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Baseline Differences and Intervention Effects of the "Gesund und Glücklich Aufwachsen (GUG-Auf)" Prevention Program for Children of Depressed Parents
- The PRODO trial -

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Zusammenfassung

Kinder eines an Depression erkrankten Elternteils weisen ein erhöhtes Risiko auf, selbst eine psychiatrische Erkrankung zu entwickeln, deshalb bilden sie eine Zielgruppe für Präventionsmaßnahmen und sollten als solche im Gesundheitswesen mit hoher Priorität berücksichtigt werden. Das kognitiv-behaviorale, familien- und gruppenbasierte Präventionsprogramm "Raising Healthy Children (RHC)" zeigte vielversprechende Ergebnisse in der Reduktion der Prävalenz von Depression und allgemeiner Psychopathologie bei einer Stichprobe von Kindern depressiver Eltern in den Vereinigten Staaten von Amerika. Das übergeordnete Ziel der vorliegenden Dissertation ist es, die deutsche Adaptation des RHC Präventionsprogramms zu evaluieren. Die vorliegende Studie ist eine Pionierarbeit, da das Präventionsprogramm in dieser Form noch nicht außerhalb der amerikanischen Forschungsgruppe auf internationaler Ebene evaluiert wurde. Im ersten Schritt wurde untersucht, ob die Psychopathologie von Kindern depressiver Eltern mit der elterlichen Depression (Anzahl depressiver Symptome, Anzahl depressiver Episoden), mit der Anzahl stressiger Lebensereignisse des Kindes sowie dem sozio-ökonomischen Status (SÖS) der Eltern assoziiert ist. Für die Baseline Unterschiede standen Datensätze von 77 Familien (welche an der Studie teilgenommen haben) zur Verfügung, die auch in die statistischen Analysen mit einbezogen wurden. Es konnte nicht gezeigt werden, dass depressive Symptome von Kindern depressiver Eltern zur Baseline mit dem Schweregrad der elterlichen Depression, mit der Anzahl depressiver Episoden, mit der Anzahl von stressigen Lebensereignissen oder dem SÖS assoziiert sind. Jedoch belegen die Ergebnisse, dass externalisierendes Problemverhalten der Kinder mit der Anzahl der elterlichen depressiven Episoden korreliert und, dass die allgemeine Psychopathologie der Kinder mit der Anzahl stressiger Lebensereignisse assoziiert ist. Es konnte kein Zusammenhang zwischen dem SÖS und der psychischen Gesundheit der Kinder festgestellt werden. Im zweiten Schritt wurde untersucht, ob das Präventionsprogramm depressive Symptome der Kinder kurzzeitig (sechs Monate nach der Baseline) reduzieren kann. Die Untersuchung vorläufiger Daten zur Inzidenz von Depression 15 Monate nach der Baseline, ermöglichte es aufzuzeigen in welchem Ausmaß das Präventionsprogramm langfristig Depressionen bei Kindern vorbeugen kann. Weitere zentrale Ziele bestanden darin zu prüfen ob das Programm kurz- und mittelfristig (sechs und neun Monate nach der Baseline) die allgemeine Psychopathologie der Kinder reduzieren, das Wissen der Kinder über Depression verbessern sowie den Erziehungsstil der Eltern positiv verändern kann. Die Studie wurde als randomisierte kontrollierte Studie durchgeführt. 77 Familien wurden randomisiert und wurden entweder der Intervention mit 12 Sitzungen oder einer Warteliste zugeteilt. Die Ergebnisse der Intervention zeigen, dass das Programm sich positiv auf internalisierende und gemischte psychopathologische Symptome der Kinder aus Sicht der Kinder (Selbstbeurteilung) auswirkt. Jedoch wurden diese Ergebnisse aus Sicht der Eltern (Fremdbeurteilung) nicht bestätigt. Aus Sicht der Eltern verbesserten sich beide Symptomskalen unabhängig von der Gruppenzuordnung (Intervention vs. Warteliste) über den Zeitraum von neun Monaten. Es konnte kein Nachweis erbracht werden, dass das Programm sich positiv auf die depressive Symptomatik der Kinder, das Wissen der Kinder über Depression oder die Wahrnehmung der Kinder im Bezug auf den elterlichen Erziehungsstil auswirkt. Vielmehr verbesserte sich das Wissen über Depression über den

Zeitraum von sechs Monaten unabhängig von der Gruppenzuordnung. Eine Evaluation des Feedbacks zeigt, dass die allgemeine Akzeptanz der Teilnehmer hinsichtlich des Präventionsprogramms hoch ist. Die Baseline Ergebnisse zeigen, dass die Psychopathologie der Kinder depressiver Eltern durch manche, aber nicht alle elterlichen Faktoren beeinflusst wird. Obwohl die vorliegende Studie keinen depressions-spezifischen Präventionseffekt aufzeigen konnte, so konnte gezeigt werden, dass manche allgemeinen psychopathologischen Symptome der Kinder mit Hilfe des Programms reduziert werden konnten. Die Studie ist ein wichtiger Schritt um zwingend erforderliche, effektivere Präventionsmethoden zu entwickeln.

Abstract

Since children with a parent suffering from depression are at heightened risk of developing psychiatric disorders themselves, they are a target group for preventive interventions and as such a major public health priority. The cognitive-behavioural, family-, and group-based prevention program "Raising Healthy Children" (RHC) has shown promising findings in reducing the prevalence of depression and general psychopathology in a sample of children of depressed parents in the United States of America. The overarching aim of the current thesis is the evaluation of a German adaption of RHC prevention program. The current study is in this form unique, because the program was not evaluated yet outside the research group on an international level. In the first step I investigated whether the psychopathology of children of depressed parents is associated with parental depression (number of symptoms, number of depressive episodes), number of stressful experiences the child experienced and the parental socio-economic status (SES). For baseline differences, data of 77 families (who took part in an intervention study) were available, which were included in the analyses. There was no evidence that children of depressed parents differ at baseline in depressive symptoms in relation to parent depression severity, number of depressive episodes, the number of stressful experiences or the SES. But evidence emerged that offspring's externalising problem behaviour is related to the number of parental depressive episodes, and offspring's general psychopathology is related to the number of stressful life events. No relation between SES and offspring's mental health could be demonstrated. In a second step, I examined whether the prevention program is effective in reducing child's depression in the short-term (six months after baseline). By investigating preliminary data on incidence of depression at 15 months after baseline, I also investigated the extent to which the intervention prevented depression in the long-term. Further key aims were to see whether the program is effective in reducing child's general psychopathology, and whether the program is effective in enhancing child's knowledge of depression as well as whether it is effective in changing the parenting style in a positive direction in the short- and medium-term (six and nine months after baseline). The study was conducted as randomised controlled trial. The 77 families were randomised to receive the twelve session intervention vs. waiting list. Intervention outcomes suggested that the program has positive effects from child's perspective on child's internalising and mixed psychopathological symptoms. However, the parental view did not confirm these findings. Parent reports indicate that independent of group (intervention vs. waiting list) both symptom scales improved over a period of nine months. No evidence was found that the program shows benefits on child's depression, knowledge of depression or child's perception of parenting style. The knowledge of depression rather enhanced in short-term independent of participation. A feedback evaluation suggested that the general acceptability of the prevention program is high. The baseline findings indicate that the psychopathology of children of depressed parents is influenced by some, but not all, parental factors. Although I could not demonstrate the prevention effect of the program for depression prevention, I could show that some general psychopathological symptoms of children can be reduced by the program. The study provides an important step in the development of more effective prevention, which is exigently required.

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Theoretical Background

Samuel's Story

Samuel is 10 years old. His mother suffers from a recurring depressive disorder. Upon arrival at the "Primary Prevention of Depression in offspring of depressed parents" (PRODO) research unit, Samuel (a fictitious name has been used) and his mother were interviewed. During the interview Samuel reported about his experience living in a family with a depressed parent.

"Sometimes my mom gets very sad and I don't know why. At that time she is just not approachable for us, neither for me nor for my father. Recently she was in hospital for several weeks and I was at home alone. At the beginning I liked it. Dad had to work and I could do what I wanted. So after school I was watching TV for several hours and I could eat as many candies as I desired. But after a while, I started to feel lonely. I had to make sandwiches on my own and no one could drive me to soccer training or to my friends. And no one really cared about me. I am scared that my mom could feel such sadness again and that she will have to go to the hospital and might stay away as long as the last time or even longer."

As in the example of Samuel, there are many children who grow up with a parent suffering from depression. The parental disorder does not only affect the parent itself, but rather the entire family, and particularly offspring is exposed to negative effects of the parental disorder.

Symptoms of Depression

Almost everyone experiences single symptoms of depression, e.g. in association with a stressful event, but major depressive disorder is a distinct experience. The disorder affects the whole organism and is characterized as a typical cluster of symptoms reflected on different levels (cognition, emotion, physiological/vegetative level and behavioural/motoric level),

which are listed below in Table 1. Symptoms have a wide variation and arriving at a diagnosis requires proper assessment ability. Symptoms have to exceed a certain period of time (persistence), intensity and severity (Beesdo-Baum & Wittchen, 2011).

Table 1. The modified presentation illustrates typical symptoms of depression reflected on four levels. The Table is modelled after Beesdo-Baum and Wittchen (2011).

Cognitive Level	**Emotional Level**	**Behavioural/Motoric Level**	**Physiological/vegetative Level**
Negative thoughts	Dolorousness	Slowed motoric and speech	Fatigue
Pessimism	Debt	Retardation or agitation	Loss of energy
Rumination	Emptiness	Suicide attempts	Listlessness
Memory problems	Fearfulness	Decreased activity ratio	Tomorrow deep
Concentration difficulties	Insensibility	Sad, rigid, mask-like,	Vegetative problems (head
Self-doubt	Despondence	tearful facial expression	pressure, stomach trouble)
Grave concern about	Irascibility	Avoidance of eye contact	Loss of appetite and weight
physical health	Desperation		Sleep disturbances
Suicidal thoughts	Dejection		Internal unrest
			Crying
			Loss of libido
			Inner tension
			Sensitive to weather changes

Specifically clinicians who work with children are exposed to the challenge of differentiating between developmental differences and the manifestation of a mental disorder (Huberty, 2012). In general, largely the same diagnostic criteria (see method section: measures: diagnostic instruments) apply to children (Ihle, Ahle, Jahnke, & Esser, 2004), adolescents and adults (Ihle et al., 2004; Lewinsohn, Petit, Joiner Jr., & Seeley, 2003).

However, the clinical picture of paediatric depressive symptoms can vary depending on child's age (Ihle et al., 2004; Sonnenmoser, 2007). An untypical manifestation in children is rather the rule than the exception (Ihle et al., 2004). Particularly younger children are not capable of verbalizing their feelings with the consequence that the disorder is more difficult to identify (Sonnenmoser, 2007). Instead of depressed mood, children and adolescents often show a cranky or irritable mood. Irritable mood in this context differs from irritable mood caused by frustration (Sass, Wittchen, & Zaudig, 1996). Other observed symptoms in children are learning deficiencies, agitation, bed-wetting, aggression (Sonnenmoser, 2007), other

behavioural problems, physical complaints (Birmaher et al., 1996) such as headache, fatigue, abdominal or muscular pains (Ihle et al., 2004), symptoms of anxiety and phobias (Birmaher et al., 1996). Thoughts of death, suicide, suicide attempts, melancholia or impaired functioning is mainly observed in adolescent persons and rises with increasing age (Birmaher et al., 1996). Beside irritability and the other reported symptoms, affected children and adolescents can also show a lack of interest in peers, social isolation, boredom, drug or substance abuse or problems in relationships (Ihle et al., 2004).

Prevalence Rates of Depression and Suicide Risk

Prevalence rates of depression in childhood and adolescence.

General prevalence rates of a unipolar affective disorder for children younger than 13 years are around 3%, while adolescents present twice as high prevalence rates of 6% (Costello, Erkanli, & Angold, 2006). When adolescents develop a depressive episode, the remission rate takes in 10% of cases longer than two years. The recurrence rate within two years is around 40%, and within five years approximately 70% (Birmaher et al., 1996), showing the long-lasting effects of the disorder, which can (lead to and) raise a high personal as well as economic burden.

The initial manifestation to develop a depression is fairly low for younger children until mid-adolescence, but is then growing continuously (Jacobi et al., 2004). The general onset of depression in youngsters reaches its peak between the age of 15 and 20 (Weissman et al., 1997, 2006) and the higher risk for females starts with puberty (Wittchen & Uhmann, 2010). Prevalence rates in adulthood are then twice as high for females than for males (Jacobi et al., 2004), although the genetic influence for both sexes does not differ (Maier, 2004).

Suicide risk associated with depression in childhood and adolescence.

The suicide risk in childhood and adolescence is increased by the occurrence of a depressive disorder (Fergusson & Woodward, 2002). Weissman et al. (1999) demonstrated in a

longitudinal study that depressed adolescents have a five-fold higher risk to attempt suicide in later life compared to non-depressed adolescents. Moreover, depression in childhood and adolescence also increases the developmental risk of other disorders (Schwartz, 2011), which in turn increases – particularly in adolescents – the rate of suicide attempts (Fergusson & Woodward, 2002) and suicide (Rohde, Lewinsohn, & Seeley, 1991). In children and adolescents, suicide is the second leading cause of death, immediately after accidents (Sonnenmoser, 2007).

The high suicide risk and high frequency of suicide attempts in children and adolescents diagnosed with depression is one reason why it is important to intervene before a depression develops and why prevention of depression should gain substantially more relevance. It is known that the disorder is often recurrent and follows chronic trends (Ihle et al., 2004), so that suicide in association with depression remains an important topic, also when children and adolescents grow up. This is reflected in the extreme high suicide rates in affected adults, showing that women are 27 times and men 20.9 times more likely to take their own life compared to common population (Ösby, Brandt, Correia, Ekbom, & Sparén, 2001).

Comorbidities

Comorbidity is the "presence of additional diseases in relation to an index disease in one individual" (Valderas, Starfield, Sibbald, Salisbury, & Roland, 2009, pp. 359), indicating that more than one disease is present at the same point of time (Zimbardo & Gerrig, 2004). Common comorbidities with depression are anxiety disorders, somatoform disorders, dysthymia, disruptive disorders, substance abuse (Angold, Costello, & Erkanli, 1999; Schmid, Fegert, & Petermann, 2010), and during adolescence particularly addictive or abusive behaviour (Fergusson & Woodward, 2002). Younger children have rather small comorbidity rates with drug- or substance-related addiction, but have high rates of concurrent anxiety disorders or somatic complaints (Sonnenmoser, 2007). Comorbidities in general are rather the rule than the exception (Schwartz, 2011), showing that the occurrence of one disorder

increases the developmental risk for other disorders. This indicates that prevention of the first occurring disorder could not only reduce the risk of this one disorder, but could rather reduce the developmental risk of a serious of other diseases.

Risk and Protective Factors

As it is known from adult studies (but is true for children and adolescents as well), unipolar affective disorders are complex disorders which underlie a strong gene-environment interaction (Maier, 2004), so that both *genetic factors* (as e.g. genetic predispositions (Kessler et al., 2003), epigenetic modifications (Maier, 2004), a disbalance of neurotransmitters (Sass et al., 1996)) as well as *environmental factors* (e.g. familial status (Kessler et al., 2003), socioeconomic status (Förstner, Nickel, Mühlbacher, & Simek, 2009) and psychosocial development (Maier, 2004)) contribute to the disease. Particularly environmental loads lend more weight in the development of depression compared to genes (Sullivan, Neale, & Kendler, 2000). The genetic influence was also demonstrated in several studies conducted with children and adolescents (see review (Schulte-Körne & Allgaier, 2008)). However, the developmental risk is also influenced by child's resilience and protective factors (Huberty, 2012), which will be discussed in this section as well.

Risk factors of depression in childhood and adolescence in general.

Diathesis-stress models.

To explain the developmental risk of mental disorders often *Diathesis-Stress Models* are used (Huberty, 2012; Ingram & Luxton, 2005). Diathesis or vulnerability is used equally and stands in relation to the presence and degree of stressors. The diathesis level of a person is determined by individual differences, reflected in e.g. individual genetic predispositions. Stress has many definitions. Essential is the definition of stress in terms of negative or aversive life events which destabilise the individual cognition, physiology and emotion.

Additive and ipsative models are quite similar in explaining a disorder onset: as illustrated in Figure 1, *additive models* assume that the psychopathological development is depending from additive effects of diathesis and stress. In case of a high vulnerability level, only a few stressors are needed to trigger a disorder onset, whereby in case of a low vulnerability level, a high load of stressors is needed to trigger a disorder onset.

Ipsative models suggest that the relationship between diathesis and stress is inverse implementing that the higher the load of one factor, the less load of the other factor is needed to lead to a psychopathological onset. This model anticipates that the sum of both factors leads to a developmental onset. Ipsative models are not necessarily different from additive models, they are rather seen as an additional explanation of existing approaches (Ingram & Luxton, 2005).

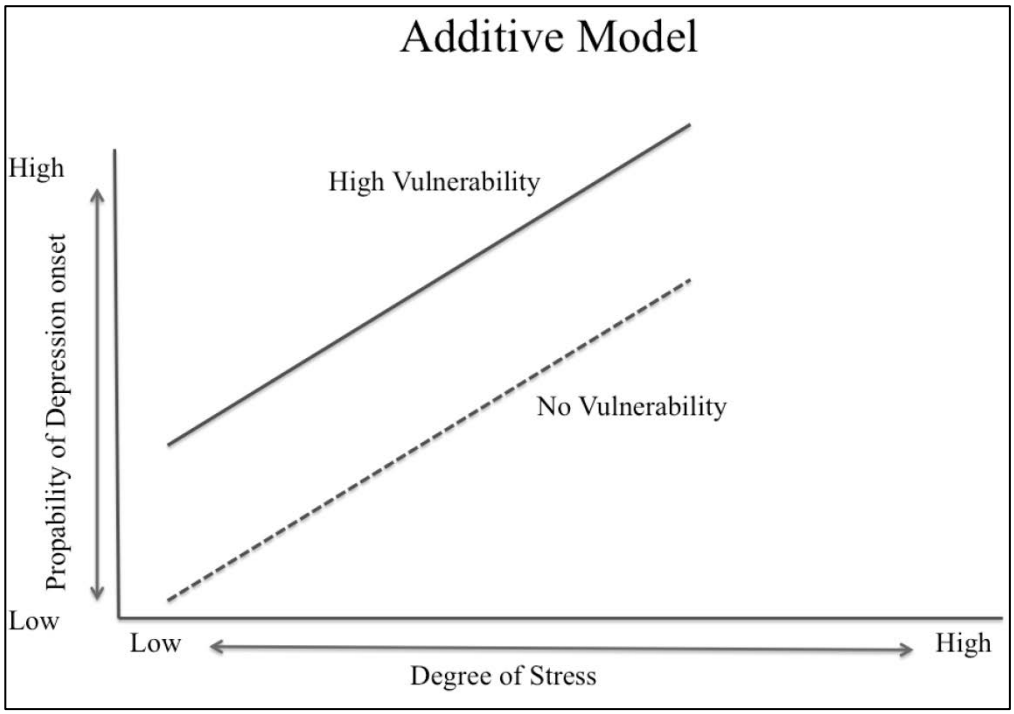

Figure 1. Modified version of the additive model of Monroe and Simons (1991) which shows the relation between stress and vulnerability.

Huberty (2012) explained the developmental risk of a disorder by the interaction of diathesis and resilience, and the inverse relationship between those two components. The child's individual resilience and vulnerability level is depending on the degree of stressors and

risk factors the child is exposed to. Huberty (2012) emphasised that not only the vulnerability level by itself, also child‘s individual ability of resilience plays a crucial role in the developmental onset of a disorder.

In summary, although different models lend different weight to certain components, the basic structure of all models is quite similar (behold Ingram and Luxton (2005)). The risk of developing a disorder is depending on the complex interplay of diathesis and stress (Ingram & Luxton, 2005) or diathesis, resilience and stress (Huberty, 2012).

Gene-environment interaction.

Genetic studies confirm that genes alone do not predict psychopathology. It is rather a complex interplay between genetic and environmental factors which contribute to a disease. Two studies have investigated the interaction between genetic and environmental factors on the development of depression in children, adolescents and young adults (Caspi et al., 2003; Kaufman et al., 2004). Researchers hypothesised that a relationship between the length of the promoterregion of the 5-HTTLPR allele and the risk for developing depression exists. The allele is especially important in the serotonergic system (Caspi et al., 2003) and serotonin is a neurotransmitter which is, among others, involved in controlling the mood (Bear, Connors, & Paradiso, 2007). Caspi et al. (2003) examined 1037 participants aged between three and 26 years and divided them into three groups, depending on genetic differences in the promoterregion of the serotonin-transporter gene 5-HTTLPR. Subgroups had either two copies of the long or short allele version or only one copy of the short and one copy of the long allele version. Results showed that individuals with the short 5-HTTLPR allele have a higher risk to develop depression when stressful life events appear compared to individuals with the long allele version (Caspi et al., 2003).

A replication of the study (Kaufman et al., 2004) that focused on neglected and maltreated children and adolescents confirmed previous findings. However, if children with

the short allele version received social support from attachment figures, the risk for developing depressive symptoms was reduced by up to 50% (Kaufman et al., 2004).

Kaufman et al. (2006) demonstrated in one other study particularly the influence of negative environmental loads on the development of depression. Therefore, ill-treated children were examined who had the same genotypes that are associated with genetic vulnerability: a short allele genotype of the 5-HTTLPR gene and simultaneously a met allele as genotype of the brain-derived neurotropic factor (BDNF). Ill-treated children were more vulnerable for a depressive disorder compared to children who had the same genotypes but were not ill-treated (Kaufman et al., 2006), confirming previous findings, namely that not genetic predispositions by itself, rather the combination of negative environmental factors and a higher genetic vulnerability act as trigger, and that the impact of negative circumstances plays a crucial role whether a depression develops or not. These findings are in line with findings of Sullivan, Neale and Kendler (2000), who emphasised that only around 30 – 40% of variability in depression is due to genetic factors, and around 60% is due to environmental loads (Sullivan et al., 2000), indicating that individual environmental circumstances lend more weight on the developmental course of a depressive disorder than genetic predispositions do.

Also studies examining heritability are in line with these findings: siblings of a person who suffers from a unipolar depressive disorder have a recurrence risk between 2.5% and 3.5% (Craddock & Forty, 2006), while concordance rates of twin studies show differences in the risk to develop a depression for monozygotics with 23 – 50% and for dizygotics with 14 – 37% (Craddock & Forty, 2006; Maier, 2004), reflecting that the disorder is more influenced by environmental loads than by genetic factors. Negative environmental loads can even lead to modifications on epigenetic level, and these changes are then also associated with a higher risk of depression (Januar, Saffery, & Ryan, 2015).

To summarize, presented studies demonstrated that the development of a depressive disorder underlies a complex gene-environment interaction. Particularly the study by Sullivan, Neale and Kendler (2000) showed that the variability of the disorder is rather explained by environmental factors than by genetic predispositions, which also fits to findings of other studies (Craddock & Forty, 2006; Maier, 2004) that investigated familial heritability. Most important is that a positive environment has protective effects, so that children and adolescents have a higher likelihood to stay healthy, despite existing genetic vulnerability. Preventive interventions begin exactly at this point: they try to create a positive environment to reduce the risk of developing depression.

Environmental risk factors.

Socioeconomic status.

It is known that parental socio-economic status (SES) influences children's general wellbeing (Topham et al., 2010) as well as offspring's depression outcomes (Anli & Karsli, 2010; Gilman, Kawachi, Fitzmaurice, & Buka, 2002; Topham et al., 2010; Wirback, Möller, Larsson, Galanti, & Engström, 2014). Most studies that investigated the influence on offspring's depression risk showed that particularly a low SES is associated with a higher developmental risk in later life (Gilman et al., 2002; Huberty, 2012; Luo & Waite, 2005; Wirback et al., 2014). Gilman et al. (2002) emphasised that the risk of developing depression is even twofold higher for children with a low SES. However, one study (Anli & Karsli, 2010) showed that not only a low, also a high SES is (compared to children from middle-class families) associated with an increased developmental risk of depression and other psychopathological outcomes in children and adolescents.

Only one study specifically investigated whether children of depressed parents are affected by SES (Reising et al., 2013). Researcher demonstrated that parental depression and economic disadvantages are associated with offspring's general psychopathology. The current

thesis has the aim to contribute to preexisting findings and therefore investigates whether sub-clinical levels of depression or general psychopathology in children of depressed parents are related to SES (prior to a preventive intervention).

Knowledge of depression.

Only a few studies (Allgaier, Schiller, & Schulte-Körne, 2011; Lenz, 2005; Schiller, Schulte-Körne, Eberle-Sejari, Maier, & Allgaier, 2014) have been conducted which *mainly* focused on child's knowledge of parental mental diseases. Particularly a child's level of knowledge of the parental disorder can either act as resilience or as stress factor for the individual. Too little or insufficient understanding can foster hazy ideas and can even cause feelings of guilt, symptoms of anxiety and helplessness (Lenz, 2005). Lenz (2005) demonstrated in a study based on semi-structured interviews, the insufficient knowledge of a mental disorder of children and adolescents with a mentally ill parent. Children and adolescents (seven – 18 years) had the task to explain possible reasons and causes which trigger the onset of a mental disorder. All participants had problems to report concrete ideas. Particularly younger children showed too little understanding of the parental disorder, reflected in insufficient explanations for the developmental onset. Older children named at least several factors contributing to the disease, whereas younger children named one reason as responsible factor (Lenz, 2005).

A lack of knowledge can lead to stigmatising beliefs in children and adolescents (Calear, Griffiths, & Christensen, 2011), to problems in identifying own depressive symptoms (Hess et al., 2004), and particularly to a reluctance of seeking help (Griffiths, Christensen, & Jorm, 2008). The fact that adolescent's level of knowledge of a depressive disorder and its' treatment possibilities can be improved, at least in short-term, was demonstrated in the study by Allgaier et al. (2011), who reported medium to high short-term effects (Φ =0.28-0.71). The positive findings were confirmed by Schiller et al. (2014), who evaluated the study. Researchers found large effects in scales capturing symptoms, suicidality, pharmacological,

and other treatment opportunities (η^2> .14), and medium effects in scales capturing helping behaviour, causes, and depression as disorder (η^2>.06). Children would like to know more about the parental disorder, about the course of the disease and about treatment possibilities. Particularly children with mentally ill parents would like to be cleared upon how to behave and support the parent (Lenz, 2005). It is known that psychoeducation for offspring of affected parents is relevant in order to convey information about the disorder, and to enable offspring to understand parental behaviour which might differ compared to before. Particularly preventive interventions (Beardslee et al., 1997, 2003, 2007; Compas et al., 2009, 2011, 2015; Clarke, Hornbrook, & Lynch, 2001; Punamäki, Paavonen, Toikka, & Solantaus, 2013; Solantaus, Paavonen, Toikka, & Punamäki, 2010) which focused on preventing depression of children with depressed parents used psychoeducation as one intervention module.

However, only a few studies (Beardslee et al., 1997, 2003, 2007; Christiansen, Anding, Schrott, & Röhrle, 2015) have yet specifically looked into the question of how effective prevention programs are for children of depressed parents in improving child's knowledge of parental depression. While the original study of Beardslee and colleagues (1997) and two replication studies (Beardslee et al., 2007; Christiansen et al., 2015) could demonstrate a significant increase of child's knolwedge level favouring the prevention program (Family Talk Intervention)[1], one replication study (Beardslee et al., 2003) showed that for both conditions (intervention as well as active control) the knowledege level increased.

The current thesis' aim is to contribute and extend to preexisting findings. Therefore one of eight investigations of the current thesis is based exactly on this question as I examine the effectiveness of the "Gesund und Glücklich Aufwachsen (GUG-Auf)" prevention program in improving offspring's knowledge of depression in short- and medium-term.

[1] More detailed information about prevention programs and other outcomes measured by the programs will be given in the section *prevention programs for children and adolescents of parents with depression*.

Parenting style.

One other factor which is known to have an influence on child's development, is the parenting style (Ajilchi & Kargar, 2013; Anli & Karsli, 2010; Compas et al., 2009, 2011; Lipps et al., 2012; Radziszewska, Richardson, Dent, & Flay, 1996; Topham et al., 2010). Parenting styles derive from the two parenting dimensions warmth and control and are subclassified into autocratic, permissive, authoritative and unengaged parenting. *Autocratic parenting* is related to no parental warmth and high level of control. *Permissive parenting* refers to very little control combined with warmth. *Unengaged parenting* neither contains parental control nor warmth. *Authoritative or positive parenting* balances between parental control and warmth (Radziszewska et al., 1996). The four parenting styles are illustrated in Figure 2. The X-axis represents the parenting dimensions *emotional warmth vs. parental control*, the Y-axis represents the *level (low vs. high)* of the two parenting dimensions.

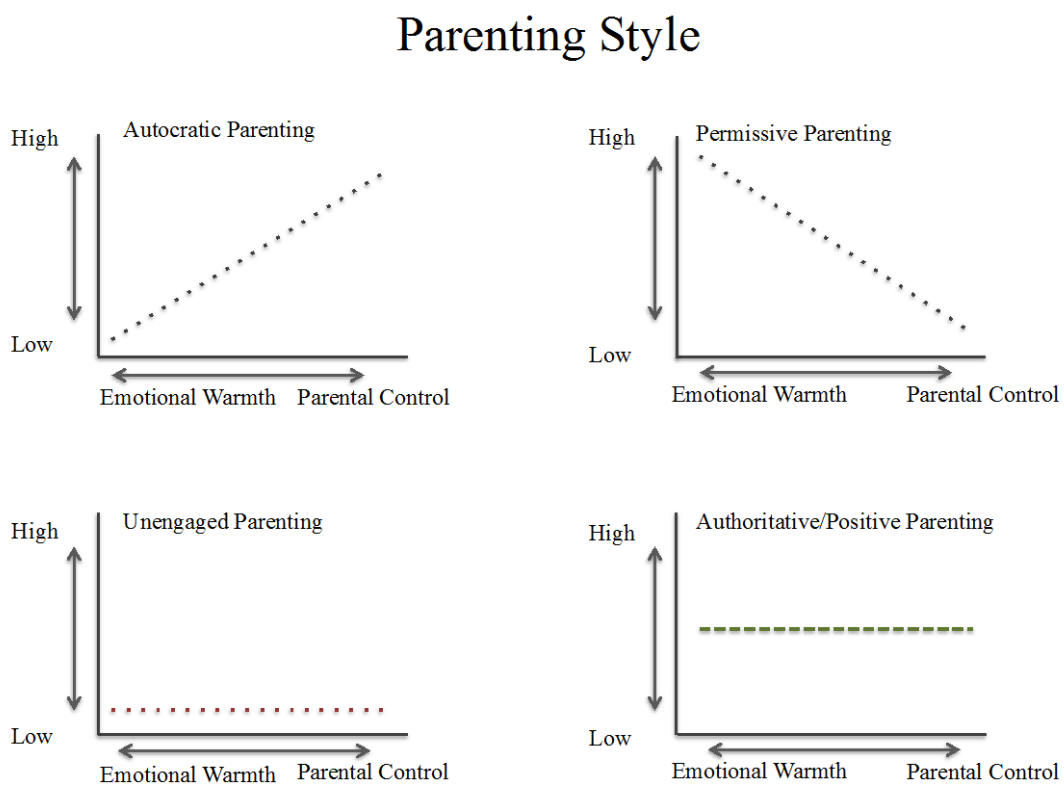

Figure 2. Modified version of the four parenting styles according to Radziszewska et al. (1996).

Research showed, that parenting in only one direction – either overprotection or rejection – increases offsprings‘ psychopathological risk (Ajilchi & Kargar, 2013; Anli & Karsli, 2010; Lipps et al. 2012; Radziszewska et al., 1996). Lowest depression outcomes were found in children and adolescents who experienced authoritative parenting (Anli & Karsli, 2010; Lipps et al., 2012; Piko & Baĺazs, 2012; Radziszewska et al., 1996; Xu, Neece, & Parker, 2014).

In summary, different parenting styles exist and parenting in only one direction increases the risk of offspring's depressive and general psychopathological outcomes. The presented studies focused on the effects of parenting styles (regardless of parental psychopathology) on child outcomes. The risk factor *parenting of a parent suffering from depression* is going to be discussed in the later section *Risk Factors of Depression in Childhood and Adolescence with a Parent suffering from Depression.*

Others.

Other environmental factors which were identified as potential risk factors of a depression onset in children and adolescents are different stressful life experiences (Shapero et al., 2014), as e.g. a poor family or school functioning, insufficient parental support, child's delinquent behaviour, child's bulimic behaviour (Seeley, Stice, & Rohde, 2009), peer stress (Axelson & Birmaher, 2001), being bullied (Axelson & Birmaher, 2001; Copeland, Wolke, Angold, & Costello, 2013; Lereya, Copeland, Costello, & Wolke, 2015; Reijntjes, Kamphuis, Prinzie, & Telch, 2010), or being a victim of cyber attacks (Kowalski, Giumetti, Schroeder, & Lattanner, 2014; Lenhart, 2015). Furthermore, when a disorder is already present, as e.g. an anxiety disorder (Lereya et al., 2015; Reijntjes et al., 2010), somatic complaints (Rigby, 2003) or other general mental health problems (Lereya et al., 2015), the risk of developing depression is increased. Children are not only affected by own stressful experiences, also parental

experiences as early parenthood, chronic difficulties (e.g. alcohol abuse) and marital problems affect child's development (Laucht, Esser, & Schmidt, 1994).

The current thesis investigates the role of five sources of environmental stress on depressive and general psychopathological outcomes *on children of depressed parents*: *1) negative affect of the depressed parent, 2) stressful life events, 3) socio-economic status, 4) limited knowledge of depression,* and *5) parenting style*. Many prevention programs have been developed to reduce offspring's developmental risk of depression and general psychopathology (Beardslee et al., 1997, 2003, 2007, 2013; Christiansen et al., 2015; Clarke et al., 2001; Compas et al., 2009, 2011, 2015; Garber et al., 2009; Sanford et al., 2003; Solantaus et al., 2010; Punamäki et al., 2013). But how exactly these environmental loads modulate offspring´s outcomes has, to my knowledge, only been investigated sparsely or not at all (as e.g. I examine *child's perception* of parenting style) for this target group.

Risk factors of depression in childhood and adolescence with a parent suffering from depression.

Parental depression.

One great risk-factor for children and adolescents to develop depression is a parent suffering from depression (Essau & Petermann, 2000; Sander & McCarty, 2005). In case a parent is or has already been depressed, the risk for offspring to develop the disorder until reaching the age of 20 is at 50% (Beardslee & Podorefsky, 1988; Beardslee & Wheelock, 1994). And if both parents are affected, the risk increases even up to 70% (Robins & Regier, 1991). Therefore children of depressed parents represent one of the greatest risk-groups (World Health Organisation, 2004). They are on the one hand exposed to the same risk factors which were described in the previous section and which are valid for children's and adolescents´ depression risk independent of parental mental health, on the other hand they have (because of parental depression) a higher genetic vulnerability (Goodman & Gotlib, 1999) and are

exposed to more environmental stress (Goodman & Gotlib, 1999) compared to children of non-depressed parents.

Children of affected parents develop the disorder earlier and more severely compared to children of mentally healthy parents (Schulte-Körne & Allgaier, 2008). Also the rate of recurrence is higher in comparison to children, who have no vulnerability in their family history (Goodman, 2007). The parental depression does not only increase offspring's depression risk, also the risk of other mental disorders is increased (Lieb, Isensee, Höfler, Pfister, & Wittchen, 2002). This is reflected in the four times higher rate to generate any affective disorder compared to children of non-depressed parents (Lavoie & Hodgins, 1994).

On the one hand parental depression acts as stressor for children and youngsters (Goodman & Gotlib, 1999), which contributes to the higher interpersonal stress level (Keenan & Hipwell, 2005), on the other hand it can foster a negative family climate (Sander & McCarty, 2005). This can result in an insufficient interpersonal communication between parent and child (Keenan & Hipwell, 2005). It is known that children of mentally ill parents develop massive worrying or feelings of guilt (Lenz, 2005). This can be in fact the result of missing communication. The familiar issues can be transferred by preadolescence into other situations and relationships (Keenan & Hipwell, 2005), so that also offspring's social life and behaviour is influenced in a negative way by the parental disorder.

Already two-year old children of mothers suffering from depression have higher rates of behavioural problems compared to children of non-depressed mothers. Moreover, it was observed that these children do not have an age-appropriate emotional, social and cognitive development (Laucht et al., 1994). This shows that children of depressed parents are negatively affected on different levels and already at early stages of development. Particularly the model by Goodman and Gotlib (1999) underlines previous findings. The model emphasises that already maternal prenatal depression can trigger a dysfunctional neuro-

regulation in offspring, increasing the vulnerability of the child (Goodman & Gotlib, 1999).

Beside the already mentioned potential mechanisms which contribute to the higher developmental risk, Goodman and Gotlib (1999) pointed out that children of depressed parents are exposed to additional environmental loads, such as negative affects, behaviour and cognitions of the affected parent. It is important to mention that the developmental risk is also influenced by the timing and course of parental depression, by the mental health of the other caregiver as well as by offspring's individual characteristics.

The main aim of this section is to point out that children of depressed parents have a general higher risk to develop depression or general psychopathology compared to children of non-depressed parents. They not only have a higher genetic vulnerability, rather they are exposed to more environmental loads compared to children of non-depressed parents, which increase the individual stress level.

This thesis focuses exactly on this target group – *children of depressed parents* – with the main aim to evaluate a program which should reduce the developmental risk of depression and general psychopathology by conveying children adequate coping strategies and training parents in positive parenting. By reducing child's stress level at home as well as the general stress level, a positive environment can be created and a positive development can be supported. On national and international level a few prevention programs (Beardslee et al., 1997, 2003, 2007; Bühler, Kötter, Jaursch, & Lösel, 2011; Clarke et al., 2001; Compas et al., 2009, 2011, 2015; Kötter, Bühler, & Jaursch, 2009; Kötter, Stemmler, Lösel, Bühler, & Jaursch, 2011; Punamäki et al., 2013; Solantaus et al., 2010) already focused on this target group. However, only one research group (Compas et al., 2009, 2011) combined different techniques and involved the entire family in a preventive intervention. The current thesis evaluates and replicates this program with a German population. Therefore I examine whether the preventive intervention is as effective as in USA in reducing offspring's depressive and

general psychopathological symptoms in short- and medium-term, and whether depression can be prevented in long-run. The evaluation and replication outside the research group of Compas and colleagues (2009, 2011) is yet unique and the current work will present first outcomes on international level.

Model of child's development of mentally ill parents.

While previous presented diathesis-stress models explained child's risk to develop depression by a gene-environment interaction, the model of Mattejat, Wüthrich, and Remschmidt (2000) (which is illustrated in Figure 3) explained offspring's development by the influence of biological/genetic predispositions on moderator variables (quality and extent of interpersonal relationships, nature and suitability of disease management, child variable, parent variable, environmental variable) as well as on child's development.

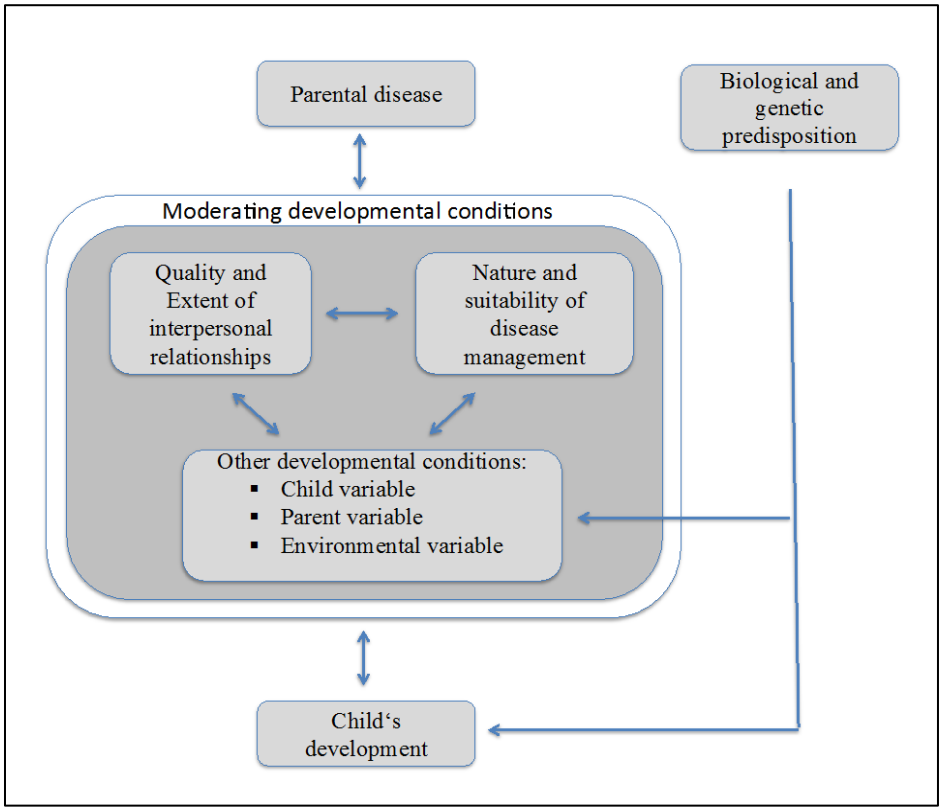

Figure 3. Modified version of the model by Mattejat et al. (2000) demonstrating psychosocial mediating processes on offspring of mentally ill parents.

The really important point is that not only genetic predispositions influence moderator variables, also *the parental mental disorder* affects – and moreover – interacts with moderator variables. In turn, moderator variables interact with child's development and influence offspring's well-being. All variables have either a direct or an indirect effect on offspring's development. And based on the idea that moderator variables are variables which are changeable, the effect on child's outcomes can be steered in different directions. The model by Mattejat et al. (2000) is one explanation how the parental disorder affects offspring's development. As in previous presented diathesis-stress models, it gets visible that offspring's well-being is not depending from one factor alone, rather multiple factors contribute to the developmental course of a child's outcome.

Parenting style of depressed parents.

Parenting (without considering parental depression) and its' influence on offspring's depression and general psychopathological outcomes, was already discussed in the section *Risk factors of depression in childhood and adolescence in general*. The same facts are true for children of depressed parents. Research that particularly examined *parental depression and parents' parenting style* in relation to offspring's psychopathological outcomes is sparse. One review (Downey & Coyne, 1990) summarized effects of parenting of depressed parents on parent-child interactions as well as on child's internalising and externalising symptoms. Researchers concluded that the parental disorder affects the individual parenting style in a negative manner, which in turn results in a poorer parent-child interaction. Particularly hostile and negative parenting skills were found to be linked to a higher likelihood of externalising symptoms in children.

Recent conducted studies that investigated effects of parenting of depressed parents on offspring's psychopathology, have in common that they are mostly based on toddlers or on elementary school children (Dette-Hagenmeyer & Reichle, 2014; Gartstein & Fagot, 2003;

Hummel, Kiel, & Zvirblyte, 2016; Xu et al., 2014). Findings show that parental depression affects parenting, visible in e.g. a reduction of parental warmth (Hummel et al., 2016), which in turn affects child's psychopathology in a negative manner (England & Sim, 2009; Hummel et al., 2016). Poor parenting skills are more likely to be found in depressed than in non-depressed mothers (Hops, 1995), which is e.g. reflected in a limited affective expression, behaviour, less positive responses and a slowdown of speech (Downey & Coyne, 1990). Particularly repellent, neglecting and passive parental behaviour is associated with parental depression (Frye & Garber, 2005; Goodman & Gotlib, 1999), reflecting the fact that children of depressed (vs. non-depressed) parents are exposed to a higher level of negative loads which come along with parental depression, and also influence child's development. One risk is that children adopt observed strategies, as e.g. negative expectations, a poor self-effectiveness or dysfunctional cognitions (Frye & Garber, 2005; Goodman & Gotlib, 1999).

Research demonstrated that parenting is changeable, and that parents´ parenting can be directed into a more authoritative/positive parenting style already by an eight week training period, visible in reductions of offspring's depression outcomes (Ajilchi & Kargar, 2013), and an increase of offspring's self-esteem (Ajilchi, Borjali, & Janbozorgi, 2011). A change of dysfunctional parenting skills can diminish later psychopathological problems in children and adolescents. Particularly high-risk children benefit from changes of dysfunctional parenting skills (Hudson, 2014).

Some prevention programs that target children of depressed parents use strategies to modify dysfunctional parenting, and thereby decrease offspring's developmental risk (Compas et al., 2009, 2011, 2015; Forehand et al., 2012; McKee et al., 2014). However, most conducted studies did not focus on whether the preventive intervention really led to positive changes of parenting or not. They rather focused on child's outcomes. Only one study (Sanford et al., 2003) investigated whether parenting of depressed parents can be modified in a positive manner by a brief parenting training. However, researchers only included depressed

parents in the rating, so that parents rated on their own whether positive changes in parenting style occurred or not.

The current thesis investigates whether the GUG-Auf prevention program is effective in changing the parenting style of the primary affected parent (and in case the other caregiver participated too also of the other caregiver). The *unique approach of the current thesis* is that not parents but *children state whether the GUG-Auf prevention program leads to positive changes of parenting style or not.* Therefore, children have the task to rate on six parenting dimensions (support, restriction, praise, blame, inconsistency, punishment) whether they perceive changes in parenting from baseline to the nine-month follow-up or not.

In summary, not many studies have been conducted which particularly investigated effects of parental depression in association with parenting on child's development. Presented studies targeted very young children, mostly with a depressed mother. Results showed that the parental mood disorder has a negative impact on parenting, which in turn affects child's later development. By modifying dysfunctional parenting into positive parenting, the psychopathological risk of children can be diminished.

This is one aim of the current study: to evaluate whether the prevention program is effective in changing the dysfunctional parenting of depressed parents into positive parenting by conveying parents positive parenting strategies, and then investigating whether changes are visible in short- and medium-term. In the current study, changes of parenting style are rated by children and adolescents, so that their perspective is in focus. Although some prevention programs used strategies to modify dysfunctional parenting, no study that focused on children of depressed parents explicitly investigated whether the parenting style can be modified in a positive manner from child's perspective by a preventive intervention.

Resiliency and protective factors.

In order to understand why some children develop depression or other psychopathologies and others do not, although they are exposed to the same risk factors, it is useful to consider

child's characteristics like resilience as well as protective factors in child's life. These factors might modify child's developmental risk in a positive manner. Resilience defines the ability of positive adaption in case a person is exposed to adverse or risky situations (Masten et al., 2004). The degree of resilience or vulnerability depends on predispositional and psychosocial factors. Each child differs in the individual threshold to develop a mental disorder, and exceeding the threshold depends on exposed risk and stress factors, resilience and vulnerability (Huberty, 2012). Protective factors are e.g. an intact familiar environment (Beach et al., 2016) or appropriate coping strategies (Huberty, 2012). They have the aim to prevent or at least to minimize negative outcomes in offspring who experience stressful situations. However, the influence of protective and risk factors is not easily explainable. Already a single negative event can cause a series of reactions creating conditions for other factors which raise the risk or trigger it (Huberty, 2012). Table 2 lists possible protective and risk factors, subclassified in individual, genetic and biological factors.

Table 2. The modified Table that is modelled after Huberty (2012), lists protective and risk factors with regard to individual, genetic and biological factors.

Context	*Protective factors*	*Risk factors*
Individual	▪ Intelligence within the normal range or higher ▪ Gender ▪ Absence of attention deficits ▪ Absence of impulse control ▪ Appropriate to the age good emotion regulation and social skills	▪ Intelligence low ▪ Not appropriate emotion regulation ▪ Self-esteem or self-efficacy low ▪ Problems with impulse control ▪ Shyness
Genetical	▪ Absence or only low impact of hereditary/genetic disorders ▪ Absence of stressors that could trigger genetic predispositions	▪ Genetic predispositions ▪ Genetic/hereditary disorders
Biological	▪ A minimal or no effect of neurological or biological problems ▪ Easy individual temperament	▪ Prenatal injury or infections ▪ In utero toxin exposure ▪ Substance abuse of the mother ▪ Poor nutrition and maternal care

As it gets visible in Table 2, individual factors like intelligence can either act as protective or risk factor for child's psychopathological development. Also the absence or

presence of impulse control or attention deficits can de- or increase the likelihood of child's psychopathology. Also genetic predispositions and neurobiological factors, as e.g. the absence or presence of neurological problems have an influence on the developmental course. These factors are difficult to control. However, it is useful to consider that some children are more vulnerable or have a higher resilience than others, so that outcomes can differ depending on these individual characteristics. The prevention program supports the creation of protective factors like e.g. approriate coping strategies and an intact familiar environment.

Treatment Possibilities for Children and Adolescents suffering from Depression

A number of treatment opportunities have evolved for children and adolescents suffering from depression (Compton et al., 2004; Horowitz, Garber, Ciesla, Young, & Mufson, 2007). Although treatment opportunities for adults apply similarly, they should not be generally transferred to children and adolescents, because treatment efficacy in minors is depending on child's age and child's development (Dolle & Schulte-Körne, 2013).

Considering these two factors, most known treatment approaches for depressed children and adolescents are nondirective supportive therapy (Zhou et al., 2015), cognitive-behavioural therapy (CBT) (Brent et al., 1997; Ihle et al., 2004; Sonnenmoser, 2007; Zhou et al., 2015), systemic behavioural family therapy (Brent et al., 1997), family therapy (FT) (Sonnenmoser, 2007; Zhou et al., 2015), problem solving therapy (Zhou et al., 2015), play therapy (Sonnenmoser, 2007; Zhou et al., 2015), interpersonal psychotherapy (IPT) (Ihle et al., 2004; Zhou et al., 2015), medication (Emslie et al., 1997, 2002) and online offers (Calear & Christensen, 2010b; Ebert et al., 2015).

A recent network meta-analysis based on 52 Randomised Controlled Trials (RCT) investigated the efficacy of nine different psychotherapeutic interventions for treating depressed children and adolescents (Zhou et al., 2015). It demonstrated highest benefits and effects for CBT (SMD:-0.46 (-0.74 to -0.18)) and IPT (SMD:-0.59 (-1.00 to -0.18)) approaches (Zhou et al., 2015), which matches with findings of other studies that were not

part of the meta-analysis (Compton et al., 2004; Dietz, Weinberg, Brent, & Mufson, 2015; Ihle et al., 2004). Particularly mild and moderate depression benefit mostly from CBT (Ihle et al., 2004). Due to the fact that both, IPT as well as CBT, lead to significant reductions of depressive symptoms, current psychotherapeutic interventions of choice for affected children (Horowitz et al., 2007; Zhou et al., 2015) and adolescents (Horowitz et al., 2007; Ihle et al., 2004; Zhou et al., 2015) are mainly based on those two methods. Play therapy is primarily used for treating very young children (Sonnenmoser, 2007).

With regard to medication, the medicine of choice for depression treatment in children and adolescents are selective serotonin reuptake inhibitors (SSRI) (Ihle et al., 2004), as e.g. the antidepressant Fluoxetine (Dolle & Schulte-Körne, 2013; Emslie et al., 1997, 2002). This is an approved and evidenced-based medication from the age of eight (Sonnenmoser, 2007). It is known to be highly effective in treating minors (Emslie et al., 1997, 2002; Ihle et al., 2004). It is well tolerated and in acute paediatric cases, a daily intake of 20 mg is recommended (Emslie et al., 1997, 2002). Children treated with tricyclic antidepressants showed no and adolescents only very little effects (Hazell, O'Connell, Heathcote, & Henry, 2002). In accordance with the guidelines for treating depressive disorders in children and adolescents (Dolle & Schulte-Körne, 2013), tricyclic antidepressants should not be prescribed to this target group.

But beside mentioned approaches, online offers also gain relevance. Available online programs for high-risk children and adolescents are e.g. CATCH-IT or MoodGYM. The example programs use CBT elements to modify online dysfunctional beliefs or thoughts, to teach reward strategies, to deliver methods of relaxation and problem solving with the aim to reduce depressive symptoms in the target population (Calear & Christensen, 2010a). Treatment opportunities for children and adolescents suffering from depression are described in detail elsewhere (Dolle & Schulte-Körne, 2013).

In summary, many different treatment approaches are available for children and adolescents who already suffer from depression. Thereby, most promising approaches are CBT and IPT. However, treatment approaches find primary use, when the disorder developed and symptoms are visible already. Particularly the chronic course and the recurrence of depression (Ihle et al., 2004) cause *high economic expenses and economic burden* (Luppa, Heinrich, Angermeyer, König, & Riedel-Heller, 2007).

The total of indirect (incapacity for work, absences) and direct (treatment) costs of depression for the society are estimated at 83.1 billion US$ in the US only (Greenberg et al., 2003). Per case, worldwide annual costs range between $200 and $400 for mortality, between $2000 and $3700 for morbidity, and between $1000 and $2500 for direct non-medical and medical costs (Luppa et al., 2007). In Germany, the annual costs for affected persons amount to 458.90 € (≈ $514.82), whereby unemployment and severity of the disorder significantly influence arising expenses. In case affected persons were diagnosed with a severe depressive disorder, the costs rose two times compared to persons with a diagnosis of moderate depression, and even five times compared to persons with a diagnosis of mild depression. 43.9% of costs are caused by inpatient treatment with annual expenses of 8782 € (≈ $9849).

Long-term consequences and costs should prompt us to adopt a different approach and to focus on preventive interventions, instead of intervening only when an outbreak occurs and people actively seek psychological and/or medical advice. Evidence suggests that depression is preventable (Jane-Llopis, 2003), therefore, in combination with treatment, the focus of the national health care system should shift to prevention. By implementing preventive interventions, the developmental risk as well as long-term costs could be diminished. Specifically high-risk groups but also health insurance companies would benefit from this procedure. Berking and Rief (2012) emphasised that preventive approaches are urgently needed, not only to reduce the enormous costs for our health care system, but also for ethical responsibility.

Prevention of Depression

Prevention introduction.

The term prevention refers to all steps that hinder health damage, reduce the likelihood of a disorder or delay its onset. Preventive interventions have the aim to reduce the number of new onsets of diseases, disabilities or premature death. They also strive to diminish relapse rates and new cases (Berking & Rief, 2012).

Differentiation of interventions regarding specificity.

Prevention approaches can be categorised as universal, selective or indicated approaches (Berking & Rief, 2012). Universal interventions address the entire population (Berking & Rief, 2012), as e.g. the Lars & Lisa program, which is used in school-based settings with the aim to prevent depressive symptoms in adolescents (Pössel, Horn, Seemann, & Hautzinger, 2004). Selective interventions address individuals with an elevated risk of depression (Berking & Rief, 2012), with the aim to reduce an already pre-existing risk (Petermann & Petermann, 2011). At intervention beginning, individuals are free of symptoms (Berking & Rief, 2012). An example of selective preventive interventions is the Raising Healthy Children program (Compas et al., 2009, 2011), which is used in preventing depression and general psychopathological outcomes in children of depressed parents. Indicated models apply to individuals who have prodromal symptoms, so that criteria are not fulfilled, but symptoms exist already (Schulte-Körne & Schiller, 2012). An example is a skill training based on interpersonal psychotherapy for adolescents with elevated depressive symptoms (Young, Mufson, & Gallop, 2010) with the aim to reduce depressive symptoms and to enhance overall functioning. All three approaches have the same aim, namely to reduce overall risk factors, among others by using psychoeducative methods, promoting social resources (Berking & Rief, 2012), strengthening social competence, modifying solving strategies, learning relaxation

techniques or by improving coping mechanisms how to deal with stressful situations (National Research Council & Institute of Medicine [NRC & IOM], 2009).

There are pros and cons of the different intervention forms. An advantage of universal approaches is that costs are comparatively low (Horowitz & Garber, 2006), but because effects vary from low to high (d = 0.30 – 1.40) (Calear & Christensen, 2010b), results are very heterogeneous. Furthermore, the generally low effect sizes mean that selective approaches have a better cost-benefit ratio (Pössel & Hautzinger, 2003). Schulte-Körne and Schiller (2012) outlined in a review the evidence of the different prevention types particularly focusing on children and adolescents for preventing depression. Based on ten systematic reviews (including in total 121 controlled studies), all intervention types showed short-term to middle-term effects (until nine months), reflected in a reduction of depressive symptoms. Long-term effects (twelve months) of universal models could not be demonstrated, but selective and indicated preventive interventions even showed effects until twelve-month follow-up. Effect sizes of selective interventions vary from d = 0.34 – 1.05, of indicated approaches from d = 0.31 – 1.00 and of universal interventions from d = 0.02 – 0.66 (Schulte-Körne & Schiller, 2012). For selective and indicated models all reviews report a significant reduction of depressive symptoms with small to moderate effect sizes (Merry et al., 2012; Stice, Shaw, Bohon, Marti, & Rohde, 2009), implementing higher efficacy compared to universal models (Merry et al., 2012; Schulte-Körne & Schiller, 2012).

Only one study (Pössel, Adelson, & Hautzinger, 2011) based on a universal approach reported long-term effects, but only for girls and not for boys. Researchers emphasised that positive effects in girls who passed through the program were relatively stable from six to twelve months, while depression scores of girls from control condition increased for the same time period (with a moderate effect size d = - 0.58 of acceleration of depressive symptoms for girls from EG vs. CG).

Individual - versus environmental - focused prevention.

In terms of intervention starting point, differentiations are made between individual-centered (behavioural prevention) and environment-oriented preventive interventions. Individual-centered prevention addresses a single person, their behaviour and traits. One example of individual-centered prevention is the reduction of consequential losses of external problem behaviour of minors by using strategies like relaxation, building up social competences and learning conflict management. Environment-oriented preventive intervention is not directly focusing on a person but rather external conditions are addressed, so that the personal life situation and circumstances are improved. Due to positive changes of social, cultural, physiological and ecological context, the individual burden is minimized and the external problem behaviour is reduced by enhanced circumstances (Berking & Rief, 2012). An analysis of successful prevention programs regarding structural and content-related conceptualisation suggests that successful interventions are the result of common characteristics: they are environment-oriented as well as individual-centered, theoretically well-founded, consider individual deficits and resources, are cross-situational usable, manualised, culturally adapted, and based on trained leaders (Berking & Rief, 2012).

Prevention programs for children and adolescents of parents with depression.

There exist only a few targeted prevention programs that have been developed for children of depressed parents. Most commonly evaluated are the four programs Parenting Training (Sanford et al., 2003), Family Talk Intervention (Beardslee et al., 1997, 2003, 2007), Coping with Depression (Beardslee et al., 2013; Brent et al., 2015; Clarke et al., 2001; Garber et al., 2009, 2016) and Raising Healthy Children (Compas et al., 2009, 2011). In terms of number of sessions and intervention duration, the programs are mostly analogical and have been developed either in Canada (Sanford et al., 2003) or in the USA (Beardslee et al., 1997; Clarke et al., 2001; Compas et al., 2009; Garber et al., 2009). Evaluations were conducted on international level, as for instance the FTI in Finland (Solantaus et al., 2010; Punamäki et al.,

2013) or Germany (Christiansen et al., 2015). Regarding inclusion criteria, the programs of Beardslee et al. (1997, 2003, 2007), Clarke et al. (2001) and Compas et al. (2009, 2011, 2015) also consider children and adolescents with a depression history. Regarding the content level, programs differ in some aspects, but have also common components.

Table 3 gives an overview of the presented prevention programs, participants targeted, number of sessions and duration of the single intervention types. The four intervention types are described in the following subsections, with the goal to show what effects can be reached with certain intervention designs and further to make clear why the current work evaluates the one certain preventive intervention of Compas and colleagues (2009, 2011).

Table 3. The Table gives an overview of the presented preventive interventions. The first column illustrates the preventive intervention (PT = Parenting Training, FTI = Family Talk Intervention, CWD = Coping With Depression, RHC = Raising Healthy Children), followed by the column published studies in which researchers who published journals regarding the intervention type are listed, followed by the column participants that describes the target group of the intervention (focus on parents alone, offspring alone or entire family), followed by the number of sessions conducted, and average intervention duration in months/years.

Preventive Intervention	**Published studies**	**Participants**	**Session number**	**Intervention duration**
PT	Sanford et al., 2003	Parents alone	8	2 months
FTI	Beardslee et al., 1997, 2003, 2007	Entire family	6-10	6-9 months
	Solantaus et al., 2010		6	2-4 months
	Punamaki et al., 2013		6	2-4 months
	Christiansen et al., 2015		6-7	3 months
CWD	Clarke et al. 2001	Offspring	15	4 months
	Garber et al., 2009, 2016	alone	14	6-8 months
	Beardslee et al., 2013		14	6-8 months
	Brent et al., 2015		14	6-8 months
RHC	Compas et al., 2009, 2011, 2015	Entire family	12	6 months
	Forehand et al., 2012		12	6 months
	McKee et al., 2014		12	6 months

Parenting Training.

The Parenting Training (PT) of Sanford et al. (2003) has its focus on depressed parents with offspring aged between six and 13 years. The two main pillars are psychoeducation and parenting training. The goal of the former is to provide knowledge of the disorder, to support

in finding problem solutions, to show the impact of the disorder on the family and to enhance communication within the family. The latter conveys cognitive and social learning strategies as well as positive parenting strategies. Contents are transferred in eight weekly two-hour sessions *to parents alone*. Via didactic materials and videos, different parenting types are shown that are subsequently discussed, and positive parenting strategies are worked out. In form of homework, these strategies are then implemented in parents' daily life. The aim of the program is to deliver information as well as strategies to parents, which are then indirectly transferred to offspring by changes of parental attitude and behaviour (Sanford et al., 2003).

The strength of the program is that it conveys psychoeducative elements as well as positive parenting, and that parents have the homework to implement acquired knowledge in their daily life. Moreover, also partners of depressed parents can participate in the program. However the weakness of the program is that only parents are included in the program, whereas children are not involved at all.

With regard to the research evidence, Sanford et al. (2003) utilised a waiting list as control group, which has the advantage to show true prevention effects (compared to an active control condition). Results showed positive trends (although not significant) for EG vs. CG, with medium effect sizes in the scales disagreement between parents (d = 0.6), parental competence (d = 0.4) and innerfamiliar conflict situations (d = 0.6). One weakness of the study is the small sample size (n = 25), which is linked to the high dropout rate (during posttreatment: 27%, during three-month follow-up measurements: 43%). The other weakness is that only parent reports were utilised as outcome measure, and self-reports of children were not considered at all. The parenting training of Sanford et al. (2003) is much less examined compared to other prevention programs.

In conclusion, the program conveys two important modules which are known to reduce the psychopathological risk in children. But these modules are solely conveyed to parents. The main limitations of the study are the small sample size, the high dropout rate and

particularly the fact that only parent reports are used as outcome measure, so that effects on child's psychopathology are not captured by child's perspective.

Family Talk Intervention.

In contrast to the PT program from Sanford et al. (2003), the Family Talk Intervention (FTI) from Beardslee et al. (1997), that has been utilised several times since the first implementation, (Beardslee, Gladstone, Wright, & Cooper, 2003; Beardslee et al., 2007; Christiansen et al., 2015; Punamäki et al., 2013; Solantaus et al., 2010) also *includes children of affected parents*. As the PT (Sanford et al., 2003), the FTI is based on psychoeducation, but it solely uses modules from the family system therapy.

The program that is conveyed to participants from EG is a clinician-facilitated program which consists of six to ten sessions, with an average of seven sessions. Lecture sessions are hold for children (aged between eight and 15 years), parents and the entire family. The purpose of the program is to reduce offspring's psychopathological risk by decreasing risk factors in child's environment. Program contents are based on sharing individual experiences with depression, gaining a better understanding of the disorder, discussing concerns and encouraging parents to talk about the disorder within the family.

The strength of the program is that children as well as parents are included in the program. One particular advantage is that all eligible children per family are invited to participate, so that more than two family members can share experiences. One weakness of the program is that it focuses on *psychoeducation only*, so that a combination of different approaches (compared to the parenting training) is not intended.

With regard to research evidence, Beardslee et al. (1997, 2003, 2007) utilised an *active control solely with parents*. The lecture format consists of two sessions with almost the same contents as the ones of the intervention group (except individual experiences with the disorder). Follow-ups were conducted one, two and a half (Beardslee et al., 2003) and four and a half (Beardslee et al., 2007) years after the treatment. Both conditions (FTI and lecture

format) could demonstrate that changes in parents' behaviour and attitudes led to changes in child's behaviour and attitudes. These changes are long-lasting and visible in an increase of protective factors and a diminution of risk factors. However, children from the intervention condition had in the original (Beardslee et al., 1997) and in one replication study (Beardslee et al., 2007) a significant better understanding of the parental disorder than children of the lecture condition. One replication study (Beardslee et al., 2003) showed that children benefit from *both* conditions, reflected in a significant better understanding of the parental disorder.

The study has several strengths: first, children and parents were assessed individually by assessors and outcomes about child's depression and general psychopathology were also rated by parents and children (Beardslee et al., 1997, 2003, 2007). A further strength is the relatively large sample size of replication studies (n = 93 (Beardslee et al., 2003); n = 105 (Beardslee et al., 2007)) with low dropout rates at two and a half (12.5% (Beardslee et al., 2003)) and four and a half years post treatment (14% (Beardslee et al., 2007)). However, in the first conducted study (Beardslee et al., 1997), five participants dropped out prior initial assessment completion, and results were based on a rather small sample size (n = 37).

In contrast to the PT (Sanford et al., 2003), the FTI was evaluated twice in Finland (Solantaus et al., 2010; Punamäki et al., 2013) and once in Germany (Christiansen et al., 2015). The main difference to the original study design is that Solantaus et al. (2010) measured offspring's outcomes solely by parental view. Moreover, researchers tested the program under real-world conditions, and confirmed promising results. Punamäki et al. (2013) set the focus slightly different, so that researchers investigated other outcome measures (e.g. child's attributional styles, child's emotional symptoms) and assessed outcomes by mothers' as well as child's view. Further, researchers used a modified active control condition ("Let's talk about the children" (LTC)). Slight deviations from the original version (Beardslee et al., 1997) are also found for session length (EG: 30-45 min; CG:15-45 min) and session number (EG: 6, CG: 1, in ¼ of cases 2). However, Punamäki et al. (2013) did not confirm

positive findings in EG, but surprisingly in CG. Christiansen et al. (2015) recently translated and adapted the FTI program into German language and compared three groups with each other (FTI vs. waiting list (WL) vs. healthy control children). Researchers reported higher psychopathological symptoms in children with depressed vs. non-depressed parents. With regard to child's knowledge level of depression, significant effects were found favouring the intervention condition. Child's view was solely considered for the variable knowledge of depression. Other psychopathological outcomes of children were assessed by parents only.

In conclusion, the FTI program conveys one important element that is known to reduce the psychopathological risk in children: psychoeducation. However, no other techniques are used in combination, which could reduce offspring's psychopathological risk (as positive parenting that was used by Sanford et al. (2003)). With regard to assessment outcomes, the conducted studies by Beardslee et al. (1997, 2003, 2007) and the replication study by Punamäki et al. (2013) used parent and child's perspective to assess child's psychopathology. Other presented replications (Solantaus et al., 2010; Christiansen et al., 2015) are limited, because they used solely parental view to assess offspring's psychopathology. One limitation of the FTI program of Beardslee and colleagues (1997, 2003, 2007) is, that true prevention effects are more difficult to demonstrate by using an active control.

Coping with Depression.

In contrast to the first two presented interventions, Clarke et al. (2001) developed a preventive intervention with the *focus solely on adolescents of depressed parents* aged between 13 and 18 years. In three separate meetings (baseline, middle, and end of intervention), parents are informed about program contents that are conveyed to adolescents, but no active participation of parents is intended. The program Coping with Depression (CWD) is a modified version of a treatment concept for depressive disorders with the two main pillars cognitive-behavioural strategies and psychoeducation. The program is manual-based and consists of 15 sessions,

using basics of the CBT. The aim is to reduce offspring's psychopathological risk by teaching adolescents in problem solving, behaviour activation and techniques of cognitive restructuring, as e.g. how to identify and change irrational or negative thoughts.

The strength of the program is that adolescents are taught in a combination of methods, so that both, psychoeducation as well as CBT techniques are delivered to participants. The weakness of the program is that it focuses on *adolescents only*, so that neither younger children nor parents are included in sessions, although they would also profit from program contents.

With regard to research evidence, Clarke et al. (2001) used *usual care as control condition* in contrast to Sanford et al. (2003) and Beardslee et al. (1997, 2003, 2007). Further, Clarke et al. (2001) focused on *offspring with subsyndromal depressive symptoms* – in contrast to Beardslee et al. (1997, 2003, 2007), who took offspring into account regardless of depression severity. From three groups (low, middle, high depression level), that were built by researchers, only participants from the low level group were randomised to EG or CG. Follow-ups were conducted directly after participants passed the program as well as 12, 18, and 24 months after treatment.

Findings demonstrate clinically significant prevention effects, reflected in reduced incidence rates for EG (9.3%) compared to CG (28.8%) at twelve-month post baseline. While control participants had 44 depressed days at the twelve months follow-up, EG participants had eleven days only, indicating that a relatively short preventive program can already reduce the developmental risk in offspring. Although diminished, significant effects sustained even at later follow-up measurements (18 and 24 months post baseline). Results emphasise that already a brief group-based preventive intervention can reduce offspring's psychopathological risk.

The study has several strengths: first, a randomised controlled trial was used which corresponds to the Gold Standard in research. Further, child's depression and other

psychopathology were assessed by parents and child's view. The study is also based on a relatively large sample size (n = 94). However, also some weaknesses have to be considered. First, from a large pool only participants with a low depression level were chosen, although researchers also had eligible participants with middle and high depression severity. This makes a generalisation of findings for participants with other depression severity level difficult. Second, also the power of the study would be stronger when all recruited participants would be included in the study. The last weakness is that no dropout rates were reported.

Like the FTI from Beardslee et al. (1997, 2003, 2007), replications of the CWD with altered versions have also been conducted (Beardslee et al., 2013; Garber et al., 2009, 2016). All replication studies used the same target group (13 – 17 years) with subliminal depressive symptoms, but also adolescents with a history of depression were included. Instead of 15, 14 sessions have been conducted. The study design with comparisons of intervention condition vs. usual care condition remained the same for all three studies. Garber et al. (2009) used follow-up assessments at three and nine months post intervention. Results demonstrated a significant reduction of depressive symptoms favouring the intervention condition (EG: 21.4% vs. CG: 32.7%). Also Beardslee and colleagues (2013) reported significant results favouring the intervention condition vs. usual care (36.8% vs. 47.7%) for a 33-month follow-up. Mainly the parental current status of depression influenced child's outcome with significant group differences when parents did not currently suffer from depression during intake, otherwise no group differences could be demonstrated. Garber et al. (2016) replicated the study once more, but this time considered in the study design participants of all three severity groups (low, middle, high). However, only for the low severity group benefits favouring the intervention program could be demonstrated.

The strengths of replication studies are that all were conducted as randomised controlled trials. Further, outcomes were based on parents and child's assessments. Moreover,

researchers investigated long-term effects from nine until 33 months follow-ups, and Garber et al. (2016) even examined differences of participants with different depression severity. All three studies had the same large sample size (n = 316). Dropout rates were relatively low (9.5% (Garber et al., 2009), 15.2% (Beardslee et al., 2013), 15.6% (Garber et al., 2016)) for all three studies. Further, all three studies were carried out at four sites.

In conclusion, the CWD program used a combination of two important elements: psychoeducation with cognitive techniques based on CBT. Further, it is a randomised controlled trial that used both, parents and child's assessments. Moreover, replications of the original study design are based on a large sample size with high power. The CWD program has the weakness that it only targets adolescent persons, so that parents are not involved in the sessions. The program is not conceptualized as family group program. One other weakness which is also valid for all studies except for the recent one of Garber et al. (2016) is that only participants with low depression severity were analysed, so that generalisabilty for other severity levels is difficult. Only Garber et al. (2016) investigated depression outcomes with regard to low, middle and high severity groups.

Raising Healthy Children.

The Raising Healthy Children (RHC) program (Compas et al., 2009) is a manualised family group-therapy program. It combines all components (psychoeducation, cognitive techniques based on CBT modules as well as parenting training) of previous presented interventions. The prevention program *targets the entire family,* so that children aged between nine and 15 years, their depressed parents and partners are involved. Also here, the purpose of the eight weekly and four monthly booster sessions is to reduce offspring's developmental risk of depression and general psychopathology. The first three sessions that focus on psychoeducation as well as the last session that is a repetition of program contents and outlook on future challenges are hold with parents and children together. Session four until eleven start and end with the whole group, but in between sessions are conducted separately for parents and children. Parents are

mainly taught in positive parenting and children in the use of appropriate coping strategies. Through homework and boosters sessions, program contents are practically trained and implemented in family's daily life. Particularly the booster sessions prepare families how to deal with stressful situations (Compas et al., 2009, 2011, 2015).

The strength of the program is that children, depressed parents and partners are included in the program. Further, different methods are combined to deepen acquired knowledge and to apply strategies in family's daily life. One particular advantage is that all eligible children and adolescents per family are invited to participate, so that more than two family members can share experiences.

When looking at research evidence, Compas et al. (2009, 2011) followed the procedure of Beardslee et al. (2003, 2007) and used an *active control group* with written information for self-study. The subject matter consists of same psychoeducative elements as the intervention program, and is adapted for each target group (children, adolescents and parents). Written information was sent to three time points by mail during the weekly sessions of the experimental group, containing a timetable until when materials should be studied from CG participants. Follow-up measures were conducted at two, four and twelve months post baseline (Compas et al., 2009). Results from twelve-month follow-up demonstrated a lower depression rate for EG compared to CG participants (8.9% vs. 20.8%) (Compas et al., 2009). Furthermore, long-term evaluations at 18 and 24 months demonstrated positive results in some, but not all outcome variables. Strongest effects were visible in questionnaires that include the whole period of 24 months, while lower effects were found for diagnostic instruments that cover a short or intermediate period of time[2]. Results from 24 months post baseline demonstrated that offspring from CG developed twofold more often a major depression compared to children from EG (32.7% vs. 14.3%) (Compas et al., 2011).

[2] For more detailed information about questionnaires utilised in the study see Compas et al. (2011).

The study has several strengths: first, it is a randomised controlled study. Further, children and parents were assessed individually with a semi-structured interview by assessors at the beginning and at the end of the study period. Outcomes of child's depression and general psychopathology were also rated by parents and children (Compas et al., 2009, 2011). A further strength is the relatively large sample size (n = 111) with a low dropout rate at twelve (14.4% (Compas et al., 2009)) and 24 (8% (Compas et al., 2011)) months. Although at the 24-month follow-up, 92% of participants remained involved, only 78% completed data collection. One point that could be seen as weakness of the study is that children and adolescents were also seen as eligible, when they had a history of depression. This could impact later outcomes. It is known that depression is recurrent and follows chronic trends (Ihle et al., 2004), which could implicate that more children develop depression, than this would be the case if mentally healthy children passed the program. True prevention effects might be easier found with children who never had a clinical diagnosis or symptoms of depression in the past.

Replications of the study were conducted by Forehand et al. (2012) and McKee et al. (2014). Forehand and colleagues (2012) set the focus slightly different and investigated the role of parental depression, its' mediating role on parenting and its' impact on childs' and adolescents' behaviour. Positive and negative parent-child situations have been videotaped at baseline and six months post baseline. Changes of parenting were then rated by researchers, who could demonstrate the mediating role of parental depression for a negative (but not positive) parenting style. Moreover, results showed that depression scores of affected parents from EG decreased after the eight weekly sessions and remained stable until six months. Results also demonstrated that a prevention program like the RHC program has positive effects on child's behaviour, even at 18 months after enrolment.

McKee et al. (2014) examined the link between parental guilt induction, offspring's cognitive style and thereby resulting internalising symptoms. Offspring's reports showed a

diminution of parental guilt induction at six months ($\beta = 0.15$, $p < .05$) and positive modulations of cognitive style at one year ($\beta = 0.25$, $p < .01$) post baseline. Offspring's internalising symptoms were even reduced 18 months post baseline ($\beta = 0.48$, $p < .001$).

In conclusion, the RHC prevention program confirms the high necessity of involving the entire family, instead of focusing on parents or children alone. And further, it shows that the combination of different approaches strengthens acquired knowledge, visible in parent and child outcomes. The study design corresponds the Gold Standard and assessment outcomes are based on trained interviewers, parents' and child's perspective. One limitation is that replications of the study (Forehand et al., 2012; McKee et al., 2014) were only conducted within the research group. Another point which could be seen as weakness is that children with a history of depression were also included in the study, which might disguise true prevention effects.

Summary of the Existing Research

In summary, there exist few interventions with the purpose to prevent depression in offspring of depressed parents. Although programs differ in their approach and also in respect of the target group they address, all showed promising results or at least trends favouring the prevention program they used. If we do not start to change our system and start to focus on prevention rather than intervening only when an outbreak occurs, depression in childhood and adolescence will remain a major issue with long-term consequences.

The consequences of such an illness do not only concern the affected person, but rather the entire family, the health care system, the society, social services, the individual career and also economic aspects (Warnke & Grimm, 2006). Existing meta-analyses and reviews that focused on depression prevention in general suggest, that the incidence of depression can be reduced and that depression is preventable (Jane-Llopis, 2003; Muñoz, Cuijpers, Smit, Barrera, & Leykin, 2010), but particularly that youth depression is preventable (Hetrick, Cox, Witt, Bir, & Merry, 2016; Mendelson & Tandon, 2017; Stockings et al., 2016).

Effects of interventions are lower when parents currently suffer from depression (Beardslee et al., 2003, 2007). Further, the severity level of the disorder influences the intervention success, reflected in higher dropout rates for parents with higher depression severity (Sanford et al., 2003). The current status of parental depression is, therefore, a relevant predictor of offspring's outcomes. Due to this knowledge, an involvement of parents is an indispensable step for positive intervention effects (Compas et al., 2009; Forehand et al., 2012; Horowitz & Garber, 2006).

Promising results were found with the RHC program by Compas and colleagues (2009, 2011) with the main advantage that it combines all techniques of previous presented programs (Beardslee et al., 1997, 2003, 2007, 2013; Clarke et al., 2001; Garber et al., 2009; Punamäki et al., 2013; Sanford et al., 2003; Solantaus et al., 2010). Moreover, it addresses both, parents and offspring. Because of the good concept of the RHC program, reflected in the approach, the contents (combination of psychoeducation and cognitive-behavioural modules) and the combination of parent, offspring and family sessions, the current work evaluates the RHC program from Compas et al. (2009, 2011). However, instead of an active control group, the current study used a waiting-list (WL) as control condition (following Sanford et al., 2003) to show true prevention effects. By evaluating the Raising Healthy Children program and comparing it with waiting list (WL), I expect to demonstrate stronger effects than Compas and colleagues (2009, 2011) could show with the written information control condition. An active control, as it was utilised by Compas et al. (2009, 2011, 2015) and several other researchers (Beardslee et al., 1997, 2003, 2007; Forehand et al., 2012; McKee et al., 2014; Punamäki et al., 2013; Solantaus et al., 2010) can limit a study, because treatment effects cannot clearly be ascribed to intervention performance. The RHC program has not yet been examined outside the research group (Compas et al., 2009, 2011, 2015; Forehand et al., 2012; McKee et al., 2014).

The Current Study

The first step in the current work is to investigate the factors which predict depressive symptoms and general psychopathology in the children of depressed parents. This includes the severity of parental depression, the number of depressive episodes, the number of stressful/negative life events that were experienced within the last year, and socio-economic status.

In a randomised controlled trial (RTC), a second step will be to investigate how effective a preventive intervention is in reducing offspring's depressive and general psychopathological symptoms in short- and medium-term, and how effective the program is in prevention depression onset in offspring in long-run. I will also investigate whether the intervention program is effective in enhancing offspring's knowledge of the disorder and in modulating parental parenting in a positive manner in short- and medium-term.

Hypotheses.

Step 1: Predictors of depressive symptoms and general psychopathology in children of depressed parents. The following hypotheses are based on a sample of non-depressed children who have a parent with depression and who were randomised to receive an intervention or waiting-list control (see Step 2). Analyses in Step 1 are conducted independent of group allocation.

Hypothesis 1: I hypothesise that the **severity of depression and the number of depressive episodes of the primary affected parent** will positively predict the child's depressive and general psychopathological symptoms.

Hypothesis 2: I hypothesise that children and adolescents who experienced a larger number (≥ five) of **stressful life events** during the last twelve months will have higher depressive and general psychopathological symptoms compared to children and adolescents without or only a few (one/two) stressful experiences. Self- and parent reports of child stressful life events were collected. It is known that the perspective of children and

adolescents' own symptoms can differ from their parent's view, and that children and adolescents stronger assess their own (specifically internalising) symptoms compared to parents (Cantwell, Lewinsohn, Rohde, & Seeley, 1997; Ihle et al., 2004). Therefore, I expect a higher coherence between offspring's report of negative events and child outcomes than between parent's reports of negative events and child outcomes.

Hypothesis 3: I expect **socio-economic status** to be associated with children's depressive symptoms and general psychopathology. Specifically, I expect that offspring's depressive and general psychopathological symptoms are more pronounced in families with low and high socio-economic background compared to families from middle-class. The stronger manifestation of participants with low and high SES will be reflected in higher scores of scales measuring depressive and general psychopathological symptoms. The SES is built following the Winkler Index (Lampert, Kroll, Müters, & Stolzenberg, 2013) which includes parental education, profession and familiar monthly net income.

Step 2: Intervention outcomes. The following hypotheses examine the effectiveness of a prevention intervention for children of depressed parents with regard to three variables of interest (offspring's mental health, offspring's knowledge of depression, offspring's perception of parenting style). Outcomes are assessed in participants who received the program (EG) vs. a waiting-list control group (CG).

Hypothesis 4: I predict that children and adolescents who received the selective preventive intervention will reach **lower scores in scales measuring depressive symptoms** across the assessment points at six and nine months than offspring of the waiting-list control condition.

Hypothesis 5: I expect the prevention program to be effective in preventing depression. Therefore, I also hypothesise that children and adolescents who received the selective preventive intervention will be **less likely to have depression at 15-month follow-up** compared to offspring from the waiting-list control condition.

Hypothesis 6: With regard to general psychopathology, I hypothesise that offspring who took part in the prevention program will have **fewer general psychopathological symptoms** at six and nine months follow-up compared to offspring of the waiting-list control condition. Like in *hypothesis 2*, I will analyse children's (Youth self-report (Döpfner et al., 1998)), and parents' (Child behaviour checklist (Döpfner, Schmeck, & Berner, 1994)) reports about offspring's internalising, externalising and mixed symptoms separately. Also here, I expect to find a higher coherence between offspring's reports about the own general psychopathological symptoms and positive effects of the preventive intervention than between parents reports about offspring's general psychopathological symptoms and positive effects of the intervention program.

Hypothesis 7: Since the program conveys psychoeducational elements about the symptoms and causes of depression, I assume that offspring of the EG will acquire **more knowledge of depression** across the assessment points at six and nine months than offspring of CG.

Hypothesis 8: Since the program focuses on positive parenting in the parent sessions (including praising their children, spending quality time (15 min per day) with each child, and having a family activity together each week), I expect that offspring of the EG will report **more positive parenting** at six and nine months follow-up compared to offspring of the waiting-list condition. Specifically in the scale 'praise' of the parenting style questionnaire (Krohne & Pulasack, 1991, 1995), I expect to find significantly higher scores in offspring from EG, whereas scores of CG I assume to be stable across all measurement points. The primary affected parent as well as the partner (in case he or she participated as well) will be analysed.

For all hypotheses of *Step 2* (except *hypothesis 5*) I expect to find best outcomes directly after intervention (T2: six months post baseline) with slight decreases at the nine months follow-up. Additionally to the presented hypotheses, the current work will describe

outcomes of individual feedback evaluation forms that were filled out from participants of the intervention condition directly after each session. These data are reported at the end of the results section.

Methods

Design

The design of the present study is a randomised controlled trial (RCT) of a family and group-based cognitive-behavioural program for prevention of depression in offspring of depressed parents. The assessment is based on families (with at least one affected parent and one healthy child) who passed the twelve session prevention program (experimental group) vs. families (with at least one affected parent and one healthy child) on the waiting list for the program who received no support in form of an intervention during the same time frame (control group). Depending on diagnostic instruments that were utilised, analyses were conducted with offspring, affected parents and/or partners.

The current work reports outcomes of families at baseline (n = 77), who provided at least some data at T2 (six months from baseline; n = 42 (54.5%)) and T3 (nine months from baseline; n = 38 (49.4%)). Out of n = 49 families, who have reached the T4 (15 months from baseline) follow-up point, n = 40 (81.6%) participated in the diagnostic interviews, where incidence of depression is assessed.

Inclusion criteria for families were met, when a) the participating parent fulfilled the criteria either for a current or a history of depression or a double depression during child's lifetime according to the Diagnostic Statistical Manual for Mental Disorders (DSM-IV-TR), and when b) the participating child/adolescent was aged between eight and 17 years, had an IQ of 85 or higher, and did not meet a psychiatrical diagnosis at the time point of participation. Children and adolescents with sub-clinical symptoms were included in the study. Eligible siblings were also included. They were allocated to the same group as the other family

members, so that members of the same family passed the program together. If both parents fulfilled the criteria for a depressive episode, both were invited to take part. But not only the depressed parent, also the partner (spouse, cohabitee or other living partner in the house with responsibilities of parenting) was asked to participate, if exclusion criteria were not met.

Families were excluded if a) the parent(s) showed symptoms of a psychosis, bipolar disorder, personality disorders, alcohol or substance abuse, was currently in crisis or suicidal or had symptoms of a disorder that hampered the ability of participation, and/or if b) offspring fulfilled the diagnostic criteria for any psychiatric disorder or took part in a therapy program for depression treatment, and/or if c) the family went to a family therapy program during the running time of the study. Children were excluded if they were in crisis or suicidal or showed symptoms of another disorder which hampered/interfered with the ability to take part in the program, such as a diagnosis of attention deficit hyperactivity disorder (ADHD). Children with ADHD might show disruptive behaviour during the two-hour sessions and disturb the entire group.

The design is in line with the SPIRIT 2013 Statement (Standard Protocol Items: Recommendations for Interventional Trials) (Chan, Tetzlaff, Altmann, & et al, 2013) and the study protocol is reported in BMC Psychiatry 2014, 14:263 (doi:10.1186/s12888-014-0263-2; Clinical Trials NCT02115880) (Platt, Pietsch, Krick, Oort, & Schulte-Körne, 2014). The study and the comprised procedure were approved by the Medical Devision Ethics Committee from the Ludwig-Maximilians-University (LMU), Munich Study ID 3 – 14.

Participants

Out of 237 interested families, 160 were either undecided, no longer interested or did not fulfil inclusion criteria. 77 were eligible, randomised blockwise and included in the current work.

Demographic information.

Table 4 lists demographic data from the 77 families who were randomised in the study and completed the assessment battery at baseline (T1). Families were randomly allocated to intervention (n = 37, 48.1%) or control (n = 40, 51.9%) condition (see section *randomisation* for detailed description of the randomisation procedure). There were no significant differences between the two groups in any of the demographic variables.

Table 4. The Table illustrates sample characteristics at baseline of the primary affected parent, the partner, the child and the family for the experimental (EG) and control group (CG) as well as for the total sample (total). The socio-economic status (SES (low/middle/high)) of families is built following the Winkler Index (Lampert et al., 2013) that considers the education level and current profession of the primary affected parent as well as the familiar net income. Reported is the valid sample size (N) of each variable (because not all families completed questionnaires), mean, standard deviation (SD), minimum-maximum (min-max), T-values or Chi-Square (χ^2), df and significance level in case of significance. T-tests and Chi-Square tests were used to check whether groups (experimental vs. control) were equal at baseline.

Demographic information	**EG**				**CG**				**Total**				**T-value/ χ^2**
Primary affected parent	N	Mean (SD)	Min-Max	%	N	Mean (SD)	Min-Max	%	N	Mean (SD)	Min-Max	%	
Age	35	45.54 (6.06)	34-56	male 43.2 %	32	47.78 (6.50)	36-60	male 35.0%	67	46.61 (6.33)	34-60	male 39.0%	age n.s. gender n.s.
Employment/ unemployment/ retirement/ others	32			75.0%/ 3.1%/ 9.4%/ 12.5%	28			82.1%/ 3.6%/ 10.7%/ 3.6%	60			78.3%/ 3.3% 10.0%/ 8.4%	n.s.
Full-time / part-time work	20/9			69.0%/ 31.0%	20/7			74.1%/ 25.9%	56			71.4%/ 28.6%	n.s.

Marital status (y/n)	26/7			78.8%/ 21.2%	21/8			72.4%/ 27.6%	62			75.8%/ 24.2%	n.s.
Nationality: German (y/n)	31/3			91.2%/8.8%	32/4			88.9%/ 11.1%	70			90.0%/ 10.0%	n.s.
Partner													
Age	23	45.43 (3.96)	38-54		17	47.12 (7.58)	35-63		40	46.15 (5.76)	35-63		age n.s.[3]
Child													
Age	37	11.89 (2.81)		male 45.9%	40	11.98 (3.02)		(male 50.0%)	77	11.94 (2.90)	8-17	(male 48.1%)	age n.s. gender n.s.
Siblings (y/n)	27/5			84.4%/15.6%	26/9			75.0%/ 25.0%	67		0-2	77.9%/22.1%	n.s.
IQ	37	103.11 (14.70)	85-141		40	109.28 (13.60)	85-133		77	106.31 (14.38)	85-141		n.s.
Family													
SES: low/middle/ high	0/6/ 22			0%/ 21.4%/ 78.6%	2/6/ 17			8.0%/ 24.0%/ 68.0%	53			3.8%/ 22.6%/ 73.6%	n.s.

[3] Data of gender of the partner were not collected.

Parent education, income and occupation.

Table 5 describes the highest level of education achieved by primary affected parents. 20.4% of parents finished their education after high school (8.5% attended "Hauptschule" and 11.9% "Realschule"). 27.1% completed the examations necessary for university entry. 42.4% obtained a university degree and a further 10.2% obtained a post-doctoral degree. 13.2 % of families reported a familiar monthly net income ≤ €2000 and 24.5% reported a monthly net income of more than €5000 (Table 5). 68.3% reported to be employees, 10.0% to be self-employed, and 3.3% to be unemployed. 10.0% were retired, 5.0% housewives, 1.7% in paternity leave and 1.7% did not specify their occupation. Out of 56 of the primary affected parents, 28.6 % are working part-time and 71.4% % full-time.

Table 5. The Table shows the valid percentage of parental education level (secondary schools (Haupt-Realschule), matriculation standard (Hochschulreife), university degree and doctor's degree). The second part illustrates the monthly net income (in Euro) of families.

Parental education level	Frequency	%
Secondary school (Hauptschule)	5	8.5
Secondary school (Mittlere Reife)	7	11.9
German matriculation standard (Hochschulreife)	16	27.1
University degree	25	42.4
Postgraduate level (doctorate)	6	10.2
Total N =	59	100.0
Familiar monthly net income (€)		
1000-2000	7	13.2
2000-3000	12	22.6
3000-4000	9	17.0
4000-5000	12	22.6
> 5000	13	24.5
Total N =	53	100.0

Offspring education.

35.8% of went to Grundschule, 4.5% to Hauptschule, 16.4% to Realschule, 41.8% to Gymnasium and 1.5% to other schools. 1.5% visited the 1st grade, most children were older and in third grade (13.8%), followed by 7th (12.3%), 9th (12.3%) and 6th (10.8%) grade. 1.5% of children went to 12th grade and 47.8% reported other grades. Around three-quarters of children (75.8%) reported to like going to school and one quarter (24.2%) negated the question. 64.1% of offspring reported to have one to five good friends.

Clinical characteristics.

Table 6 illustrates average values of clinical characteristics of the primary affected parent, the partner, or both parents, and the child separately for experimental and control condition as well as for the total sample.

Primary affected parent.

With regard to the primary affected parent it gets visible that from the total sample 67.5% were currently depressed at enrolment and 32.5% were currently remitted. Out of all cases, independent of current or past depression, 12.1% had a single episode. 84.4% were diagnosed at baseline (T1) with a recurrent depressive episode (DSM-IV: 296.3x) of whom 13.0% had a mild, 29.9% a moderate, and 10.4% a severe recurrent depressive episode. 1.3% was partially remitted, 29.9% were fully remitted and 15.5% had other depressive episodes. The average score of the Beck Depression-Inventory (BDI-II) (Hautzinger, Keller, & Kühner, 2009) that assesses the severity of depression was 17.60 (SD: 10.98) and ranged from zero to 53. The Structured Clinical Interview for Axis II DSM disorders (SCID-II) (Wittchen, Zaudig, & Fydrich, 1997) gives a first statement about conspicuousness with regard to personality disorder. Here, 19.4% of primary affected patients were assessed as sub-clinical conspicuous. The average number of psychiatric disorders within the family was 2.93 (SD: 1.64) and ranged from zero to ten. 46.8% of affected parents had a comorbid diagnose. Of those, 47.5%

fulfilled criteria for an anxiety disorder, 11.2% for a post-traumatic stress disorder, 8.4% for an obsessive compulsive disorder and 8.4% for an eating disorder. 24.5% reported to have other comorbidities.

Psychotherapeutic experience was reported by 91.4% of primary affected parents. Only 2% were in treatment less than four weeks, 8% less than three months, 16% less than one year and 50% reported treatment durations over two years. 76.8% made psychiatric experiences with 4.9% less than four weeks, 14.6% less than three months, 14.6 % less than one year and 46.3% reported treatment durations over two years. 69.6% received inpatient treatment, whereas 30.4% reported that they have never been in inpatient treatment. 63.9% had only one and 27.8% two visits. 8.3% reported having three or more clinic stays (Mean = 1.50; SD = 0.85). Regarding treatment satisfaction, 76.0% reported to benefit from psychotherapy, 78.6% from psychiatric therapy and 84.2% from inpatient treatment. 85.7% of patients reported of medical treatment and 14.3% negated the question. Regarding support by others, 48.3% seeked friends' help, 20.7% used consulting services, and 31.0% reported to make use of other support.

Partner.

In 10.4% of cases both parents fulfilled criteria of a major depression, and in 4.5% of valid cases the partner was currently depressed. The average BDI-II score of the partner was 5.33 (SD: 5.59) and ranged from zero to 22. In 4.2% the partner was assessed in the SCID-II as sub-clinical conspicuous.

Child.

Table 6 illustrates child's depressive and general psychopathological symptoms at enrolment. Due to the fact that children were only seen as eligible for participation when no psychiatric disorder was diagnosed, scores of depressive measurements (Beck Depression-Inventory (BDI-II) (Hautzinger et al., 2009), Depressions-Inventar für Kinder und Jugendliche (DIKJ)

(Stiensmeier-Pelster, Schürmann, & Duda, 2000)) and general psychopathological measurements (Youth Self-Report (YSR) (Döpfner et al., 1998), Child Behaviour Checklist (CBCL) (Döpfner et al., 1994)) were at T1 on average within normal limits. For YSR and CBCL, the three scales that measure internalising, externalising and mixed psychopathological symptoms are listed separately (for more detailed information about questionnaires see section measures).

At enrolment, 65.3% of the interviewed parents perceived offspring's general mental health as inconspicuous. In 28.0% of cases parents stated to register mild conspicuouities and in 6.7% of cases parents were concerned about offspring's mental health. Based on diagnostic interviews conducted by trained study members, 85.5% of children showed no signs of any psychiatric disorder (n = 65) and 14.5% showed sub-clinical symptoms of depression (n = 11).

Child's general outcomes.

Child's general outcomes which were measured at baseline are displayed in Table 7. This includes average scores of offspring's knowledge of depression (that was assessed with the German questionnaire about knowledge of Depression (Allgaier et al., 2011)), offspring's perception of parenting style from the primary affected parent and the partner (Erziehungsstil Inventar (ESI) (Krohne & Pulasack, 1991, 1995)) which was measured by six dimensions (support, restriction, praise, blame, inconsistency, punishment) as well as the number of stressful life events, which was assessed with the Child and Adolescent Survey of Experiences (CASE-C/P) (Allen, Rapee, & Sandberg, 2012).

Table 6. The Table illustrates clinical sample characteristics at baseline of the primary affected parent (current depression, number of depressive episodes, recurrent depressive episodes, BDI-II score of the Beck Depression-Inventory (BDI-II) (Hautzinger et al., 2009), Structured Clinical Interview for Axis II DSM disorders (SCID-II) (Wittchen et al., 1997), number of psychiatric disorders in family, comorbidity, in treatment (psychotherapy, psychiatry), clinic stay, number (No.) of clinic stays, medication, other support), the other caregiver (partner (current depression, number of depressive episodes, BDI-II score, SCID-II)), both parents (both depressed) and the child (BDI-II, Depressions-Inventar für Kinder und Jugendliche (DIKJ) (Stiensmeier-Pelster et al., 2000), Diagnostic Interview of Psychiatric Disorders for children and adolescents (K-DIPS) (Unnewehr, Schneider, & Margraf, 2008), Youth Self-Report (YSR (internalising, externalising, mixed)) (Döpfner et al., 1998), Child Behaviour Checklist (CBCL (internalising, externalising, mixed)) (Döpfner et al., 1994)). All variables are listed separate for experimental (EG) and control (CG) condition as well as for the total sample. Reported is the valid sample size (N) for each variable (because not all families completed questionnaires), mean, standard deviation (SD), minimum-maximum (min-max), T-values or Chi-Square (χ^2), df and significance level, in case of significance. T-tests and Chi-Square tests were used to check whether groups were equal at baseline.

Clinical characteristics	**EG**				**CG**				**Total**				**T-value / χ^2**	**Df**	**p-value**
Primary affected parent	N	Mean (SD)	Min-Max	%	N	Mean (SD)	Min-Max	%	N	Mean (SD)	Min-Max	%			
Current depression (y/n)	24/13			64.9%/ 35.1%	28/12			70.0%/ 30.0%	77			67.5%/ 32.5%	n.s.		
Number of depressive episodes	29	6.10 (5.07)	1-20		29	5.86 (6.20)	1-20		58	5.98 (5.61)	1-20	12.1 % single episode	n.s.		
Recurrent depressive episode (296.3x) (y/n)	32/5			86.5%/13.5%	33/7			82.5%/ 17.5%	65/12			84.4%/15.6%	n.s.		
BDI-II score	35	17.31			37	17.86			72	17.60	0-53				

		(9.89)				(12.06)				(10.98)		
SCID-II sub-clinical conspicuous (y/n)	8/27			22.9%/77.1%	6/31	6/31		16.2%/ 83.8%	14/58			19.4%/ 80.6%	n.s.
No. of psych. disorders in family	27	3.11 (1.99)	0-10		28	2.75 (1.24)			55	2.93 (1.64)	0-10		n.s.
Comorbidity	17			45.9%	19			47.5%	36			46.8%	n.s.
In treatment Psychotherapy (y/n)	27/2			93.1%/6.9% treatment > 2 years: 55.6%	26/3			89.7%/ 10.3% treatment > 2 years: 43.5%	53/5			91.4 %/ 8.6%; treatment > 2 years: 50 %	n.s.
In psychiatric treatment (y/n)	24/5			82.8%/17.2% treatment > 2 years: 59.1%	19/8			70.4%/ 29.6% treatment > 2 years: 31.6%	43/13			76.8%/ 23.2%; treatment > 2 years: 46.3 %	n.s.
Clinic stay (y/n)	19/10			65.5%/34.5%	20/7			74.1%/ 25.9%	39/17			69.6%/30.4%	n.s.
Number of clinic stays	17	1.59 (0.62)	1-3	47.1% one	19	1.42 (1.02)	1-5	78.9% one	36	1.50 (0.85)	1-5	63.9 % one visit	n.s.

				visit				visit							
Medication (y/n)	32			86.5%	34			85.0%	66			85.7%	n.s.		
Other support	15			66.7 % friends, 13.3 % consulting, 20.0% others	14			28.6 % friends, 42.9 % consulting, 28.5% others	29			48.3 % friends, 20.7 % consulting, 31.0% others	8.88	3	0.031
Partner															
Current depression (y/n)	1/29			3.3%/ 96.7%	2/34			5.6%/ 94.4%	3/63			4.5%/95.5%	n.s.		
Number of depressive episodes	4	3.0 (1.41)	2-5		2	2.00 (1.41)	1-3		6	2.67 (1.37)	1-5		n.s.		
BDI-II score	24	4.04 (4.78)	0-21		18	7.06 (6.24)			42	5.33 (5.59)	0-22		n.s.		
SCID-II sub-clinical conspicuous (y/n)	1/10			9.1%/ 90.9%	0/13	1.00 (0.00)		0/%/ 100%	1/23			4.2%/95.8%	n.s.		
Both parents															
Both parents depressed (y/n)	4/33			10.8%/ 89.2%	4/36			10.0%/ 90.0%	8/69			10.4%/ 89.6%	n.s.		

Child

BDI-II	11	6.45 (4.84)	2-18		15	6.73 (5.66)	0-16		26	6.62 (5.23)	0-18		n.s.	
DIKJ	27	46.37 (7.90)	36-69		26	47.65 (8.47)	33-69		53	47.00 (8.13)	33-69		n.s.	
K-DIPS (inconspicuous/ sub-clinical conspicuous)	32/5			86.5%/13.5%	33/6			84.6%/ 15.4%	65/11			85.5%/14.5%	n.s.	
YSR internalising	31	55.16 (9.79)	40-80		26	48.35 (9.95)	34-67		57	52.05 (10.36)	34-80		2.60	55	0.012
YSR externalising	31	51.58 (7.04)	37-66		26	50.00 (7.44)	40-71		57	50.86 (7.20)	37-71		n.s.	
YSR mixed	31	55.48 (8.22)	41-80		26	50.46 (8.67)	35-69		57	53.19 (8.72)	35-80		2.24	55	0.029
CBCL internalising	30	59.23 (10.44)	38-76		27	57.30 (8.48)	42-76		57	58.32 (9.53)	38-76		n.s.	
CBCL externalising	30	53.63 (7.44)	37-71		27	48.96 (7.12)	36-62		57	51.42 (7.60)	36-71		2.41	55	0.019
CBCL mixed	30	57.67 (7.42)	43-71		27	53.00 (7.44)	40-68		57	55.46 (7.73)	40-71		2.37	55	0.021

Table 7. The Table illustrates child's general outcome measures (German questionnaire about knowledge of Depression (Allgaier et al., 2011), Erziehungsstil Inventar (ESI) (Krohne & Pulasack, 1991, 1995) of affected parent (support (US), restriction (ES), praise (LS), blame (TS), inconsistency (IK), punishment (SI)), ESI of partner (support (US), restriction (ES), praise (LS), blame (TS), inconsistency (IK), punishment (SI)), Child and Adolescent Survey of Experiences (CASE-C/P) (Allen et al., 2012)). All variables are listed separate for experimental (EG) and control (CG) condition as well as for the total sample. Reported is the valid sample size (N) for each variable (because not all families completed questionnaires), mean, standard deviation (SD), minimum-maximum (min-max), T-values or Chi-Square (χ^2), df and significance level, in case of significance. T-tests and Chi-Square tests were used to check whether groups were equal at baseline.

Child's general outcomes	**EG**				**CG**				**Total**				**T-value / χ^2**	**Df**	**p-value**
	N	Mean (SD)	Min-Max	%	N	Mean (SD)	Min-Max	%	N	Mean (SD)	Min-Max	%			
Knowledge of Depression	30	33.77 (4.71)	22-40		27	32.41 (3.62)	24-38		57	33.12 (4.25)	22-40		n.s.		
ESI affected parent															
ESI support (US)	31	37.10 (6.66)	24-48		28	34.18 (7.02)	16-48		59	35.71 (6.93)	16-48		n.s.		
ESI restriction (ES)	31	16.42 (4.38)	12-30		28	16.14 (3.09)	13-27		59	16.29 (3.79)	12-30		n.s.		
ESI praise (LS)	31	36.84 (6.31)	22-44		28	35.46 (7.58)	15-44		59	36.19 (6.92)	15-44		n.s.		

ESI blame (TS)	31	25.55 (7.80)	12-42	28	23.71 (5.50)	15-38	59	24.68 (6.81)	12-42	n.s.
ESI inconsistency (IK)	31	18.39 (6.14)	12-40	28	18.39 (4.31)	12-26	59	18.39 (5.30)	12-40	n.s.
ESI punishment (SI)	31	9.48 (2.90)	5-15	28	9.32 (2.64)	5-15	59	9.41 (2.76)	5-15	n.s.
ESI partner										
ESI support (US)	30	36.63 (5.93)	27-48	25	36.12 (5.07)	26-44	55	36.40 (5.51)	26-48	n.s.
ESI restriction (ES)	30	16.77 (3.51)	12-27	25	17.44 (3.29)	12-27	55	17.07 (3.40)	12-27	n.s.
ESI praise (LS)	30	35.03 (6.46)	25-44	25	36.80 (5.51)	26-44	55	35.84 (6.06)	25-44	n.s.
ESI blame (TS)	30	24.80 (7.38)	13-42	25	26.84 (8.08)	16-45	55	25.73 (7.70)	13-45	n.s.
ESI inconsistency (IK)	30	17.93 (4.58)	12-27	25	19.24 (4.45)	13-33	55	18.53 (4.53)	12-33	n.s.
ESI punishment (SI)	30	9.30 (2.55)	5-15	25	10.60 (2.81)	5-17	55	9.89 (2.73)	5-17	n.s.
CASE negative events	28	3.68 (2.45)	0-9	26	3.27 (2.55)	0-10	54	3.48 (2.49)	0-10	n.s.

Procedure

Recruitment.

The recruitment of eligible families was conducted via multiple sources in urban settings in and around Munich. Several methods were used in order to ensure optimal recruitment strategies: Participants were contacted over affected siblings of the Department of Child and Adolescent Psychiatry, Psychosomatics and Psychotherapy of the University Clinic Munich. Also patients from Adult Psychiatries in and around Munich who fulfilled inclusion criteria were informed about the PRODO study either by team members or by responsible medical staff or therapists. Moreover, recruitment was extended to general practices, paediatricians, newspapers, employer's liability insurance coverages, occupational rehabilitation, helplines, information centres, pharmacies and the internet (homepage: www.prodo-studie.de), facebook, twitter, online newsletter. Children, adolescents and parents who have taken part in previous studies and who were interested to participate in other studies were also invited in case of eligibility.

Over a period of two and a half years 759 different institutions were contacted. 56 remained core cooperating partner institutions and were contacted and visited periodically. As illustrated in Figure 4, most of the included families have been recruited via clinics and therapists (35.7%), followed by public adverts (33.9%), paediatricians (7.1%), previously conducted studies in the clinic (5.4%), colleagues (5.4%), community and advice centres (3.6%), council data base (3.6%), information evenings (1.8%) and others (3.6%).

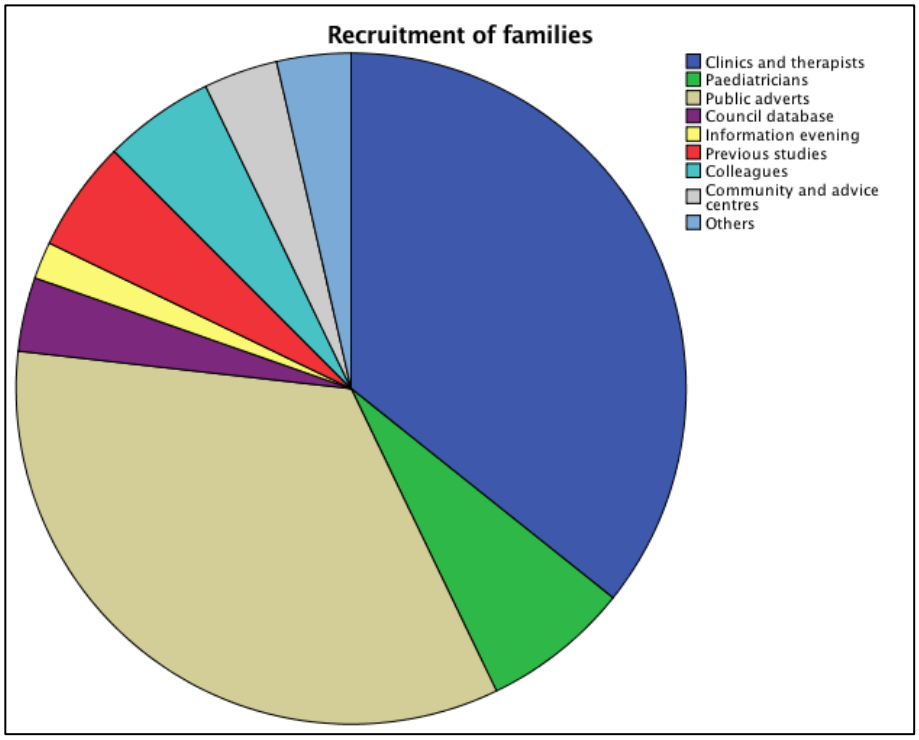

Figure 4. Illustration of cooperating institutions that support the recruitment of families.

Before families could take part in the study, the eligibility was assessed by phone (n = 237). In this first contact, interested families also received detailed information about the study. Families who were suitable and interested were invited for a personal appointment in which they received an overview of study procedure, including the topics of random allocation of families to either experimental or control condition, about audio-recording of the screening sessions (T1 and T4), and audio- or video-recording of the intervention sessions. The recording of screening sessions served on a random basis as quality control of diagnostic assessments. The recording of intervention sessions was used for a random assessment of the quality of the manual conduction to ensure that all team leaders implemented the manual similarly. Further, participants were informed that (independent of group allocation) self-report questionnaires have to be completed at four assessment points (T1 – T4), which are sent by post. For the last outcome measure, families have been informed that they will be invited back for a personal appointment 15 months after baseline (T4) in order to assess the final outcome by using the DIPS (Schneider & Margraf, 2011) as diagnostic instrument. Furthermore, families were elucidated about the voluntariness of participation and about the

opportunity to quit at any time. Written informed consent was taken from each participant. Children and adolescents within the age range as well as the affected parent were interviewed simultaneously with the clinical instrument “Diagnostisches Interview psychischer Störungen” (DIPS) (Schneider & Margraf, 2011). Therefore, one interviewer per participant was planned and interviews were conducted in separate rooms. If both parents reported to be affected from a depressive disorder (independent of a current or past episode), both were interviewed separately and received same questionnaires at assessment points. With the DIPS, potential families were screened for exclusion criteria, which made it possible to verify suitability and to assess the current mental health status of each participant. After the first appointment, interviewers discussed family’s suitability for taking part. In case inclusion criteria were not met or families were no longer interested, they were excluded from the study. In case families required professional help (because e.g. children already developed a depression or some other psychopathology or parents required a therapist), proper institutions were recommended such as counselling centres, psychiatries, therapists or other outpatient departments. The list of recommendations is illustrated in Appendix A.

In case of suitability and interest, families were included. They received questionnaires that had to be completed within four weeks, at least before starting the program, independent of group allocation. If only one parent attended the first screening session, nevertheless the other parent or current spouse was asked to fill in self-report questionnaires. These questionnaires enquired symptoms of depression and also symptoms of general psychopathology (see section *outcome measures*).

Further, families received two 25 € payments: at the beginning of the study period after filling out and sending back baseline questionnaires (T1) and at the end of the study period (T4: 15 months after baseline) under the same conditions. The aim was that parents deliver the money to offspring for motivating them to fill out questionnaires completely.

Randomisation.

After recruitment of eight to ten suitable families, a randomisation was performed by a statistician (Frans Oort, University of Amsterdam). The study leader (Dr. Belinda Platt) matched families into pairs based on offspring's age and current parental health status (currently or previously depressed) to ensure a balance between conditions. The statistician generated random numbers which decided whether the first or second family in each pair would be allocated to the experimental condition.

Figure 5 illustrates the screening and randomisation procedure and gives an overview of how many families from EG and CG reached each follow-up time point and how many provided at least some data to each assessment point. T1 until T3 are based on questionnaires, T4 is based on diagnostic interviews (Diagnostic Interview of Psychiatric Disorders for children and adolescents (Unnewehr et al., 2008)) conducted with children.

When looking at the EG, 37 families reached T2 and 20 (54.1%) provided at least some data. T3 was reached by 32 families and 16 (50.0%) provided some data. T4 was reached by 23 families and 19 (82.6%) provided data. When looking at families from the CG, 40 reached T2 and 22 (55.0%) provided some data. T3 was reached by 35 families and 22 (62.9%) provided some data. T4 was reached by 26 families and 21 (80.8%) provided data.

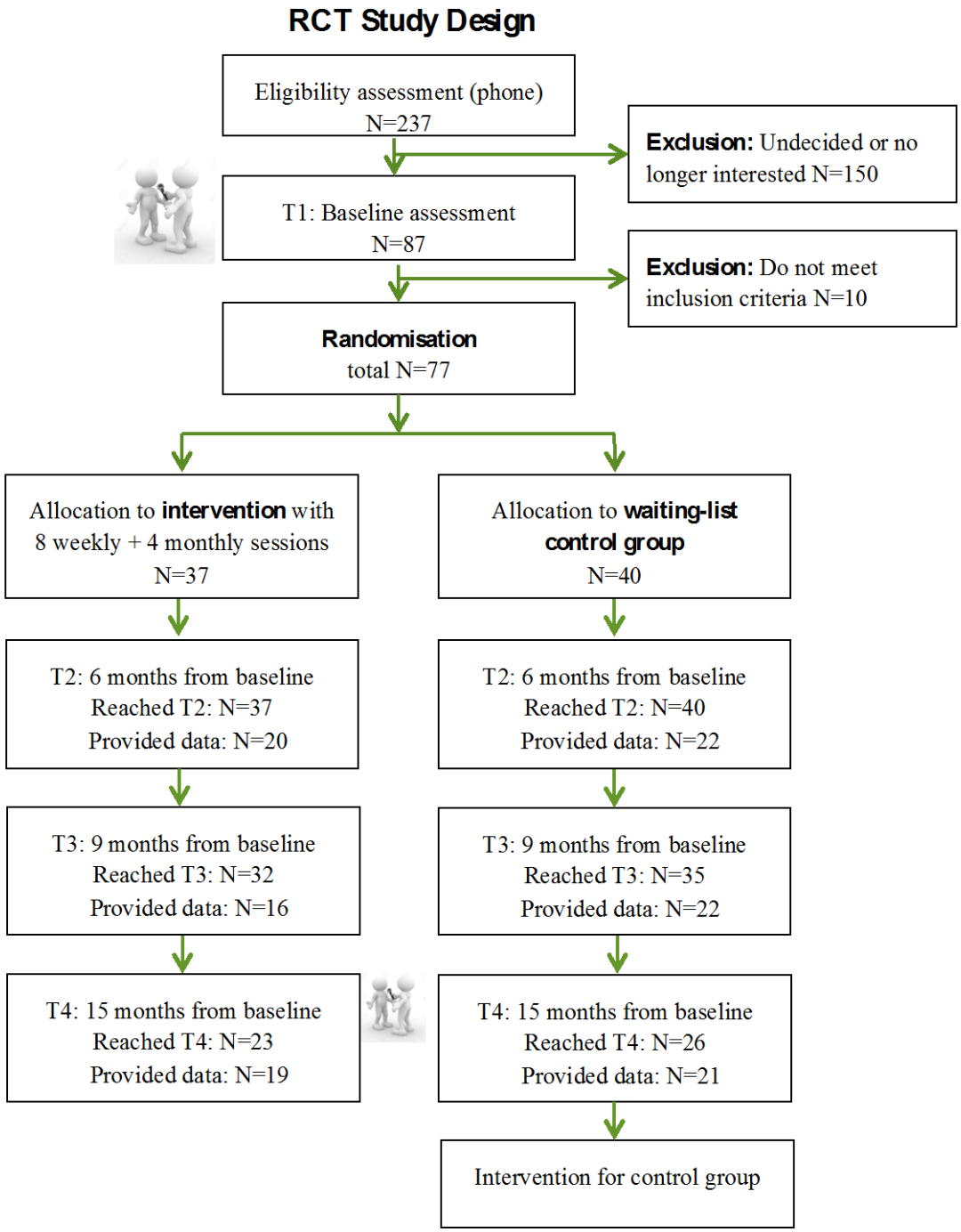

Figure 5. Illustration of the study design.

Intervention Program

The prevention program "GUG-Auf: Gesund und Glücklich Aufwachsen" is a modified version of the Raising Healthy Children (RHC) intervention, which was developed and evaluated by Compas et al. (2009, 2011). The program is manualised and based on cognitive-behavioural modules and includes affected parents, their mentally healthy offspring and the

entire family if partners were willing to participate and siblings fell into the same age range. The intervention is based on eight weekly and four monthly booster sessions, which took place in the conference rooms of the Child and Adolescent Psychiatry, Psychosomatic and Psychotherapy of the University Hospital Munich. Most sessions took place on weekdays in the late afternoon or during evening hours.

Cultural adaptation.

Materials from the original version (parental and offspring's workbooks, leaders manual, training sheets for homework and feedback evaluation forms) were translated into the German language and adapted to German culture. An example of the original manual for the strategy *acceptance of uncontrollable stressors* was conveyed to children and adolescents by the nerve-racking waiting process to get fast food. However, German children would wait hours to get fast food, therefore, this example was not useful to understand the definition of acceptance. Thus, a different example was implemented. Another example that was replaced was football. Instead, soccer was used as an example. Moreover, in the German version the training for at home was not called homework but training so that children and adolescents did not get the feeling of being in a school setting.

In order to create a good atmosphere right from the beginning, the first session for each group contained a video told from a perspective of a child with an affected mother. A German-language video was created because the original video from the RHC program was in English and with the youngest participating children being eight years old might have had problems in understanding and following the contents. The story of the German video was similar to the original version and was also based on a ten-year-old boy with an affected mother who reported from his point of view what it is like to live with a depressed parent. The story content and happy end made it possible to discuss depression, symptoms, individual coping mechanisms and therapy opportunities. By means of presenting the video and

subsequent discussion, a friendly atmosphere was created (the video is available at the following link: https://www.youtube.com/watch?v=VbYD_v3afko).

In the beginning of the study regular Skype sessions with Bruce Compas and colleagues were conducted to clarify open questions and to adapt the program correctly.

Training in using the manual.

The intervention sessions were provided by two doctoral students, two medical doctors as well as psychologists (with at least a Bachelor's degree) or team members from related fields (with at least a Bachelor's degree). All team members of the study have been trained by the project leader (a qualified post-doctoral researcher in clinical psychology) on how to conduct the manual.

The program in detail.

An overview of the session contents is shown in Figure 6. Each session is two hours long and composes training tasks for children and parents as homework which were explained at the end of each session. Completed training tasks were discussed in the beginning of the next session. Over the course of the program different aspects of psychoeducation, key coping strategies (used A'APP), parents' parenting skills and problem solving strategies were presented and discussed. Psychoeducation contained causes of depression, symptoms, how depression is defined and which role stress plays on the developmental risk. By this method each participant received a similar level of knowledge of the disorder. The key aim was to break the taboo, to talk about the disease within the group, to make the behaviour of the affected parent comprehensible and transparent for offspring as well as for the partner, and to show that different ways exist to deal with stressful situations. The first three sessions were conducted with the entire group together. From session four until eleven the group was split into two groups (parents and children separately), after discussing one part of the homework – the FUN ("Familienunternehmung") activity. At the end of the session both groups were

brought together and families were asked to talk about session contents, particularly of the children session. Training tasks for at home were explained at the end of each session by the group leader. Participants were asked to complete homework until the next session. The FUN activity was a regular homework for the entire family and contained the task to undertake a positive activity with the entire family each week for the time of the intervention program. The idea was that children and adults learn to perceive the difference of the individual status of well-being prior and after the family activity. Ideally, everyone felt better after the FUN activity. In the separate sessions, parents learned parenting skills, how to parent positively, which parenting styles exist, how parenting impacts offspring's behaviour, which influence offspring's age has and how to adapt parenting for current challenges. The implementation of knowledge was enhanced by role plays, group activities and lively debates.

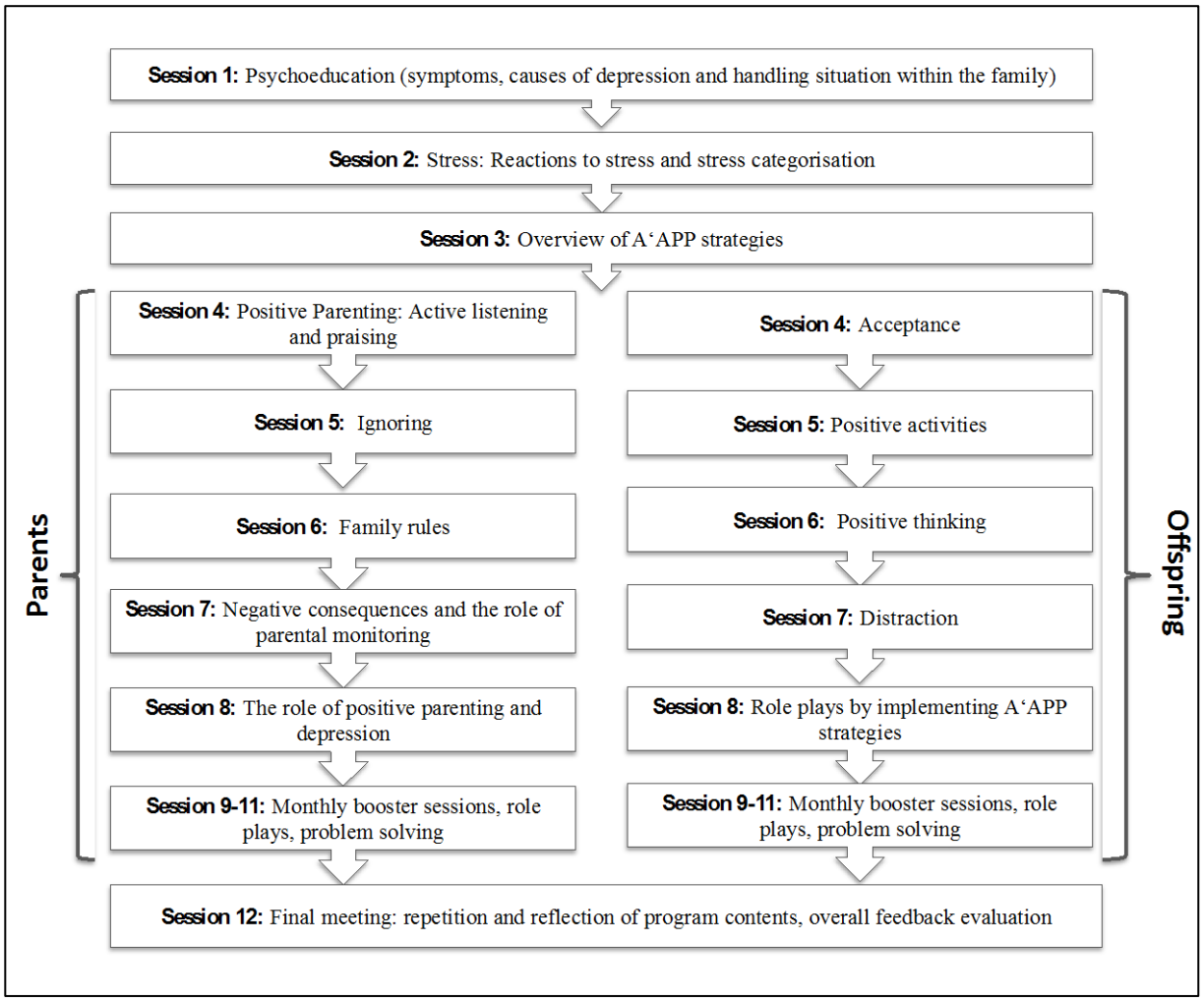

Figure 6. Session contents of the eight weekly and four monthly sessions inspired by Compas et al. (2009).

Children and adolescents deepened coping strategies during that time which were also topic of the first three sessions (*acceptance, distraction, positive activities, positive thinking*). In the original version, the strategies were called *ADAPT* strategies, while the current study renamed it into *A'APP* ("*Akzeptanz, Ablenkung, Positive Aktivität, Positives Denken*") strategies, and explained children and adolescents that the A'APP strategies will be installed on their internal hard disc (their brain) for future challenges, specifically for stressful events associated with parental depression. These coping strategies were explained to children and adolescents and like in parental sessions, strategies were practiced via group activities and role plays. In both groups, role plays consisted of stressful familiar situations with the aim to either implement positive parenting or for offspring to cope with the stressor. In both groups, particularly booster sessions had the aim of problem solving, looking how good strategies have been implemented in daily routine, what kind of stressors could arise in the future and what strategies would be useful for future challenges. Session twelve was a resume of the first eight sessions and the implemented strategies of both groups (children/adolescents and parent) were passed in review. In a playful way by using the game "Who wants to be a millionaire", knowledge of families was queried. Each family built one team and families played against each other. The winner received a small present ("celebration chocolate package") and at the end of the session an overall feedback evaluation form was filled out by each participant. Conduction time for each session was around 120 minutes.

Intervention fidelity.

Of 16 families no data were available of the attendance of the intervention sessions. Of those who provided data, parents (n = 21) attended on average 9.48 (standard deviation (SD) = 1.81) and children (n = 21) 9.29 (SD = 2.15) of the twelve sessions. Within the sessions, affected parents made their homework with a mean of 6.67 (SD = 2.33) and offspring with a mean of 7.38 (SD = 2.48).

The fidelity of sessions was checked with a checklist (see Appendix B) that was filled out by group leaders after each session. In general, sessions consisted of eight or nine subsections, except session twelve, which consisted of five subsections. For each section, group leaders had to state whether the section was conducted or not. From the eight groups that passed the program until now, data of seven groups (group two to eight) are available, from group one the data are only partially available (from session five to twelve).

Regarding session one (*contents: introduction, symptoms/ definition/ causes of depression, group activity, watching video, discussion video contents, explanation of homework principles and training sheets, session evaluation*), 87.5% were fully conducted but data of 12.5% (group one) are missing. The same was true for session two (*contents: discussion of homework, psychoeducation about stress related reactions, A'APP, positive activity, group activity, training sheets, session evaluation*) with 87.5% of session contents being completely conducted and 12.5% missing.

Regarding session three, group three did not pass the sections *positive and negative thinking*, all other sections (*discussion of homework, psychoeducation about acceptance, and distraction, a short summary of A'APP strategies, explanation of training sheets, session evaluation)* were conducted from the group. The other groups passed all contents, so that in total 75.0% were fully and 12.5% were partially conducted. From 12.5% (group one) data are missing.

For session four, group five and six did not pass the parent sections that discussed different *parenting styles* and *training of positive time*. In the children session, the section *implementing acceptance* was only partially conducted. All other parts (*discussion of homework, parents (positive time, praise, role-plays), children (uncontrollable/controllable stressors, definition of acceptance), training sheets, session evaluation*) were conducted by the group and all other groups. In total, 37.5% were fully and 37.5% were partially conducted, 12.5% were not conducted and 12.5% (group one) of data are missing.

For session five, data of group five were not available for the parent session (*discussion of homework, psychoeducation about ignoring, role play ignoring and praise, support network*), but for the children session (*positive activity, categories and exercise of positive activity*, *training sheets, session evaluation*). Group one did not conduct the subsection *praise and ignoring* in the parent session and also the *session evaluation* was not conducted. All other groups passed session contents, resulting in 75.0 % that were fully conducted, 12.5 % that were not conducted and 12.5% of missing data.

For session six, data of group two were not available for the section *discussing homework* from the children session. Group three, five and six did not conduct an *optional role play* in parent sessions with the focus on *house rules*. In the children session, the group activity *modulating negative thoughts in positive thoughts* was not conducted by group three. All other sections (*discussion of homework, parents (house rules, reward, target table), children (negative/positive thinking*)) were conducted by groups, resulting in 25.0% that were fully conducted, 50.0% that were partially conducted, 12.5% that were not conducted and 12.5% of missing data.

For session seven, data from group three from the parent session were not available, and data were also missing from group six for parents and children. Group two did not conduct the subsection *negative consequences* in the parent session and in the children session, the subsection *when distraction is a useful strategy* was not conducted. Group four partially conducted in the parent session the subsections *communicating consequences* and *homework*. From all other groups, session contents were conducted (*discussion homework, parents (knowing where children are, negative consequences*), *children (acceptance, distraction), training sheets, session evaluation*), resulting in 50.0% that were completely conducted, 12.5% that were partially conducted, 12.5% that were not conducted and 25.0% that were missing data.

For session eight, group two did not conduct the *role play with the A'APP strategies* in the children session, but all other sections were delivered by group leaders. In the parent session, group seven partially conducted the section that deals with the topic *how to support children by using the A'APP strategies*. All other groups conducted all sections (*discussion of homework, parents (positive parenting, recognising depressive symptoms and personal limits), children (repetition of A'APP strategies and role plays*), so that in total 75.0% of program contents were entirely delivered to groups, 12.5% were partially delivered and 12.5% were not delivered.

For session nine, group one did not pass *the role plays with future stressors* and *homework* in the parent session. Group three and five did not pass *role plays with future stressors* in the children session. Group seven did not pass the sections *discussing homework from last week* and *role plays with future stressors* in the parent session. All other groups passed all session contents (*homework, parents (repetition of positive parenting, future stressors and parenting), children (repetition of A'APP strategies, role plays with stressors in relation to a depressed parent), role-plays with the entire family*), so that 50.0% of program contents were fully, 25.0% were partially and 25.0% were not conducted.

Session ten and eleven consist exactly of the same program contents as session nine. In session ten, group one did not pass the *role play with future stressors* in the children session, all other program contents were conducted. Group three did not pass the section f*uture stressors in relation to parenting* in the parent session and the *role play with future stressors* in the children session. For group four, data of the parent session were not available and for group seven, no data of this session were available. All other groups passed all session contents. In total 37.5% were entirely, 25.0% were partially, 12.5% were not conducted and 25.0% of data were missing.

For session eleven, group one did not pass the section f*uture stressors in relation to parenting* in the parent session and in the children session the optional section *role play with*

future stressors was not passed. Further, the *family homework* was not explained to participants. Moreover, in the children session of group three, the optional section *role play with future stressors* was not conducted, but all other sections were delivered to participants. Group five did not pass the sections *future stressors and role plays with future stressors* in the parent session, and the *optional role play* in the children session. Group seven was partially conducted so that parents did not pass the *homework* and children did not pass the *optional role play*. Groups two, four, six and eight passed all program contents, so that in total 50.0% of program contents were entirely, 25.0% were partially and 25.0% were not conducted.

For session twelve, group one did not discuss the topic *personal aims and finding individual solutions for problems*, all other sections were conducted (*discussion of homework, the individual progress, the game who wants to be a millionaire, closing, final session evaluation*). Group two to six passed all program contents. Group seven did not pass the section *discussion of homework from last week.* Further data of the other sections were not available of this group. Group eight did not pass the last session. Therefore, data are not available here either. In total, 62.5% of program contents were fully delivered, 12.5% were partially delivered and 25.0% of data were not available.

In case not all program contents were delivered to participants in a session, a maximum of three (except session six of group one, in which more than three subsections were not conducted) subsections were not or only partially conducted. Due to the fact that the majority of sessions were fully conducted and single sessions consisted of eight or nine subsections, most program contents were delivered to participants.

Control Group

Whereas Compas et al. (2009, 2011) provided the control group written information, the current study followed the method of Sanford et al. (2003), who compared the experimental group with a waiting-list control group. By using this procedure, it is possible to test true prevention effects as well as the relevance of the program for clinical context. The effects of

prevention programs are highly complex and include the leader-group relationship, group effects and active elements that are used during sessions. A comparison of the intervention with an active control (which is usually a self-study), would make it difficult to show true prevention effects, as Compas and colleagues (2011) already discussed as one limitation of their study procedure. Further, an active control is not able to control placebo effects.

In the current study, prior randomisation participants were informed that in case of being allocated to the control condition, they would be able to receive the contents of the program after the study time period was over. At the beginning of the study, the plan was to deliver this in the form of written information although during the study the Deutsche Gesellschaft für Verhaltenstherapie (DGVT) offered to deliver the program close to its original format.

Measures

Table 8 gives an overview of screening and outcome measures. Outcome measures are subdivided into baseline and intervention outcomes. The time points of data collection (T1-T4) are also provided.

Table 8. The first row gives an overview of the function (screening measures, baseline outcomes, intervention outcomes). The second row illustrates the measures used in the current work and if measures were collected of offspring, parents or both. The third row lists diagnostic instruments with abbreviations (Diagnostic Interview of Psychiatric Disorders (DIPS) (Schneider & Margraf, 2011), Structured Clinical Interview for Axis II DSM disorders (SCID-II) (Wittchen et al., 1997), Diagnostic Interview of Psychiatric Disorders for children and adolescents (K-DIPS) (Unnewehr et al., 2008), Culture Fair Intelligence Test 20-R (CFT 20-R) (Weiß, 2006), Beck Depression-Inventory (BDI-II) for parents and adolescents (age ≥13) (Hautzinger et al., 2009), Depressions-Inventar für Kinder und Jugendliche (DIKJ) (Stiensmeier-Pelster et al., 2000), Youth Self-Report (YSR) (Döpfner et al., 1998), Child Behaviour Checklist (CBCL) (Döpfner et al., 1994), Child and Adolescent Survey of Experiences (CASE) (Allen et al., 2012), socio-economic status (SES) (Lampert et al., 2013), German questionnaire about knowledge of Depression (Allgaier et al., 2011), Erziehungsstil Inventar (ESI) (Krohne & Pulasack, 1991, 1995). Row four until seven give an overview of assessment points from baseline (T1), six months (T2), nine months (T3), and 15 months follow-up (T4), and which instruments were used at which time points.

Function	Measure	Diagnostic Instrument	T1	T2	T3	T4
Screening measures	Psycho-Diagnostic (Parent)	DIPS	x			
		SCID-II	x			
	Psycho-Diagnostic (Child)	K-DIPS	x			
	IQ (Child)	CFT-20-R	x			
	Psychopathological and depressive symptoms of partner	BDI-II, SCID-II	x			
Baseline outcomes	Child's psychopathology	BDI-II, DIKJ, YSR (self-report), CBCL (parent report)	x			
	Stressful life events (Child)	CASE	x			
	Socio-economic status	SES	x			
Intervention outcomes	Psycho-Diagnostic (Child)	K-DIPS	x			x
		Depression: BDI-II, DIKJ	x	x	x	
	General Psychopathology (Child)	YSR	x	x	x	
		CBCL	x	x	x	
	Knowledge of Depression (Child)	German questionnaire about knowledge of Depression	x	x	x	
	Parenting Style (Child)	ESI (affected parent and partner)	x	x	x	

Diagnostic instruments.

In order to make a proper diagnosis, researchers and clinicians apply specific diagnostic tools. A diagnosis is therefore either based on criteria of the Diagnostic and Statistical Manual of

Mental Disorders (Sass et al., 1996) or on criteria of the International Classification of Diseases (Dilling, Mombour, & Schmidt, 2015). Both are standard classification systems which allow a classification of mental disorders and both are structured similarly. With regard to unipolar affective disorders, they agree textually in many points, but differ in some diagnostic criteria. For research purposes (including this study) the DSM diagnostic manual is commonly applied. For this study, diagnostic instruments were used to assess whether families met inclusion criteria and did not meet exclusion criteria (see section *design*).

According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), a major depression is separated into major depression with a single episode (296.20-296.24) and major depression with a recurrent episode (296.30-296.34). Both, the single as well as the recurrent episode differentiate between unspecified, mild, moderate and severe depression with or without psychotic symptoms. From the two core symptoms (*loss of pleasure or interest in daily activities* or *depressed mood*), at least one plus four other symptoms (*changes in appetite or weight (5%), changes in activity (retardation or psychomotoric agitation), changes in sleep (hyper- or insomnia), changes in concentration (indecisiveness, reduced ability of concentrating or thinking), feelings of guilt or worthlessness, loss of energy or fatigue, suicidality (recurring thoughts of death or suicide, suicidal ideation without a concrete plan or a concrete suicide plan)* have to be present at least for two weeks for nearly every day and most of the day, and induce a changed functioning compared to the functioning before. Further, symptoms have to cause impairment in occupational or social areas or other scopes of functioning or cause a clinical relevant suffering. It is important that symptoms are not created by medical reason, by intake of psychotropic substances or caused by special circumstances, like e.g. bereavement, that criteria for a manic or hypomanic disorder are not fulfilled and the disorder cannot be better explained by schizophrenia, schizo-affective, schizotypic or delusional disorders or other not specified disorders. The number of symptoms provides information about the severity level.

The DSM-IV considers in case of a recurring depressive episode also the aspect of partial (296.35) or full (296.36) remission. Further, a diagnosis of double depression can be given, which means that within a phase of a dysthymic disorder (300.4), a major depression can be diagnosed simultaneously. A double depression can only be coded when a dysthymic disorder persists for at least two years (in children one year), and after this period the criteria of a major depressive disorder (MDD) are fulfilled. In case a major depressive episode occurs within those two years and criteria of a MDD are fulfilled, a MDD would be diagnosed (Sass et al., 1996).

The DSM-IV and the International Classification of Diseases (ICD-10) have a similar structure and both are used as diagnostic instruments. However, they also have slight differentiations in diagnosing a major depression. On the one hand they differ in the diagnostic codes (ICD-10: F32.xx, F33.xx (Dilling et al., 2015), DSM-IV: 296.2x, 296.3x (Sass et al., 1996)), on the other hand in some parts of classification criteria. According to the DSM-IV, one of two core symptoms plus four other symptoms have to be fulfilled, while the ICD-10 presupposes two of three core symptoms (*joylessness/loss of interest, depressed mood, listlessness/increased weariness*) plus at least two other symptoms which are equivalent with the other listed symptoms of the DSM-IV. Furthermore, the DSM-IV subclassifies between partial and full remission for a recurrent depressive episode, and also the diagnosis of a double depression is found only in DSM-IV. However, the ICD-10 differentiates between the cases *with and without somatic syndrome* in case of a mild or moderate depressive episode, and in case of a severe depression with psychotic symptoms between *parathym* or *synthym symptoms*; in the DSM-IV such a subclassification cannot be found.

Screening measures.

Diagnostic status (parents).

In order to decide whether the participating parent(s) fulfil(s) inclusion criteria by having a previous or current episode of depression as defined by DSM-IV, trained psychologists with at least a Bachelor´s degree or study nurses who received further training conducted the Diagnostic Interview for Psychiatric Disorders (DIPS) (Schneider & Margraf, 2011). The DIPS is a diagnostic instrument that enables the interviewer to diagnose retrospective as well as current episodes of depression, but also other mental disorders are screened with the tool. It is a semi-structured and clinician-administered interview which is applied to ensure different diagnosis in population samples as well as in psychiatric patients. The average implementation time is ≈ 120 minutes. In case both parents reported depressive symptoms, both were interviewed. If exclusion criteria (current symptoms of psychosis, suicidal risk, bipolar disorder, substance, alcohol abuse) were met, the participant was excluded from the study and individual recommendations were made (as proper institutions, counseling centres, doctors or therapists (see Appendix A).

To check for reliability of made DIPS diagnosis, an interviewer who is trained and has experience in performing psychiatric assessments, but who was not involved in providing intervention sessions to the families, listened to 20% of audio-recorded interviews and made a separate diagnosis. The level of agreement[4] between two assessors of the same interview resulted in a reliability level of 73.9%.

Symptoms of personality disorder.

The Structured Clinical Interview for Axis II DSM disorders (SCID) is a standardised screening instrument which consists of two major test sections (SCID-I, SCID-II) (Wittchen

[4] The level of agreement is based on parent and child interviews (n = 46). As agreement counted the precise type of MDD (single vs. remitted episode, double depression or severity) with parents interviews and the precise type of diagnosis or no diagnosis with child interviews.

et al., 1997). The first test part is a semi-structured interview (equivalent to the DIPS interview that was used in the current work) and it screens for individuals' psychopathology. The second part is a two-step instrument based on 117 yes/no questions that are formulated in form of statements about the own attitude, behaviour and experience. The questions cover the period from the last five to ten years. For assessing whether participants had a personality disorder (which would exclude them from the study), the second part of the Structured Clinical Interview for Axis II DSM-IV disorders (SCID-II) was conducted. In case questions were answered with yes, an interview followed as second step with detailed requests in order to be able to assess participants' eligibility. The instrument is used for notification of symptoms of twelve different personality disorders (according to Axis II of DSM-IV), but results should only be intended for guidance. This diagnostic instrument can be applied in out-patient as well as clinical settings. The general implementation time varies from a few minutes until one hour.

Diagnostic status (child).

For the assessment of offspring's mental health at baseline and to ensure that children and adolescents did not meet exclusion criteria (by having one or more previous or current psychiatric diagnosis according to DSM-IV criteria) the Diagnostic Interview of Psychiatric Disorders for children and adolescents (K-DIPS) (Unnewehr et al., 2008) was conducted. The instrument is standardised and it is designed for clinical diagnoses of children and adolescents aged between six and 18 years. By using the semi-structured interview, offspring's general psychopathological symptoms were assessed. Children and adolescents were only included in the study in case they were either free of psychopathological symptoms or symptoms occurred on sub-clinical level, so that diagnostic criteria were not fulfilled. The semi-structured interview was delivered by team members who received training in the manual use. With parents, a DIPS in relation to offspring's mental health was additionally conducted in order to receive an external view. In case discrepancies between parental and offspring's reports were

observed, child's statements gained more weight. It is known that parent and offspring can differ in their perspectives about offspring's health status and offspring particularly stronger assesses own internalising symptoms compared to parents (Cantwell et al., 1997; Ihle et al., 2004). The K-DIPS was also conducted at 15 months follow-up (T4) in order to be able to assess how many children developed depression from EG vs. CG. For the last interview, the analysis and conduction of screening sessions has been carried out by someone who is trained and has experience in performing psychiatric assessments, but who was not involved in providing intervention sessions to the families of the experimental group.

Fluid intelligence.

For the assessment of offspring's basic intelligence or the individual fluid intelligence, the German version of the Culture Fair Intelligence Test (CFT 20-R) (Weiß, 2006) was utilised. The test is developed for children and adolescents aged between eight and 19 years. It is a language-free and vivid method, and it is free of cultural and social influences. The CFT 20-R is a revised version of the CFT 20 with an enhanced differentiation in the upper performance range. The intelligence test is built on two similar parts, each with four subtests (continuous rows, object classification, matrices, topological concluding). In total, both test parts consist of 101 items (part one = 56 items, part two = 45 items) and the answer format is delivered as multiple choice version. Part one has two different time indications (subtest one and two = 4 min. each; subtest three and four = 3 min. each) to enable the diagnostician to get an impression of participants working speed. Test duration inclusive test introduction is around 60 minutes. The duration of the short test version (test part one only) takes around 35 - 40 minutes. Group and single tests are possible.

In the current study, participants conducted the short test version. Within a preset time, offspring had the task to dissolve formal-logical reasoning issues and figural relationships with different severity levels. Based on the CFT 20-R it was possible to ascertain whether children and adolescents met inclusion criteria by having an adequate cognitive power (IQ ≥

85) to understand program contents and to perform the intervention. The test-retest reliability (0.80 – 0.82) as well as the internal consistency (0.95) of the CFT-20R is very good. Correlations with other intelligence tests vary from r = 0.57 – 0.73, such as with the intelligence test Prüfsystem für Schul- und Bildungsberatung (0.60 – 0.63) (Weiß, 2006).

Assessment of depression severity (adolescents and adults).

The German version of the revised Beck Depression-Inventory (BDI-II) (Hautzinger et al., 2009) is a diagnostic instrument that enables the assessment of depression severity of minors (age ≥ 13) and adults. The self-report questionnaire consists of 21 items and depressive symptoms are rated depending on answers given on a four-point Likert-scale. The internal consistency is independent on the diverse subsamples with values ≥ 0.89 and a test-retest reliability = 0.78 indicating a very good internal consistency and test-retest reliability. Also the conduction with depressed patients receiving treatment reach high reliability (Cronbach's Alpha = 0.93). Validity varies for self-report from r = 0.72 – 0.89 and for external assessment from r = 0.68 – 0.70. Single and group tests are possible. Conduction time varies from five to ten minutes (Hautzinger et al., 2009).

Baseline outcomes.

Baseline outcomes of the current work are assessed by the depressive symptom severity in offspring which is measured with the BDI-II questionnaire (Hautzinger et al., 2009) and the "Depressionsinventar für Kinder und Jugendliche" questionnaire (Stiensmeier-Pelster et al., 2000), as well as by general psychopathology which is measured with child's self- (Döpfner et al., 1998) and parent reports (Döpfner et al., 1994). Because these measures are also part of intervention outcomes, they are described below in the section intervention outcomes. Other baseline outcomes are related to Child and Adolescent Survey of Experiences (Allen et al., 2012), and child's psychopathology in relation to the socio-economic status. The socio-economic status is classified following the Winkler-Index scale of Lampert et al. (2013).

Experiences with stressful and pleasant events (child).

The German version of the Child and Adolescent Survey of Experiences (CASE) (Allen et al., 2012) is a checklist that provides information about child's and adolescent's experiences with stressful and pleasant events. The checklist captures the time period of the last twelve months and is based on 38 items which describe life events that might have occurred within that time period. Participants were asked to rate whether the life event occurred (yes/no), and if yes, they rated between six answer options how positive or negative they felt the event was (really good, quite good, a little good, a little bad, quite bad, really bad). Unpleasant and pleasant life events that are queried concern e.g. job changes, school changes, experiences in school (winning prize, bullying), house moving, familiar relationships, health, cases of death and other happy or upsetting events. Due to standardised test instructions as well as standard values for test conduction, evaluation and interpretation, test objectivity can be assumed. The test-retest reliability (one week) is good ($r_{tt} = 0.75$) with accordance rates of 60% between mother and child. Further, the CASE correlates well with an interview-based measurement of individual stressful life events (Allen et al., 2012). The current work classified the number of stressful events in none (zero), a few (one/two), several (three/four) and many (five or more) events in order to examine whether offspring's depressive and general psychopathological symptoms stand in relation to the number of stressful events that were experienced.

Socio-economic status.

For measuring the socio-economic status (SES), guidelines of the Winkler-Index scale (that was also utilised by other researchers (Lampert et al., 2013; Ravens-Sieberer, Wille, Bettge, & Erhart, 2007)) were followed. The SES-Index is calculated as sum score based on the single dimensions parent education, parent profession and the familiar net income. The current study used for the dimensions parent education and parent profession data of the primary affected parent.

For each dimension, one to seven (low-high) points can be awarded and because the three scales are weight equally, values range from three to 21 (Lampert et al., 2013). An example for awarded points with regard to the dimension education is that secondary school is awarded with two points, university degree with six points and doctorate with seven points.

In order to receive a three-stage scale with low, middle and high SES from the numeric score, achieved scores (measured by a cumulation of education, income and professional status) are classified into three scales. Three to eight points define a low SES, nine to 15 points a middle SES and 16 to 21 a high SES, enabling a comparison between the 20% of upper and lower population with a wide middle range that captures 60% of the population (Lampert et al., 2013). Also other researchers refer to the three stages of SES for comparisons (Anli & Karsli, 2010; Ravens-Sieberer et al., 2007; Topham et al., 2010). The SES-Index has a high degree of conformity with other measures of the SES ranging from r = 0.63 – 0.87 (see Lampert et al. (2013)).

Intervention outcomes.

Intervention outcomes of the current work are assessed by the depressive symptom severity in offspring which is measured with the BDI-II questionnaire (Hautzinger et al., 2009) and the "Depressionsinventar für Kinder und Jugendliche" questionnaire (Stiensmeier-Pelster et al., 2000). The K-DIPS (Unnewehr et al., 2008) is used to assesses absence or presence of depression by comparing screening measures from baseline with outcomes from the 15 months follow-up. Thereby it is possible to show whether the prevention program is effective in depression prevention in children. Due to the fact that too little data from all other outcome measures from the 15 months follow-up have yet been collected, all other intervention outcomes are based on the six (T2) and nine (T3) months follow-up assessments. Symptom severity of offspring's general psychopathology is assessed with the questionnaires Youth Self-Report (Döpfner et al., 1998) from child's and adolescents' view and the Child-Behaviour Checklist (Döpfner et al., 1994) from parental view. Child's knowledge of

depression is assessed with the German questionnaire about depression (Allgaier et al., 2011) and offspring's perception about parenting style is assessed with the Erziehungsstil-Inventar (Krohne & Pulasack, 1991, 1995) questionnaire.

Assessment of depression severity (child).

The Depressionsinventar für Kinder und Jugendliche (DIKJ) (Stiensmeier-Pelster et al., 2000) is a self-report questionnaire that measures depressive symptom severity in children and adolescents aged between eight and 17 years. It is the German version of the Children's Depression Inventory (CDI) that was developed by Kovacs (1992). The original item "hypochondria" of the CDI was replaced in the DIKJ by the item "problem solving". The questionnaire is developed as child-friendly version and it covers all main symptoms of the depressive disorder according to the DSM-IV as well as accompanying symptoms. It consists of 26 items with a three-point Likert-scale (0-2) and the conduction time varies between ten and 15 minutes. The evaluation, implementation and interpretation of the test as well as the test instruction are standardised, so that the objectivity of the diagnostic instrument can be assumed. The DIKJ is an established measure of symptom severity of depression and shows in an unselected sample of students an internal consistency varying from $\alpha = 0.82 – 0.85$ and in a clinical sample $\alpha = 0.91$, showing a very high internal consistency. The DIKJ provides a good discriminate and convergent validity, which was demonstrated by having more correlations with construct-related (stability of self-esteem) than with construct-unrelated (capability of self-conceptualisation) questionnaires.

General psychopathology (self-report).

The Youth Self-Report questionnaire (Döpfner et al., 1998) for adolescents aged between eleven and 18 years was developed in German version in cooperation between the German working group Child Behaviour Checklist (Döpfner et al., 1994) and Thomas Achenbach who developed the original Youth Self-Report (Achenbach, 1991). It consists of two scales and it

captures child's statements about own competences, emotional and behavioural disorders. The first scale measures the competence by enquiring school achievements, sports activity and social competences. The second scale (the syndrome scale) consists of 120 items with a three-point Likert-scale and measures eight syndrome scales (categorized in internalising, externalising and mixed disorders) by capturing somatic complaints, emotional and behavioural disorders. The conduction time varies between 15 and 20 minutes. Due to a standardised test instruction and standard values, the instrument can be seen as objective concerning conduction, interpretation and evaluation. The reliability of the syndrome scales was largely confirmed by testing in a clinical sample (n = 292). For the overall conspicuity and the internalising and externalising scales, very good internal consistencies ($r \geq 0.86$) were reported. For the syndrome scales anxiety/depression, aggressive behaviour, somatic complaints, attention problems and dissocial behaviour only sufficient internal consistencies ($r > 0.70$) were found. In Germany, the standard values were examined in a nationwide sample of children and adolescents (n = 1800). Separate standard values for age and gender are reported, either in percentile ranks or in T-values. By using the main component analysis with subsequent varimax rotation, the factorial validity of the scales could be proven. In a clinical sample, the construction of scale was largely confirmed, except the scale social withdrawal. This scale was already not factorially confirmed in the original version.

General psychopathology (parent report).

For the assessment of parental judgement about offspring's competence skills behavioural and emotional psychopathological symptoms, the German version of the Child Behaviour Checklist (Döpfner et al., 1994) was utilised. This questionnaire is largely analogue to the YSR (Döpfner et al., 1998), it measures the same competence and syndrome scales, and is constructed for parents with children and adolescents aged between four and 18 years, but parental statement is in focus. The test is divided into two test sections. The first test section, the competence scale, consists of seven items capturing three scales (sport activity, school

achievement, social competence). The second test section, the syndrome scale, consists of 120 items measuring the same eight syndrome scales (internalising (anxiety/depression, social withdrawal, somatic complaints), externalising (aggressive and dissocial behaviour) and mixed (attention problems, social problems, schizoid-obsessive tendencies)). The conduction time varies between 15 and 20 minutes. Due to standardised test instructions as well as standard values of test conduction, evaluation and interpretation, test objectivity can be assumed. Reliability of global scales (internalising, externalising, mixed disorders) and syndrome scales could largely be confirmed in a German non-clinical (n = 1622) and clinical (n = 1653) sample. For the global scales internalising and externalising behaviour, good to very high internal consistencies (r >.85) were found in both samples. Separate standard values for age (four to eleven years; twelve to 18 years) and gender (male/female) are reported, either in percentile ranks or in T-values. In a clinical sample, except of the two syndrome scales "social withdrawal" and "social problems", the factor validity could be confirmed. Based on confirmatory analysis, the factorial structure was confirmed in 29 cultures, inclusive a German non-clinical sample (n = 2900) (Döpfner et al., 1994). Because the CBCL questionnaire is largely analogue to YSR (Döpfner et al., 1998), answer comparisons between the two questionnaires are possible which enables to capture behavioural competences and disorders from several perspectives.

Knowledge of depression.

A validated questionnaire for adolescents about knowledge and attitude of depression that was developed by colleagues (Allgaier et al., 2011) was utilised to establish whether offspring's knowledge of depression improved by the PRODO intervention. The questionnaire contains 50 items and covers statements about depression, symptoms, causes and treatment possibilities. Based on a four-point Likert-scale, children and adolescents rated the statements from strongly disagree (0) to strongly agree (3). At all four assessment points (T1-T4), data of knowledge have been collected. The current work presents T1-T3 measures.

Parenting style from child's view.

The Erziehungsstil-Inventar (ESI) questionnaire (Krohne & Pulasack, 1991, 1995) is used for the assessment of how offspring aged between eight and 16 years experiences parental parenting, so that information of problematic maternal or paternal parenting is provided. The ESI is a useful tool for finding possible reasons of offspring's problematic behaviour, exploration, for the interpretation of further test data and for monitoring success of interventions such as parental training. The questionnaire is applicable in clinics, at practices, for psychologists, in child and adolescent psychiatrists, for counselling services and it also finds use for school-psychological screenings.

Two identical test versions (mother/father) exist (Krohne & Pulasack, 1991, 1995). The current work classified the test versions of mothers and fathers parenting style in the parenting style of the affected parent and the parenting style of the other caregiver in order to examine whether the parenting of the depressed parent (and in case the other caregiver participated as well of the other parent) was modified by the program in a positive manner. Changes of parenting of both parents were then reported by offspring.

The test is divided into two test sections which assess six parenting style dimensions. The first test section consists of 60 items and captures the five parenting styles support (US), restriction (ES), praise (LS), blame (TS), and inconsistency (IK). The assessment of each parenting style is based on twelve items with answer options on a four-point Likert-scale. The second test section captures the intensity of punishment (SI) with five items with answer options on a six-point Likert-scale. An example of the support scale is: "my mother (my father) understands that I have a different opinion than she (he) has." Single and group tests are possible. The conduction time takes ≈ 20 minutes. The internal consistencies of the five scales support, restriction, praise, blame, inconsistency (test section one) vary from $\alpha = 0.77 – 0.92$. The internal consistency of the punishment intensity scale (test section two) varies from $\alpha = 0.65 – 0.71$. The test-retest coefficient (three weeks interval) relies between $r_{tt} = 0.51$ and

r_{tt} = 0.72. Separate standard values for age and gender are reported, either in percentile ranks or in T-values. Results of internal discriminate and convergent validity are provided as well as the relation to external validity criteria, e.g. intelligence, social competence, school performance or aggressiveness (Krohne & Pulasack, 1991, 1995).

Feedback Evaluation

Each participant (both children and their parents) received a feedback questionnaire directly after each session with the aim to evaluate the general comprehensiveness and acceptance of the program and training sheets. This is an important point because only if families accept such an offer and rate it as useful will they make use of it.

The feedback evaluation questionnaire is the German version of the original feedback evaluation form of Compas et al. (2009, 2011) and consists of six items that are rated on a five-point Likert-scale. Questions focus on program contents, usefulness of the session, and if the person felt comfortable within the setting. The current work will present outcomes of the five items comprehensiveness, active participation, comfort, understanding training sheets and usefulness of training.

Data Preparation and Analytic Strategy

Data preparation.

The current study included one child per family in the analysis, and in case more than one child per family participated in the study, the oldest child was included in the analysis. The reason for this lies in the general onset of depression in adolescent persons, which reaches its peak in the age range of 15 to 20 years (Weissman et al., 1997, 2006), meaning the oldest child per family is probably the child at highest risk.

With regard to depression measures: because adolescents by the age of 13 or older filled out the BDI-II questionnaire (Hautzinger et al., 2009) and children below the age of 13 the DIKJ questionnaire (Stiensmeier-Pelster et al., 2000), a BDI-II/DIKJ composite was

created by z-transforming both questionnaires to one composite. Both questionnaires were also analysed separately as outcome measures of depressive symptoms.

Analytic strategy.

The analysis strategy for individual hypotheses is reported below. For describing sample characteristics and analysing hypotheses, the statistic-software IBM SPSS Statistic Version 23 was used. By using the Shapiro-Wilk test (S-W) it was possible to check whether variables of interest were normally distributed, although histograms were also used to visually assess the normality of distributions. In case the S-W revealed a non-normal distribution, the variable of interest was log-transformed or statistical tests were used that are relatively robust against breaches of normal distributions. Variance homogeneity was tested by using the Levene-test. Spherictiy was assessed by using the Mauchly-test. In case sphericity could not be assumed, a Greenhouse-Geisser correction was utilised. Mean scores (M), standard deviations (SD), minimum, maximum, frequencies and percentages were calculated.

Effect sizes of univariate ANOVAs and repeated measures ANOVAs are reported with Eta-square (η_p^2), with small effects when $\eta_p^2 = 0.01$, medium effects when $\eta_p^2 = 0.06$ and strong effects when $\eta_p^2 \geq 0.14$. In case of a significant interaction or main effect, post-hoc t-tests were conducted with the Bonferroni correction and the mean difference (md) as well as the significance level is reported. Effect sizes of post-hoc t-tests are reported by using Cohen's d. Here effects are small when d = 0.2, medium when d = 0.5 and strong when d = 0.8. At the end of the results section I will report descriptive statistics of the feedback evaluation based on averages across all sessions.

Baseline outcomes.

Hypothesis 1: It is hypothesised that the severity of parental depression (measured by BDI-II score of the primary affected parent) and the number of depressive episodes of the primary affected parent correlate positively with child's depressive (BDI-II, DIKJ) and general

psychopathological (YSR, CBCL) symptoms. To test this, a Pearson correlation was conducted.

Hypothesis 2: To test whether children and adolescents who experienced many stressful life events (five or more) during the last twelve months will have higher scores in scales measuring depressive (BDI-II, DIKJ) and general psychopathological (YSR, CBCL) symptoms at baseline compared to offspring without or only a few (zero or one/two) stressful experiences, univariate ANOVAs were performed. This was done firstly with the depression composite (z-transformed BDI-II and DIKJ), the BDI-II and DIKJ separately as dependent variable, secondly with offspring's self-report about general psychopathological symptoms (YSR (Döpfner et al., 1998)) as well as with parental report about offspring's general psychopathological symptoms (CBCL (Döpfner et al., 1994)). As independent variable, the sum score of negative events of the CASE (Allen et al., 2012) questionnaire was utilised, which was classified in none (zero), a few (one/two), several (three/four) and many (five or more) stressful life events.

Hypothesis 3: Originally the plan was to examine differences of all three SES types, to test whether children and adolescents with low and high SES have stronger pronounced psychopathological symptoms compared to children and adolescents from middle-class. But since less people were recruited with low SES (n = 2), the original plan of analysing data was not possible. Therefore, the analysis was conducted with the two scales middle vs. high SES. As previous presented studies showed depressive or general psychopathological conspicuities in children with low or high SES and none with middle-SES and since the current sample consists solely of families with middle or high SES, it is hypothesised that offspring's depressive and general psychopathological symptoms are more pronounced in families with high SES compared to families with middle SES. Therefore, univariate ANOVAs were conducted separately with offspring's BDI-II/DIKJ composite (z-transformed BDI-II and DIKJ), offspring's BDI-II and DIKJ, the three scales (internalising, externalising, mixed) of

the YSR (Döpfner et al., 1998) and CBCL (Döpfner et al., 1994), each as dependent variable. The SES was built following the Winkler Index (Lampert et al., 2013) as independent variable.

Particular data preparation of intervention outcomes.

The analytic approach of intervention outcomes is based on the comparison of 77 families, who were stratified, randomised blockwise and allocated either to the preventive intervention (EG, n = 37) or to a waiting-list control condition (CG: WL, n = 40). Examined was the effectiveness of the program in depression prevention as well as intervention effects on offspring's depressive symptoms, general psychopathological symptoms, knowledge of depression and parenting style. Pairwise t-tests or Chi-Square tests were utilised to ensure equal distributions of data at baseline. As illustrated in Tables 4, 6 and 7, groups did not differ in terms of the following key variables of the primary affected parent (age, gender, status of employment, working time, marital status, nationality, current depression, number of depressive episodes, recurrent depressive episode, BDI-II scores, SCID-II, number of psychiatric disorders in family, comorbidity, in treatment (psychotherapy, psychiatry), clinic stay, number of clinic stays, medication), the partner (age, current depression, number of depressive episodes, BDI-II scores, SCID-II scores), when both parents are affected (both depressed), the child (age, gender, number of siblings, IQ, BDI-II, DIKJ, K-DIPS, YSR externalising, CBCL internalising, knowledge of depression, offspring's perception of the primary affected parent or partner (ESI affected parent, ESI partner), negative events (CASE)) or the familial socio-economic status (all $ps > 0.05$). However, significant differences between groups were seen for the following variables (other support, YSR internalising, YSR mixed, CBCL externalising, CBCL mixed (Table 6)) with $p < 0.05$. Therefore, the current work used statistical operations that are relatively robust against breaches of group differences or normal distributions (Kähler, 2008).

During follow-up measurements, only complete questionnaires were included, incomplete questionnaires were not considered.

Intervention outcomes.

To investigate whether the program is effective in preventing a depression onset in children in the long-term (*Hypothesis 5*), a Chi-Square test was conducted with data of the 15 months follow-up. Therefore, depression outcomes of EG vs. CG participants were compared. Because the Pearson Chi-Square test revealed that 50% of expected counts were less than 5, outcomes are based on the Fisher's exact test. Percentage of participants who developed a depression is going to be reported for both groups as well as the valid n.

The other four intervention outcomes were run with four 2 x 3 mixed ANOVAs with group (EG, CG) as the between-subjects factor and time (T1, T2, T3) as the within-subjects factor. To assess the effect of depressive symptoms, the BDI-II/DIKJ composite was analysed, but also the two questionnaires BDI-II (Hautzinger et al., 2009) and DIKJ (Stiensmeier-Pelster et al., 2000) were analysed separately as outcome measures of depressive symptoms in offspring (*Hypothesis 4*). Since too little data were available of the T3 assessment point (BDI-II = 8, DIKJ = 12) for this hypothesis, the analysis was run with 2 x 2 mixed ANOVAs with group (EG, CG) as the between-subjects factor and time (T1, T2) as the within-subjects factor. To assess the effect on general psychopathology, offspring's self-reports (YSR (Döpfner et al., 1998)) and parent reports (CBCL (Döpfner et al., 1994)) were used as outcomes (*Hypothesis 6*). To assess offspring's knowledge of depression, the German questionnaire about knowledge of depression (Allgaier et al., 2011) was used as outcome measure (*Hypothesis 7*). To assess whether offspring from EG perceived parents´ parenting style differently and more positive compared to offspring from CG, the ESI questionnaire was utilised (Krohne & Pulasack, 1991, 1995) as outcome measure (*Hypothesis 8*).

An interaction between time and group would suggest an influence of the prevention program. Significant interaction of time and group or main effects will be followed up with post-hoc t-tests. Two sample t-tests will be performed to investigate at which time points

groups differ and to test the direction of effects. Bonferroni correction was applied for alpha inflation of multiple comparisons.

Results

Hypothesis Testing

Baseline outcomes.

Hypothesis 1: Parental depression and offspring's mental health.

Table 9 gives an overview of correlations between parent BDI-II scores, number of depressive episodes and offspring's depressive and general psychopathological symptoms. Mean scores and standard deviations are listed in Table 6 in the method section.

Table 9. Child depressive (BDI-II, DIKJ) and general psychopathological outcomes (YSR (internalising, externalising, mixed), CBCL (internalising, externalising, mixed)) in association with parent BDI-II scores as well as with the number of parental depressive episodes. The scores of the BDI-II and DIKJ are the original scores and not the log-transformed[5] ones. *The correlation is on the level of $\alpha \leq 0.05$ (2-tailed) significant.

Child depressive and general psychopathological outcomes	Parent BDI-II	Parent No. of episodes
Child BDI-II	0.07	-0.17
Child DIKJ	-0.02	0.16
YSR internalising	-0.09	0.26
YSR externalising	0.03	0.345*
YSR mixed	-0.08	0.26
CBCL internalising	0.05	0.16
CBCL externalising	0.08	0.21
CBCL mixed	0.03	0.19

[5] Ln transformation of child's BDI-II and DIKJ variables.

By using the Shapiro-Wilk (S-W) test there was evidence that child's BDI-II (S-W statistic = 0.91, df = 26, p = 0.029) and DIKJ (S-W statistic = 0.92, df = 53, p = 0.001) were not normally distributed. However, the histograms of the BDI-II and DIKJ showed a normal distribution. All other measures (YSR (internalising, externalising, mixed), CBCL (internalising, externalising, mixed)) were normally distributed according to the S-W test. The Pearson correlation that was performed to test whether depression severity (measured by BDI-II scores) and the number of depressive episodes of the primary affected parent correlate with offspring's depressive and general psychopathological symptoms showed that neither BDI-II scores (child's BDI-II n = 26, r = 0.07, p > 0.05; DIKJ: n = 51, r = -0.02, p > 0.05) nor the number of depressive episodes (child's BDI-II: n = 19, r = -0.17, p > 0.05; DIKJ: n = 44, r = 0.16, p > 0.05) of the primary affected parent correlate with variables that measure offspring's depressive symptoms. Because child's BDI-II and DIKJ scores were according to the S-W test not normally distributed, the same analyses were conducted with log-transformed variables. Here, child's BDI-II or DIKJ scores also did not correlate with parental depression severity (child's BDI-II n = 24, r = 0.17, p > 0.05; DIKJ: n = 52, r = -0.04, p > 0.05) or the number of depressive episodes (child's BDI-II: n = 17, r = -0.09, p > 0.05; DIKJ: n = 44, r = 0.14, p > 0.05).

When looking at child's general psychopathological symptoms, results demonstrated that the number of depressive episodes of the primary affected parent positively correlate with the externalising scale of the YSR (n = 46, r = 0.345, p = 0.019), indicating that the more episodes the primary affected parent experienced, the more externalising psychopathological symptoms in offspring were observed, such as aggressive or dissocial behaviour. However, neither the internalising (n = 46, r = 0.26, p > 0.05) nor the mixed (n = 46, r = 0.26, p > 0.05) YSR scale correlated with the number of depressive episodes. No scale of the YSR correlated with parental BDI-II (YSR internalising: n = 57, r = - 0.09, p > 0.05; externalising: n = 57, r = 0.03, p > 0.05; mixed: n = 57, r = -0.08, p > 0.05).

No scale of the CBCL correlated with parental BDI-II (internalising: n = 57, r = 0.05, p > 0.05; externalising: n = 57, r = 0.08, p > 0.05; mixed: n = 57, r = 0.03, p > 0.05) or the number of depressive episodes (internalising: n = 46, r = 0.16, p > 0.05; externalising: n = 46, r = 0.21, p > 0.05; mixed: n = 46, r = 0.19, p > 0.05).

Hypothesis 2: Aversive events and offspring's mental health.

Table 10 gives an overview of the three scales of the YSR in association with the number of stressful or aversive events, the child experienced in the last twelve months.

Table 10. The Table illustrates means, standard deviations (SD) and sample size (n) of all three scales of the YSR (internalising, externalising, mixed) across offspring of affected parents at baseline**.** The stressful life events are measured with the CASE questionnaire (Allen et al., 2012). Differentiations were made between none (0), a few (1-2), several (3-4) and many (≥ 5) stressful life events.

			CASE			
			0	1-2	3-4	≥ 5
YSR	Internalising	M	47.80	47.72	52.21	59.86
		SD	10.76	9.58	6.93	11.13
		N	5	18	14	14
	Externalising	M	48.40	49.33	48.57	57.29
		SD	3.05	6.53	4.11	8.89
		N	5	18	14	14
	Mixed	M	50.40	50.06	50.71	61.07
		SD	6.35	7.92	6.84	9.42
		N	5	18	14	14

Because for the Shapiro-Wilk test too little data were available within *no negative events*, the normal distribution was assessed visually with histograms. All measures showed normal distributions within no negative events. In case of *few (one/two) negative events*, the S-W test revealed that all measures were normally distributed. In case of *several (three/four) negative events*, the externalising scale of the CBCL was not normally distributed (S-W

statistic = 0.86, df = 13, p = 0.036), however the histogram showed a normal distribution. All other measures were normally distributed. In case of *many (five or more) negative events*, all measures were normally distributed.

Regarding offspring's depressive symptoms, there was no evidence of significant group differences between those who experienced none, those who experienced a few, those who experienced several and those who experienced many stressful life events measured by using a BDI-II/DIKJ composite ($F_{3,48}$ = 1.61, p > 0.05, η_p^2 = 0.091). The same was true when the BDI-II ($F_{3,16}$ = 1.05, p > 0.05, η_p^2 = 0.165) with n = 20 and the DIKJ ($F_{3,39}$ = 2.34, p > 0.05, η_p^2 = 0.153) with n = 43 were analysed separately.

Regarding offspring's general psychopathological symptoms, there was evidence of a significant effect of stressful life events on all scales of the YSR (n = 51), reflected on internalising ($F_{3,47}$ = 4.70, p = 0.006, η_p^2 = 0.231), externalising ($F_{3,47}$ = 5.60, p = 0.002, η_p^2 = 0.263) and mixed ($F_{3,47}$ = 6.13, p = 0.001, η_p^2 = 0.281) level. Regarding the scale that measures internalising symptoms (YSR internalising), post-hoc t-tests with the Bonferroni corrected α (p < 0.0125) revealed significant differences between those participants who had a few (one/two) stressful life events and those who had many (five or more) stressful life events (mean difference (md) = 12.14, p = 0.005, d = 1.18). All other effects were non-significant. For the scale that measures externalising psychopathological symptoms, the same difference was true (md = 7.95, p = 0.008, d = 1.04), but additionally also a significant difference was found between those participants who experienced several (three/four) and those who experienced many (five or more) stressful life events (md = 8.71, p = 0.006, d = 1.26). Other significant differences were not observed. In the scale that measures mixed psychopathological symptoms, there were also significant differences between those participants who experienced a few (one/two) and those who experienced many (five or more) stressful life events (md = 11.02, p = 0.002, d = 1.28). Additionally, also a significant difference was found between those participants who experienced several (three/four) and

those who experienced many (five or more) stressful life events (md = 10.36, $p = 0.007$, d = 1.26). A comparison of mean values (see Table 10) demonstrates that with many stressful life events (≥ five) the scores of all three scales are generally higher compared to a few (one/two) stressful life events in the past twelve months, indicating that offspring's general psychopathological symptoms stand in relation to the number of aversive experiences.

Results of the parental view on offspring's general psychopathological symptoms (CBCL) revealed (with n = 51) no significance, neither for the internalising ($F_{3,47} = 0.78$, $p > 0.05$, $\eta_p^2 = 0.048$), nor for the externalising scale ($F_{3,47} = 1.04$, $p > 0.05$, $\eta_p^2 = 0.062$) and nor for the mixed scale ($F_{3,47} = 1.11$, $p > 0.05$, $\eta_p^2 = 0.066$). Furthermore, when the log-transformed externalising CBCL scale was utilised, no significance was found ($F_{3,47} = 0.89$, $p > 0.05$, $\eta_p^2 = 0.054$).

Hypothesis 3: SES and offspring's mental health.

All symptom measures were normally distributed within the SES scales middle vs. high. The analysis that compared the two scales, revealed no evidence that SES was associated with offspring's depressive symptoms, reflected in the BDI-II/DIKJ composite ($F_{1,48} = 0.09$, $p > 0.05$, $\eta_p^2 = 0.002$). The same was true when the BDI-II ($F_{1,19} = 0.01$, $p > 0.05$, $\eta_p^2 = 0.001$) with n = 21 and the DIKJ ($F_{1,39} = 0.53$, $p > 0.05$, $\eta_p^2 = 0.013$) with n = 41 were analysed separately. When looking at offspring's general psychopathological symptoms, the conduction of univariate ANOVAs revealed neither significance for the internalising ($F_{1,45} = 0.19$, $p > 0.05$, $\eta_p^2 = 0.004$), nor the externalising ($F_{1,45} = 0.07$, $p > 0.05$, $\eta_p^2 = 0.001$) and nor the mixed ($F_{1,45} = 0.17$, $p > 0.05$, $\eta_p^2 = 0.004$) scale of offspring's self-report (YSR) with n = 47, nor for the internalising ($F_{1,45} = 1.11$, $p > 0.05$, $\eta_p^2 = 0.024$), the externalising ($F_{1,45} = 0.45$, $p > 0.05$, $\eta_p^2 = 0.010$) and nor the mixed ($F_{1,45} = 0.62$, $p > 0.05$, $\eta_p^2 = 0.014$) scale of parent report (CBCL) about offspring's general psychopathology (with n = 47), indicating that there is no difference between children with middle vs. high SES.

Intervention outcomes.

Hypothesis 4: Offspring's depressive symptoms.

Table 11 and 12 illustrate mean, standard deviations and sample size (n) of both questionnaires over time (T1-T2) separately for EG and CG and the total sample.

Table 11. The Table illustrates means (M), standard deviations (SD) and sample size separately for EG, CG and the total sample over assessment points T1 and T2 of the BDI-II questionnaire.

	Group	**EG**		**CG**		**Total**	
	Time	T1	T2	T1	T2	T1	T2
BDI-II	M	7.83	6.83	7.20	9.60	7.44	8.56
	SD	6.37	5.27	5.55	5.99	5.67	5.72
	N	6	6	10	10	16	16

Table 12. The Table illustrates means (M), standard deviations (SD) and sample size separately for EG, CG and the total sample over assessment points T1-T2 of the DIKJ questionnaire.

	Group	**EG**		**CG**		**Total**	
	Time	T1	T2	T1	T2	T1	T2
DIKJ	M	46.27	45.91	45.00	46.00	45.74	45.95
	SD	7.30	12.07	4.90	9.18	6.27	10.66
	N	11	11	8	8	19	19

By using the Shapiro-Wilk test, there was evidence to suggest that changes of depressive symptoms over time were normally distributed within groups. The Levene-test revealed evidence that variance homogeneity was given for both, the BDI-II and DIKJ. The Mauchly-test showed that sphericity was given for all three conducted analyses. The BDI-II/DIKJ composite, the BDI-II and DIKJ were equally distributed between groups. The three 2 x 2 mixed ANOVAs that were performed (BDI-II/DIKJ composite, BDI-II (Hautzinger et al.,

2009), DIKJ (Stiensmeier-Pelster et al., 2000)) to test whether differences in offspring's depressive symptoms can be found between the intervention and waiting-list control group revealed neither a significant interaction ($F_{1,33} = 0.08$, $p > 0.05$, $\eta_p^2 = 0.002$) nor a significant main effect of time ($F_{1,33} = 0.10$, $p > 0.05$, $\eta_p^2 = 0.003$) with regard to the BDI-II/DIKJ composite (n = 35). The same was true for separate analyses of the BDI-II (n = 16) with no evidence of an interaction ($F_{1,14} = 1.11$, $p > 0.05$, $\eta_p^2 = 0.074$) or main effect of time ($F_{1,14} = 0.19$, $p > 0.05$, $\eta_p^2 = 0.013$) and the DIKJ (n = 19) with no evidence of an interaction ($F_{1,17} = 0.10$, $p > 0.05$, $\eta_p^2 = 0.006$) or main effect of time ($F_{1,17} = 0.02$, $p > 0.05$, $\eta_p^2 = 0.001$).

Hypothesis 5: Prevention of depression in offspring.

Results of the Fisher's exact test that was conducted to compare the incidence of depression in the 40 families who had reached the 15 month follow-up, showed a trend for the preventive effects of the intervention (0/19; 0%) versus the control group (4/21; 19%). However, this trend was not statistically significant ($p > 0.05$).

Hypothesis 6: Offspring's general psychopathology.

The Shapiro-Wilk test showed evidence to suggest that the externalising YSR scale was not normally distributed for CG participants at T1 (S-W statistic = 0.83, df = 15, $p = 0.008$). Also the mixed YSR scale was not normally distributed for EG participants at T2 (S-W statistic = 0.69, df = 13, $p < 0.001$). All other collected data of self- (YSR) and parent reports (CBCL) were normally distributed within conditions over the three time points. Because the majority of collected data was normally distributed and the ANOVA is robust of infringing the premises of normal distribution, the analyses were conducted with repeated measures ANOVA. The Levene-test revealed evidence that variance homogeneity was given for all scales of the YSR (internalising, externalising, mixed) and the CBCL (internalising, externalising, mixed). The Mauchly-test that was performed to check for sphericity revealed evidence that sphericity was given for all scales of the YSR (internalising, externalising,

mixed). For the CBCL, sphericity was given for externalising and mixed scales. For the internalising scale (Mauchly-W = 0.76, df = 2, p = 0.030), a Greenhouse-Geisser correction was performed. The YSR externalising scale and the CBCL internalising scale were equally distributed across groups. No equal distribution was found for the YSR internalising (t_{55} = 2.60, p = 0.012) and mixed (t_{55} = 2.24, p = 0.029) scales. The same was true for the CBCL externalising (t_{55} = 2.41, p = 0.019) and mixed (t_{55} = 2.37, p = 0.021) scales (see Table 6).

Internalising symptoms (self-report).

Figure 7 illustrates the course of internalising symptoms over time for both groups (EG, CG) separately. It can be seen how mean scores of internalising symptoms of participants who took part in the prevention program (EG) decreased significantly in short-term (from T1 to T2).

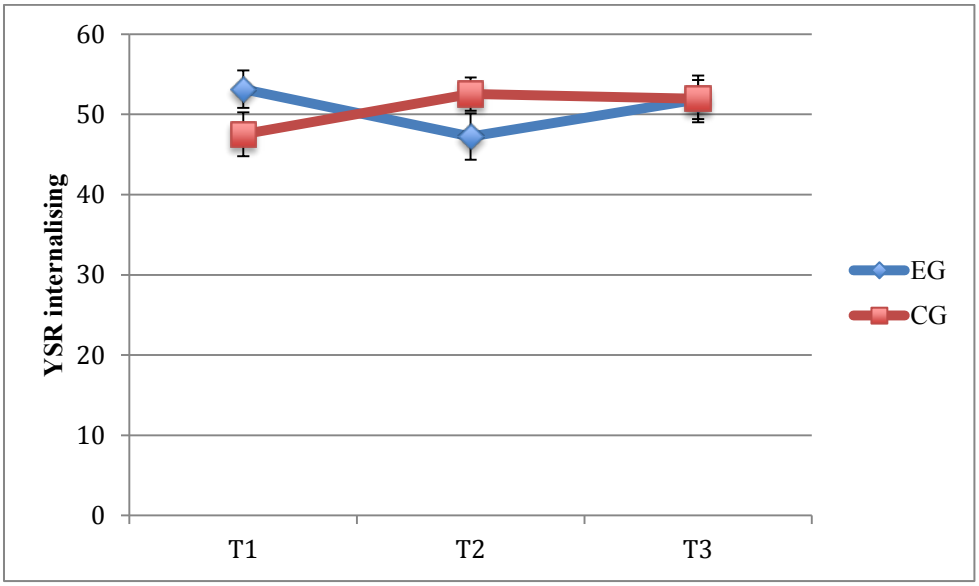

Figure 7. The graph illustrates scores of the YSR scale internalising symptoms for both groups over the three assessment points (T1-T3). Error bars indicate standard errors of mean (SEM).

Table 13. The Table illustrates means (M), standard deviations (SD) and sample size (N) separately for EG and CG and also for the total sample over assessment points T1-T3 for each scale of the YSR (internalising, externalising, mixed) questionnaire.

YSR	Group	EG			CG			Total		
	Time	T1	T2	T3	T1	T2	T3	T1	T2	T3
Internalising	M	53.15	47.23	51.85	47.53	52.53	51.93	50.14	50.07	51.89
	SD	8.40	10.42	8.76	10.56	7.98	11.27	9.86	9.41	10.00
	N	13	13	13	15	15	15	28	28	28
Externalising	M	51.54	48.23	48.38	49.47	52.93	48.67	50.43	50.75	48.54
	SD	6.68	7.41	6.51	7.54	10.16	10.93	7.10	9.14	8.99
	N	13	13	13	15	15	15	28	28	28
Mixed	M	54.46	46.81	51.31	50.33	54.20	54.27	52.25	50.77	52.89
	SD	7.16	13.30	7.84	8.02	8.82	10.55	7.78	11.54	9.34
	N	13	13	13	15	15	15	28	28	28

Table 13 describes scores of self-reported internalising symptoms across the three assessment points (T1 (baseline), T2 (six months), T3 (nine months)). The 2 x 3 mixed ANOVA that was performed to test whether differences in internalising symptoms can be found between intervention and control groups in offspring's self-reports (YSR) revealed evidence of a significant interaction between time and group ($F_{2,52} = 5.02$, $p = 0.010$) with a large effect size of $\eta_p^2 = 0.162$. The main effect of time ($F_{2,52} = 0.74$, $p > 0.05$, $\eta_p^2 = 0.028$) revealed no significance. When performing post-hoc t-tests, the adjustment for multiple comparisons with Bonferroni revealed no significant difference for EG between the time points T1 and T2 (mean difference (md) = 5.92, $p > 0.05$), T1 and T3 (md = 1.31, $p > 0.05$) or T2 and T3 (md = -4.62, $p > 0.05$). The same was true for CG (T1 and T2 (md = -5.00, $p > 0.05$), T1 and T3 (md = -4.40, $p > 0.05$), T2 and T3 (md = 0.60, $p > 0.05$)). By using standard t-tests as post-hoc tests with the Bonferroni corrected α, significant differences for EG participants could be observed between T1 and T2 ($t_{15} = 2.68$, $p = 0.016$, d = 0.49), reflected

in a decrease of internalising symptoms. Between the time points T1 and T3 (t_{13} = 0.50, p > 0.017, d = 0.09) and T2 and T3 (t_{12} = -2.58, p > 0.017, d = - 0.48) no significant differences were found. For CG participants, no differences between T1 and T2 (t_{16} = -1.30, p > 0.017, d = - 0.42), T1 and T3 (t_{16} = - 2.16, p > 0.017, d = - 0.49) or T2 and T3 (t_{16} = - 0.13, p > 0.017, d = - 0.03) could be demonstrated.

Internalising symptoms (parent report).

Table 14 displays means and standard deviations of the CBCL across the three assessment points separately for EG and CG, but also for the total sample.

Table 14. The Table illustrates means (M), standard deviations (SD) and sample size (N) separately for EG and CG and also for the total sample over assessment points T1-T3. Scores of all three scales (internalising, externalising, mixed) of the CBCL questionnaire are illustrated.

CBCL	**Group**	**EG**			**CG**			**Total**		
	Time	T1	T2	T3	T1	T2	T3	T1	T2	T3
Internalising	M	59.17	56.42	52.83	59.13	57.63	55.25	59.14	57.11	54.21
	SD	10.32	10.21	10.90	7.91	9.54	9.30	8.84	9.67	9.90
	N	12	12	12	16	16	16	28	28	28
Externalising	M	53.92	52.92	51.42	48.06	49.00	47.44	50.57	50.68	49.14
	SD	7.01	6.26	9.39	8.19	8.11	7.94	8.12	7.51	8.66
	N	12	12	12	16	16	16	28	28	28
Mixed	M	58.33	54.58	52.75	52.88	53.81	51.63	55.21	54.14	52.11
	SD	6.89	8.12	7.45	7.90	7.59	6.72	7.85	7.68	6.93
	N	12	12	12	16	16	16	28	28	28

The 2 x 3 mixed ANOVA that was performed to assess internalising psychopathological symptoms of offspring from parental view (CBCL) revealed no evidence of an interaction ($F_{1.61, 41.78}$ = 0.36, p > 0.05, η_p^2 = 0.014) but a significant main effect of time ($F_{1.61, 41.78}$ = 6.34, p = 0.007, η_p^2 = 0.196), indicating that independent of the program,

differences across time occurred. The Bonferroni adjustment for multiple comparisons revealed a significant difference between the time points T1 and T3 (md = 5.10, $p = 0.018$), reflecting a decrease of offspring's internalising symptoms from parent view over time, independent of the program. No significant difference between the other time points could be observed (T1 and T2 (md = 2.13, $p > 0.05$), T2 and T3 (md = 2.98, $p > 0.05$)). Standard t-tests as post-hoc tests with the Bonferroni corrected α even revealed significant differences between T1 and T2 ($t_{32} = 2.88$, $p = 0.007$, d = 0.42) as well as between T1 and T3 ($t_{31} = 3.37$, $p = 0.002$, d = 0.55), reflecting a decrease of symptoms independent of group allocation over time. Between the time points T2 and T3 ($t_{28} = 2.19$, $p > 0.017$, d = 0.32), no significant difference could be observed.

Externalising symptoms (self-report).

Scores of externalising symptoms of the YSR are illustrated in Table 13. Regarding scales that measure externalising psychopathological symptoms (aggressive, delinquent behaviour) with the self-report questionnaire (YSR), analysis revealed no significant interaction between time and group ($F_{2,52} = 2.80$, $p > 0.05$, $\eta_p^2 = 0.097$). There was also no evidence of a significant main effect of time on the YSR ($F_{2,52} = 1.29$, $p > 0.05$, $\eta_p^2 = 0.047$).

Externalising symptoms (parent report).

Scores of externalising symptoms of the parent report (CBCL) are illustrated in Table 14. The CBCL of offspring's externalising psychopathological symptoms revealed no significant interaction ($F_{2,52} = 0.38$, $p > 0.05$, $\eta_p^2 = 0.014$) or main effect of time ($F_{2,52} = 1.00$, $p > 0.05$, $\eta_p^2 = 0.037$) either.

Mixed symptoms (self-report).

Figure 8 illustrates the course of mixed psychopathological symptoms of the YSR across both groups. Scores of mixed symptoms of the YSR are illustrated in Table 13.

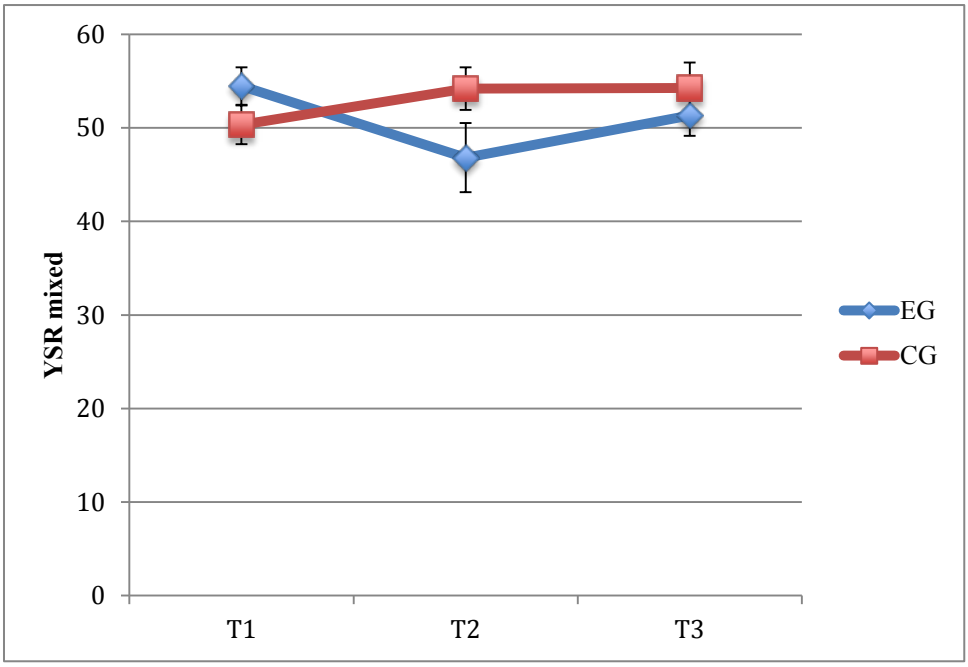

Figure 8. The graph illustrates scores of the YSR scale mixed symptoms for both groups over the three assessment points (T1-T3). Error bars indicate standard errors of mean (SEM).

The self-report questionnaire (YSR) that measures mixed psychopathological symptoms (attention deficits, social problems, schizoid/obsessive behaviour) revealed a significant interaction between time and group ($F_{2,52} = 3.55$, $p = 0.036$) with an almost strong effect size of $\eta_p^2 = 0.120$, indicating effects of the preventive intervention on child's mixed psychopathological symptoms. There was no evidence of a main effect of time on the YSR ($F_{2,52} = 0.63$, $p > 0.05$, $\eta_p^2 = 0.024$). The Bonferroni adjustment for multiple comparisons revealed no significant difference between T1 and T2 (md = 7.65, $p > 0.05$), T1 and T3 (md = 3.15, $p > 0.05$) or T2 and T3 (md = - 4.50, $p > 0.05$) for the EG. The same was true for CG participants (T1 and T2 (md = - 3.87, $p > 0.05$), T1 and T3 (md = - 3.93, $p > 0.05$), T2 and T3 (md = - 0.07, $p > 0.05$)). Standard t-tests as post-hoc tests with a Bonferroni corrected α revealed no significant differences for EG participants between T1 and T2 ($t_{15} = 1.73$, $p > 0.017$, d = 0.55), T1 and T3 ($t_{13} = 1.72$, $p > 0.017$, d = 0.35) or T2 and T3 ($t_{12} = - 1.05$, $p > 0.017$, d = - 0.41). For CG participants the same was true, meaning that no differences between T1 and T2 ($t_{16} = - 1.27$, $p > 0.017$, d = - 0.35), T1 and T3 ($t_{16} = - 1.63$, $p > 0.017$, d = - 0.51) or T2 and T3 ($t_{16} = - 0.31$, $p > 0.017$, d = - 0.05) became significant.

Mixed symptoms (parent report).

Scores of the parent report (CBCL mixed) of offspring's mixed psychopathological symptoms are illustrated in Table 14. The parent report questionnaire (CBCL) that measures mixed psychopathological symptoms in offspring revealed no significant interaction between time and group ($F_{2,52} = 2.72$, $p > 0.05$, $\eta_p^2 = 0.095$). But there was evidence of a main effect of time ($F_{2,52} = 4.71$, $p = 0.013$, $\eta_p^2 = 0.153$), indicating changes of child's mixed psychopathological symptoms independent of group allocation. The Bonferroni adjustment for multiple comparisons revealed a significant difference between the time points T1 and T3 (md = 3.42, $p = 0.023$), reflecting a decrease of mixed psychopathological symptoms independent of group allocation. There was no evidence of significant differences between the other time points (T1 and T2 (md = 1.41, $p > 0.05$), T2 and T3 (md = 2.01, $p > 0.05$)). By using standard t-tests as post-hoc tests with a Bonferroni corrected α level, the significant difference between T1 and T3 ($t_{31} = 2.67$, $p = 0.012$, d = 0.41) was confirmed, reflecting a decrease of mixed psychopathological symptoms over the assessment points. Between other time points, no significant differences were found (T1 and T2 ($t_{32} = 1.71$, $p > 0.017$, d = 0.24), T2 and T3 ($t_{28} = 2.05$, $p > 0.017$, d = 0.28).

Hypothesis 7: Offspring's knowledge of the mood disorder.

Scores of the knowledge questionnaire are displayed in Table 15 for EG, CG and the total sample.

Table 15. The Table illustrates means (M), standard deviations (SD) and sample size (N) separately for EG and CG and the total sample over assessment points T1-T3 of the knowledge questionnaire.

	Group	**EG**			**CG**			**Total**		
	Time	T1	T2	T3	T1	T2	T3	T1	T2	T3
Knowledge	M	33.79	35.71	37.79	32.40	35.40	34.20	33.07	35.55	35.93
	SD	4.66	3.17	8.60	3.33	2.87	3.67	4.02	2.97	6.67
	N	14	14	14	15	15	15	29	29	29

The Shapiro-Wilk test revealed evidence that the majority of collected data were distributed normally within conditions over the three time points. However, the knowledge variable revealed significance at T3 for EG participants (S-W statistic = 0.69, df = 4, $p < 0.001$). Due to the robustness of ANOVA of infringing premises of normal distribution and since all other data were normally distributed within conditions and to different time points (T1-T3), an ANOVA was performed. The Levene-test revealed evidence that variance homogeneity was given. The Mauchly-test showed that sphericity could not be assumed (Mauchly-W = 0.65, df = 2, $p = 0.004$), therefore a Greenhouse-Geisser correction was performed. The measure was equally distributed between groups (see Table 7).

The 2 x 3 mixed ANOVA that was conducted to test whether the prevention program is effective in enhancing child's knowledge of the mood disorder in medium-term revealed no significant interaction ($F_{1.48,39.96} = 1.09$, $p > 0.05$, $\eta_p^2 = 0.039$). There was evidence of a main effect of time on the knowledge questionnaire ($F_{1.48,39.96} = 3.81$, $p = 0.042$, $\eta_p^2 = 0.124$), indicating that, independent of group allocation, the knowledge changed over time. The Bonferroni adjustment of multiple comparisons revealed a significant difference between T1 and T2 (md = -2.46, $p = 0.010$), reflecting an increase of knowledge over the two assessment points independent of group allocation. Between other time points (T1 and T3 (md = -2.90, $p > 0.05$), T2 and T3 (md = -0.44, $p > 0.05$)), no significant difference could be observed. Standard t-tests as post-hoc tests confirmed these findings and showed a significant increase of knowledge from T1 to T2 ($t_{33} = -3.49$, $p = 0.001$, d = - 0.68). Significant differences between other time points (T1 and T3 ($t_{30} = -2.11$, $p > 0.017$, d = - 0.51), (T2 and T3 ($t_{29} = -0.35$, $p > 0.017$, d = - 0.08) could not be demonstrated.

Hypothesis 8: Offspring's perception of parenting style.

The Shapiro-Wilk test revealed evidence to suggest that the majority of collected data were normally distributed within conditions over the three time points. However, also non-normal

distributions were found for single time points within some scales and conditions[6]. When looking on descriptive data, the histograms showed a normal distribution in all scales. Due to the fact that the majority of collected data were normally distributed in all scales and based on the fact that ANOVA statistics are robust against non-normal distribution, the analyses were performed with repeated measures ANOVA.

The Levene-test revealed evidence that variance homogeneity was given for all scales of the ESI questionnaire (support (US), restriction (ES), praise (LS), blame (TS), inconsistency (IK), punishment (SI)) of the primary affected parent as well as of the partner. The Mauchly-test that was performed to check for sphericity revealed evidence that sphericity was given for the scales restriction, blame, inconsistency and punishment of the primary affected parent. The scales support (Mauchly-W = 0.78, df = 2, p = 0.031) and praise (Mauchly-W = 0.72, df = 2, p = 0.011) of the primary affected parent had to be Greenhouse-Geisser corrected.

For the scales support, restriction, blame and inconsistency of the partner questionnaire, sphericity was given. However, the scales praise (Mauchly-W = 0.70, df = 2, p = 0.012) and punishment (Mauchly-W = 0.60, df = 2, p = 0.002) had to be Greenhouse-Geisser corrected. All collected data were equally distributed between groups (see Table 7).

[6] When looking at the primary affected parent, CG participants were not normally distributed within condition at T2 (S-W statistic = 0.88, df = 18, p = 0.025) in the support scale. EG participants were not normally distributed at T3 (S-W statistic = 0.84, df = 13, p = 0.023). In the restriction scale, CG participants were not normally distributed at T3 (S-W statistic = 0.86, df = 18, p = 0.010). All data of the scales praise and blame were normally distributed. The scale measuring inconsistency revealed significance for EG participants at T2 (S-W statistic = 0.71, df = 13, p = 0.001). All data of the punishment scale were normally distributed. When looking at the other caregiver, the support scale showed that CG participants were not normally distributed at T2 (S-W statistic = 0.85, df = 15, p = 0.017). The restriction scale showed that EG participants were not normally distributed within the condition at T1 (S-W statistic = 0.83, df = 13, p = 0.014) and T3 (S-W statistic = 0.78, df = 13, p = 0.004). All collected data of the praise, blame and punishment scale were normally distributed within conditions and to different time points. In the scale that measures inconsistency, EG participants were not normally distributed at T3 (S-W statistic = 0.75, df = 13, p = 0.002).

Primary affected parent.

Scores of all six scales of the ESI questionnaire of the primary affected parents are displayed in Table 16.

Table 16. The Table illustrates means (M), standard deviations (SD) and sample size (N) separately for EG and CG and the total sample over assessment points T1-T3. All six scales (support (US), restriction (ES), praise (LS), blame (TS), inconsistency (IK), punishment (SI)) of the ESI questionnaire (Krohne & Pulasack, 1991, 1995) are listed. Scores are displayed from child's view on the parenting style of the primary affected parent.

ESI	**Group**	**EG**			**CG**			**Total**		
Primary affected Parent	Time	T1	T2	T3	T1	T2	T3	T1	T2	T3
Support (US)	M	37.23	37.46	35.23	34.89	35.00	33.22	35.87	36.03	34.06
	SD	4.85	7.32	10.03	5.09	6.16	8.24	5.04	6.67	8.93
	N	13	13	13	18	18	18	31	31	31
Restriction (ES)	M	15.31	15.15	15.31	16.50	16.06	16.11	16.00	15.68	15.77
	SD	2.50	2.12	2.72	3.50	2.62	3.68	3.13	2.43	3.28
	N	13	13	13	18	18	18	31	31	31
Praise (LS)	M	36.92	35.58	32.50	36.44	35.00	34.22	36.63	35.23	33.53
	SD	6.87	9.24	11.69	5.50	7.99	7.89	5.97	8.36	9.43
	N	12	12	12	18	18	18	30	30	30
Blame (TS)	M	23.08	24.00	23.92	24.56	24.28	24.56	23.94	24.16	24.29
	SD	6.34	5.02	7.25	5.96	5.81	4.74	6.07	5.40	5.82
	N	13	13	13	18	18	18	31	31	31
Inconsistency (IK)	M	17.15	16.62	16.31	18.39	18.44	18.72	17.87	17.68	17.71
	SD	4.49	5.84	4.50	4.39	4.16	5.20	4.40	4.93	4.99
	N	13	13	13	18	18	18	31	31	31
Punishment (SI)	M	9.08	9.00	8.00	9.44	9.06	8.39	9.30	9.03	8.23
	SD	3.23	2.63	3.41	2.79	2.75	2.81	2.93	2.66	3.01
	N	12	12	12	18	18	18	30	30	30

The six 2 x 3 mixed ANOVAs that were conducted separately with each scale of the ESI questionnaire, revealed no evidence of a significant interaction (US, ES, LS, TS, IK, SI with $p > 0.05$) or main effect of time (US, ES, LS, TS, IK, SI with $p > 0.05$) for the primary affected parent, indicating that the hypothesis could not be confirmed.

The other caregiver (partner).

Scores of all six scales of the ESI questionnaire of the other caregiver are displayed in Table 17. The six 2 x 3 mixed ANOVAs that were conducted separately with each scale of the ESI questionnaire also revealed no evidence of a significant interaction (US, ES, LS, TS, IK, SI with $p > 0.05$) for the other caregiver, reflecting that the participation in the program had no effects on parenting style from child's view. There was also no evidence of a main effect of time for any scale of the ESI questionnaire (US, ES, LS, TS, IK, SI with $p > 0.05$), indicating that independent of group allocation, children perceived no differences in parenting style over time.

Table 17. The Table illustrates means (M), standard deviations (SD) and sample size (N) separately for EG and CG and the total sample over assessment points T1-T3. All six scales (support (US), restriction (ES), praise (LS), blame (TS), inconsistency (IK), punishment (SI)) of the ESI questionnaire (Krohne & Pulasack, 1991, 1995) are listed. Scores are displayed from child's view on the parenting style of the other caregiver.

ESI	**Group**	**EG**			**CG**			**Total**		
Partner										
	Time	T1	T2	T3	T1	T2	T3	T1	T2	T3
Support (US)	M	38.00	37.38	35.62	35.93	35.73	33.47	36.89	36.50	34.46
	SD	5.16	7.75	11.03	4.65	6.53	5.94	4.92	7.04	8.57
	N	13	13	13	15	15	15	28	28	28
Restriction (ES)	M	16.23	14.92	14.46	17.93	16.93	17.00	17.14	16.00	15.82
	SD	2.83	2.90	1.94	3.92	2.69	5.44	3.50	2.92	4.32
	N	13	13	13	15	15	15	28	28	28
Praise (LS)	M	37.62	37.23	34.92	36.60	34.87	34.53	37.07	35.96	34.71
	SD	5.36	8.42	11.63	5.96	8.94	8.49	5.61	8.62	9.88
	N	13	13	13	15	15	15	28	28	28
Blame (TS)	M	24.08	21.92	22.69	27.87	27.27	27.87	26.11	24.79	25.46
	SD	7.59	4.79	7.11	8.21	7.35	6.85	8.02	6.75	7.33
	N	13	13	13	15	15	15	28	28	28
Inconsistency (IK)	M	17.38	16.15	15.54	20.43	19.86	19.79	18.96	18.07	17.74
	SD	3.73	4.76	4.43	5.29	3.37	5.04	4.78	4.44	5.14
	N	13	13	13	14	14	14	27	27	27
Punishment (SI)	M	9.25	8.67	7.75	10.13	9.07	8.93	9.74	8.89	8.41
	SD	2.45	2.67	3.62	3.00	2.02	3.41	2.75	2.29	3.49
	N	12	12	12	15	15	15	27	27	27

Feedback Evaluation

After each session of the program, parents and children were asked to complete feedback evaluation forms. Figure 9 illustrates the feedback provided by parents and children averaged across all sessions. Mean values, standard deviations, minimum and maximum scores as well as the number of feedback evaluations (N) that were filled out after single sessions are shown in Table 18. Parents' as well as children's scoring across all the variables of interest (comprehensiveness, active participation, comfort during sessions, understanding training sheets, usefulness of training sheets) ranged from 3.99 to 4.63 on a total scale of 1 – 5 (1 = not at all – 5 = very good).

As visible in the graph, children and parents gave quite similar feedback in most variables. Study contents were understood from both very well (parents: M=4.63, SD=0.62; children: M=4.49, SD=0.71). This is reflected in the variable comprehensiveness that reached highest scores. Lowest scores were reached in the variable active participation with an average of 4.00 (parents: M=4.01, SD=0.75; children: M=3.99, SD=1.08), which is still high. In the variable comfort, children reported to feel slightly more comfortable during sessions compared to parents (parents: M=4.27, SD=0.74; children: M=4.47, SD=0.82), but again, scores are high and reflect an overall positive feedback. On average, both understood the training sheets well (parents: M=4.44, SD=0.77; children: M=4.37, SD=0.91) and children rated training sheets to be more useful than parents did (parents: M=4.14, SD=0.81; children: M=4.31, SD=0.83).

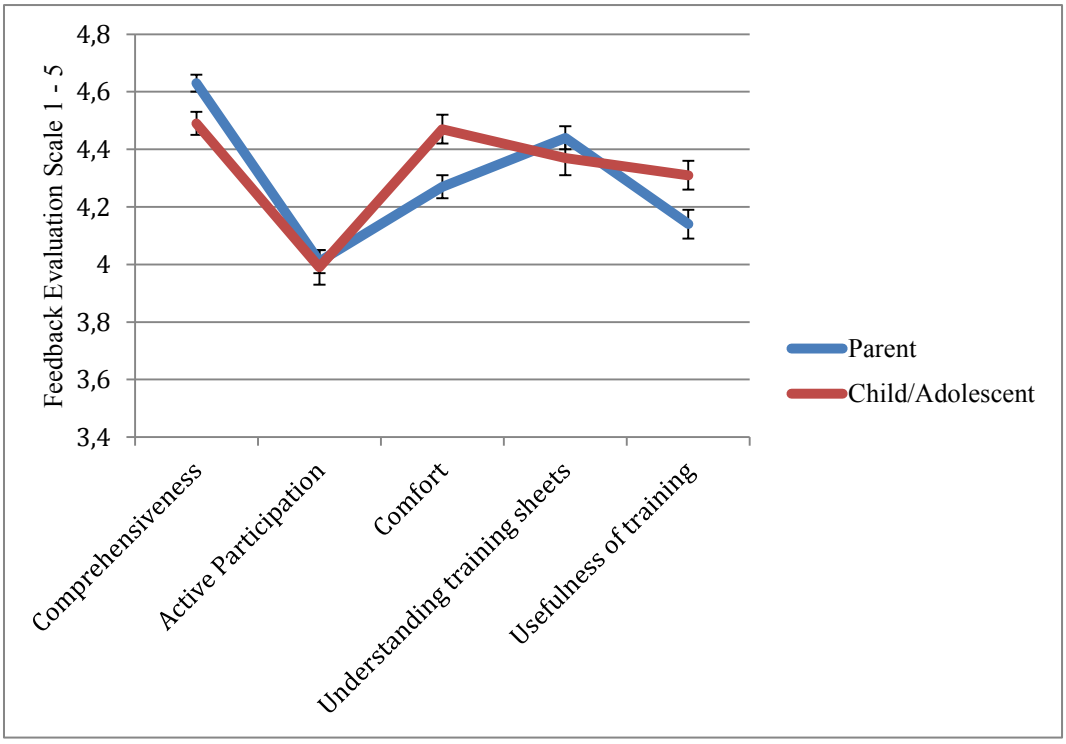

Figure 9. The graph illustrates scores of the feedback evaluation that was filled out after each session from all attendant participants. Evaluation scored from 1(= not at all) to 5 (= very good). Error bars indicate standard errors of mean (SEM).

Table 18. The Table illustrates means (M), standard deviations (SD) and sample size (N) of all participants who passed the program (EG) and evaluated single sessions. As visible, participants evaluated the active participation with lowest scores that represented still good ratings. Highest scores were reached in the scale comprehensiveness.

Feedback evaluation	**Comprehensiveness**			**Active participation**			**Comfort**			**Understanding training sheets**			**Usefulness of training sheets**		
	Parent	Child	Total	Parent	Child	Total	Parent	Child	Total	Parent	Child	Total	Parent	Child	Total
Mean	4.63	4.49	4.56	4.01	3.99	4.00	4.27	4.47	4.37	4.44	4.37	4.41	4.14	4.31	4.23
SD	0.62	0.71	0.67	0.75	1.08	0.92	0.74	0.82	0.78	0.77	0.91	0.84	0.81	0.83	0.82
Min-Max	1-5	1-5	1-5	2-5	1-5	1-5	1-5	1-5	1-5	1-5	1-5	1-5	1-5	1-5	1-5
N	339	295	634	339	295	634	341	295	636	303	254	557	314	287	601

Discussion

Summary of Findings

The current work provides first international results on the effectiveness of the GUG-Auf intervention program in terms of reducing the psychopathological risk or preventing the development of psychopathological symptoms in children and adolescents. The prevention program is an evaluation of the Raising Healthy Children program by Compas et al. (2009, 2011), is conceptualized as family-group setting and utilizes psychoeducation as well as cognitive-behavioural elements with the aim to reduce the developmental risk of depression and general psychopathology of offspring with a parent suffering from depression. To gain further insight into the role of parental depression on psychopathological outcomes in their offspring, my first part of the analysis focused on explaining variability in the overall sample prior to the intervention. I first investigated offspring's mental health at baseline and examined whether offspring's depressive and general psychopathological symptoms have a relation to parental mental health. Additionally, I investigated how stressful or aversive experiences affect offspring's well-being, but also whether the socio-economic background plays a role in observed differences in depressive and general psychopathological symptoms. The main focus of this work was the evaluation of the intervention outcome, i.e. estimating the efficacy of the intervention program in reducing and preventing the depressive and general psychopathological risk of offspring with an affected parent, and further to test the effectiveness of the program in enhancing children's knowledge of the disorder and to test whether positive changes in parental parenting can be observed. Lastly, data about the general acceptance of the intervention program were presented.

Interpretation of Findings

Baseline outcomes.

Parental depression and offspring's mental health.

Previous literature shows that parental depression impacts offspring's mental health (Lieb et al., 2002). Children of affected parents develop the disorder earlier, more seriously and have a higher recurrence rate than children from mentally healthy parents (Goodman, 2007). Children with one affected parent have a risk of 50% to develop the disorder (Beardslee & Wheelock, 1994), whereas if both parents are affected, the developmental risk increases to up to 70% (Robins & Regier, 1991). This high percentage allows assuming positive correlations between offspring's mental health and parental depression scores as well as between offspring's mental health and the number of depressive episodes.

The investigation of parent depression on offspring's depression had the purpose to show that children of depressed parents differ already at baseline on sub-clinical depression level, which stands in relation to parent depression severity and the number of depressive episodes the parent experienced. Therefore, in the current sample offspring's mental health was correlated with parental depression severity (primary affected parent's depression scores measured with the BDI-II questionnaire) and the number of depressive episodes. Surprisingly, there was no evidence of a correlation between parental depression severity and offspring's depressive symptoms. The same was true for the number of depressive episodes and offspring's depressive symptoms. The lack of findings could be explained by the rather small variation in BDI-II and DIKJ scores between children, which in turn could be explained with the fact that only children were included who were either free of depressive symptoms or showed only depressive symptoms on sub-clinical level, so that variations between children were rather small. However, it is also possible that parental depression severity and the number of depressive episodes simply do not predict child depressive outcomes.

Previous literature (Keller et al., 1986; Orvaschel, Walsh-Allis, & Ye, 1988) emphasised that children of depressed parents have generally higher prevalence rates for other psychopathological symptoms compared to children of non-depressed parents. I suggested that children do not necessarily develop a depression by itself but rather general psychopathological conspicuities could occur. Therefore I also examined whether offspring of depressed parents differ already at baseline on sub-clinical level in their general psychopathology.

This was also found in current data: offspring's self-reports of externalising symptoms correlated positively with the number of depressive episodes of the primary affected parent ($r = 0.35$, $p < 0.05$) such that a higher number of depressive episodes were related to higher levels of aggressive or dissocial behaviour in offspring from offspring's view. However, there was no evidence that self-reports of internalising and mixed psychopathology correlated with the number of episodes of the depressed parent. It might be that a parent with more depressive episodes makes a child more vulnerable to externalising problem behaviour, because children are exposed to e.g. more inconsistent parental behaviour (Goodman & Gotlib, 1999) as well as by poor family functioning (Seeley et al., 2009), which might influence child's behaviour patterns in a negative manner. It might also be that children want to arrest attention of their parents which they probably also get by being more aggressive or dissocial. Surprisingly, children did not state internalising symptoms in relation to parent depression severity or the number of depressive episodes. It is possible that externalising symptoms were already so strong pronounced (although only on sub-clinical level), making it easier to identify those compared to internalising symptoms on sub-clinical level.

Parent reports of general psychopathology of offspring did not correlate with the number of depressive episodes of the parent. Also BDI-II scores of the parent did not correlate with child general psychopathology. It is known that children stronger assess own internalising symptoms compared to parents, whereas discrepancies in the assessment of

externalising symptoms are not as large (Lohaus & Vierhaus, 2014). The discrepancies between parents' and offspring's view in the current study could be explained in the way that depressed parents might trivialise offspring's symptoms and do not want to see that children already developed some psychopathological symptoms on sub-clinical level. It is also possible that parents are, because of depression, so preoccupied with themselves that they do not even recognise that children developed some psychopathological symptoms on subclinical level. However, it is also possible that parents underestimate child's symptoms, because they compare the level of their own psychopathology with child's psychopathology.

In summary, although preliminary evidence emerged that the number of depressive episodes of the parent is correlated with child's externalising symptoms, these findings were not replicated across all measures (see above).

Aversive events and offspring's mental health.

A further investigation was whether the number of stressful events a child of a depressed parent has experienced influenced their depressive symptom severity. It is known that the risk of developing depressive symptoms during childhood and adolescence is increased by stressful life experiences (Bouma, Ormel, Verhulst, & Oldehinkel, 2017) and that parental depression in itself can already be considered as stressful life event (Goodman & Gotlib, 1999). Stress experiences in this relation might be due to changed parental role models, reflected by inconsistent parental behaviour (Goodman & Gotlib, 1999) or by poor family functioning (Seeley et al., 2009). Children of affected parents also experience other stress factors, induced by environmental loads (Goodman & Gotlib, 1999) as chronic problems or marital problems (Laucht et al., 1994). Findings of Hammen and Brennan (2003) underline the elevated depression risk of offspring with a depressed parent. Researchers showed that maternal depression severity and family stress significantly interact with each other, and that offspring of depressed mothers are exposed to more stress factors than offspring of non-depressed mothers, which in turn elevates offspring's depression risk.

But also other environmental factors such as moving away, sickness, an injured pet, struggle or death of a loved person are factors that increase the stress level of a child. These negative events were measured with the CASE questionnaire (Allen et al., 2012), to test whether offspring who experienced many stressful life events reached higher scores in depression and general psychopathological scales compared to those who experienced only a few or no stressful life events.

Contrary to expectations, in the current sample there was no evidence that the number of stressful life events was associated with offspring's depressive symptoms. Neither the BDI-II/DIKJ composite, nor separate analyses of BDI-II and DIKJ revealed significance. As in the previous hypothesis, it is possible that the lack of findings is a result of the rather small variation in the BDI-II and DIKJ scores between children and adolescence. But it is also possible that stress experiences have to last longer to affect specifically offspring's depressive symptoms.

However, the current findings demonstrated significant effects of stressful life events in offspring's self-reports' of general psychopathological symptoms (all scales, internalising; externalising, mixed) (all *ps* < 0.01). Effect sizes were large and ranged from $\eta_p^2 = 0.231$ to $\eta_p^2 = 0.281$. Post-hoc tests revealed that when children and adolescents experienced a few (one/two) stressful events during the last year, mean scores of internalising, externalising and mixed symptoms were significantly lower compared to children and adolescents who experienced many (five or more) stressful events. Externalising and mixed scales also revealed significant differences between participants who experienced several (three/four) and many (five or more) stressful life events. However, the internalising scale of self-reports revealed no significant difference between participants who experienced several (three/four) and many (five or more) stressful life events. Also no significant differences were observed in self-reports between none and a few, none and several, and none and many stressful life events. Parent reports of general psychopathology of offspring did not reveal any significance,

neither for internalising or externalising nor for mixed symptoms. The findings show that parents and children can differ in the way how they assess offspring's psychopathology, which is known particularly for child's internalising symptoms, as anxiety or social withdrawal (Cantwell et al., 1997; Ihle et al., 2004), as was already discussed in the previous section. The current data showed that child's and parents' perspective can also differ in offspring's externalising symptoms. It is possible that depressed parents are so stressed that they do not recognise sub-clinical general psychopathological symptoms in their children. One other explanation could be that although parents recognise these changes, they trivialise and refuse to believe the situation. The trivialisation could be linked to feelings of guilt, making it more difficult for parents to accept the worsening of their offspring's mental health.

Interestingly, children stated differences in general psychopathological outcomes between a few and many or between a few and several stressful life events, but not between none and few, none and several or none and many stressful life events.

To my knowledge, only a few studies (Adrian & Hammen, 1993; Bouma, Ormel, Verhulst, & Oldehinkel, 2008; Hammen, 1988) have yet investigated the effects of stressful life events on depression and other psychopathological outcomes of children of depressed parents. The current thesis, which investigated *the effect of the number of stressful life events on child's depressive and general psychopathological outcomes*, contributes to previous findings and shows that children recognise differences in own general psychopathology already at baseline, even though only *children without a diagnosis* were included in the study. Because children of depressed parents are at heightened risk for any psychiatric disorder, it is a first important step to make these children (but also their parents) aware of potential risk factors. This was also one aim of the GUG-Auf prevention program: making families aware of stressors in their daily life and conveying appropriate coping strategies. Particularly children who experienced more stressful events could profit from such a program.

However, one point should also be mentioned and discussed: not only the number, but also the impact of stressful life events plays a role in offspring's developmental risk of a psychiatric disorder. Stressful experiences are subjective experiences. It is doubtful whether moving, mobbing and death of a loved person are comparably stressful experiences, and whether not only one stressful experience with a strong impact, such as a divorce of parents or the death of a loved person is related to stronger psychopathological outcomes in offspring than the sum of a few events with a smaller impact. Future research should therefore also consider the impact as well as the number of stressful experiences to show effects on child's psychopathology at baseline.

Differences in child's depression and general psychopathology in association with SES.

When looking at the socio-economic status, existing literature suggests that the familial SES is associated with offspring's wellbeing (Ajilchi & Kargar, 2013; Anli & Karsli, 2010; Reising et al., 2013; Topham et al., 2010) and that high and very low familial SES (compared to middle-class) are linked to more frequent mental health problems in offspring, reflected in significantly stronger pronounced psychopathological symptoms in children (Anli & Karsli, 2010). The current work investigated in the question *whether children of depressed parents are affected by SES*, which could be reflected already at baseline on sub-clinical level in depression and general psychopathological outcomes.

Unfortunately, most families that have been recruited had either a very high or middle socio-economic background and only two families with low socio-economic background participated in the study. The reason therefore is that it was not planned originally to investigate the SES, because otherwise the recruitment strategy would be handled differently.

With regard to child's depressive and general psychopathological outcomes, a comparison was therefore useful between middle vs. high SES. Based on these findings, I

expected that child's depressive and general psychopathological symptoms are significantly stronger pronounced in families with high SES compared to families with middle SES.

Unexpectedly, no significant differences in offspring's mental well-being (self- and parent reports of depression and general psychopathology) were found. The results indicate that SES is not associated with child's depressive symptoms or child's general psychopathology. It is possible that parental depression plays such a big role in offspring's life that the additional stress, which is associated with high SES, plays a too little role in these children. One other explanation is based on findings of other studies: the majority of studies (Gilman et al., 2002; Huberty, 2012; Luo & Waite, 2005; Wirback et al., 2014) demonstrated only for children with low SES an association with offspring's depression risk in later life. Only the study by Anli and Karsli (2010) demonstrated that additionally to a low SES, also a high SES is (compared to middle SES) associated with an increased risk of child's psychopathology. It is possible that a replication of the study by Anli and Karsli (2010) with a different sample would lack in confirming previous findings, and that only in children with low SES psychopathological symptoms would be found. But it is also possible that the variation in depression and general psychopathological scores is too small to show that child's psychopathology is associated with high SES. One other explanation could be that cultural differences play a role. The study of Anli and Karsli (2010) was conducted with a Turkish sample, whereas the current study was conducted with a German sample. It is conceivable that discrepancies between my findings and the findings of Anli and Karsli (2010) are linked to different lifestyles of the two societies.

Summary of baseline outcomes.

In summary, although there was no evidence that parental depression severity was associated with child depression severity or child psychopathology, there was preliminary evidence that the number of depressive episodes of the primary affected parent is linked to child's externalising psychopathological symptoms from child's perspective. Other scales did not

correlate with the number of depressive symptoms, neither in self- nor in parent reports. It is possible that a higher recurrence rate of parental depression makes the child more vulnerable for externalising problem behaviour. That child's sub-clinical depressive symptoms did not correlate with parents' depression severity or number of depressive episodes might be due to the rather small variation in the BDI-II and DIKJ scores, because only children without a diagnosis were included in the study. However, it is also possible that parental depression severity and the number of depressive episodes simply do not predict child depressive outcomes.

Although there was no evidence that the number of stressful life events experienced by offspring in the last twelve months is related to child's depression severity, there was preliminary evidence that the number of stressful life events is negatively linked to offspring's general psychopathological outcomes. This was reflected in all three scales of offspring's self-reports, however, not in parent reports. That children and parents differ in their view could be explained in the way that parents trivialise offspring's symptoms or even do not recognise them because they are preoccupied with themselves, whereas children might better assess the relation between stressful life events and their own mental health status.

When looking on baseline differences in child's depression and general psychopathology in association with SES, the current study could not demonstrate that offspring's depression or general psychopathological outcomes are related to SES. It is possible that parental depression plays such a big role in children's lives, so that the additional stress, which is related to high SES, plays a smaller role in these children. But it is also possible that cultural differences are the reason, why findings differ to the study of Anli and Karsli (2010).

Intervention outcomes.

Outcomes of the GUG-Auf prevention program on child's depressive symptoms.

Because of the high risk level for children of depressed parents, researchers developed and evaluated different prevention programs with the aim to reduce incidence rates of the high-risk individuals. The first evidence that prevention programs are effective in reducing depressive and general psychopathological symptoms in offspring of depressed parents was reported by Beardslee and colleagues (1997) with the Family Talk Intervention (FTI). This is one of the *four prevention programs* that have been developed particularly for *depression prevention in offspring of depressed parents*. Three programs (FTI (Beardslee et al., 2003, 2007; Christiansen et al., 2015; Punamäki et al., 2013; Solantaus et al., 2010), Coping with Depression (Beardslee et al., 2013; Clarke et al., 2001; Garber et al., 2009, 2016) and Raising Healthy Children (Clarke et al., 2001; Compas et al., 2009, 2011, 2015; Forehand et al., 2012; McKee et al., 2014)), reported promising findings. The Parenting Training (Sanford et al., 2003) showed at least positive trends favouring the preventive intervention (vs. waiting-list). E.g. Compas and colleagues (2009, 2011) demonstrated that children who passed the RHC program vs. an active control had significant lower incidence rates in medium- (twelve months follow-up (8.9% vs. 20.8%)) and long-term (24 months follow-up (14.3% vs. 32.7%)).

The current work evaluated the RHC program by Compas et al. (2009, 2011), which so far has only been replicated within the research group (Forehand et al., 2012; McKee et al., 2014). But with this work, the RHC program is evaluated and replicated for the first time on an international level. Moreover, in Germany no program for this target group has yet been replicated as randomised controlled trial. This program was chosen because of the positive outcomes that have been reported and because of the combination of different approaches in comparison to other preventive interventions (Beardslee et al., 1997, 2003, 2007, 2013; Clarke et al., 2001; Garber et al., 2009).

The current work focused on the questions whether children with a parent suffering from depression who took part in the prevention program are less likely to develop a depression and have lower scores in scales measuring depressive and general psychopathological symptoms compared to offspring of affected parents who had no comparable treatment during the same time frame and were allocated to a waiting-list control condition. The hypothesis was that already in offspring‘s self-reports, lower depression scores will be found immediately after the intervention (six months post baseline) as well as nine months post baseline. Because too little data of offspring's self-reports of depression were available to test prevention effects at nine months, I report the analysis of the six months outcomes.

Results revealed no significant difference between the intervention and control group with regard to offspring's changes in self-report depressive symptoms over time ($p > 0.05$). This remained the case when the depression composite (BDI-II/DIKJ) was analysed as well as when the measures were analysed separately. It is possible that the parental depression status influences offspring's outcomes. Beardslee et al. (2013) argued that offspring's mental health is influenced by the current status of parental depression and when parents are remitted, the effects of the intervention are stronger. In our sample, 67.5% of primary depressed parents were currently depressed, which could be taken as one possible reason why no effects could be demonstrated in self-reports. However, the sample wasn't large enough to test whether status of parental depression (current or remitted) predicted outcomes. On the other hand, a six-month follow-up is not a long time period to show a reduction of depressive symptoms. Also Compas and colleagues (2009) did not find significant differences in offspring's self-reports of depressive symptoms at six months between the intervention and active control condition. Effects became visible in medium- (twelve months) and long-term (24 months), reflected in lower incidence rates of children who passed the preventive intervention (Compas et al., 2009, 2011). One other point is that the current work included only *children and*

adolescents without a diagnosis. It is possible that these children do not necessarily develop a depression in the assessment period rather to a later point of time, which could also be a reason why no effects were visible within the time frame.

From the long-term follow-up (15 months (T4)), data of the standardized semi-structured K-Dips interview of Unnewehr et al. (2008) were collected (n = 40), which give an overview of the developmental course of depression from both groups. I expected a significantly lower proportion of children in the EG to be depressed compared to children in the CG. However, no significant differences could be observed. When looking on descriptive data, it became visible that no child of the EG developed a depressive disorder, whereas 19.0 % (n = 4) of offspring from the waiting-list control condition developed a depression. The analysis should be conducted again after all participants passed the 15 months follow-up to show that depression is in long-run preventable, as Compas et al. (2009) demonstrated in the twelve months follow-up (EG: 8.9% vs. CG: 20.8% with d = 0.42).

In conclusion, the prevention program showed no significant differences in depression outcomes, neither at six nor at 15 months follow-up. However, descriptive data demonstrated a first trend that children who passed the program are less likely to develop a depression compared to children from the waiting-list.

Outcomes of the GUG-Auf prevention program on child's general psychopathology.

Children from depressed parents do not necessarily develop a depression instead other psychiatric disorders may be developed. Therefore, general psychopathological symptoms were analysed, separately for offspring's self-reports by utilizing the YSR (Döpfner et al., 1998) and for parent reports by using the CBCL (Döpfner et al., 1994) questionnaire. Separate analyses were conducted with the scales that measure internalising (social withdrawal, somatic complaints, anxiety, depression), externalising (aggressive, dissocial behaviour) and mixed (attention deficits, social problems, schizoid/obsessive behaviour) psychopathological symptoms. The YSR as well as the CBCL were also utilised by Compas and colleagues (2009,

2011), so that the breadth of measures and effect sizes were comparable for offspring's psychopathology. By using self-reports of offspring's internalising and externalising psychopathological symptoms (YSR), Compas et al. (2009) found a significant decrease of internalising symptoms directly after the eight weekly sessions, favouring the prevention program. The significant difference between intervention and the active control remained stable at six, and twelve months follow-ups. Externalising psychopathological symptoms also decreased in short-term (six months follow-up), but effects could not be demonstrated in the twelve months follow-up. From parents' view (CBCL), offspring's externalising symptoms decreased significantly at six months favouring the prevention program. But significant changes of internalising symptoms could not be demonstrated.

The current study demonstrated in offspring's self-reports significant interaction effects on internalising ($p < 0.05$, $\eta_p^2 = 0.162$) and mixed ($p < 0.05$, $\eta_p^2 = 0.120$) scales, favouring the family prevention program. However, after correction for multiple comparisons with the Bonferroni method for both scales, no significant differences between any of the time points (T1-T2, T1-T3, T2-T3) could be found. Due to the fact that the Bonferroni correction is a very conservative measure (Kähler, 2008), the analysis may have lacked the statistical power to detect any differences. This was confirmed by conducting the test with standard t-tests as post-hoc tests. Significant differences – at least – for internalising symptoms of the self-report questionnaire (YSR (Döpfner et al., 1998)) could be demonstrated for EG participants between the two time points T1 and T2 ($t_{15} = 2.68$, $p < 0.017$), with a decrease of symptoms and a medium effect size of d = 0.49. Compas and colleagues (2009) found for this scale at six months follow-up a smaller effect of d = 0.37. That effects diminished from six to nine months follow-up is not surprising. During the prevention program (six months period), families had regular meetings and regular training tasks. After the program finished, families probably trained less and also forgot some program contents over time, so that intervention effects diminished over the assessment period.

The fact that no significant difference was found for mixed psychopathological symptoms after Bonferroni correction and conduction of standard t-tests could be explained by the large variance in the scale. Therefore, results should be interpreted carefully.

However, in the scale that measures externalising psychopathological symptoms, neither a significant interaction nor a significant main effect could be demonstrated in short- or medium-term. These findings are contrary to the findings made by Compas et al. (2009), who at least demonstrated a significant reduction of externalising psychopathological symptoms from offspring's view in short-term.

When looking at parent reports about offspring's general psychopathology, no significant interaction was found. But for offspring's internalising symptoms a significant main effect of time ($p < 0.01$, $\eta_p^2 = 0.196$) was observed, indicating that independent of group allocation, changes in offspring's internalising psychopathological symptoms occurred over time. After correcting with Bonferroni, it became visible that parents reported a significant decrease in offspring's internalising symptoms from baseline to the nine months follow-up. Standard t-tests confirmed these findings with a medium effect size of $d = 0.55$. Moreover, t-tests even showed significant differences between T1 and T2 with $d = 0.42$. For mixed psychopathological symptoms a main effect of time ($p < 0.05$, $\eta_p^2 = 0.153$) was also found, indicating that independent of group allocation, changes in offspring's mixed psychopathological symptoms occurred over time. The Bonferroni correction revealed significance between assessment points T1 and T3 ($p = 0.023$). By using standard t-tests, a significant difference between the two time points could be demonstrated with a decrease of mixed psychopathological symptoms over time and an effect of $d = 0.41$.

However, here the scale that measured offspring's externalising psychopathological symptoms also revealed neither a significant interaction nor a significant main effect. Compas et al. (2009) demonstrated in the externalising scale from parents' view at least significant benefits for participants who passed the prevention program in short-term.

Interestingly, parents and children differed in their views on whether the program showed benefits for participants who passed the program or not. While from self-reports it became visible that internalising and mixed psychopathological symptoms decreased favouring the prevention program, from parents' perspective those symptoms decreased independent of group allocation. It is possible that families who were willing to participate in the study talked more about depression within the family independent of group allocation, so that this open dialogue conveyed parents the impression that offspring can better deal with the parental disorder, because e.g. children get the understanding that it is not their fault that parents are depressed, which is reflected in a symptom decrease independent of participation. Because it is known that children's ability to assess their own internalising psychopathological symptoms is better compared to parents' ability, and that discrepancies between parents' and offspring's view can occur (Cantwell et al., 1997; Ihle et al., 2004), I expected to find a greater coherence between offspring's reports about their own psychopathological symptoms and positive effects of the GUG-Auf prevention program than between parent reports and positive effects of the prevention program. This was also confirmed by the current findings.

In conclusion, the prevention program demonstrated benefits on child's internalising and mixed psychopathological symptoms, but only from child's perspective. Although parents also reported a decrease of those symptoms, they reported the decrease independent of group allocation. Externalising symptoms showed no change, neither from offspring's nor from parents' perspective.

Outcomes of the GUG-Auf prevention program on child's knowledge.

The further analysis focused on one other relevant outcome that was conveyed through the GUG-Auf prevention program, namely the knowledge of depression. In general, the level of knowledge of a parental disorder can either act as stress or resilience factor, depending on whether the child has a low or a high knowledge level (Lenz, 2005). A high knowledge level

increases the resilience. The fact that the knowledge level of depression can be increased in youngsters, at least in short and medium-term, was already demonstrated by Allgaier et al. (2011) and Schiller et al. (2014). That is why programs that target children of depressed parents use psychoeducation as one element of the prevention program like the CWD (Beardslee et al., 2013; Clarke et al., 2001; Garber et al., 2009) or the RHC (Compas et al., 2009, 2011, 2015; Forehand et al., 2012; McKee et al., 2014). The FTI (Beardslee et al. 1997, 2003, 2007), for example, is solely based on psychoeducation, and although only this method was used, it still showed that incidence rates of children developing depression were reduced and child's knowledge level about parental disorder increased.

In summary, knowledge acquisition is a relevant method in order to reduce the depression risk in children at risk. Therefore prevention programs which focus on depression prevention in children with depressed parents use psychoeducation as one module. However, to my knowledge, only a few studies (Beardslee et al., 1997, 2003, 2007; Christiansen et al., 2015) have yet specifically investigated the question of how effective prevention programs are for children of depressed parents in improving child's knowledge of depression. These studies were solely based on the FTI prevention program. To my knowledge no other prevention program focused on the knowledge acquisition of children. Whereas Beardslee et al. (1997, 2007) and Christiansen et al. (2015) found a significant higher level of child's knowledge favouring the prevention program, Beardslee et al. (2003) reported for children of the intervention program as well as for children of the active control group a significant better understanding of parental disorder.

The current thesis examined this question by testing whether the GUG-Auf preventive intervention improved offspring's knowledge of depression in short- and medium-term. Strongest effects were expected to be found immediately after the intervention with significant benefits for children who took part in the preventive intervention, whereas the knowledge level of control participants was expected to remain relatively stable during the

assessment period, because they did not receive any intervention or active control in form of written information about the disorder.

Contrary to expectations, there was no indication that children and adolescents in the experimental group had benefits in knowledge acquisition across the six and nine months assessment points compared to participants from CG. However, independent of the program, the knowledge level of both groups increased from baseline to the six months follow-up with a medium effect of d = - 0.68. This knowledge increase independent of group allocation could be explained in the way that families who decided to participate talked more about the disorder within the family compared to before, which became visible at least in short-term.

The reasons why no differences between groups occurred, could also be explained by the rather small sample size (n = 29 with EG: 14, CG: 15) and a too short assessment period, so that true effects could not be observed and previous promising findings (Beardslee et al., 1997, 2007; Christiansen et al., 2015) could not be confirmed.

Outcomes of the GUG-Auf prevention program on child's perception of parenting style.

A further aim of the study was to investigate whether offspring who received the intervention program perceived parenting style (across the assessment points at six and nine months) more positively compared to offspring from the waiting-list control condition from whom no changed perception was expected. From literature it is known that the parenting style is influenced by parental mental health (Lipps et al., 2012; Radziszewska et al., 1996) and that parenting style has an influence on offspring's wellbeing (Ajilchi & Kargar, 2013; Lipps et al., 2012; Radziszewska et al., 1996; Topham et al., 2010). Specifically parenting in only one direction (overprotection vs. rejection) increases offspring's psychopathological risk, whereas a positive parenting style that balances between parental control and emotional warmth has protective effects (Anli & Karsli, 2010).

One aim of the current study was to modify dysfunctional parenting strategies of depressed parents into positive parenting. The GUG-Auf prevention program did therefore not only provide separate sessions for children and adolescents in which they learned, e.g., how to cope with negative thoughts, parents also received separate sessions in which information about the different parenting styles and their effects on offspring's behaviour and wellbeing were transferred. The session contents specifically focused on the individual parenting style and on how positive parenting can best be implemented.

Although some preventive interventions (Compas et al., 2009, 2011; Sanford et al., 2003) already used strategies to change dysfunctional parenting of depressed parents, to my knowledge, no study that focused on children of depressed parents explicitly investigated whether the parenting style was really modified in a positive manner by a prevention program and whether these changes of parenting are perceived by their children in short- and medium-term. The current study examined exactly this question and measured changes in parenting style with the ESI questionnaire (Krohne & Pulasack, 1991, 1995) at baseline as well as six and nine months after baseline. Thereby children and adolescents stated at all three assessment points how they perceived parenting of both parents in the six dimensions support, restriction, praise, blame, inconsistency and punishment. I assumed that children from the intervention program perceived and therefore will also report positive changes of parenting, whereas I did not expect a changed perception and different reports from offspring of the waiting-list control condition. Particularly in the dimension praise I expected significant changes in EG compared to CG participants. Analyses were conducted separately for the depressed parent and for the other caregiver in case he or she participated in the study.

With regard to the primary affected parent, not one of the six scales of the ESI questionnaire (Krohne & Pulasack, 1991, 1995) revealed a significant interaction, indicating that primary affected parents who took part in the prevention program did not show a changed parenting style compared to primary affected parents from the waiting-list control condition.

At least children and adolescents did not perceive the parenting style of their parents as different compared to before. Also no main effect was found, showing that also independent of the prevention program, no changes in parenting style could be observed by children over the assessment period.

Due to the fact that parents' regular task was to praise their children whenever children showed desirable behaviour and to support them with daily life concerns, these findings are surprising. Particularly, as parents reported during sessions that they praise their children much more compared to before. At least as long as families participated in the study, had regular trainings within the sessions and also training tasks in form of homework (such as praising children, spending daily positive time with each other and planning one family activity per week), positive outcomes on all scales were expected. As soon as the regularity of training was not given any more, a diminution of effects was assumed. However, no changes were found. It is possible that the implementation of new parenting strategies takes depressed parents longer than the assessment period really is. It might also be that children are used to fluctuations in the parenting style, so that their tolerance for different parenting strategies is so high that they do not recognise positive changes. But it is also possible that the parenting style simply did not change, although parents reported that it changed, so that children could not recognise any positive changes.

When looking at the other caregiver, no scale revealed significance either, reflecting that children did not perceive the parenting style of the other caregiver differently over the assessment period of nine months compared to children from the waiting-list control condition. It is possible that mentally healthy parents are generally more consistent in their parenting style, so that slight differences in behaviour were not recognised by offspring.

It is also possible that no significant effects were found because the sample size of parents (n = 30-31) and partners (n = 27-28) who took part in the prevention program and

from whom data of the ESI questionnaire were available, is yet too small and effects will become visible as soon as all families from T1 passed the assessment period of nine months.

Summary of the efficacy of the GUG-Auf prevention program.

In summary, intervention outcomes which reflect the effectiveness of the *GUG-Auf prevention program* did not demonstrate significant differences between participants of experimental and control groups regarding depressive symptoms. Neither the self-report measures nor the semi-structured interviews that were conducted at 15 months post baseline revealed significance. It is possible that the parent depression status influences child's depression outcomes. It is known that intervention effects are stronger when parents are remitted. And since in the current sample 67.5% of primary affected parents were currently depressed, this could be one possible explanation why no significance was reached. Further, the current study solely included children without a diagnosis, whereas other studies also included children with a history of depression. For children without a diagnosis, the probability of a depression onset within 15 months is lower than for children who had previous diagnoses. However, based on the interviews, at least first trends became visible, reflected in a depression onset in four cases of CG and zero cases of EG from a total of 40 participants.

When looking at general psychopathological symptoms, the prevention program showed positive effects on internalising and mixed psychopathological symptoms, visible in offspring's self-reports. Internalising symptoms like social withdrawal and anxiety significantly decreased for EG participants from baseline to the six-month follow-up, favouring the preventive intervention, whereas for CG participants such differences could not be demonstrated. That the positive course of symptoms could not be found at nine-month follow-up could be explained in the way that there was no regular training anymore and the acquired strategies were less used by children. Parent reports did not confirm child's self-reports. Instead, parents reported that independent of the program, offspring's internalising

and mixed psychopathological symptoms like social problems or compulsive behaviour decreased from baseline to the nine-month follow-up. It is possible that parents who participated in the study (independent of allocation) talked more within the family about depression and its effects on the family system, so that the open dialogue gave parents the impression that children come along better with the parental disorder, reflected in a decrease of own symptoms independent of participation. No significant differences could be observed in offspring's externalising psychopathological symptoms, neither from offspring's nor from parents' perspective.

When looking at knowledge acquisition, no benefits were found favouring the prevention program. Rather an increase of knowledge was demonstrated independent of group allocation from baseline to the six-month follow-up. This could also be explained in the way that parents talked more with their children about causes and symptoms of the disorder and families dealt more with the topic in general.

Benefits of participating in the program were also not demonstrated in offspring's reports of parenting style. It is possible that the implementation of the new parenting strategies takes longer than the assessment period captured. But it is also conceivable that the parenting style simply did not change.

Feedback Evaluation

With regard to the feedback evaluation forms that were filled out by participants from the intervention condition directly after each session, it became visible that both, children and parents, had a generally high acceptability favouring the prevention program. The overall ratings regarding the scale measuring comprehensiveness were strongest (mean = 4.56, SD = 0.67), followed by ratings on the scales understanding training sheets (mean = 4.41, SD = 0.84), comfort (mean = 4.37, SD=0.78), usefulness of training sheets (mean = 4.23, SD = 0.82) and active participation (mean = 4.0, SD= 0.92). Participants rated on a categorical scale between good and very good, reflecting that there is a need for families to get offers like the

conducted prevention program and that families profit from the theoretical and practical input that is conveyed by program contents. The feedback evaluation is an important output, because the program has been utilised in Germany for the first time. In case families would not accept the program and would not benefit from its contents, it would not be useful to have the aim of implementing the program for long-term treatment in the national health care system.

Methodological Differences between the Current and the Original Study Design

When looking at the study design of the current thesis, in most points the design was identical to the original RHC program (Compas et al., 2009). However, some differences occurred which should be mentioned and discussed.

Whereas Compas et al. (2009, 2011) utilised an active control as control condition following the written information condition by Beardslee et al. (1997, 2003, 2007), the current work followed the procedure by Sanford et al. (2003) with a waiting-list control condition in order to demonstrate true prevention effects. The non-active control group could be one reason why I found slightly bigger effects in internalising symptoms compared to Compas et al. (2009), who used an active control group.

Regarding inclusion criteria, slightly different age groups were included. While in the current work, children and youngsters aged between eight and 17 were seen as eligible, Compas et al. (2009, 2011) included children aged between nine and 15 years. Researchers argued that this age range is most useful, because children and adolescents were involved prior to the higher depression rates that are found in early and mid-adolescence. The reason of the extended age range in the current study was to appeal to as many families as possible. As Compas et al. (2009) included only children by the age of nine, the current study set the age of eight to include only children who were old enough to understand the rather complex program contents and coping skills that were conveyed by the prevention program.

When looking at other inclusion criteria, Compas et al. (2009) also included children when they met criteria of a MDD in the past, but had no current diagnosis. In the current work only children were included who did not fulfil diagnostic criteria of any psychiatric disorder. This could explain why children did not develop any depressive symptoms in short-term, and why incidence rates in the 15 months follow-up were rather low. As last point, Compas et al. (2009) included several children per family in the analysis, whereas the current study included only one child per family in the analysis, even though some families had more than one child that took part in the program.

Strengths

The current work is the first replication of the RHC program by Compas and colleagues (2009, 2011) outside the research group and the first evaluation of its kind in Germany. It extends the growing body of work that has been conducted in this research area. The study is based on diverse features which strengthen our findings of beneficial prevention effects. Well established measurements and a multiaxial approach (questionnaires (self- and parent reports), semi-structured diagnostic interviews) were utilised to collect data of all variables of interest. A multiaxial diagnostic is on the one hand relevant to enhance the reliability of diagnosed psychiatric disorders and on the other hand important to enhance the assessment of comorbid diagnosis (Essau & Petermann, 1999; Ihle et al., 2004). Reports about offspring's psychopathological symptoms were collected from both, parental and offspring's view. The recruitment was conducted in diverse clinics, institutions, counselling services, communication media and the regional administration office ("Kreisverwaltungsreferat") in and around Munich in order to raise attention to families with diverse socio-economic backgrounds and different degrees of disorder severity. Although it was rather difficult to recruit families, the families who took part in the intervention mostly responded positively to the prevention program. One of the strengths of the program is that the entire family is

included and that, apart from the group sessions, children and parents also receive separate sessions of their own, so that program contents can address individual needs.

In order to assess and enhance the quality of sessions, checklists were used that were filled out by group leaders after single sessions. Feedback evaluations that were filled out by participants immediately after each session reflected an overall satisfaction from families and showed that program contents as well as training sheets were well understood and accepted.

As comparison condition, a waiting-list was chosen in order to test whether the program is more efficient than what families would usually receive. Compas et al. (2009, 2011) utilised an active control, whereas I assume that with waiting-list true prevention effects will get more visible, especially when data collection is completed.

Limitations

Despite overall promising findings, the current work has also several limitations that should be noted. First of all, most participants were Caucasian (98.4%) which raises generalising issues for other population groups. Secondly, it was very hard work to find families who were willing to participate in the study. The low uptake of the program raises concern about familial interest in prevention services in general. It is possible that Compas et al. (2009, 2011) could recruit more families because the approach and cost coverage of the American health care system differs from the German one, and financial costs cause another hurdle to take for people struggling with depression. This might be one explanation why prevention programs in the USA seem to have a greater demand and seem to be more popular and attractive to American families compared to German families. The recruitment of families with a parent suffering from a depressive disorder was conducted systematically and families were not actively seeking help, which might also be a reason why response rates were low. Further, depression is still a taboo topic, even today, and families might be insecure about how to communicate the theme within the family and how to convince family members to take part in a study. Subjectively, sometimes it seems easier not to talk about things than to

break the taboo. The six months time period of the program demands a long-term commitment and with additional effort and strains on daily life. Thirdly, although families who participated in the study were highly motivated, a lot of data was still lost at follow-up assessments, which makes it difficult to generalise findings. It is possible that four assessment points, each with a relatively large number of questionnaires that had to be completed by participants, simply meant too much effort for families.

Fourthly, most families who took part had a high socio-economic background, which is not representative for the overall population. 42.4% of parents had a university degree, and almost one out of four families (24.5%) reported to have a monthly familial net income of 5000 € or higher. Fifthly, no intervention effects were found on offspring's self-reports about depressive symptoms. This might be due to the six-months-assessment. Longer-term assessments are relevant to capture whether the intervention is beneficial in the long-run, and to test whether children who took part in the intervention condition have significantly lower incidence rates of depression compared to waiting-list participants. The study is still running and data collection includes a 15-months-follow-up, enabling us to see at least a first tendency of the program efficacy in depression prevention. But since the presented data are preliminary data (K-DIPS n = 40), interpretations should be drawn with caution.

Future Research

Future research should already investigate baseline differences in children of depressed parents. This would provide a first insight into the complexity between child's psychopathology and the number of stressful life experiences, the SES and parental depression severity. It would be useful to examine whether children who differ in some of the mentioned factors at baseline, profit from the prevention program in short-, medium- and long-run in different ways.

Therefore, future research should also address follow-up-assessment points with a longer term in order to investigate the stability of intervention effects. By assessing, for

example, comparisons of mental health outcomes from children and adolescents who took part in the prevention program vs. waiting-list at 24 months, it would become evident if depressive and/or general psychopathological symptoms still differ between groups so that benefits are visible after two years. Further, also mediator variables should be considered and analysed in future works. Due to the fact that children were taught in coping strategies and parents in parenting skills, it would be interesting to see if changes in offspring's depressive and general psychopathological symptoms are mediated by one or both targets. A mediator analysis is only useful when the sample size is large enough and the study has enough power. Research with the focus on prevention of depression is still a relatively young research field which is continuously in progress.

Overall Summary

To summarize, it could be demonstrated that children of depressed parents differ already at baseline in some but not all outcome measures. Although child's depression outcomes were not affected by the investigated factors (parental depression severity, number of depressive episodes, number of stressful life events, SES), there was preliminary evidence that the number of parental depressive episodes is related to offspring's externalising symptoms. It is possible that child's sub-clinical depressive symptoms had too little variance between children, so that no significant correlations became visible. But it could also be that parental depression simply does not predict specifically child's depressive symptoms. However, children reported that the number of parental depressive episodes is linked to own externalising symptoms. It might be that children are, because of the recurrence of parental depression, more vulnerable to aggressive or dissocial behaviour than to other symptoms as anxiety or social withdrawal. Depending on the number of stressful life events, self-reports showed that all scales that measured child's general psychopathology were affected. That parent reports did not confirm child's self-reports could be explained in the way that parents trivialise child's symptoms. Or they do not even recognise child's general psychopathology, because they are so preoccupied

with their own mental health. The SES did not stand in relation to offspring's psychopathology, which is contrary to previous findings. But the current study only included middle and high SES families, whereas other studies also included families with low SES, showing that child's psychopathology is specifically in relation to low SES. Only one study (Anli & Karsli, 2010) showed that also a high SES is related to child's psychopathology, which could not be confirmed with the current data, but which could be due to cultural differences between Germany and Turkey. The baseline outcomes contribute to pre-existing findings, but also demonstrate some new findings, namely that specifically the recurrence of parental depression is linked to child's externalising symptoms.

When looking at intervention effects, the current work is the first evaluation of the RHC program by Compas et al. (2009, 2011) that was conducted outside of the research group on an international level. The findings provide no evidence that the program is effective in the prevention or reduction of offspring's depressive symptoms, neither in short- nor in long-term. Data of the semi-structured interviews conducted by trained psychologists showed at least first positive trends of the developmental course of depression favouring the prevention program, although not significantly. That no significant reduction or even prevention of offspring's depressive symptoms was found, might be due to the fact that the current study solely included children without a diagnosis, whereas other studies also included children with a history of depression. The probability that children without a diagnosis develop a depression within 15 months is comparably low compared to children who are remitted. Also the status of parental depression (current vs. past) could play a role in the effectiveness of the program.

Self-reports of children about general psychopathology demonstrated that internalising and mixed psychopathological symptoms can be reduced by the prevention program. However, parent reports did not confirm child's self-reports. Instead, parents reported an overall reduction of those two symptom scales, independent of participation in the program. It

is possible that parents who participated in the program, generally talked more about the disorder with their children and that because of the open dialogue, parents had the impression that children deal better with the disorder, reflected in a reduction of own general psychopathological symptoms, independent of group allocation. However, from child's perspective only, benefits for internalising and mixed psychopathological symptoms were found for children of the intervention condition, favouring the prevention program.

There was no evidence that the program was effective in enhancing child's level of knowledge of depression. However, the knowledge level increased independent of group allocation in short-term. It might be that parents talked more about causes and symptoms of the disorder with their children, independent of group allocation, which would explain why the knowledge level increased independent of participation in the program.

There was also no evidence that from child's perspective, the parenting style could be changed in a positive manner by the program. It is conceivable that implementing the parenting strategies takes depressed parents longer than the assessment period really captured. But of course, it is also possible that the parenting style simply did not change, so that children could not recognise positive changes in the parenting style.

The results of the intervention outcomes contribute to existing literature, but also demonstrate some new findings, showing that the knowledge level and the parenting style could not be modified by the prevention program in short- and medium-term. Further research with the GUG-Auf program is needed in order to establish the efficacy of the preventive intervention and therefore, especially a larger sample size as well as families with different ethnic and socio-economic backgrounds are needed to enable a generalisation of findings.

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Appendices

Appendix A

Therapieempfehlungen für Kinder und Jugendliche

Klinik und Poliklinik für Kinder- und Jugendpsychiatrie, Psychosomatik und Psychotherapie

Nussbaumstraße 5a, 80336 München (Wartezeit ca. 4 Wochen)

Tel.: 089/4400 55911

Kinderzentrum München

Heiglhofstraße 63, 81377 München

Tel.: 089 710090

Adressen Verhaltenstherapie

CIP

Terminvereinbarung unter Tel.: 089-13 07 93- 30

Rotkreuzplatz 1, 80634 München, U1/U7 bis Haltestelle Rotkreuzplatz

AVM

Leopoldstraße 21 (Eingang über den Innenhof), 80802 München

Tel.: (0 89) 3 88 88 47-0

Di.: 16:00 - 18:00 Uhr, Do.: 08.00 - 10.00 Uhr, Fr.: 12:00 - 14:00 Uhr

VFKV

Lindwurmstr. 117 / 4. und 5. St., 80337 München

Sprechzeiten der Institutsambulanz:

Montag bis Donnerstag 10.00 - 12.00 Uhr

Telefon: 089-45 24 166 50

DGVT

Candidplatz 9/ 1. St., 81543 München

Sprechzeiten der Institutsambulanz:

Montag 11.00 - 13.00 Uhr

Donnerstag 14.00 - 16.00 Uhr

Telefon: 089-62230704

Adressen Psychodynamische Psychotherapie

MAP

Rosenheimerstraße 1, Müllersches Volksbad (im 2. Stock), 81667 München

Telefon Nummer 089 - 44 14 15 55

Mo, Di, Do 9 – 12 Uhr

Do 18 – 20 Uhr

Kassenärztliche Vereinigung Bayern

http://www.kvb.de/patienten/therapieplatzvermittlung/

Tel.: 0921 787765-40410

Montag bis Donnerstag 09:00-17:00 Uhr

Freitag 09:00-13:00 Uhr

Appendix B

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 Gedanken 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		

Eidesstattliche Versicherung

Kornelija Laura Starman

_____

Name, Vorname

Ich erkläre hiermit an Eides statt,
dass ich die vorliegende Dissertation mit dem Thema:

Baseline Differences and Intervention Effects of the "Gesund und Glücklich Aufwachsen (GUG-Auf)" Prevention Program for Children of Depressed Parents
- The PRODO trial -

selbstständig 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 17.05.2018

_____ Kornelija Starman

Ort, Datum Unterschrift Doktorandin