediatrics*, *112*(2), e119 LP-e131.

Beardslee, W. R., Schultz, L. H., & Selman, R. L. (1987). Level of Social-Cognitive Development , Adaptive Functioning , and DSM-III Diagnoses in Adolescent Offspring of Parents With Affective Disorders : Implications of the Development of the Capacity for Mutuality. *Developmental Psychology*, *23*(6), 807–815. https://doi.org/10.1037//0012-1649.23.6.807

Beardslee, W. R., Versage, E. M., & Gladstone, T. R. (1998). Children of affectively ill parents: a review of the past 10 years. *Journal of the American Academy of Child and Adolescent Psychiatry*, *37*(11), 1134–1141. https://doi.org/10.1097/00004583-199811000-00012

Beardslee, W. R., Versage, E. M., Wright, E. J., Salt, P., Rothberg, P. C., Drezner, K., & Gladstone, T. R. (1997). Examination of preventive interventions for families with depression: evidence of change. *Development and Psychopathology*, *9*(1), 109–130. https://doi.org/10.1017/S0954579497001090

Beardslee, W. R., Wright, E. J., Gladstone, T. R. G., & Forbes, P. (2007). Long-term effects from a randomized trial of two public health preventive interventions for parental depression. *Journal of Family Psychology*, *21*, 703–713.

Beardslee, W. R., Wright, E. J., Salt, P., Drezner, K., Gladstone, T. R., Versage, E. M., & Rothberg, P. C. (1997). Examination of children's responses to two preventive intervention strategies over time. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*(2), 196–204. https://doi.org/doi: 10.1097/00004583-199702000-00010

Beck, A. T. (1967). *Depression: Clinical, Experimental, and Theoretical Aspects.* New York, N: Harper & Row.

Beck, A. T., Rush, A. K., Shaw, B. F., & Emery, G. (1979). Cognitive therapy of depression.

Beck, R., & Perkins, T. S. (2001). Cognitive content-specificity for anxiety and depression: A meta-analysis. *Cognitive Therapy and Research*, *25*(6), 651–663. https://doi.org/10.1023/A:1012911104891

Belsky, J. (1984). The Determinants of Parenting: A Process Model. *Child Development*, *55*(1), 83–96. https://doi.org/10.2307/1129836

Billings, A. C., & Moos, R. H. (1982). Psychosocial theory and research on depression: An integrative framework and review. *Clinical Psychology Review*, *2*(2), 213–237. https://doi.org/10.1016/0272-7358(82)90013-7

Birmaher, B., Axelson, D., Goldstein, B., Strober, M., Gill, M. K., Hunt, J., … Keller, M. (2009). Four-year longitudinal course of children and adolescents with bipolar spectrum disorders: The course and outcome of bipolar youth (COBY) study. *American Journal of Psychiatry*, *166*(7), 795–804. https://doi.org/10.1176/appi.ajp.2009.08101569

Birmaher, B., Ryan, N. D., Williamson, D. E., Brent, D. A., & Kaufman, J. (1996). Childhood and Adolescent Depression: A Review of the Past 10 Years. Part II. *Journal of the American Academy of Child & Adolescent Psychiatry*, *35*(12), 1575–1583. https://doi.org/10.1097/00004583-199612000-00008

Boszormenyi-Nagy, I., & Spark, G. M. (1981). Unsichtbare Bindungen. Die Dynamik familiärer Systeme.

Braet, C., Vlierberghe, L. Van, Vandevivere, E., Theuwis, L., & Bosmans, G. (2013). Depression in early, middle and late adolescence: Differential evidence for the cognitive diathesis-stress model. *Clinical Psychology and Psychotherapy*, *20*(5), 369–383. https://doi.org/10.1002/cpp.1789

Braet, C., Wante, L., Van Beveren, M. L., & Theuwis, L. (2015). Is the cognitive triad a clear marker of depressive symptoms in youngsters? *European Child and Adolescent Psychiatry*, *24*(10), 1261–1268. https://doi.org/10.1007/s00787-015-0674-8

Brent, D. A., Brunwasser, S. M., Hollon, S. D., Weersing, V., Clarke, G. N., Dickerson, J. F., … Garber, J. (2015, November). Effect of a cognitive-behavioral prevention program on depression 6 years after implementation among at-risk adolescents: a randomized clinical trial. *JAMA Psychiatry*. https://doi.org/10.1001/jamapsychiatry.2015.1559

Brent, D. a., Brunwasser, S. M., Hollon, S. D., Weersing, V. R., Clarke, G. N., Dickerson, J. F., … Garber, J. (2015). Effect of a Cognitive-Behavioral Prevention Program on Depression 6 Years After Implementation Among At-Risk Adolescents. *JAMA Psychiatry*, *72*(11), 1110. https://doi.org/10.1001/jamapsychiatry.2015.1559

Breznitz, Z., & Sherman, T. (1987). Speech patterning of natural discourse of well and depressed mothers and their young children. *Child Development*, *58*(2), 395–400. https://doi.org/10.1016/S0163-6383(86)80344-7

Bühler, A., Kötter, C., Stemmler, M., Jaursch, S., & Lösel, F. (2013). EFFEKT-E: Wirksamkeit eines Präventionsprogramms für Kinder emotional belasteter Mütter. *Das Gesundheitswesen*, *77*(S 01), S64–S65. https://doi.org/10.1055/s-0032-1333244

Calear, A. L., & Christensen, H. (2010). Systematic review of school-based prevention and

early intervention programs for depression. *Journal of Adolescence*, *33*(3), 429–438. https://doi.org/10.1016/j.adolescence.2009.07.004

Calvete, E., & Cardeñoso, O. (2005). Gender differences in cognitive vulnerability to depression and behavior problems in adolescents. *Journal of Abnormal Child Psychology*, *33*(2), 179–192. https://doi.org/10.1007/s10802-005-1826-y

Chang, J. J., Halpern, C. T., & Kaufman, J. S. (2007). Maternal depressive symptoms, father's involvement, and the trajectories of child problem behaviors in a US national sample. *Archives of Pediatrics & Adolescent Medicine*, *161*(7), 697–703. https://doi.org/10.1001/archpedi.161.7.697

Cho, Y., & Telch, M. J. (2005). Testing the cognitive content-specificity hypothesis of social anxiety and depression: An application of structural equation modeling. *Cognitive Therapy and Research*, *29*(4), 399–416. https://doi.org/10.1007/s10608-005-2081-9

Choudhoury, M. S., Pimentel, S. S., & Kendall, P. C. (2003). Childhood anxiety disorders: parent-child (dis)agreement using a structured interview for the DSM-IV. *Journal of the American Academy of Child and Adolescent PsychiatryAdolescent Psychiatry*, *42*(957–964).

Christiansen, H., Anding, J., & Donath, L. (2014). Interventionen für Kinder psychisch kranker Eltern. In M. Kölch, U. Ziegenhain, & J. M. Fegert (Eds.), *Kinder psychisch kranker Eltern. Herausforderung für eine interdisziplinäre Kooperation in Betreeung und Versorgung* (pp. 80–105). Weinheim: Beltz Juventa.

Christiansen, H., Anding, J., Schrott, B., & Röhrle, B. (2015). Children of mentally ill parents-A pilot study of a group intervention program. *Frontiers in Psychology*, *6*(OCT), 1–8. https://doi.org/10.3389/fpsyg.2015.01494

Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology*, *103*(1), 103–116. https://doi.org/10.1037/0021-843X.103.1.103

Clarke, G. N., Hornbrook, M., Lynch, F., et al., Polen, M., Gale, J., … Seeley, J. (2001). A randomized trial of a group cognitive intervention for preventing depression in adolescent offspring of depressed parents. *Archives of General Psychiatry*, *58*(12), 1127–1134. https://doi.org/10.1001/archpsyc.58.12.1127

Clarke, G. N., Hornbrook, M., Lynch, F., Polen, M., Gale, J., Beardslee, W. R., … Seeley, J. (2001). A randomized trial of a group cognitive intervention for preventing depression in adolescent offspring of depressed parents. *Archives of General Psychiatry*, *58*(12), 1127–1134. https://doi.org/http://dx.doi.org/10.1001/archpsyc.58.12.1127

Cohen-Woods, S., Craig, I. W., & McGuffin, P. (2013). The current state of play on the molecular genetics of depression. *Psychol Med*, *43*(4), 673–687. https://doi.org/S0033291712001286 [pii]\r10.1017/S0033291712001286 [doi]

Colodro-Conde, L., Couvy-Duchesne, B., Zhu, G., Coventry, W. L., Byrne, E. M., Gordon, S., … Martin, N. G. (2017). A direct test of the diathesis–stress model for depression. *Molecular Psychiatry*, (April), 1–7. https://doi.org/10.1038/mp.2017.130

Compas, B. E., Connor-Smith, J. K., Saltzman, H., Thomsen, a H., & Wadsworth, M. E. (2001). Coping with stress during childhood and adolescence: problems, progress, and

potential in theory and research. *Psychological Bulletin*, *127*(1), 87–127. https://doi.org/10.1037//0033-2909.127.1.87

Compas, B. E., Forehand, R., Keller, G., Champion, J. E., Rakow, A., Reeslund, K., … Cole, D. A. (2009). Randomized controlled trial of a family cognitive-behavioral preventive intervention for children of depressed parents. *Journal of Consulting and Clinical Psychology*. https://doi.org/10.1037/a0016930

Compas, B. E., Forehand, R., Keller, G., Champion, J. E., Rakow, A., Reeslund, K. L., … Cole, D. a. (2010). NIH Public Access, *77*(6). https://doi.org/10.1037/a0016930.Randomized

Compas, B. E., Forehand, R., Thigpen, J. C., Keller, G., Hardcastle, E. J., Cole, D. A., … Roberts, L. (2011). Family group cognitive-behavioral preventive intervention for families of depressed parents: 18- and 24-month outcomes. *Journal of Consulting and Clinical Psychology*, *79*(4), 488–99. https://doi.org/10.1037/a0024254

Compas, B. E., Forehand, R., Thigpen, J., Hardcastle, E., Garai, E., Mckee, L., … Sterba, S. (2015). Preventive Intervention for Children of Depressed Parents. *Journal of Consulting and Clinical Psychology*, *83*(3), 541–553. https://doi.org/10.1037/a0039053

Compas, B. E., Forehand, R., Thigpen, J., Hardcastle, E., Garai, E., McKee, L., … Sterba, S. (2015). Efficacy and moderators of a family group cognitive–behavioral preventive intervention for children of parents with depression. *Journal of Consulting and Clinical Psychology*. https://doi.org/10.1037/a0039053

Compas, B. E., Forehand, R., Thigpen, J., Keller, G., Hardcastle, E., Cole, D. A., … Roberts, L. (2011). Family group cognitive-behavioral preventive intervention for families of depressed parents: 18- and 24-month outcomes. *Journal of Consulting and Clinical Psychology*, *79*(4), 488–99. https://doi.org/10.1037/a0024254

Copeland, W. E., Angold, A., Shanahan, L., & Costello, E. J. (2014). Longitudinal patterns of anxiety from childhood to adulthood: The great smoky mountains study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *53*(1), 21–33. https://doi.org/10.1016/j.jaac.2013.09.017

Copeland, W. E., Wolke, D., Angold, A., & Costello, E. J. (2013). Adult psychiatric outcomes of bullying and being bullied by peers in childhood and adolescence. *JAMA Psychiatry*, *70*(4), 419–26. https://doi.org/10.1001/jamapsychiatry.2013.504

David-Ferdon, C., & Kaslow, N. J. (2008). *Evidence-based psychosocial treatments for child and adolescent depression. Journal of Clinical Child and Adolescent Psychology* (Vol. 37). https://doi.org/10.1080/15374410701817865

Dawson, G. (1994). Frontal Electroencephalographic Correlates of Individual Differences in Emotion Expression in Infants: a Brain Systems Perspective on Emotion. *Monographs of the Society for Research in Child Development*, *59*(2–3), 135–151. https://doi.org/10.1111/j.1540-5834.1994.tb01281.x

Dearing, K. F., & Gotlib, I. H. (2009). Interpretation of ambiguous information in girls at risk for depression. *Journal of Abnormal Child Psychology*, *37*(1), 79–91. https://doi.org/10.1007/s10802-008-9259-z

de Veld, D. M. J., Riksen-Walraven, J. M., & de Weerth, C. (2012). The relation between

emotion regulation strategies and physiological stress responses in middle childhood. *Psychoneuroendocrinology*, *37*(8), 1309–1319. https://doi.org/10.1016/j.psyneuen.2012.01.004

Depression, W. (2012). Fact sheet N 369. *World Health Organizastion.[Online] October*.

Dietz, L. J., Weinberg, R. J., Brent, D. A., & Mufson, L. (2015). Family-based interpersonal psychotherapy for depressed preadolescents: Examining efficacy and potential treatment mechanisms. *Journal of the American Academy of Child and Adolescent Psychiatry*, *54*(3), 191–199. https://doi.org/10.1016/j.jaac.2014.12.011

Dolle, K., Schulte-Körne, G., von Hofacker, N., Izat, Y., & Allgaier, A.-K. (2012). Übereinstimmung von klinischer Diagnose, strukturierten Interviews und Selbstbeurteilungsfragebögen bei Depression im Kindes- und Jugendalter. *Zeitschrift Für Kinder- Und Jugendpsychiatrie Und Psychotherapie*, *40*(6), 405–414. https://doi.org/10.1024/1422-4917/a000200

Döpfner, M., Berner, W., & Lehmkuhl, G. (1994). Handbuch: Fragebogen für Jugendliche. Forschungsergebnisse zur deutschen Fassung der Youth Self-Report Form (YSR) der Child Behavior Checklist.

Döpfner, M., Schmeck, K., & Berner, W. (1994). Handbuch: Elternfragebogen über das Verhalten von Kindern und Jugendlichen. Forschungsergebnisse zur deutschen Fassung der Child Behavior Checklist (CBCL/4-18).

Downey, G., & Coyne, J. C. (1990). Children of depressed parents: An integrative review. *Psychological Bulletin*, *108*(1), 50–76. https://doi.org/10.1037/0033-2909.108.1.50

Downey, G., & Walker, E. (1989). Social cognition and adjustment in children at risk for psychopathology. *Developmental Psychology*, *25*(5), 835–845. https://doi.org/10.1037/0012-1649.25.5.835

Ehring, T., Tuschen-Caffier, B., Schnülle, J., Fischer, S., & Gross, J. J. (2010). Emotion regulation and vulnerability to depression: spontaneous versus instructed use of emotion suppression and reappraisal. *Emotion (Washington, D.C.)*, *10*(4), 563–572. https://doi.org/10.1037/a0019010

Elgar, F. J., Mills, R. S. L., McGrath, P. J., Waschbusch, D. A., & Brownridge, D. A. (2007). Maternal and paternal depressive symptoms and child maladjustment: The mediating role of parental behavior. *Journal of Abnormal Child Psychology*, *35*(6), 943–955. https://doi.org/10.1007/s10802-007-9145-0

Emory, E. K., Hatch, M., Blackmore, C., & Strock, B. (1993). *Psychophysiological responses to stress during pregnancy*. Atlanta, Ga: Centers for Disease Control and Prevention, Division of Reproductive Health.

England, M. J., & Sim, L. J. (2009). Associations Between Depression in Parents and Parenting, Child Health, and Child Psychological Functioning. *Depression in Parents, Parenting, and Children: Opportunities to Improve Identification, Treatment, and Prevention*, 119–182.

Evans, L. D., Kouros, C., A., F. S., McCauley, E., Diamond, G. S., Schloredt, Kelly, A., & Garber, J. (2015). Longitudinal Relations between Stress and Depressive Symptoms in Youth: Coping as a Mediator. *Journal of Abnormal Child Psychology*, *43*(2), 355–368.

https://doi.org/10.1007/s10802-014-9906-5.Longitudinal

Ezpeleta, L., Keeler, G., Erkanli, A., Costello, E. J., & Angold, A. (2001). Epidemiology of psychiatric disability in childhood and adolescence. *J Child Psychol Psychiatry*, *42*(7), 901–914. https://doi.org/10.1111/1469-7610.00786

Foley, D. L., Goldston, D. B., Costello, E. J., & Angold, A. (2006). Proximal Psychiatric Risk Factors for Suicidality in Youth. The Great Smoky Mountains Study. *Archives of General Psychiatry*, *63*, 1017–1024. https://doi.org/10.1001/archpsyc.63.9.1017

Folkman, S., & Moskowitz, J. T. (2004). Coping: pitfalls and promise. *Annual Review of Psychology*, *55*, 745–774. https://doi.org/10.1146/annurev.psych.55.090902.141456

Franke, G. H. (2002). SCL-90-R: The Symptom Checklist by L.R. Derogatis.

Fried, E., & Nesse, R. M. (2015). Depression sum-scores don't add up: Why analyzing specific depression symptoms is essential. *BMC Medicine*.

Garber, J. (2006). Depression in Children and Adolescents. Linking Risk Research and Prevention. *American Journal of Preventive Medicine*, *31*(6 SUPPL. 1), 104–125. https://doi.org/10.1016/j.amepre.2006.07.007

Garber, J., Clarke, G. N., Weersing, V., Beardslee, W. R., Brent, D. A., Gladstone, T. R., … Iyengar, S. (2009). Prevention of depression in at-risk adolescents: a randomized controlled trial. *JAMA Psychiatry*, *301*, 2215–2224.

Garber, J., Clarke, G. N., Weersing, V. R., Beardslee, W. R., Brent, D. A., Gladstone, T. R. G., … Shamseddeen, W. (2009). Prevention of Depression in At-Risk Adolescents: A Randomized Controlled Trial. *NIH Public Access*, *301*(21), 2215–2224. https://doi.org/10.1001/jama.2009.788.Prevention

Garber, J., & Dodge, K. A. (1991). *The developmen of emotion regulation and dysregulation.* New York.

Garber, J., & Flynn, C. (2001). Predictors of depressive cognitions in young adolescents. *Cognitive Therapy and Research*, *25*(4), 353–376. https://doi.org/10.1023/A:1005530402239

Garber, J., Weersing, V., Hollon, S. D., Porta, G., Clarke, G. N., Dickerson, J. F., … Brent, D. A. (2016). Prevention of depression in at-risk adolescents: moderators of long-term response. *Prevention Science*, 1–10. https://doi.org/10.1007/s11121-015-0626-z

Garber Robinson, N.S., J., Garber, J., & Robinson, N. S. (1997). Cognitive vulnerability in children at risk for depression. *Cognition and Emotion*, *11*(5), 619–635. https://doi.org/10.1080/026999397379881b

Gibb, S. J., Fergusson, D. M., & Horwood, L. J. (2011). Relationship separation and mental health problems: findings from a 30-year longitudinal study. *The Australian and New Zealand Journal of Psychiatry*, *45*(2), 163–169. https://doi.org/10.3109/00048674.2010.529603

Glaesmer, H., Brähler, E., & Lersner, U. (2012). Kultursensible Diagnostik in Forschung und Praxis. *Psychotherapeut*, *57*(1), 22–28. https://doi.org/10.1007/s00278-011-0877-5

Goodman, S. H., & Garber, J. (2017). Evidence-based interventions for depressed mothers

and their young children. *Child Development*, *88*(2), 368–377. https://doi.org/10.1111/cdev.12732

Goodman, S. H., & Gotlib, I. (1999a). Risk for Psychopathology in the Children of Depressed Mothers: A Developmental Model for Understanding Mechanisms of Transmission. *Psychological Review*, *106*(3), 458–490. https://doi.org/10.1037/10449-000

Goodman, S. H., & Gotlib, I. H. (1999b). Risk for Psychopathology in the Children of Depressed Mothers : A Developmental Model for Understanding Mechanisms of Transmission, *106*(3), 458–490.

Goodman, S. H., Rouse, M. H., Connell, A. M., Broth, M. R., Hall, C. M., & Heyward, D. (2011). Maternal Depression and Child Psychopathology: A Meta-Analytic Review. *Clinical Child an*, *14*(1), 1–27.

Green, J. G., McLaughlin, K. A., Berglund, P. A., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2010). Childhood adversities and adult psychopathology in the National Comorbidity Survey Replication (NCS-R) I: Associations with first onset of DSM-IV disorders. *Archives of General Psychiatry*, *67*(2), 113. https://doi.org/10.1001/archgenpsychiatry.2009.186

Grillon, C., Warner, V., Hille, J., Merikangas, K. R., Bruder, G. E., Tenke, C. E., … Weissman, M. M. (2005). Families at high and low risk for depression: a three-generation startle study. *Biological Psychiatry*, *57*(9), 953–960. https://doi.org/10.1016/j.biopsych.2005.01.045

Grob, A. Smolenski, C. (2005). Fragebogen zur Erhebung der Emotionsregulation bei Kindern und Jugendlichen (FEEL-KJ). [Questionnaire to assess children and adolescents' emotion regulation].

Gundelfinger, R. (1997). Welche Hilfen brauchen Kinder psychisch kranker Eltern? *Kindheit Und Entwicklung*, (6), 147–151.

Hammen, C. (1988). Self-cognitions, stressful events, and the prediction of depression in children of depressed mothers. *Journal of Abnormal Child Psychology*, *16*(3), 347–360. https://doi.org/10.1007/BF00913805

Hammen, C., Adrian, C., Gordon, D., Burge, D., Jaenicke, C., & Hiroto, D. (1987). Children of depressed mothers: Maternal strain and symptom predictors of dysfunction. *Journal of Abnormal Psychology*. US: American Psychological Association. https://doi.org/10.1037/0021-843X.96.3.190

Hammen, C., Burge, D., & Adrian, C. (1991). Timing of mother and child depression in a longitudinal study of children at risk. *Journal of Consulting and Clinical Psychology*, *59*(2), 341–345.

Handley, S. L., Dunn, T. L., Waldron, G., & Baker, J. M. (1980). Tryptophan, cortisol and puerperal mood. *British Journal of Psychiatry*, *136*(5), 498–508. https://doi.org/10.1192/BJP.136.5.498

Hankin, B., & Abela, J. (2005). Development of Psychopathology: A Vulnerability Stress Perspective.

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. a, McGee, R., & Angell, K. E.

(1998). Development of depression from preadolescence to young adulthood: emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology*, *107*(1), 128–140. https://doi.org/10.1037/0021-843X.107.1.128

Hankin, B. L., Oppenheimer, C., Jenness, J., Barrocas, A., Shapero, B. G., & Goldband, J. (2011). NIH Public Access, *65*(12). https://doi.org/10.1002/jclp.20625.Developmental

Hart, C. H., Newell, L., & Olsen, S. (2003). Parenting skills and social-communicative competence in childhood. *Handbook of Communication and Social Interaction Skills*, (January 2003), 753–797.

Hautzinger, M., Bailer, M., Worall, H., & Keller, F. (1994). *Beck-Depressions-Inventar (BDI). German version. Test manual.(Bearbeitung der deutschen Ausgabe. Testhandbuch.)*. Göttingen: Huber.

Hayden, E. P., Hankin, B. L., Mackrell, S. V. M., Sheikh, H. I., Jordan, P. L., Dozois, D. J. A., … Badanes, L. S. (2014). Parental depression and child cognitive vulnerability predict children's cortisol reactivity. *Development and Psychopathology*, *26*(4), 1445–1460. https://doi.org/10.1017/S0954579414001138

Heitmann, D., & Bauer, U. (2007). Kinder psychisch erkrankter Eltern – Forschungsdesiderata und psychiatrischer Interventionsbedarf. *Zeitschrift Für Pflegewissenschaft Und Psychische Gesundheit*, *1*(1), 5–16. Retrieved from http://www.kindernetzwerk.de

Henggeler, S. W., & Borduin, C. M. (1990). Family therapy and beyond: a multisystemic approach to treating the behavior problems of children and adolescents, 376.

Hetrick, S. E., Cox, G. R., Fisher, C. A., Bhar, S. S., Rice, S. M., Davey, C. G., & Parker, A. G. (2015). Back to basics: Could behavioural therapy be a good treatment option for youth depression? A critical review. *Early Intervention in Psychiatry*, *9*(2), 93–99. https://doi.org/10.1111/eip.12142

Hetrick, S. E., Cox, G. R., Witt, K. G., Bir, J. J., & Merry, S. N. (2016). Cognitive behavioural therapy (CBT), third-wave CBT and interpersonal therapy (IPT) based interventions for preventing depression in children and adolescents. *The Cochrane Database of Systematic Reviews*, (8), CD003380. https://doi.org/10.1002/14651858.CD003380.pub4

Hjemdal, O., Vogel, P. A., Solem, S., Hagen, K., & Stiles, T. C. (2011). The relationship between resilience and levels of anxiety, depression, and obsessive-compulsive symptoms in adolescents. *Clinical Psychology and Psychotherapy*, *18*(4), 314–321. https://doi.org/10.1002/cpp.719

Hops, H., Biglan, a, Sherman, L., Arthur, J., Friedman, L., & Osteen, V. (1987). Home observations of family interactions of depressed women. *Journal of Consulting and Clinical Psychology*, *55*(3), 341–346. https://doi.org/10.1037/0022-006X.55.3.341

Horowitz, J., & Garber, J. (2006). The Prevention of Depressive Symptoms in Children and Adolescents: A Meta-Analytic Review. *J Consult Clin Psychol*, *74*(3), 401–415. Retrieved from http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=paovfth&NEWS=N&AN=00004730-200606000-00001

Horowitz, J. L., & Garber, J. (2006). The prevention of depressive symptoms in children and adolescents: A meta-analytic review. *Journal of Consulting and Clinical Psychology*, *74*(3), 401–415. https://doi.org/10.1037/0022-006X.74.3.401

Horowitz, J. L., Garber, J., Ciesla, J. A., Young, J. F., & Mufson, L. (2007). Prevention of depressive symptoms in adolescents: a randomized trial of cognitive-behavioral and interpersonal prevention programs. *Journal of Consulting and Clinical Psychology*, *75*(5), 693–706. https://doi.org/10.1037/0022-006X.75.5.693

Hosman, Clemens, M. H., van Doesum, K. T. H., & van Santvoort, F. (2009). Prevention of emotional problems and psychiatric risks in children of parents with a mental illness in the Netherlands: I. The scientific basis to a comprehensive approach. *Advances in Mental Health*, *8*(3), 264–276. https://doi.org/10.5172/jamh.8.3.264

Hosman, C. (2009). Prevention of emotional problems and psychiatric risks in children of parents with a mental illness in the Netherlands: I. The scientific basis to a comprehensive. *Australian E-Journal for …*.

Huberty, T. J. (2012). Anxiety and Depression in Children and Adolescents. *Springer*.

Huizink, A. C., Robles de Medina, P. G., Mulder, E. J. H., Visser, G. H. A., & Buitelaar, J. K. (2003). Stress during pregnancy is associated with developmental outcome in infancy. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *44*(6), 810–818.

Hurrelmann, K., Klotz, T., & Haisch, J. (2009). Lehrbuch Prävention und Gesundheitsförderung.

Ihle, W., & Esser, G. (2002). Epidemiologie psyhischer störungen im kindes- und jugendalter: Prävalenz, verlauf, komorbidität und geschlechtsunterschiede. *Psychologische Rundschau*, *53*(4), 159–169. https://doi.org/10.1026//0033-3042.53.4.159

In-Albon, T., Suppiger, A., Schlup, B., Wendler, S., Margraf, J., & Schneider, S. (2008). Validity of the "Diagnostisches interview bei psychischen Störungen" (DIPS für DSM-IV-TR) | Validität des Diagnostischen Interviews bei Psychischen Störungen (DIPS für DSM-IV-TR). *Zeitschrift Fur Klinische Psychologie Und Psychotherapie*, *37*(1), 33–42. https://doi.org/10.1026/1616-3443.37.1.33

Ingram, R. E., & Luxton, D. D. (2005). Vulnerability-Stress Models. *Development of Psychopathology: A Vulnrerably -Stress Perspective*, 32–46. https://doi.org/10.4135/9781452231655

Jacobs, L., & Joseph, S. (1997). Cognitive Triad Inventory and its association with symptoms of depression and anxiety in adolescents. *Personality and Individual Differences*, *22*(5), 769–770. https://doi.org/10.1016/S0191-8869(96)00257-7

Jaffee, S.R., Moffitt, T.E., Caspi, A., Fombonne, E., Poulton, R., and Martin, J. (2002). Differences in Early Childhood Risk Factors for Juvenile-Onset and Adult-Onset Depression. *Archives of General Psychiatry.*, *59*, 215–222.

Jane Costello, E., Erkanli, A., & Angold, A. (2006). Is there an epidemic of child or adolescent depression? *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *47*(12), 1263–1271. https://doi.org/10.1111/j.1469-7610.2006.01682.x

Januar, V., Saffery, R., & Ryan, J. (2015). Epigenetics and depressive disorders: a review of

current progress and future directions. *International Journal of Epidemiology*, *44*(4), 1364–1387. https://doi.org/10.1093/ije/dyu273

Jarosz, A. F., & Wiley, J. (2014). What are the odds? A practical guide to computing and reporting Bayes Factors. *The Journal of Problem Solving*, *7*, 2–9. https://doi.org/10.7771/1932-6246.1167

Jaser, S. S., Langrock, A. M., Keller, G., Merchant, M. J., Benson, M. a, Reeslund, K., … Compas, B. E. (2005). Coping with the stress of parental depression II: adolescent and parent reports of coping and adjustment. *Journal of Clinical Child and Adolescent Psychology : The Official Journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53*, *34*(1), 193–205. https://doi.org/10.1207/s15374424jccp3401_18

Jessor, R., Van Den Bos, J., Vanderryn, J., Costa, F. M., & Turbin, M. S. (1995). Protective factors in adolescent problem behavior: Moderator effects and developmental change. *Developmental Psychology*, *31*(6), 923–933. https://doi.org/10.1037/0012-1649.31.6.923

Joiner, T. E., & Wagner, K. D. (1995). Attributional style and depression in children and adolescents: A meta-analytic review. *Clinical Psychology Review*, *15*(8), 777–798. https://doi.org/10.1016/0272-7358(95)00046-1

Koole, S. L. (2009). The psychology of emotion regulation: An integrative review. *Cognition & Emotion*, *23*, 4–41.

Kovacs, M. (1992). *The Children's Depression, Inventory (CDI)*. North Towanda, NY: Multi-Health System. https://doi.org/10.3724/SP.J.1041.2015.01004

Kovacs, M. (1996). Presentation and course of major depressive disorder during childhood and later years of the life span. *Journal of the American Academy of Child and Adolescent Psychiatry*, *35*(6), 705–15. https://doi.org/10.1097/00004583-199606000-00010

Kovacs, M., Feinberg, T., Crouse-Novak, M., Paulauskas, S., Pollock, M., & Finkelstein, R. (1984). Depressive disorders in childhood.II. A longitudinal study of the risk for a subsequent major depression. *Arch Gen Psychiatry*, *41*, 643–649.

Kraaij, V., Garnefski, N., De Wilde, E. J., Dijkstra, A., Gebhardt, W., Maes, S., & Ter Doest, L. (2003). Negative Life Events and Depressive Symptoms in Late Adolescence: Bonding and Cognitive Coping as Vulnerability Factors? *Journal of Youth and Adolescence*, *32*(3), 185–193. https://doi.org/10.1023/A:1022543419747

Kühner, C. (1997). Fragebogen zur Depressionsdiagnostik nach DSM-IV.

Lazarus, R. S. (1993). From Psychological stress to the emotions.pdf. *Annual Review of Psychology*, (44), 1–21.

Lee, L. C., Halpern, C. T., Irva, H. P., & Martin, S. L. (2006). Child care and social support modify the association between maternal depressive symptoms and early childhood behaviour problems: a US national study. *Journal of Epidemiology & Community Health*, *60*(4), 305–310.

Lenz, A. (2005). Vorstellungen der Kinder ??ber die psychische Erkrankung ihrer Eltern: Eine explorative Studie. *Praxis Der Kinderpsychologie Und Kinderpsychiatrie*, *54*(5), 382–

398.

Lenz, A. (2009). Riskante Lebensbedingungen von Kindern psychisch und suchtkranker Eltern – Stärkung ihrer Resilienzressourcen durch Angebote der Jugendhilfe. *Sachverständigenkommision Des 13. Kinder- Und Jugendberichts*, 1–51.

Lenz, A., & Kuhn, J. (2011). Was stärkt Kinder psychisch kranker Eltern und fördert ihre Entwicklung ? Überblick über die Ergebnisse der Resilienz- und Copingforschung. *Kinder Mit Psychisch Kranken Eltern. Klinik Und Forschung.*, 269–298. Retrieved from http://www.pedocs.de/

Lewinsohn, P. M., & Clarke, G. N. (1999). Psychosocial treatments for adolescent depression. *Clinical Psychology Review*, *19*(3), 329–342. https://doi.org/10.1016/S0272-7358(98)00055-5

Lewinsohn, P. M., Rohde, P., Klein, D. N., & Seeley, J. R. (1999). Natural course of adolescent major depressive disorder: I. Continuity into young adulthood. *Journal of the American Academy of Child and Adolescent Psychiatry*, *38*(1), 56–63. https://doi.org/10.1097/00004583-199901000-00020

Lieb, R., Isensee, B., Höfler, M., Pfister, H., & Wittchen, H. (2002). Parental major depression and the risk of depression and other mental disorders in offspring: A prospective-longitudinal community study. *Archives of General Psychiatry*, *59*(4), 365–374. Retrieved from http://dx.doi.org/10.1001/archpsyc.59.4.365

Lodge, J., Harte, D. K., & Tripp, G. (1998). Children's self-talk under conditions of mild anxiety. *Journal of Anxiety Disorders*, *12*(2), 153–176. https://doi.org/10.1016/S0887-6185(98)00006-1

Loechner, J., Starman, K., Galuschka, K., Tamm, J., Schulte-Körne, G., Rubel, J., & Platt, B. (n.d.). Preventing depression in the offspring of parents with depression: A systematic review and meta-analysis of randomised controlled trials. *Under Review*.

Lonigan, C. J., Elbert, J. C., & Bennett-Johnson, S. (1998). Empirically supported psychosocial interventions for children: An overview. *Journal of Clinical Child Psychology*, *27*, 138–145.

Lüdtke, O., Robitzsch, A., Rautwein, U., & Köller, O. (2007). Umgang mit fehlenden Werten in der psychologischen Forschung: Probleme und Lösungen. *Psychologische Rundschau*, *58*(2), 103–117.

Lyons, M. J., Eisen, S. A., Goldberg, J., True, W., Lin, N., Meyer, J. M., … Tsuang, M. T. (1998). A registry-based twin study of depression in men. *Arch Gen Psychiatry*, *55*(5), 468–472. https://doi.org/http://dx.doi.org/10.1001/archpsyc.55.5.468

Manuscript, A., Depression, C., & Predicts, E. (2013). NIH Public Access, *71*(1), 15–21. https://doi.org/10.1016/j.biopsych.2011.09.023.Cumulative

Mason, W. A., Haggerty, K. P., Fleming, A. P., & Casey-Goldtein, M. (2012). Family intervention to prevent depression and substance use among adolescents of depressed parents. *Journal of Child and Family Studies*, *21*(6), 891–905. https://doi.org/10.1007/s10826-011-9549-x

Masten, A. S. (2001). Ordinary magic: Resilience processes in development. *American*

Psychologist, *56*(3), 227–238. https://doi.org/10.1037//0003-066X.56.3.227

Mathers, C., Fat, D. M., & Boerma, J. T. (2008). *The global burden of disease: 2004 update: World Health Organization.* (World Health Organization, Ed.).

Mathews, A., & Macleod, C. M. (2005). Cognitive vulnerability to emotional disorders. *Annual Review of Clinical Psychology*, *1*, 167–95. https://doi.org/10.1146/annurev.clinpsy.1.102803.143916

Mattejat, F., Lenz, A., & Wiegand-Grefe, S. (2012). Kinder mit psychisch kranken Eltern Klinik und Forschung. *Kinder Mit Psychisch Kranken Eltern. Klinik Und Forschung.*, 13–24.

Mattejat, F., & Remschmidt, H. (2008). The children of mentally ill parents. *Deutsches Ärzteblatt International*, *105*(23), 413–8. https://doi.org/10.3238/arztebl.2008.0413

McLaughlin, K. A. (2011). The public health impact of major depression: a call for interdisciplinary prevention efforts. *Prevention Science : The Official Journal of the Society for Prevention Research*, *12*(4), 361–71. https://doi.org/10.1007/s11121-011-0231-8

Mclaughlin, K. A., Gadermann, A. M., Hwang, I., Sampson, N. A., Al-hamzawi, A., Andrade, L. H., … Kessler, R. C. (n.d.). Parent psychopathology and offspring mental disorders : results from the WHO World Mental Health Surveys. https://doi.org/10.1192/bjp.bp.111.101253

Mendelson, T., & Tandon, S. D. (2016). Prevention of Depression in Childhood and Adolescence. *Child and Adolescent Psychiatric Clinics of North America*, *25*(2), 201–18. https://doi.org/10.1016/j.chc.2015.11.005

Meyer, S., Chrousos, G. P., & Gold, A. (2001). Major depression and the stress system: A life span perspective. *Development & Psychopathology*, *13*, 565–580.

Micco, J. A., Henin, A., & Hirshfeld-Becker, D. R. (2014). Efficacy of interpretation bias modification in depressed adolescents and young adults. *Cognitive Therapy and Research*, *38*(2), 89–102. https://doi.org/10.1007/s10608-013-9578-4

Miranda, R., Fontes, M., & Marroquín, B. (2008). Cognitive content-specificity in future expectancies: Role of hopelessness and intolerance of uncertainty in depression and GAD symptoms. *Behaviour Research and Therapy*, *46*(10), 1151–1159. https://doi.org/10.1016/j.brat.2008.05.009

Miranda, R., & Mennin, D. S. (2007). Depression, generalized anxiety disorder, and certainty in pessimistic predictions about the future. *Cognitive Therapy and Research*, *31*(1), 71–82. https://doi.org/10.1007/s10608-006-9063-4

Monroe, S. M., & Hadjiyannakis, K. (2002). The social environment and depression: focusing on severe life stress. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of Depression* (pp. 314–340). Guilford Press.

Monroe, S. M., & Harkness, K. L. (2005). Life stress, the "kindling" hypothesis, and the recurrence of depression: considerations from a life stress perspective . *Psychological Review*, *112*(2), 417–445. https://doi.org/10.1037/0033-295X.112.2.417

Monroe, S. M., & Simons, A. D. (1991). Diathesis-stress theory in the context of life stress

research: Implications for depressive disorders. *Psychological Bulletin*, *110*, 406–425.

Monroe, S. M., Slavich, G. M., Torres, L. D., & Gotlib, I. H. (2007). Severe life events predict specific patterns of change in cognitive biases in major depression. *Psychological Medicine*, *37*(6), 863–871. https://doi.org/10.1017/S0033291707000281

Montgomery, S. A., & Asperg, M. (1979). A new depression scale designed to be sensitive to change. *British Journal of Psychiatry*, *134*(4), 382–389.

Mrazek, P. J., & Haggerty, R. J. (1994). *Reducing the risks for mental disorders: Frontiers for preventive intervention research*. Washington DC.: National Academy Press.

Muris, P., & Van Der Heiden, S. (2006). Anxiety, depression, and judgments about the probability of future negative and positive events in children. *Journal of Anxiety Disorders*, *20*(2), 252–261. https://doi.org/10.1016/j.janxdis.2004.12.001

National Institute for Clinical Excellence. (2005). Depression in Children and Young People. Identification and management in primary, community and secondary care. *East*, (28).

Oppenheimer, C. W., Hankin, B. L., & Young, J. (2017). Effect of Parenting and Peer Stressors on Cognitive Vulnerability and Risk for Depression among Youth. *Journal of Abnormal Child Psychology*, 1–16.

Perrino, T., Beardslee, W., & Bernal, G. (2015). Toward scientific equity for the prevention of depression and depressive symptoms in vulnerable youth. *Prevention …*.

Petermann, F., & Petermann, U. (2011). Prävention. *Kindheit Und Entwicklung*, *20*(4), 197–200.

Phikala, H., & Johansson, E. E. (2008). Longing and fearing for dialogue with children: depressed parents' way into Beardslee's preventive family intervention. *Nordic Journal of Psychiatry*, *62*(5), 399–404.

Pine, D. S., Cohen, P., & Gurley, D. (1998). The Risk for Early-Adulthood Anxiety and Depressive Disorders in Adolescents With Anxiety and Depressive Disorders. *Archives of General Psychiatry*, *55*(1), 56–64. https://doi.org/10.1001/archpsyc.55.1.56

Pine, D. S., Cohen, P., Gurley, D., Brook, J., & Ma, Y. (2007). The risk for early-adulthood anxiety and depressive disorders in adolescents with anxiety and depressive disorders. *Archives of General Psychiatry*, *55*(1), 56–64. https://doi.org/10.1001/archpsyc.55.1.56

Porges, S. W., Doussard-Roosevelt, J. A., & Maiti, A. K. (1994). Vagal Tone and the Physiological Regulation of Emotion. *Monographs of the Society for Research in Child Development*, *59*(2–3), 167–186. https://doi.org/10.1111/j.1540-5834.1994.tb01283.x

Pound, A., Puckering, C., And, T. C., & Mills, M. (1988). The Impact of Maternal Depression on Young Children. *British Journal of Psychotherapy*, *4*(3), 240–252. https://doi.org/10.1111/j.1752-0118.1988.tb01026.x

Puig-Antich, J., Lukens, E., Davies, M., Goetz, D., Brennan-Quattrock, J., & Todak, G. (1985). Psychosocial functioning in prepubertal major depressive disorders. II. Interpersonal realtionships after sustained recovery from affective episode. *Arch Gen Psychiatry*, *42*(511–517).

Punamäki, R.-L., Paavonen, J., Toikka, S., & Solantaus, T. (2013). Effectiveness of

preventive family intervention in improving cognitive attributions among children of depressed parents: A randomized study. *Journal of Family Psychology*, *27*(4), 683–690. https://doi.org/10.1037/a0033466

Radke-Yarrow, M., & Sherman, T. (1990). Hard growing: children who survive. *Risk and Protective Factors in the Development of Psychopathology*, 97–119.

Rao, U. (2006). Development and natural history of pediatric depression: treatment implications. *Clin Neuropsychiatry: J Treat Eval.*, *3*, 194–2.

Rasing, S., Creemers, D., Stikkelbroek, Y., Kuijpers, Y., & Engels, R. (2016). Adolescent Depression Prevention Growing Up: Challenges and Future Directions. *ISSBD Bulletin*, (2), 20–22.

Reck, C. (2007). [Postpartal depression: possible effects on early mother-child interaction and psychotherapeutical treatment approach]. *Prax Kinderpsychol Kinderpsychiatr*, *56*, 234–244. https://doi.org/10.13109/prkk.2007.56.3.234

Reck, C., Nonnenmacher, N., & Zietlow, A. L. (2016). Intergenerational Transmission of Internalizing Behavior: The Role of Maternal Psychopathology, Child Responsiveness and Maternal Attachment Style Insecurity. *Psychopathology*, *49*(4), 277–284. https://doi.org/10.1159/000446846

Reinecke, M. A., Ryan, N. E., & DuBois, D. L. (1998). Cognitive-behavioral therapy of depression and depressive symptoms during adolescence: A review and meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry*, *37*(1), 26–34. https://doi.org/http://dx.doi.org/10.1097/00004583-199801000-00012

Romens, S. E., Mcdonald, J., Svaren, J., & Pollak, S. D. (2015). Associations Between Early Life Stress and Gene Methylation in Children. *Child Development*, *86*(1), 303–309. https://doi.org/10.1111/cdev.12270

Ronsaville, D. S., Municchi, G., Laney, C., Cizza, G., Meyer, S. E., Haim, A., … Martinez, P. E. (2006). Maternal and environmental factors influence the hypothalamic-pituitary-adrenal axis response to corticotropin-releasing hormone infusion in offspring of mothers with or without mood disorders. *Development and Psychopathology*, *18*(1), 173–194. https://doi.org/10.1017/S095457940606010X

Rost, D. (2007). Interpretation und Bewertung pädagodisch-psychologischer Studien. Eine Einführung. *Weinheim:Beltz*.

Rost, D. (2009). Hochbegabte und hochleistende Jugendliche. Befunde aus dem Marburger Hochbegabtenprojekt. *Waxmann, Münster*, *2*.

Roza, S. J., Hofstra, M. B., van der Ende, J., & Verhulst, F. C. (2003). Stable predictions of mood and anxiety disorders based on behavioral and emotional problems in childhood: A 14-year follow-up during childhood, adolescence, and young adulthood. *American Journal of Psychiatry*, *160*(12), 2116–2121. https://doi.org/10.1176/appi.ajp.160.12.2116

Rutter, M., & Quinton, D. (1984). Parental psychiatric disorder: Effects on children. *Psychological Medicine*, *14*, 853–880.

Sandberg, S., Rutter, M., Giles, S., Owen, A., Champion, L., Nicholls, J., … Drinnan, D. (1993). Assessment of psychosocial experiences in childhood: Methodological issues

and some illustrative findings. *Journal of Child Psychology and Psychiatry*, *34*, 879–897. https://doi.org/10.1111/j.1469-7610.1994.tb01176.x

Sanford, M., Byrne, C., Williams, S., Atley, S., Ridley, T., Miller, J., & Allin, H. (2003a). A pilot study of a parent-education group for families affected by depression. *Canadian Journal of Psychiatry*, *48*(2), 78–86. https://doi.org/10.1177/070674370304800203

Sanford, M., Byrne, C., Williams, S., Atley, S., Ridley, T., Miller, J., & Allin, H. (2003b). A pilot study of a parent-education group for families affected by depression. *The Canadian Journal of Psychiatry*, *48*(2), 78–86. https://doi.org/10.1177/070674370304800203

Schäfer, J. Ö., Naumann, E., Holmes, E. A., Tuschen-Caffier, B., & Samson, A. C. (2016). Emotion Regulation Strategies in Depressive and Anxiety Symptoms in Youth: A Meta-Analytic Review. *Journal of Youth and Adolescence*. https://doi.org/10.1007/s10964-016-0585-0

Scherrmann, T. E., Seizer, H.-U.-, Rutow, R., & Vieten, C. (1992). Psychoedukatiove Angehörigengruppe zur Belastungsreduktion und Rückfallprohylaxe in Familien schizophrener Patienten. *Psychiatrische Praxis*, *19*, 66–71.

Schneider, S., In-Albon, T., Nuendel, B., & Margraf, J. (2013). Parental panic treatment reduces children's long-term psychopathology: A prospective longitudinal study. *Psychotherapy and Psychosomatics*, *82*(5), 346–348. https://doi.org/10.1159/000350448

Schneider, S., Margraf, J., Spörkel, H., & Franzen, U. (1992). Therapiebezogene Diagnostik: Reliabilität des Diagnostischen Interviews bei psychischen Störungen (DIPS). *Diagnostica*, *38*(3), 209–227.

Schulte-Körne, G., & Schiller, Y. (2012). Wirksamkeit universeller und selektiver pr??vention von depression im kindes- und jugendalter. Ein systematischer review. *Zeitschrift Fur Kinder- Und Jugendpsychiatrie Und Psychotherapie*, *40*(6), 385–397. https://doi.org/10.1024/1422-4917/a000198

Sheeber, L. B., Allen, N. B., Leve, C., Davis, B., Shortt, J. W., & Katz, L. F. (2009). Dynamics of affective expereince and behaviour in depressed adolescents. *Journal of Child Psychology and Psychiatry*, *50*(11), 1419–1427.

Siddaway, A. P., Wood., A. M., & Cartwright-Hatton, S. (2014). Involving parents in cognitive-behavioral therapy for child anxiety problems: a case study. *Clinical Case Studies*, *13*(4), 322–335.

Sidebotham, P., & Heron, J. (2006). Child maltreatment in the "children of the nineties": a cohort study of risk factors. *Child Abuse Negl.*, *30*(5), 497–522. https://doi.org/S0145-2134(06)00103-7 [pii];10.1016/j.chiabu.2005.11.005 [doi]

Silk, J., Shaw, D., Forbes, E., Lane, T., & Kovacs, M. (2006a). Maternal depression and child internalizing: The moderating role of child emotion regulation. *Of Clinical Child and*, *35*(1), 116–126. https://doi.org/10.1207/s15374424jccp3501

Silk, J., Shaw, D., Forbes, E., Lane, T., & Kovacs, M. (2006b). Maternal depression and child internalizing: The moderating role of child emotion regulation. *Of Clinical Child and*, *35*(1), 116–126. https://doi.org/10.1207/s15374424jccp3501

Smart, C., Strathdee, G., Watson, S., Murgatroyd, C., & McAllister-Williams, R. H. (2015). Early life trauma, depression and the glucocorticoid receptor gene--an epigenetic perspective. *Psychological Medicine*, *45*(16), 3393–3410. https://doi.org/10.1017/S0033291715001555

Solantaus, T., Paavonen, E. J., Toikka, S., & Punamäki, R. L. (2010). Preventive interventions in families with parental depression: children's psychosocial symptoms and prosocial behaviour. *European Child and Adolescent Psychiatry*, 1–10. https://doi.org/10.1007/s00787-010-0135-3

Solantaus, T., Paavonen, E. J., Toikka, S., & Punamäki, R. L. (2010). Preventive interventions in families with parental depression: children's psychosocial symptoms and prosocial behaviour. *European Child and Adolescent Psychiatry*, *19*(12), 883–92. https://doi.org/10.1007/s00787-010-0135-3

Solantaus, T., Toikka, S., Alasuutari, M., Beardslee, W. R., & Paavonen, E. J. (2009). Safety, Feasibility and Family Experiences of Preventive Interventions for Children and Families with Parental Depression. *International Journal of Mental Health Promotion*, *11*(4), 15–24. https://doi.org/10.1080/14623730.2009.9721796

Somerville, L., Jones, R., & Casey, B. (2010). A time of change: behavioral and neural correlates of adolescent sensitivity to appetitive and aversive environmental cues. *Brain Cogn.*, *1*, 124–133.

Spillane, V., Byrne, M. C., Byrne, M., Leathem, C. S., O'Malley, M., & Cupples, M. E. (2007). Monitoring treatment fidelity in a randomized controlled trial of a complex intervention. *Journal of Advanced Nursing*, *60*(3), 343–352. https://doi.org/10.1111/j.1365-2648.2007.04386.x

Sroufe, L. a, Carlson, E. a, Levy, a K., & Egeland, B. (1999). Implications of attachment theory for developmental psychopathology. *Development and Psychopathology*, *11*(1), 1–13. https://doi.org/10.1017/S0954579499001923

Stark, K. D., Schmidt, K. L., & Joiner, T. E. (1996). Cognitive triad: relationship to depressive symptoms, parents' cognitive triad, and perceived parental messages. *Journal of Abnormal Child Psychology*, *24*(5), 615–31. https://doi.org/10.1007/BF01670103

Stephens, M., Smith, N. J., & Donnelly, P. (2001). A new statistical method for haplotype reconstruction from population data. *American Journal of Human Genetics*, *68*(4), 978–989. https://doi.org/10.1086/319501

Stice, E., Shaw, H., Bohon, C., & Marti, C. N. (2010). A Meta-Analytic Review of Depression Prevention Programs for Children and Adolescents: Factors that Predict Magnitude of Intervention Effects. *J Consult Clin Psychol*, *77*(3), 486–503. https://doi.org/10.1037/a0015168.A

Stieglitz, R.-D. (2002). Familientherapie aus verhaltenstherapeutischer Sicht. In *Paar- und Familientherapie* (pp. 121–135). Berlin, Heidelberg, New Yorl: Springer.

Stiensmeier-Pelster, J., Schürmann, M., & Duda, K. (2000). *Depressionsinventar für Kinder- und Jugendliche (DIKJ).* Hogrefe.

Stiensmeier-Pelster, J., Schürmann, M., Eckert, C., & Pelster, A. (1994). *Attributionsstil-Fragebogen für Kinder und Jugendliche (ASF)*. Göttingen: Hogrefe.

Stockings, E. A., Degenhardt, L., Dobbins, T., Lee, Y. Y., Erskine, H. E., Whiteford, H. A., & Patton, G. (2016). Preventing depression and anxiety in young people: a review of the joint efficacy of universal, selective and indicated prevention. *Psychological Medicine*, *46*(1), 11–26. https://doi.org/10.1017/S0033291715001725

Stringaris, A., Maughan, B., Copeland, W. S., Costello, E. J., & Angold, A. (2013). Irritable mood as a symptom of depression in youth: Prevalence, developmental, and clinical correlates in the Great Smoky Mountains study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *52*(8), 831–840. https://doi.org/10.1016/j.jaac.2013.05.017

Sullivan, P. F., Neale, M. C., & Kendler, K. S. (2000). Genetic Epidemiology of Major Depression: Review and Meta-Analysis. *American Journal of Psychiatry*, *157*(10), 1552–1562. https://doi.org/10.1176/appi.ajp.157.10.1552

Summerfelt, W. T. (2003). Program Strength and Fidelity in Evaluation. *Applied Developmental Science*, *7*(2), 55–61. https://doi.org/10.1207/S1532480XADS0702_2

Suppiger, A., In-Albon, T., Herren, C., Bader, K., Schneider, S., & Margraf, J. (2008). Reliabilit??t des diagnostischen interviews bei psychischen st??rungen (DIPS f??r DSM-IV-TR) unter klinischen routinebedingungen. *Verhaltenstherapie*, *18*(4), 237–244. https://doi.org/10.1159/000169699

Thompson, R. A. (1994). Emotion regulation: a theme in search of definition. *Monographs of the Society for Research in Child Develop*, *2–3*, 25–52.

Tompson, M. C., O Connor, E. E., Kemp, G. N., Langer, D. A., & Asarnow, J. R. (2015). Depression in Childhood and Early Adolescence: Parental Expressed Emotion and Family Functioning. *Annals of Depression and Anxiety*, *2*(7).

Tronick, E., Als, H., Adamson, L., Wise, S., & Brazelton, T. B. (1978a). The Infant's Response to Entrapment between Contradictory Messages in Face-to-Face Interaction. *Journal of the American Academy of Child Psychiatry*, *17*(1), 1–13. https://doi.org/http://dx.doi.org/10.1016/S0002-7138(09)62273-1

Tronick, E., Als, H., Adamson, L., Wise, S., & Brazelton, T. B. (1978b). The Infant's Response to Entrapment between Contradictory Messages in Face-to-Face Interaction. *Journal of the American Academy of Child Psychiatry*, *17*(1), 1–13. https://doi.org/http://dx.doi.org/10.1016/S0002-7138(09)62273-1

Tsuang, M. T., & Faraone, S. V. (1990). *The genetics of mood disorders.* Baltimore: Johns Hopkins University Press.

Van Beveren, M. L., McIntosh, K., Vandevivere, E., Wante, L., Vandeweghe, L., Van Durme, K., … Braet, C. (2016). Associations Between Temperament, Emotion Regulation, and Depression in Youth: The Role of Positive Temperament. *Journal of Child and Family Studies*, *25*(6), 1954–1968. https://doi.org/10.1007/s10826-016-0368-y

Warwick., H., Reardon, T., Cooper, P., Murayama, K., Reynolds, S., Wilson, C., & Creswell, C. (2017). Complete recovery from anxiety disorders following Cognitive Behavioural Therapy in children and adolescents: a meta analysis. *Clinical Psychology Review*, *52*, 77–91.

Watanabe, N., Hunot, V., Omori, I. M., Churchill, R., & Furukawa, T. A. (2007).

Psychotherapy for depression among children and adolescents: A systematic review. *Acta Psychiatrica Scandinavica*, *116*(2), 84–95. https://doi.org/10.1111/j.1600-0447.2007.01018.x

Weersing, V. R., Shamseddeen, W., Garber, J., Hollon, S. D., Clarke, G. N., Beardslee, W. R., … Brent, D. A. (2016). Prevention of Depression in At-Risk Adolescents: Predictors and Moderators of Acute Effects. *Journal of the American Academy of Child and Adolescent Psychiatry*, *55*(3), 219–226. https://doi.org/10.1016/j.jaac.2015.12.015

Weiß., R. (2006). Grundintelligenztest Ksala 2 - Revision (CFT 20-R). *Göttingen*, *Hogrefe*.

Weissman, M. (1997). Offspring of depressed parents: 10 years later. *Archives of …*.

Weissman, M. M., Pilowsky, D. J., Wickramaratne, P. J., Talati, A., Wisniewski, S. R., Fava, M., … STAR*D-Child Team, for the. (2006). Remissions in Maternal Depression and Child Psychopathology. *Jama*, *295*(12), 1389. https://doi.org/10.1001/jama.295.12.1389

Weissman, M. M., SCHUENGEL, C., BAKERMANS–KRANENBURG, M. J., Cicchetti, D., Kovacs, M., WALLIS, J. M., … Hewitt, J. K. (1997). Offspring of Depressed Parents. *Archives of General Psychiatry*, *54*(10), 932. https://doi.org/10.1001/archpsyc.1997.01830220054009

Weissman, M. M., Warner, V., Wickramaratne, P., Moreau, D., & Olfson, M. (1997). Offspring Depressed. *Arch Gen Psychiatry*, *54*, 932–940.

Weissman, M. M., Wickramaratne, P., Nomura, Y., Warner, V., Pilowsky, D., & Verdeli, H. (2006). Offspring of depressed parents: 20 Years later. *American Journal of Psychiatry*, *163*(6), 1001–1008. https://doi.org/10.1176/appi.ajp.163.6.1001

Weisz, J. R., McCarty, C. A., & Valeri, S. M. (2006). Effects of psychotherapy for depression in children and adolescents: a meta-analysis. *Psychological Bulletin*, *132*(1), 132–49. https://doi.org/10.1037/0033-2909.132.1.132

West., S. G., Finch, J. F., & Curran, P. J. (1995). Structural equatation models with nonnormal variables: Problems and remedies. *In R.H.Hoyle (Hg.). Structural Equatation Modeling: Concepts. Issues and Apllications. Thousand Oakes. CA:Sage.*

WHO. (2004). Prevention of mental disorders: Effective interventions, and policy options. Summary report. *Geneva: WHO.*

WHO. (2015). Depression Fact Sheet N°369. *http://www.who.int/mediacentre/factsheets/fs369/en/*.

Wiegand-Grefe, S., Werkmeister, S., Bullinger, M., Plass, A., & Petermann, F. (2012). Gesundheitsbezogene Lebensqualit??t und soziale Unterst??tzung von Kindern psychisch kranker Eltern: Effekte einer manualisierten Familienintervention. *Kindheit Und Entwicklung*, *21*(1), 64–73. https://doi.org/10.1026/0942-5403/a000071

Wittchen, H.-U., Jacobi, F., Klose, M., & Ryl, L. (2010). Gesundheitsberichterstattung des Bundes Heft 51: Depressive Erkrankungen, 43. https://doi.org/10.1016/j.khinf.2010.03.002

Wittchen, H.-U., & Uhmann, S. (2010). The timing of depression: An epidemiological perspective. *Medicographia*, *32*(2), 115–124. Retrieved from http://www.medicographia.com/2010/10/the-timing-of-depression-an-epidemiological-

perspective/

Wittchen, H.-U., Zaudig, M., & Fydrich, T. H. (1997). *SKID - Strukturiertes Klinisches Interview für DSM-IV. Achse I und II Handanweisungen [Structured Clinical Interview for DSM-IV]*. Göttingen: Hogrefe. https://doi.org/10.1026//0084-5345.28.1.68

World Health Organization. (1993). *ICD-10, the ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research.* Geneva.

Wunderlich, U., Wiedemann, G., & Buchkremer, G. (1996). Sind psychosoziale Interventionen bei schizophrenen Patienten wirksam? Eine Metaanalyse. *Verhaltenstherapie1*, *6*, 4–13.

Yalom, I. (1989). *Existentielle Psychotherapie*. (H. Psychologie, Ed.). Köln.

Zhou, X., Hetrick, S. E., Cuijpers, P., Qin, B., Barth, J., Whittington, C. J., … Xie, P. (2015). Comparative efficacy and acceptability of psychotherapies for depression in children and adolescents: A systematic review and network meta-analysis. *World Psychiatry : Official Journal of the World Psychiatric Association (WPA)*, *14*(2), 207–222. https://doi.org/10.1002/wps.20217

Zietlow, A. L., Schlüter, M. K., Nonnenmacher, N., Müller, M., & Reck, C. (2014). Maternal Self-confidence Postpartum and at Pre-school Age: The Role of Depression, Anxiety Disorders, Maternal Attachment Insecurity. *Maternal and Child Health Journal*, *18*(8), 1873–1880. https://doi.org/10.1007/s10995-014-1431-1

Eigenständigkeitserklärung

Ich erkläre hiermit an Eides statt, dass ich die vorliegende Dissertation mit dem Thema „*Transmission and Prevention of Depression in Offspring of Depressed Parents – Differences and changes in psychopathology, emotion regulation and attributional style"* selbständig verfasst, mich außer der angegebenen keiner weiteren Hilfsmittel bedient und alle Erkenntnisse, die aus dem Schrifttum ganz oder annähernd übernommen sind, als solche kenntlich gemacht und nach ihrer Herkunft unter Bezeichnung der Fundstelle einzeln nachgewiesen habe. Ich erkläre des Weiteren, dass die hier vorgelegte Dissertation nicht in gleicher oder in ähnlicher Form bei einer anderen Stelle zur Erlangung eines akademischen Grades eingereicht wurde.

München, den 10.07.2018 Dipl.-Psych. Johanna Löchner

Zustimmung zur Veröffentlichung nach § 6 Abs. 1 UrhG

Hiermit stimme ich ausdrücklich zu, dass meine durch Prof. Dr. Gerd Schulte-Körne und Dr. Belinda Platt betreute bzw. begutachtete Doktorarbeit mit dem Titel „*Transmission and Prevention of Depression in Offspring of Depressed Parents – Differences and changes in psychopathology, emotion regulation and attributional style“* nach Beendigung der Doktorprüfung wissenschaftlichen Zwecken zugänglich gemacht und in der Bibliothek der Ludwigs-Maximilians Universität, München aufgestellt wird (Veröffentlichung nach § 6 Abs. 1 UrhG), sowie dass hieraus im Rahmen des § 51 UrhG zitiert werden kann.

Sämtliche Verwertungsrechte nach §15 UrhG verbleiben davon unberührt bei der Verfasserin der Doktorarbeit.

München, den 10.07.2018 Dipl.-Psych. Johanna Löchner