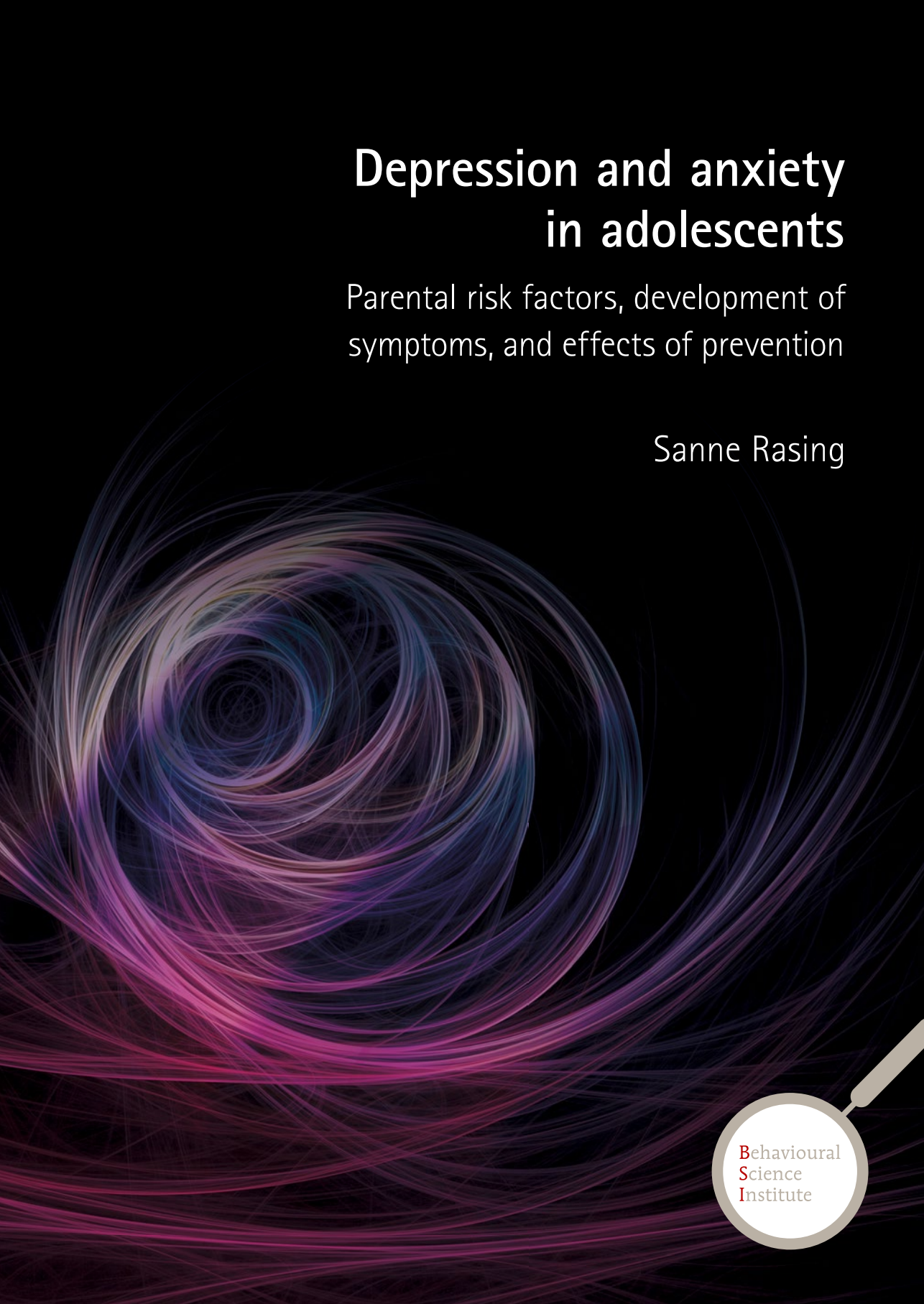
Depression and anxiety in adolescents

Parental risk factors, development of symptoms, and effects of prevention

Sanne Rasing



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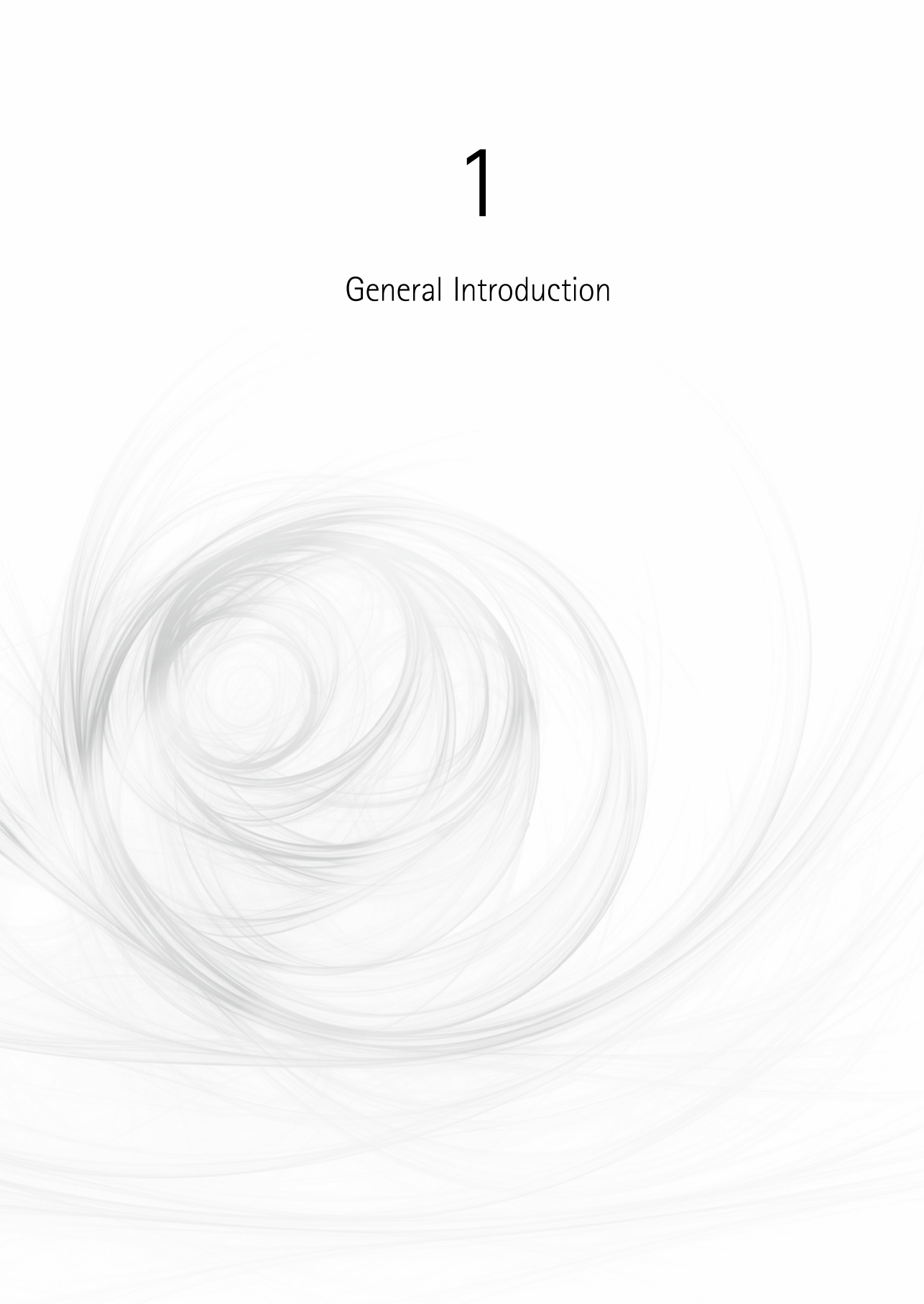
General Introduction



Depression and anxiety in adolescents

Adolescence is characterized by emotional and social challenges (Hurrelmann & Quenzel, 2015). Peer groups are becoming crucial and parents and teachers as adult are relatively losing importance. Adolescence is also the phase during which youngsters become more emotionally and intellectually independent, and become more autonomous compared to childhood (Iannotti & Bush, 2014; Oudekerk, Allen, Hessel, & Molloy, 2015). This life phase is particularly of interest considering the heightened vulnerability to mental illness and behavioral problems (Paus, Keshavan, & Giedd, 2008).

Depression and anxiety

Depression and anxiety are the most common mental health disorders during adolescence (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012; Verhulst, Van der Ende, Ferdinand, & Kasius, 1997). The disorders often co-occur and symptoms are strongly alike (Cummings, Caporino, & Kendall, 2014). Despite the strong overlap in symptoms, it is suggested that depression and anxiety are not completely similar constructs, nor are they two forms of one construct (Seligman & Ollendick, 1998).

Depression is defined by persistent unhappiness or a loss of interest or pleasure in most activities. In adolescents, irritability, rather than sadness, may be more prominent, accompanied by anger and social isolation (American Psychiatric Association, 2013; Thapar, Collishaw, Pine, & Thapar, 2012; World Health Organization, 2010). Depression can also be accompanied by vital complaints, such as disturbances in sleep and eating patterns, tiredness, and physical complaints such as stomach aches or headaches.

Anxiety disorders are characterized by extensive anxiety, physiological anxiety symptoms, excessive worrying and expecting the worst. People with anxiety disorders are constantly anticipating disaster and are overly concerned. Anxiety in adolescents is manifested by more or less the same characteristics as in adults. In adolescents, however, classification needs a fewer number of symptoms, shorter duration and anxiety does not have to be unreasonable (American Psychiatric Association, 2013; World Health Organization, 2010).

Adolescent depression and anxiety are known to be strongly related to each other and to have a high comorbidity. This is shown in high comorbidity rates in diagnoses; 25-50% of depressed adolescents had a comorbid anxiety disorder and 10-15% of adolescents with an anxiety disorders had a comorbid depression (Axelson & Birmaher, 2001; Sørensen, Nissen, Mors, & Thomsen, 2005; Weersing, Gonzalez, Campo, & Lucas, 2008). High comorbidity of depression and anxiety was also found in highly correlated symptoms of both problems, with up to 75% of comorbid symptoms (Cohen, Young, Gibb, Hankin, & Abela, 2014; Cummings et al., 2014). Further, the co-occurrence of depression and anxiety symptoms is known to be related to more severe symptoms (O'Neil, Podell, Benjamin, & Kendall, 2010), with greater impairment and more suicide attempts (Fichter, Quadflieg, Fischer, & Kohlboeck, 2010; Franco, Saavedra, & Silverman, 2007). Despite the overlap in

characteristics, depression and anxiety are different disorders with distinct features (Hale III, Raaijmakers, Muris, Van Hoof, & Meeus, 2009). Nonetheless, depression and anxiety do overlap and share similar dysfunctional cognitive processes, negative automatic thoughts, maladaptive beliefs (Ehring & Watkins, 2008; Harvey, 2004; McEvoy, Watson, Watkins, & Nathan, 2013; Muris, Roelofs, Rassin, Franken, & Mayer, 2005), and negative affect as latent factors underlying the disorders (Brown, Chorpita, & Barlow, 1998; Trosper, Whitton, Brown, & Pincus, 2012).

Depression and anxiety during adolescence

Depression and anxiety symptoms rise dramatically during adolescence (Kessler et al., 2012; Roza, Hofstra, Van der Ende, & Verhulst, 2003). Pre-adolescence depression prevalence is estimated at 4-8% (Birmaher et al., 1996) and anxiety is estimated at 20% (Chavira, Stein, Bailey, & Stein, 2004), although cautiousness is required because of the lack of reliability of lifetime estimates in children (Merikangas & He, 2014). Meaningful risk in mental health problems start during childhood, and continue to rise dramatically during adolescence (Kessler, Avenevoli, & Merikangas, 2001). For adolescents in the age of 13 to 17, the lifetime prevalence of depression and anxiety disorders is estimated at 12.6% and 32.4%, respectively (Kessler et al., 2012). Females are more likely to suffer from depression and anxiety disorders than males. These differences become more visible during adolescence, where ratios reach 2:1 to 3:1 (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Nolen-Hoeksema & Girgus, 1994; Pine, Cohen, Gurley, Brook, & Ma, 1998; Wittchen, Nelson, & Lachner, 1998).

Depression and anxiety during adolescence can have detrimental individual consequences. Both disorders are related to negative consequences in several areas. It is known that depression and anxiety are related to more problems in social and family functioning (Jaycox et al., 2009; Verboom, Sijtsema, Verhulst, Penninx, & Ormel, 2014), poor academic performance and higher school dropout (Balazs et al., 2013; Fergusson & Woodward, 2002; Quiroga, Janosz, Bisset, & Morin, 2013; Verboom et al., 2014), and unemployment (Fergusson, Horwood, & Woodward, 2001). Most detrimental are the consequences for physical and mental health on the longer term. Depression and anxiety are known to be associated with increased risk for substance abuse (Merikangas, Dierker, & Szatmari, 1998), suicide attempts and completed suicide (Bolton et al., 2008; Glied & Pine, 2002). These negative outcomes are not only associated with full-blown depression and anxiety disorders, but also subclinical levels of depression or anxiety are associated with significant distress and dysfunction. They are also a risk for future disorders, and with depression and anxiety disorders in later life (Aalto-Setala, Marttunen, Tuulio-Henriksson, Poikolainen, & Lonnqvist, 2002; Beesdo, Knappe, & Pine, 2009; Copeland, Angold, Shanahan, & Costello, 2014; Lewinsohn, Solomon, Seeley, & Zeiss, 2000; Pine, Cohen, Cohen, & Brook, 1999). Given the high prevalence of depression and anxiety disorders and the high rates of depression and anxiety symptoms during adolescence, and considering their impact on

the quality of life, it is a priority to identifying developmental predictors and prevent these disorders.

Besides the individual consequences, depression and anxiety disorders also have important societal consequences. Mental health problems accounted for 13% of the total global burden of disease in 2004. Depression is the largest contributor to the burden of mental health problems, and also anxiety contributes significantly to this burden of mental health problems (Baxter et al., 2014; Ferrari et al., 2013; World Health Organization, 2013). In adolescents and young adults, depression and anxiety disorders are also a major factor in the disability burden (Mathews, Hall, Vos, Patton, & Degenhardt, 2011). Evidently, the economic consequences of these mental health problems are large. Depression accounted for 40.5% of the disease burden caused by mental and substance use disorders, while anxiety accounted for 14.6% of the disease burden (World Health Organization, 2013).

According to the Netherlands mental health survey and incidence study, 37,400 (3.8%) adolescents between 13 and 17 suffer yearly from a depression, and 114,000 (11.6%) from an anxiety disorder (Verhulst et al., 1997). Consistent with the global numbers, depression and anxiety are a major concern in the Dutch public health. According to conservative estimations, depression costs the Dutch society 660 million euro per year in direct costs of care for people with depression disorders. Indirect costs, such as absenteeism at work, are 953 million euro per year (Meijer, Smit, Schoemaker, & Cuijpers, 2006; Smit et al., 2006). For anxiety, the direct costs are estimated at 275 million euro per year, and indirect costs at 3.9 million euro per year (Meijer et al., 2006; Smit et al., 2006).

Risk factors for development of depression and anxiety

With respect to depression and anxiety disorders, several factors increase the probability of developing one of these disorders. In this dissertation, the focus is on family and child related risk factors.

It is known that children of parents with a mental illness have a higher risk for developing mental health problems themselves (Beardslee, Gladstone, & O'Connor, 2011; Garber, 2006; Schwartz et al., 2012). When one of the parents suffers from depression, children are three times more likely to develop depression compared to children with healthy parents (Bijl, Cuijpers, & Smit, 2002; Knappe, Beesdo-Baum, & Wittchen, 2010; Lieb, Isensee, Höfler, Pfister, & Wittchen, 2002; Sander & McCarty, 2005). For anxiety, numbers show that 68% of children with a parent with an anxiety disorder show symptoms of anxiety as well (Capps, Sigman, Sena, Henker, & Whalen, 1996). Moreover, these children are up to seven times more likely to develop an anxiety disorder (Beidel & Turner, 1997; Kashani et al., 1990; Merikangas et al., 1998; Micco et al., 2009; Van Dorsselaer et al., 2006). This intergenerational transmission of disorders can be partly explained by a genetic predisposition; some estimate this influence to be 30-50% of the total variance in symptoms (Merikangas, Lieb, Wittchen, & Avenevoli, 2003; Ogliari et al., 2010; Rice, 2009; Sullivan, Neale, & Kendler, 2000).

Also, negative parenting behavior has been associated with an increased risk for depression and anxiety disorders in offspring (Knappe, Lieb, et al., 2009; Needham, 2008). Low parental emotional support was found to be most strongly associated with depression in adolescents (McLeod, Weisz, & Wood, 2007). Further, parental control is strongly associated with offspring anxiety disorders (McLeod, Wood, & Weisz, 2007), where a high level of control, also conceptualized as low respect for autonomy, may thwart opportunities for normative exploration of the environment and the development of self-efficacy, resulting in offspring's vulnerability for anxiety disorders (Rapee, 1997). Nonetheless, the specific relations between depression and anxiety symptoms in adolescents on the one hand and parental psychopathology, parenting behavior and their interaction on the other hand need to be further specified.

Besides parent-related factors, there are also child-related characteristics that increase the risk on developing depression and anxiety disorders. Having elevated symptoms of depression and anxiety is known as a strong precursor of later depression and anxiety disorders (Seeley, Stice, & Rohde, 2009). Onset of depression symptoms at younger age and with longer duration proved to be a risk factor for the development of a depressive disorder or even chronic depression (Hölzel, Härter, Reese, & Kriston, 2011). Therefore, early identification and prevention of depression and anxiety are needed to prevent direct and long term consequences (Siu & US Preventive Services Task Force, 2016).

Also, gender is a risk factor in the development of depression and anxiety symptoms. Female adolescents have been identified as having a higher risk for symptoms of depression and anxiety than male adolescents (Chaplin, Gillham, & Seligman, 2009; De Graaf, Ten Have, & Van Dorsselaer, 2010). The differences between males and females in prevalence of anxiety, that is, anxiety being more prevalent in females, start during childhood, and this difference continues to consist during adolescence. The difference in depression prevalence between males and females starts during adolescence, with also depression being more prevalent in females than in males (Lewinsohn, Gotlib, Lewinsohn, Seeley, & Allen, 1998). This difference does not only exist in levels of actual depression and anxiety disorders, but subclinical symptoms of depression and anxiety are also more present in female than in male adolescents (Angold, Erkanli, Silberg, Eaves, & Costello, 2002; Hankin, 2009; Nolen-Hoeksema, Larson, & Grayson, 1999). Particularly in female adolescents, stress and depression are closely related (Ge, Lorenz, Conger, Elder, & Simons, 1994; Larson & Ham, 1993; Rudolph & Hammen, 1999), and earlier research showed that increased depression and anxiety symptoms are often preceded by stressful life events or stress (Garber, Keiley, & Martin, 2002; Ge et al., 1994). Given the high risk female adolescents have to develop depression and anxiety, gender should be taken into account as risk factor when depression and anxiety prevention programs are offered.

Cognitive behavioral therapy based prevention

Cognitive behavioral therapy demonstrated to be an effective approach in treating a wide range of psychological problems, including depression and anxiety disorders (Butler, Chapman, Forman, & Beck, 2006; Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012). Cognitive behavioral therapy aims to modify several mechanisms that underlie depression and anxiety symptoms, such as dysfunctional emotions, maladaptive behaviors, response style, and cognitive errors (Calvete, Orue, & Hankin, 2013; Cole et al., 2008; Trosper et al., 2012). During therapy, dysfunctional or negative thought are identified and replaced by more positive thoughts and behaviors, causing a decrease in depression and anxiety symptoms (Beck, 1967; Beck, 2005; Nolen-Hoeksema, 2001).

Whereas treatment aims to reduce existing depression and anxiety symptoms, prevention in high risk populations aims to decrease the likelihood of the onset of a depressive or anxiety disorder or a decrease of symptoms (Garber & Weersing, 2010). So, treatment and prevention of depression and anxiety disorders seem to have comparable goals. Based on the successful effects of cognitive behavioral therapy in treatment of depression and anxiety and based on parallel goals between treatment and prevention, cognitive behavioral therapy seems to be suitable to use in the prevention of depression and anxiety in high-risk adolescents. Similar to treatment interventions, prevention based on cognitive behavioral therapy should contain elements that include problem solving, cognitive restructuring, family communication skills training, exposure, pleasant activity scheduling, and behavioral activation, with a focus on the interplay between thoughts, feelings, and behaviors, and practicing adaptive responses to difficult events (Compton et al., 2004). By integrating these elements accurately in prevention programs, depression and anxiety symptoms will reduce, as is intended by indicated depression and anxiety prevention programs.

Effects of depression and anxiety prevention

Gaining insight into the risk factors of depression and anxiety in adolescents, pro-active screening on risk factors, and organizing early detection in adolescents are a few steps toward improving mental health in adolescents. To actually reduce the risk on depression and anxiety disorders and to decrease depression and anxiety symptoms, depression and anxiety prevention for adolescents needs to be organized on a large scale. Prevention of depression and anxiety in young age has the potential to reduce serious individual consequences, but can also reduce the societal burden of mental health problems (Cuijpers, Beekman, & Reynolds, 2012; Kessler & Greenberg, 2002).

Prevention programs utilize different types of prevention strategies and focus on populations with different levels of risk on developing depression and anxiety (Mrazek & Haggerty, 1994). In all types of prevention, the goal is to reduce the number of new cases of depression or anxiety or reduce subclinical symptoms of depression or anxiety. Prevention strategies aimed at all individuals from the general population, regardless of their level of

risk are known as universal prevention. Two other levels of prevention strategies can be distinguished, in addition to universal prevention. Targeted prevention strategies are prevention programs aimed at populations at risk. First, when prevention programs target a population with a general risk factor, for example, children with parents with a mental illness or children from lower socio-economical environments (Garber et al., 2009; Hyun, Chung, & Lee, 2005), it is called selective prevention. Second, when programs are aimed at individuals with already existing depression or anxiety symptoms, but the symptoms do not qualify for a clinical diagnosis, they are indicated prevention (Stallard et al., 2012; Wijnhoven, Creemers, Vermulst, Scholte, & Engels, 2014). An indicated prevention strategy implies two phases. First, at-risk adolescents have to be identified by means of screening on their depression and anxiety symptoms. Based on their elevated symptom level, adolescents are directed towards appropriate health care. Second, prevention programs are offered to adolescents with elevated symptoms in order to reduce their risk on developing a clinical disorder.

Universal prevention showed mixed results in reducing symptoms or preventing the onset of depression or anxiety disorders. Review studies found less evidence for effects of universally delivered depression prevention programs than for targeted prevention programs (Brunwasser, Gillham, & Kim, 2009; Calear & Christensen, 2010; Christensen, Pallister, Smale, Hickie, & Calear, 2010; Horowitz & Garber, 2006; Merry et al., 2012; Merry & Spence, 2007; Stice, Shaw, Bohon, Marti, & Rohde, 2009). Further, universally delivered anxiety prevention programs have shown to be less promising than targeted anxiety prevention. Previous research has pointed out that effects of universally delivered prevention programs are small and particularly smaller than effects of selective and indicated prevention programs (Christensen et al., 2010; Corrieri et al., 2014; Fisak Jr, Richard, & Mann, 2011; Neil & Christensen, 2009; Teubert & Pinquart, 2011).

With regard to treatment of depression and anxiety disorders, cognitive behavioral therapy is, by far, the most commonly used treatment approach. Cognitive behavioral therapy has shown to be effective in treating depression and anxiety disorders (Butler et al., 2006; Cartwright-Hatton, Roberts, Chitsabesan, Fothergill, & Harrington, 2004; Compton et al., 2004), and has also shown to have enduring effects that reduce the risk for symptoms to return (Hollon, Stewart, & Strunk, 2006). Knowing that depression and anxiety share some underlying characteristics and cognitive processes, targeting these disorders with a similar approach at the same time might have some advantages (Chu, 2012; Chu, Johns, & Hoffman, 2015; Dozois, Seeds, & Collins, 2009; Taylor & Clark, 2009), especially when prevention programs target subclinical symptoms and not clearly defined clinical disorders. Because of these positive results of cognitive behavioral therapy in treating depression and anxiety disorders, these techniques are also often used in prevention programs of depression and anxiety.

Depression and anxiety prevention programs based on cognitive behavioral therapy are in general found to be effective (Christensen et al., 2010; Fisak Jr et al., 2011; Merry et

al., 2012). Prevention programs can show effectiveness by means of reducing the severity of depression and anxiety symptoms or reducing the prevalence of the disorders (McGorry, Purcell, Goldstone, & Amminger, 2011). Up to now, several prevention programs have shown to be effective in reducing symptoms (Barrett, Lock, & Farrell, 2005; Wijnhoven et al., 2014), but also in preventing the onset of actual disorders in the longer term (Beardslee et al., 2013; Brent et al., 2015).

The therapeutic techniques that are used in cognitive behavioral therapy, but also in prevention programs based on this approach, are known to target mechanisms underlying depression and anxiety (Chu & Harrison, 2007). These mechanisms, that is, dysfunctional emotions, maladaptive behaviors, and other cognitive errors, are known to be related to depression and anxiety (Calvete et al., 2013; Cole et al., 2008). More specific, stronger dysfunctional emotions, maladaptive behaviors, and cognitive errors, are related to higher levels of depression and anxiety (Brown et al., 1998; Trosper et al., 2012). The concepts response style and cognitive errors, reflecting the underlying mechanisms, are, therefore, assumed to mediate the relation between prevention programs and symptoms of depression and anxiety (Johnson & Miller, 1990; LaGrange et al., 2011). It is, however, unknown whether dysfunctional emotions, maladaptive behaviors, response style, and cognitive errors are related to subclinical symptoms of depression and anxiety, and whether depression and anxiety prevention programs have sufficient impact to reduce the underlying mechanisms.

Besides underlying mechanisms, there are also moderating factors to take into account. It is known that parental psychopathology, parental emotional support, and parental control play a role in depression and anxiety symptoms in their offspring (Bögels & Phares, 2008; Bosco, Renk, Dinger, Epstein, & Phares, 2003; Brennan, Hammen, Katz, & Le Brocque, 2002; Connell & Goodman, 2002; Kane & Garber, 2004).

Parental psychopathology is a known risk factor in adolescents' mental health, and maternal and paternal psychopathology have shown to be related to higher levels of depression and anxiety (Brennan et al., 2002; Connell & Goodman, 2002; Kane & Garber, 2004). It is, however, unclear which role maternal and paternal psychopathology actually play. These inconsistencies might be explained by the different influence maternal and paternal psychopathology have during childhood and adolescence (Bögels & Phares, 2008; Connell & Goodman, 2002). It is suggested that maternal psychopathology might be more influential during childhood, whereas paternal psychopathology might have more of a detrimental impact on the development of depression and anxiety during adolescence (Connell & Goodman, 2002). Earlier research suggested that preventive interventions have no impact on reducing symptoms in adolescent offspring of parents with mental health problems compared to adolescents with healthy parents (Brent et al., 2015; Weersing et al., 2016). Nevertheless, there are prevention programs specifically aimed at children and adolescent of parents with mental illness. In conclusion, results are mixed and effectiveness of prevention programs for offspring of parents with mental illness needs to be further specified (Beardslee, Wright, Gladstone, & Forbes, 2007; Siegenthaler, Munder, & Egger, 2012).

Parenting behavior is also known to be related to depression and anxiety symptoms. Especially emotional support and respect for autonomy are parenting behavior known to be associated with adolescents' depression and anxiety symptoms (Knappe, Lieb, et al., 2009; Needham, 2008). Emotional support and respect for autonomy from parents are suggested to increase their children's confidence and might serve as protective factor in the development of depression and anxiety disorders. Review studies showed that a lack of parental emotional support is related to depression symptoms in offspring (McLeod, Weisz, et al., 2007), and that low respect for autonomy is associated with offspring's anxiety (McLeod, Wood, et al., 2007). How these dimensions of parenting are related to the effectiveness of depression and anxiety prevention is unknown and moderating effects need to be explored.

Aim and relevance of this dissertation

The overall aim of the current dissertation was to contribute to the existing literature on risk factors for the development of depression and anxiety symptoms and the effectiveness of prevention in adolescent girls with a high familial risk. Assessing the effects of parent related risk factors on the development of depression and anxiety symptoms may contribute to selecting high risk adolescents who, in light of the negative consequences of elevated depression and anxiety symptoms, may strongly benefit from an intervention in young age. It is known that early detection of adolescents with high risk is highly important, because undetected and untreated depression and anxiety symptoms are associated with negative consequences at the individual level. Further, it is important to determine which prevention programs are effective in reducing symptoms or in preventing symptoms of depression and anxiety from developing. In the current dissertation we examined whether the prevention program 'Een Sprong Vooruit' [A Leap Forward] causes benefits for adolescents who have a high risk on developing depression or anxiety disorders. Since we know that having elevated depression and anxiety symptoms is a precursor for depression and anxiety disorders in later life, early intervention could potentially reduce costs of mental health care, and, more important, could prevent a lifetime of distress.

Overview of this dissertation

This dissertation is divided in two parts. Part one consists of two studies on the effects of parent related risk factors on depression and anxiety symptoms in female adolescents and how these factors contribute to the level or development of depression and anxiety symptoms of these female adolescents. In Chapter 2, a study is presented on the influence of perceived maternal and paternal psychopathology on the level of depression and anxiety

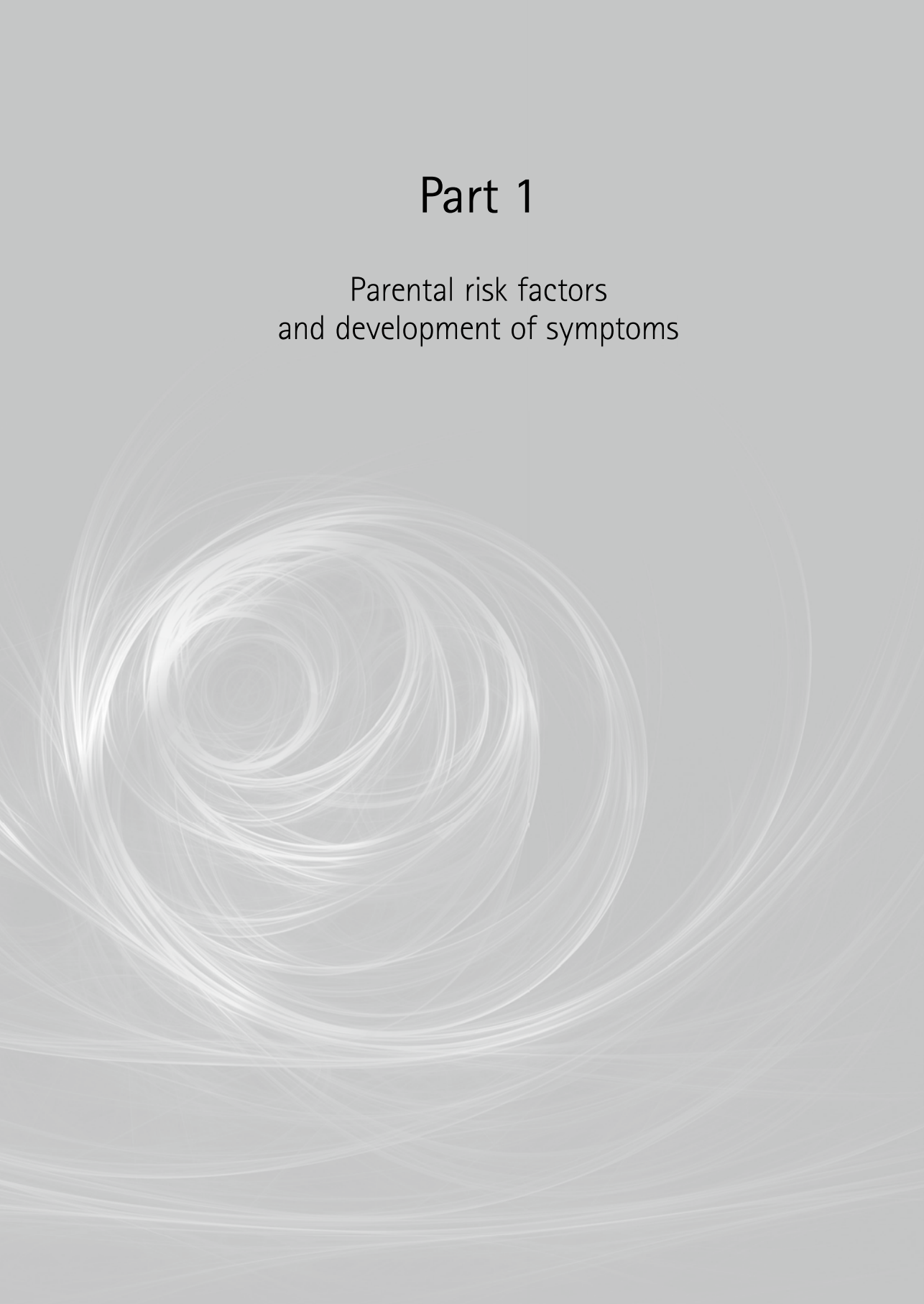
symptoms in a general population of adolescent girls. The data in this study represents the screening assessment for the randomized controlled trial reported on in following chapters. We tested whether the influence of perceived maternal psychopathology was different from the influence of perceived paternal psychopathology, and whether these predictors had an interaction effect on depression and anxiety symptoms of female adolescents. Chapter 3 presents a study on the effect of maternal and paternal psychopathology, emotional support and respect for autonomy on the level and development of depression and anxiety symptoms in high-risk adolescent girls. We used data of a non-blinded randomized controlled trial on the effectiveness of depression and anxiety prevention in female adolescents with elevated symptoms of depression and anxiety, from baseline assessment, post-intervention, 6-month and 12-month follow-up assessments. We examined whether parental psychopathology in interaction with emotional support and respect for autonomy influence depression and anxiety symptoms.

Part two of this dissertation focuses on the effectiveness of depression and anxiety prevention in high-risk adolescent populations. In Chapter 4, a meta-analysis is presented of the effects of school-based and community-based prevention programs that are based on cognitive behavioral therapy aimed at preventing depression and anxiety in high-risk adolescents. We included results of 23 randomized controlled trails on targeted depression and/or anxiety prevention to perform a meta-analysis. The studies we used in this meta-analysis were published before July 2013. We analyzed whether depression and anxiety prevention for high risk adolescents is effective on the short-term and on the long-term. Chapter 5 addresses the study protocol of a randomized controlled trial aiming to study the effects of depression and anxiety prevention in adolescent girls with a high familial risk. In Chapter 6, results of the randomized controlled trial are presented. We examined the effectiveness of the prevention program 'Een Sprong Vooruit' in reducing depression and anxiety symptoms in adolescent girls. Participants were 142 female adolescents with elevated symptoms of depression and/or anxiety and with parents with mental health problems. The participants were randomly assigned to the intervention or control condition. The intervention was a depression and anxiety prevention program aimed at adolescents with parents with mental health problems. For this, assessments at baseline, after two sessions, after four sessions, post-intervention, 6-month and 12-month follow-up were used. We tested whether the depression and anxiety symptoms were different between participant in the intervention and control condition on short- and long-term, and whether the symptom development is different between the participants in the different conditions. Chapter 7 presents the moderating effects of parental psychopathology, parenting behavior and parenting stress on the effects of 'Een Sprong Vooruit' in reducing depression and anxiety symptoms in female adolescents. Further, we tested whether the prevention program had effect on underlying mechanism of depression and anxiety, that is, response style consisting of distraction, problem solving and rumination, and cognitive errors, consisting of underestimation of the ability to cope, personalizing without mind reading,

selective abstraction, overgeneralizing, and mind reading. Finally, Chapter 8 presents a summary and general discussion of the main findings. In addition, clinical implications related to the studies and directions for future research will be discussed.

Part 1

Parental risk factors and development of symptoms



2

The Association between Perceived Maternal and Paternal Psychopathology and Depression and Anxiety Symptoms in Adolescent Girls

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Abstract

Exposure to parental depression and anxiety is known to heighten the risk of internalizing symptoms and disorders in children and adolescents. Ample research has focused on the influence of maternal depression and anxiety, but the contribution of psychopathology in fathers remains unclear. We studied the relationships of perceived maternal and paternal psychopathology with adolescents' depression and anxiety symptoms in a general population sample of 862 adolescent girls (age $M = 12.39$, $SD = .79$). Assessments included adolescents' self-reports of their own depression and anxiety as well as their reports of maternal and paternal psychopathology. We found that perceived maternal and paternal psychopathology were both related to depression and anxiety symptoms in adolescent girls. A combination of higher maternal and paternal psychopathology was related to even higher levels of depression and anxiety in adolescent girls. Our findings showed that adolescents' perceptions of their parents' psychopathology are significantly related to their own emotional problems.

Introduction

The association between parents' psychopathology and their children's psychopathology has been well studied (Hosman, Van Doesum, & Van Santvoort, 2009; Maybery, Reupert, Patrick, Goodyear, & Crase, 2009). Especially the link between parental depression or anxiety and children's internalizing problems has been a focus of much research (Connell & Goodman, 2002; England & Sim, 2009; Goodman et al., 2011; Goodman & Tully, 2006). It is known that exposure to parental depression or anxiety disorders heightens children's vulnerability to internalizing symptoms and disorders (Bijl et al., 2002; Lieb et al., 2002) and that particularly mothers' depression and anxiety are a risk factor for adolescents' depression or anxiety (Singh et al., 2011). The contribution of fathers' psychopathology has received increasing attention over the last years and seems to be of equal importance when compared to mothers' psychopathology (Connell & Goodman, 2002; Ramchandani et al., 2011). To our knowledge, the potential risk to adolescents when both parents suffer from mental health problems compared to one of the parents has not been extensively studied. Therefore, this study focuses on the relationship of parental psychopathology with symptoms of depression and anxiety in adolescents.

Depression and anxiety are common in adolescence, with a prevalence of 5.6% for depression (Costello, Erkanli, & Angold, 2006) and 3-20% for anxiety (Albano, Chorpita, & Barlow, 2003). Mental disorders, with depression and anxiety the most prevalent and often not recognized, contribute largely to internalizing problems in young people (Mathews et al., 2011). The disorders and particularly depression, besides their consequences during adolescence and young adulthood, are marked by a recurrent course, and it has been found that the onset of adult depression and anxiety often emerges during adolescence (Albano et al., 2003; Beesdo et al., 2009; Birmaher et al., 1996; Costello et al., 2006; Dobson, Hopkins, Fata, Scherrer, & Allan, 2010). Knowing that internalizing disorders have the highest lifetime prevalence, estimated to be 3.3-21.4% for mood disorders and 4.8-31.0% for anxiety disorders (Kessler et al., 2007), it is important to identify risk factors for development of these disorders in adolescents. It is known that adolescent girls are more vulnerable to develop a depressive or anxiety disorder than boys (Garber et al., 2002; Nolen-Hoeksema, 2001). Also elevated symptoms of depression or anxiety symptoms are more present in girls than in boys during adolescence (Hankin & Abramson, 2001). Because girls have a higher risk on developing depression or anxiety, we focused specifically on adolescent girls.

Children of parents with a mental disorder have an elevated risk of developing psychopathology themselves (Lieb et al., 2002; Merikangas et al., 1998; Micco et al., 2009). More specifically, in case of parental depression, children are three times more likely to develop depression compared to children of healthy parents (Weissman, Wickramaratne, et al., 2006). In case of parental anxiety, children are two to seven times more likely to develop an anxiety disorder (Beidel & Turner, 1997). Thus, intergenerational transmission of mental health problems is rather consistent (Goodman & Gotlib, 1999). Further, research showed

that there was no interaction between gender and parental depression and that transmission of depression from parents to children was comparable for boys and girls (Bouma, Ormel, Verhulst, & Oldehinkel, 2008). Models describing the intergenerational transmission refer to several factors, such as heritability, exposure to parental maladaptive behavior and cognitions, and exposure to a stressful dysfunctional family situation (Garber & Cole, 2010; Goodman & Gotlib, 1999). This, however, describes the contribution of parents in general and does not explain the extent to which mothers or fathers, uniquely and in combination, influence the development of depression or anxiety in adolescents.

The maternal and paternal influences on the normal development of children have been studied extensively, and it is known that both parents have their own unique contribution (Lamb, 2010). In the development of children's psychopathology, fathers' contribution has long been underestimated and therefore not well understood. Most adolescents' problems were attributed to the influence of mothers (Phares, 1992). Recently, the contribution of fathers has been increasingly recognized, although mostly in the development of externalizing problems rather than internalizing problems (Phares & Compas, 1992).

Beside the influence of mothers' and fathers' psychopathology as single risk factor, the presence of paternal psychopathology has also been conceptualized as a moderator of maternal psychopathology as suggested based on previous studies (Goodman & Gotlib, 1999; Weissman, Leckman, Merikangas, Gammon, & Prusoff, 1984). This implies that psychopathology of fathers may additionally increase the risk for psychopathology in children, especially when mothers suffer from mental health problems. However, whether having two parents – instead of one – with psychopathology indeed increases the level of symptoms in adolescents needs to be studied more carefully.

As a measure of parental psychopathology, earlier studies on the relationship between parental psychopathology and children's psychopathology used mostly the symptom level or clinical diagnosis as reported by the parents. However, it has been suggested that children are best informants of their own internalizing symptoms (Bird, Gould, & Staghezza, 1992; Kazdin, 1994), and several studies used children as informants of parental characteristics as well, such as parent-child relationships and parenting behavior (Forehand & Nousiainen, 1993). To our knowledge, no studies have used adolescents' perceptions to further unravel the relationships between maternal, paternal, and children's mental health. Thus, this study would contribute to the understanding of whether parental psychopathology can be measured based on adolescents' perception, and whether the same relationships hold as those between parent-rated parental psychopathology and adolescents' psychopathology.

The first aim of the current study was to examine whether parental psychopathology, as perceived by adolescent girls, was related to adolescent psychopathology. More specifically, we studied whether maternal and paternal psychopathology were related to depression and anxiety symptoms in adolescent girls. The second aim was to explore whether the presence of both maternal and paternal psychopathology, rather than only maternal or paternal symptomatology, was related to higher depression and anxiety

symptoms in adolescent girls. We hypothesized that higher perceived maternal and paternal psychopathology were separately related to a higher level of depressive and anxiety symptoms in adolescent girls, and that maternal and paternal psychopathology would have an additive effect on depression and anxiety symptoms in adolescent girls.

Method

For this study, data of the screening procedure of a Dutch randomized controlled trial (Dutch Trial Register NTR3720) on the prevention of depression and anxiety in adolescent girls with high familial risk were used (Rasing, Creemers, Janssens, & Scholte, 2013). The medical ethics committee CMO Region Arnhem-Nijmegen, The Netherlands, has approved this study.

Participants and procedure

Female students in the first and second grade of secondary school received written information about the study together with an opt-out form, which allowed them and their parents to refuse the participation. After passive consent was received, 862 female adolescents completed questionnaires about symptoms of depression or anxiety and about their perceptions of psychopathology in their parents. These students were selected from five schools ranging from vocational education up to pre-university education in rural area as a representative sample of the general adolescent female population. The age of the adolescents ranged from 11 to 15, with a mean age of 12.39 (*SD* = .79). Most adolescents were of Dutch origin (96.6%).

Measures

Depression

The Dutch version of the Children's Depression Inventory 2 (CDI 2) (Kovacs, 2012), which consists of 28 items, was used to measure depression symptoms. Each item consists of three statements rated in severity from 0 to 2. Sample statements include, "Sometimes I feel sad", "Most of the times I feel sad", and "I always feel sad". Cronbach's alpha was .87.

Anxiety

The Dutch version of the Spence Children's Anxiety Scale (SCAS) (Spence, 1997) was used to measure anxiety symptoms. This 44-item self-report questionnaire measures the frequency of symptoms on a 4-point scale ranging from never to always. Sample statement is, "I worry about things". Cronbach's alpha was .85.

Adolescents' perception of parental psychopathology

Students responded to seven statements about parental psychopathology for both mother and father. Adolescents indicated whether the following statements are true for their parents: "My parent received treatment from a psychologist or psychiatrist", "My parent had a depressed mood for more than two weeks", "My parent had a decreased interest or pleasure in most or all activities", "My parent had a period of fatigue or loss of energy", "My parent had excessive worry and anxiety about general events for at least six months", "My parent had excessive and unreasonable fear of a specific situation (e.g., elevators) or object (e.g., spiders)" and "My parent had recurrent panic attacks, with or without fear to leave his/her home or safe environment". In all statements, wording of parent was replaced by either mother or father. Answers were rated as not present (0) or present (1). Because the seven items were related to several concepts – three items were related to parental depression, three to parental anxiety and one to parental treatment – we could not assume that the seven items were highly related to each other and that the seven items could be interpreted as a unidimensional scale. Therefore, we did not compute a classic Cronbach's alpha. To assess a general indication for parental psychopathology, we counted the number of items for which their adolescent daughters indicated parental psychopathology.

Statistical analyses

First, means, standard deviations, and bivariate correlations were computed for all study variables. Second, hierarchical regression analyses were performed to examine the relationships and the interaction of maternal and paternal psychopathology with adolescents' depression and anxiety. All predictor variables were continuous and centered in all analyses before testing interactions (Aiken & West, 1991).

In the first regression analysis, we used adolescent depression as an outcome variable, and entered perceived maternal psychopathology as predictor variable in step 1, perceived paternal psychopathology as predictor variable in step 2 and their interaction in step 3. In the second analysis, adolescent anxiety was the outcome variable and again, we entered the perceived maternal psychopathology as predictor variable in step 1, perceived paternal psychopathology as predictor variable in step 2 and their interaction in step 3.

Results

Descriptive statistics and correlations

Descriptive statistics were computed for adolescents' depression symptoms (M = 8.13, SD = 6.06), anxiety symptoms (M = 26.69, SD = 13.89), perceptions of maternal psychopathology (M = .89, SD = 1.18) and perceptions of paternal psychopathology (M = .52, SD = 1.00). Bivariate correlations among study variables (Table 1) showed that adolescents' depression and anxiety symptoms were highly correlated. Further, adolescents' depression and anxiety

Table 1 Correlations Between Model Variables

	1.	2.	3.	4.
1. Depression symptoms adolescents	-	.72***	.34***	.25***
2. Anxiety symptoms adolescents		-	.34***	.26***
3. Perceived maternal psychopathology			-	.43***
4. Perceived paternal psychopathology				-

Note. *** $p < .001$

symptoms were positively related to the adolescents' perceptions of maternal and paternal psychopathology. Additionally, perceived maternal and paternal psychopathologies were positively related.

As can be seen in Table 2, perceived maternal and paternal psychopathology were significant predictors of depression symptoms in adolescents. Additionally, interaction between perceived maternal and paternal psychopathology was a significant predictor of depression symptoms in adolescents. This interaction can also be seen in Figure 1, where the predictors maternal and paternal psychopathology are divided in low (below mean) and high (above mean).

Table 2 Regression Analyses of Perceived Maternal and Paternal Psychopathology and Their Interaction Effect on Adolescents' Depression Symptoms

	B	*SE*	β	*t*	*F*(*F*-change)	Δ R^2
Step 1					104.78	.11***
Perceived maternal psychopathology	1.71	.17	.33	10.24***		
Step 2					59.76 (13.21)	.01***
Perceived maternal psychopathology	1.42	.18	.28	7.71***		
Perceived paternal psychopathology	.79	.22	.13	3.63***		
Step 3					44.62 (12.70)	.01***
Perceived maternal psychopathology	1.59	.19	.31	8.42***		
Perceived paternal psychopathology	1.13	.24	.19	4.78***		
Interaction perceived maternal psychopathology × perceived paternal psychopathology	-.41	.12	-.14	-3.56***		

Note. *** $p < .001$

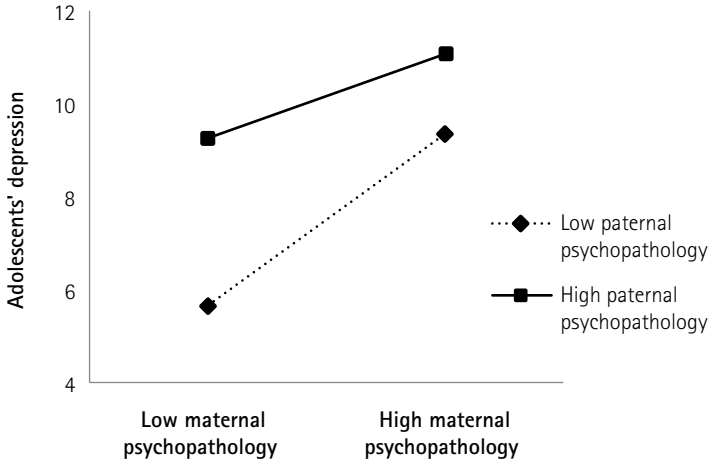

Figure 1 Adolescents' depression symptoms predicted by the interaction of maternal and paternal psychopathology

Table 3 Regression Analyses of Perceived Maternal and Paternal Psychopathology and Their Interaction Effect on Adolescents' Anxiety Symptoms

	B	*SE*	β	*t*	*F* (*F*-change)	Δ R^2
Step 1					113.32	.12***
Perceived maternal psychopathology	4.08	.38	.34	10.65***		
Step 2					64.28 (13.54)	.01***
Perceived maternal psychopathology	3.40	.42	.29	8.07***		
Perceived paternal psychopathology	1.84	.50	.13	3.69***		
Step 3					45.03 (5.81)	.01*
Perceived maternal psychopathology	3.67	.44	.31	8.44***		
Perceived paternal psychopathology	2.36	.54	.17	4.35***		
Interaction perceived maternal psychopathology × perceived paternal psychopathology	-.64	.27	-.09	-.09*		

Note. * $p < .05$ *** $p < .001$

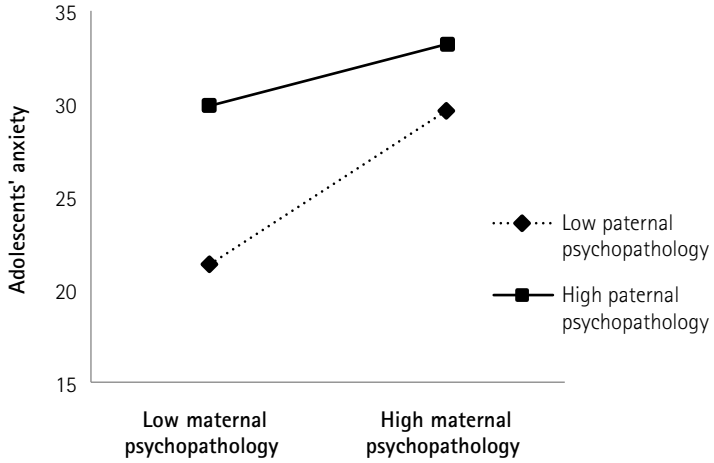

Figure 2 Adolescents' anxiety symptoms predicted by the interaction of maternal and paternal psychopathology

The same relationships were found between perceived maternal and paternal psychopathology and anxiety symptoms in adolescents, as can be seen in Table 3. The interaction between perceived maternal and paternal psychopathology also significantly predicted adolescents' anxiety symptoms. This interaction is also shown in Figure 2, where again the predictors maternal and paternal psychopathology are divided in low (below mean) and high (above mean).

This means that higher perceived maternal psychopathology was associated with higher levels of adolescents' depression and anxiety symptoms and that higher perceived paternal psychopathology was also associated with more depression and anxiety symptoms in adolescents. Moreover, the combination of higher perceived maternal and paternal psychopathology had an additive effect on symptoms of depression and anxiety in adolescents.

Discussion

The present study examined whether perceived parental psychopathology was related to symptoms of depression and anxiety in adolescent girls. Furthermore, we explored whether the presence of both perceived maternal and perceived paternal psychopathology was related to higher depression and anxiety symptoms in adolescent girls compared to when only one of the parents was reported to show psychopathology.

Our findings showed that perceived maternal and paternal psychopathology were related to depression and anxiety symptoms in adolescent girls, in accordance with previous studies, which demonstrated the same relationships (Brennan et al., 2002; McClure, Brennan, Hammen, & Le Brocque, 2001). We also found that a combination of higher perceived maternal psychopathology and higher perceived paternal psychopathology was related to even higher depression and anxiety symptoms in adolescent girls. This is consistent with previous studies, which showed relations among maternal, paternal, and offspring symptoms (Klein, Lewinsohn, Rohde, Seeley, & Olino, 2005; Renk et al., 2007). In Goodman and Gotlib's (1999) model of intergenerational transmission, paternal mental health is described as a moderator in the development of the child's psychopathological symptoms. Healthy fathers could compensate for genetic risk and provide healthy cognitions, behavior, and affect. They could provide substitute caregiving and could provide support for mothers. When only one parent suffers from mental health problems, the healthy parent can be seen as a protective factor by acting as a positive role model (Connell & Goodman, 2002; Goodman & Gotlib, 1999; Ramchandani & Psychogiou, 2009). When both parents show symptoms of psychopathology; thus, the protective factor is not present, children are likely to experience more psychopathology. Earlier studies also confirmed that parental psychopathology in both parents had an additive effect and resulted in a higher symptom level symptoms in children (Dierker, Merikangas, & Szatmari, 1999; Goodman, Brogan, Lynch, & Fielding, 1993).

In this study, we used adolescent girls' perceptions to assess parental psychopathology. Our findings showed strong similarities with earlier studies, which used parents as informants of parental psychopathology. Given our findings, using adolescent girls' perceptions seems a promising way to measure parental psychopathology. However, there also might be some limitations using adolescents as informants; according to the distortion hypothesis (Richters & Pellegrini, 1989), the informants' psychopathology influences the report of symptoms of a different person. Richters and colleagues studied the accuracy of depressed mothers' reports informants of their child's symptoms of psychopathology (Richters, 1992; Richters & Pellegrini, 1989). They hypothesized that a negative perceptual bias related to mothers' own depression results in over reporting of their children's symptoms. This might also be the case for our adolescents' reports of their parents' psychopathology. Another limitation is that we studied these relationships only in adolescent girls. The relationship between perceived maternal psychopathology and perceived paternal psychopathology with depression and anxiety symptoms might be different in adolescent boys than in girls, because girls are known to be more responsive to depression than boys (Garber et al., 2002; Nolen-Hoeksema, 2001). Although earlier research suggested that transmission of depression from parents to children was comparable for boys and girls, future studies should also focus on boys instead of only girls.

Since the present study used a cross-sectional design, we were not able to test causal pathways in the intergenerational transmission of psychopathology. Future studies should

therefore examine these processes utilizing a longitudinal study design in order to determine how these processes develop and in what sequence. Further, studies should consider using disorder-specific questionnaires. For our measures of perceived parental psychopathology, we used seven statements related to three different concepts, parental depression, anxiety and treatment instead of using unidimensional scales for each of these three concepts. Measuring parental depression and anxiety using for each a unidimensional instrument may result in more valid and reliable measurements of specific types of parental psychopathology.

The clinical implication of this screening method is that adolescents at risk for depression or anxiety can be identified in clinical practice. This makes it possible to intervene early and to prevent them from developing a clinical disorder. Selecting an appropriate depression and anxiety prevention program and testing its effectiveness should be considered as the next step.

In conclusion, the present study showed that perceived maternal and paternal psychopathology were related to symptoms of depression and anxiety in their adolescent daughters. Depression and anxiety symptoms in adolescent girls were even higher when both parents had higher psychopathology, which underlines the relevance of parental psychopathology in both parents. The causal pathways, however, are unclear and need to be studied in longitudinal studies. Future research should examine how the intergenerational transmission of depression and anxiety unfolds over time as well as from an earlier age through adolescence into young adulthood.

3

Development of Depression and Anxiety Symptoms in Female Adolescents: The Role of Parental Psychopathology and Parenting Behavior

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Rasing, S. P. A., Braam, M. W. G., Brunwasser, S. M., Janssens, J. M. A. M., Creemers, D. H. M., & Scholte, R. H. J. Development of depression and anxiety symptoms in female adolescents: The role of parental psychopathology and parenting behavior

Abstract

Parental psychopathology and parenting behavior are known to be related to depression and anxiety in adolescents, but research findings have not been clear about the unique roles of mothers and fathers. The aim of our study was to examine the effect of maternal and paternal psychopathology, emotional support, and respect for autonomy, and in addition, whether parental psychopathology and emotional support and respect for autonomy interacted with each other in affecting the level and development of depression and anxiety symptoms in female adolescents. In total, 142 adolescent females (age M = 12.87, SD = .69) participated in this study, together with 137 mothers (age M = 43.89, SD = 4.41) and 113 fathers (age M = 47.10, SD = 4.58). Data were analyzed using latent growth curve modeling in Mplus. Paternal emotional support was negatively related to adolescent baseline level of depression and anxiety symptoms. No relationships were found between maternal psychopathology or maternal parenting behavior and depression and anxiety symptoms in adolescents. Further, we found a significant interaction effect of paternal psychopathology and respect for autonomy on depression symptoms in adolescents. Our findings suggest that the association between paternal respect for autonomy and depression symptoms in adolescents was negative at lower levels of paternal psychopathology, and that the association between paternal respect for autonomy and depression symptoms in adolescents was positive for higher levels of paternal psychopathology.

Introduction

Depression and anxiety rates rise dramatically during adolescence. For adolescents aged between 13 and 17, lifetime prevalence is estimated at 12.6% for depression and 32.4% for anxiety disorders (Kessler et al., 2012). Females are more likely to develop a depression and anxiety disorders during childhood, and these gender differences become even more visible during adolescence, reaching ratios of 2:1 to 3:1 (Costello et al., 2003; Nolen-Hoeksema & Girgus, 1994; Pine et al., 1998; Wittchen et al., 1998). For females aged 13 to 17, lifetime prevalence of depression and anxiety disorders is estimated at 16.8% and 38.8%, respectively (Kessler et al., 2012). Suffering from a depression or anxiety disorder during adolescence can have serious consequences for the quality of life. Both disorders are related to poor psychological wellbeing, social impairment, poor academic functioning (Balazs et al., 2013; Fletcher, 2008; Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2003; Verboom et al., 2014), increased risk for substance abuse (Merikangas et al., 1998), and both completed suicide and suicide attempts (Bolton et al., 2008; Glied & Pine, 2002). Furthermore, experiencing depression or anxiety disorders during adolescence is associated with recurrent depression and anxiety disorders later in life (Aalto-Setala et al., 2002; Copeland et al., 2014; Pine et al., 1999). Given the high prevalence of these disorders during adolescence and their impact on the quality of life, identifying the developmental predictors of these disorders is a priority.

Depression and anxiety disorders are highly familial disorders (Knappe et al., 2010; Sander & McCarty, 2005). Parental psychopathology and parenting behavior are associated with adolescent depression and anxiety disorders and symptom severity (Garber, 2006; Schwartz et al., 2012). However, previous research has not focused on the interaction between parental psychopathology and parenting behavior in relation to adolescents' depression and anxiety symptoms. It has also not been examined whether maternal and paternal psychopathology or parenting behavior have a differential impact on the development of adolescent depression and anxiety disorders or symptoms. In this study, we examined the association between maternal and paternal psychopathology in combination with maternal and paternal parenting behavior on the development of female adolescent depression and anxiety symptoms.

Earlier research suggests that parental psychopathology is an important risk factor for the development of depression and anxiety disorders in offspring (Connell & Goodman, 2002; England & Sim, 2009; Goodman et al., 2011; Goodman & Tully, 2006). Children of depressed parents are three times more likely to develop depression than children of parents without depression (Birmaher et al., 1996; Lieb et al., 2002). Also, children of parents with an anxiety disorder are even two to seven times more likely to develop an anxiety disorder themselves (Micco et al., 2009). The increased risk for depression and anxiety disorders in offspring can partly be explained by a high genetic predisposition (Boomsma, Van Beijsterveldt, & Hudziak, 2005; Middeldorp et al., 2005; Sullivan et al., 2000). In addition,

environmental influences, such as the negative emotional climate that often pervades a child's home life when a parent has a depression or anxiety disorder (Goodman & Gotlib, 1999), and the gene-environment interaction contribute to depression and anxiety disorders in offspring (Rutter, 2002; Sullivan et al., 2000). Depressed parents often have negative cognitions, affect, and behaviors that compromise their ability to meet the child's social and emotional needs. This negatively affects children's development of social skills and cognitive styles through social learning and modeling (Schwartz et al., 2012). Together, these deficient social skills and negative cognitive styles place children at elevated risk for developing depression (Goodman & Gotlib, 1999). For anxiety, it is theorized that social learning mechanisms, such as acquiring parental attitudes, the imitation of parental actions and parental modeling of anxious or avoidance behavior, mediate the influence of parental psychopathology on the development of anxiety in children (Beesdo et al., 2009).

Maternal and paternal psychopathology have been found to be independently related to depression and anxiety disorders during childhood (Kane & Garber, 2004). Some studies found that maternal depression was more strongly related to offspring internalizing problems than paternal depression (Brennan et al., 2002; Connell & Goodman, 2002). Other studies reported a more pervasive impact of paternal psychopathology on levels of anxiety and depression symptoms in female adolescents (Bosco et al., 2003). These inconsistencies might be explained by a different role of maternal and paternal psychopathology during the lifespan (Bögels & Phares, 2008; Connell & Goodman, 2002). Maternal psychopathology might be more influential during childhood, whereas paternal psychopathology might have more of a detrimental impact on the development of depression and anxiety during adolescence (Connell & Goodman, 2002).

Besides parental psychopathology, negative parenting behavior has been associated with an increased risk for depression and anxiety disorders in offspring (Knappe, Lieb, et al., 2009; Needham, 2008). Traditionally, research has focused on two broad categories of parenting behavior, emotional support and control. A lack of parental emotional support was found to be most strongly associated with offspring depression (McLeod, Weisz, et al., 2007). Parents who show low emotional support may increase children's negative perceptions about themselves and the world, which can lead to negative schemas that increase the vulnerability to depression symptoms (Hammen, 1992). High parental control was strongly associated with offspring anxiety disorders (McLeod, Wood, et al., 2007). Parents who show a high level of control, also conceptualized as low respect for autonomy, may thwart opportunities for normative exploration of the environment and the development of self-efficacy. This, in turn, might increase their offspring's vulnerability for anxiety disorders (Rapee, 1997). In contrast, higher parental respect for autonomy is likely to increase children's confidence and can serve as a protective factor against the development of anxiety disorders (McLeod, Wood, et al., 2007).

Research findings have not been clear about the unique roles of maternal and paternal parenting behavior in the development of adolescent depression and anxiety disorders.

Concerning adolescent depression, several studies found that a lack of maternal and paternal emotional support was equally important (Needham, 2008; Van Roekel, Engels, Verhagen, Goossens, & Scholte, 2011). Concerning anxiety disorders, low paternal respect for autonomy was found to have more impact on the development of anxiety disorders than low maternal respect for autonomy (Bögels & Perotti, 2011; Pereira, Barros, Mendonça, & Muris, 2014). It was also suggested that a lack of maternal and paternal respect for autonomy were more strongly related to offspring's anxiety disorders during childhood than during adolescence (Verhoeven, Bögels, & Van der Bruggen, 2012).

Parenting behavior has also been found to interact with parental psychopathology in predicting depression and anxiety in offspring. Children with parents suffering from psychopathology were found to be more resilient when parents provided high levels of emotional support or high respect for autonomy (Brennan, Le Brocque, & Hammen, 2003). In contrast, a combination of parental psychopathology and low emotional support or low respect for autonomy was found to account for the highest risk of anxiety in offspring (Knappe, Lieb, et al., 2009). Moreover, it has been found that whereas parental psychopathology is crucial for the onset of a disorder, an unfavorable family environment predicts higher persistence of the disorder (Knappe, Beesdo, et al., 2009).

The aim of the present study was to investigate whether maternal and paternal psychopathology and parenting behavior were related to the development of depression and anxiety symptoms in adolescent girls. We hypothesized that higher levels of parental psychopathology and low levels of parental emotional support and respect for autonomy would be related to higher baseline levels and an increase in offspring's depression and anxiety symptoms over time. In addition, we examined whether parental psychopathology and parenting behavior interacted with each other in affecting the development of depression and anxiety symptoms in female adolescents. More specifically, we expected that a combination of high parental psychopathology and low emotional support and low respect for autonomy would be related to a higher baseline level and an increase in depression and anxiety symptoms over time.

Method

Ethics

This study was approved by the medical ethics committee CMO Region Arnhem-Nijmegen, The Netherlands. The trial was registered in the Dutch Trial Register as NTR3720. All participants provided written informed consent.

Procedure

The present study was part of an effectiveness trial on depression and anxiety prevention in adolescents with a high familial risk (Rasing et al., 2013). In total, 862 female adolescents

in first and second grades from five secondary schools participated in the screening with passive consent of their parents. They were screened on depression symptoms using the Children's Depression Inventory 2 (CDI 2) (Kovacs, 2012), on anxiety symptoms using the Spence Children's Anxiety Scale (SCAS) (Spence, 1998), on suicidal ideation using one item from the CDI 2, and on perceived parental psychopathology using seven self-developed items.

Adolescents meeting our eligibility criteria, which included having elevated depression symptoms (CDI 2 ≥ 15) or elevated anxiety symptoms (SCAS ≥ 39), having at least one parent with perceived parental psychopathology, absence of prominent suicidal ideation, and having parental permission to participate, participated in an effectiveness trial for depression and anxiety prevention. For the effectiveness trial, the adolescents were randomly assigned to the intervention condition or control condition, stratified on school, grade and educational level. Participants in the intervention condition attended six weekly meetings of a depression and anxiety prevention program and completed online questionnaires. Participants in the control condition only completed online questionnaires. Parents were informed about the study and invited to fill out four online questionnaires themselves.

For this study, we used data of the effectiveness trial that was gathered at baseline (T0), immediately after the intervention (T1), and follow-up at 6 months (T2) and at 12 months (T3)[1].

Participants

In total, 142 female adolescents with elevated levels of depression and/or anxiety symptoms participated in this study, together with both (77.5%), one (21.8%), or none (0.7%) of their parents. The adolescents, aged 11-14 years (M = 12.87, SD = .69), were in first or second grade of secondary school in various educational levels; vocational training (18.3%), vocational training/ high school training (17.6%), high school training (15.5%), high school training/ pre-university training (30.3%) and pre-university training (18.3%). Most of the adolescents were of Dutch nationality (97.2%), while the remaining adolescents (2.8%) were of different European and non-European origin.

Of the parents, 138 mothers participated (biological mothers, stepmothers and foster mother), aged between 28 and 56 (M = 43.89, SD = 4.41). Most of the mothers were of Dutch nationality (93.5%), while the other mothers (6.5%) were of different European or non-European origin. The 113 fathers who participated (biological father, stepfathers and foster father) were aged between 37 and 61 (M = 47.10, SD = 4.58). Also the majority of

1 It should be noted that in Chapter 3 we used data from measurements at baseline, post-intervention, 6 months, and 12 months follow-up, which we presented as T0, T1, T2, and T3, respectively. In Chapter 5, 6, and 7, we used data from measurements at baseline, after 2 sessions, after 4 sessions, post-intervention, 6 months, and 12 months follow-up, which we presented as T0, T1, T2, T3, T4, and T5, respectively.

fathers were of Dutch nationality (92.0%), while the other fathers (8.0%) were of different European or non-European origin.

Measures

Depression symptoms were assessed using the Dutch version of the CDI 2 (Kovacs, 2012). This questionnaire consisted of 28 items, each consisting of 3 statements graded in severity of 0 (absent), 1 (sometimes present), or 2 (always present). Sample statements include "Sometimes I feel sad", "Most of the times I feel sad", and "I always feel sad". The scores on all 28 items were added together to compute sum scores. Cronbach's alpha was 0.81 on T0, 0.89 at T1, 0.91 at T2 and 0.92 at T3.

Anxiety symptoms were assessed using the Dutch version of the SCAS (Spence, 1998). The questionnaire consisted of 44 items, rating the frequency of anxiety symptoms on a 4-point Likert scale ranging from never to always. Sample statements were "I worry about things", "I am scared of the dark". Sum scores were computed by summing up all items, excluding the filler items 11, 17, 26, 31, 38 and 43 (e.g. 'I am popular amongst other kids my own age'). Cronbach's alpha was 0.82 at T0, 0.89 at T1, 0.89 at T2, and 0.90 at T3.

Perceived parental psychopathology was measured with a self-developed instrument with seven different statements about each parent (Rasing, Creemers, Janssens, & Scholte, 2015). Sample statements were "My parent received treatment from a psychologist or psychiatrist" and "My parent had a depressed mood for more than 2 weeks". With at least one statement answered positively, we defined this as the presence of perceived parental psychopathology. This instrument was used to select adolescents for the study, but was not included in further analyses.

Parental emotional support and respect for autonomy were assessed at baseline using subscales of the Dutch version of the Relational Support Inventory (RSI), 'Warmth versus Hostility' and 'Respect for autonomy versus setting limits' respectively (Scholte, Van Lieshout, & Van Aken, 2001). Each subscale consisted of six items rated on a 5-point scale ranging from "very untrue" to "very true". Both subscales were rated by the adolescents for each parent, resulting in measures of maternal emotional support, maternal respect for autonomy, paternal emotional support and paternal respect for autonomy. Sample statements were "My mother/ father supports me in what I am doing" and "My mother/ father sets strict rules, demands, and limits". Mean scores were computed by averaging all items. Cronbach's alpha was 0.86 for maternal emotional support, 0.73 for maternal respect for autonomy, 0.87 for paternal emotional support and 0.72 for paternal autonomy.

Maternal and paternal psychopathology was assessed at baseline using the Dutch version of the Brief Symptom Inventory (BSI) (Derogatis & Melisaratos, 1983). The BSI is a short version of the Symptom Checklist-90 (SCL-90) (Derogatis, 1975b), and consisted of 53 items rated on a 5-point Likert scale ranging from 0 (not at all) to 4 (extremely). Sample statements were "Feeling no interest in things" and "Feeling tense or keyed up". Questionnaires were rated by each participating parent, resulting in maternal and paternal

psychopathology. Mean scores were computed by averaging all items. Cronbach's alpha was 0.94 for maternal psychopathology and 0.93 for paternal psychopathology

Statistical Analyses

We applied latent growth curve modeling (LGCM) using Mplus (version 6.11) to examine individual development of depression and anxiety symptoms at baseline (i.e., the intercept) and the change in depression and anxiety symptoms over time (i.e., slope). Parameters in the models were estimated by applying the maximum likelihood estimator with robust standard errors (MLR). Robust maximum likelihood methods take violations of non-normality in dependent variables into account by adjusting standard errors and fit indices (Muthén & Muthén, 2010; Yuan & Bentler, 2000). Model fit was assessed by χ^2, CFI (preferably .95 or higher), RMSEA (preferably .05 or lower, and satisfactory between .05 and .08), and SRMR (preferably .08 or lower) (Hu & Bentler, 1998). To examine the development of depression and anxiety symptoms, we first tested growth models without predictors separately for depression and anxiety. The mean of the intercept in these models provides information about the average level of depression or anxiety symptoms at baseline and the mean of the slope represents the average change in depression or anxiety symptoms across the four time-points. Condition (i.e., whether the adolescents participated in the intervention or control condition) was added to these models to account for non-independence due to nesting within experimental condition. Second, we tested in separate models for depression and anxiety whether maternal psychopathology, maternal emotional support, maternal respect for autonomy, paternal psychopathology, paternal emotional support and paternal respect for autonomy were related to baseline level and change of depression or anxiety symptoms over time. This was done by regressing the intercept (baseline level) and slope (change over time) of depression and anxiety symptoms on these parental variables. Again, condition was added to the models to account for non-independence. Subsequently, we tested the interaction effects of parental psychopathology and parental emotional support and the interaction effects of parental psychopathology and parental respect for autonomy on the intercept and slope separately for mothers and fathers. To limit multicollinearity, all predictor variables were centered before computing the interaction terms. Models were tested for depression and anxiety symptoms separately. Again we added condition to the models to account for non-independence.

Results

Descriptive statistics

Means, standard deviations and Pearson correlations of the model variables were calculated and are presented in Table 1 and Table 2. Average levels of depression and anxiety symptoms in adolescents were mild to moderate relative to the norming sample in the Netherlands.

Table 1 Means and Standard Deviations of Model Variables

	M	*SD*
Depression symptoms T0	14.44	6.50
Depression symptoms T1	13.66	8.06
Depression symptoms T2	13.04	8.96
Depression symptoms T3	12.38	9.12
Anxiety symptoms T0	37.77	13.57
Anxiety symptoms T1	33.19	16.03
Anxiety symptoms T2	32.81	16.15
Anxiety symptoms T3	29.39	16.81
Maternal psychopathology	.33	.30
Maternal emotional support	4.18	.80
Maternal respect for autonomy	3.74	.73
Paternal psychopathology	.25	.25
Paternal emotional support	4.08	.88
Paternal respect for autonomy	3.83	.76

Maternal and paternal psychopathology were low. Maternal and paternal emotional support and respect for autonomy were moderate to high. Both depression and anxiety symptoms decreased significantly over time (Wilks' $\lambda = .91$, $F(3,119) = 3.99$, $p = .01$ and Wilks' $\lambda = .69$, $F(3,114) = 16.81$, $p < .001$, respectively). Depression and anxiety symptoms were highly correlated. Depression symptoms were negatively related to maternal and paternal emotional support, which means that depression symptoms tended to be lower at higher levels of emotional support. Anxiety at baseline was negatively related to paternal respect for autonomy, which means that anxiety symptoms tended to be lower at higher levels of respect for autonomy. Maternal emotional support and respect for autonomy were positively related to paternal emotional support and respect for autonomy. Paternal psychopathology was only related negatively to paternal emotional support.

Model findings

The model for depression symptoms and the model for anxiety symptoms without predictors were tested first. The growth of depression symptoms was best fitted in a linear model (χ^2 [7, $N = 138$] = 7.14, CFI = 1.00, RMSEA = .01; 90% CI [.00, .11], and SRMR = .06). The intercept ($B = 13.68$, $p < .001$) and slope ($B = -.67$, $p = .04$) of depression symptoms were both significant, showing that participants on average scored 13.68 on depression symptoms with an expected linear decrease of 0.67 points per year. The growth of anxiety was best fitted in a non-linear model in which the first two factor loading scores for the

Table 2 Correlations Between Model Variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1. Depression symptoms T0	-	.69***	.60***	.61***	.61***	.47***	.40***	.42***	-.03	-.25**	-.15	.13	-.34***	-.14
2. Depression symptoms T1		-	.66***	.61***	.42***	.60***	.52***	.42***	-.05	-.16	-.09	.09	-.26**	-.11
3. Depression symptoms T2			-	.65***	.39***	.46***	.63***	.55***	.13	-.16	-.10	.21*	-.17	-.01
4. Depression symptoms T3				-	.33***	.38***	.41***	.69***	-.01	-.13	-.11	.15	-.18*	-.04
5. Anxiety symptoms T0					-	.72***	.59***	.49***	-.04	-.15	-.16	.04	-.26**	-.15
6. Anxiety symptoms T1						-	.73***	.65***	-.03	-.04	-.03	-.05	-.18	.03
7. Anxiety symptoms T2							-	.68***	-.01	-.08	-.03	.04	-.15	-.03
8. Anxiety symptoms T3								-	.01	-.03	.03	.13	-.13	.05
9. Maternal psychopathology									-	-.01	.01	.15	-.05	.10
10. Maternal emotional support										-	.68***	-.16	.74***	.55***
11. Maternal respect for autonomy											-	-.05	.60***	.64***
12. Paternal psychopathology												-	-.21*	-.04
13. Paternal emotional support													-	.71***
14. Paternal respect for autonomy														-

Note. * $p < .05$ ** $p < .01$ *** $p < .001$

slope factor were set at 0 and 1, respectively, and the remaining factor scores were freely estimated (Bollen & Curran, 2006) (χ^2 [6, N = 138] = 6.28, CFI = 1.00, RMSEA = .02; 90% CI [.00, .11], and SRMR = .04). The intercept (B = 41.67, p < .001) and slope (B = -2.01, p = .003) were both significant, showing that participants scored an average of 41.67 on anxiety symptoms which decreased over time.

Parental psychopathology, parental emotional support and parental respect for autonomy

Subsequently, we tested for main effects of maternal psychopathology, maternal emotional support, maternal respect for autonomy, paternal psychopathology, paternal emotional support, and paternal respect for autonomy in two separate models, one for depression symptoms (χ^2 [19, N = 103] = 29.70, CFI = .95, RMSEA = .07 ; 90% CI [.00, .12], and SRMR = .06) (Table 3) and one for anxiety symptoms (χ^2 [18, N = 103] = 24.00, CFI = .97, RMSEA = .06; 90% CI [.00, .11], and SRMR = .03) (Table 4). Paternal emotional support was related to the baseline level of depression symptoms (B = -3.62, p = .003), indicating that high levels of paternal emotional support were related to lower depression symptoms at baseline. No relationships were found with the rate of change in depression symptoms. Paternal emotional support was also found to be significantly related to baseline anxiety symptoms (B = -5.22, p = .02), indicating that higher levels of paternal emotional support were related to lower anxiety symptoms at baseline. Paternal respect for autonomy was found to be related to the rate of change in anxiety symptoms (B = .89, p = .05), indicating that higher paternal respect for autonomy was related to an increase in anxiety symptoms.

Interaction between parental psychopathology and parental emotional support and respect for autonomy

Additionally, we tested interaction effects between maternal psychopathology and emotional support, maternal psychopathology and respect for autonomy, paternal psychopathology and emotional support, and paternal psychopathology and respect for autonomy on depression and anxiety symptoms, again in two separate models. In the model for depression symptoms (χ^2 [37, N = 103] = 48.49, CFI = .95, RMSEA = .06; 90% CI [.00, .09], and SRMR = .04) (Table 3), none of the interactions between parental psychopathology and parenting behavior were significant predictors of the slope (i.e., there were no three-way interactions with time). Given that the interactions as predictors of slope worsened the model fit considerably and none approached significance, they were dropped from final model. In the model for anxiety symptoms (χ^2 [26, N = 103] = 33.31, CFI = .97, RMSEA = .05; 90% CI [.00, .10], and SRMR = .03) (Table 4), the intercept and slope were regressed on main and interaction effects. The interaction between paternal psychopathology and paternal respect for autonomy was related to depression symptoms at baseline (B = 11.95, p = .03), indicating that the magnitude of the negative association between paternal respect for autonomy and depression symptoms in adolescents was larger at lower levels of paternal

Table 3 Regression Estimates for Initial Level (Intercept) and Rate of Change (Slope) in Adolescents' Depression Symptoms on Main Effects of and Interactions Between Parental Psychopathology, Parental Emotional Support, and Parental Respect for Autonomy

	Model with main effects				Model with main and interaction effects			
Predictor	**Intercept**		**Slope**		**Intercept**		**Slope**	
Maternal psychopathology	.37	(1.99)	-.02	(.52)	-7.34	(10.25)	-	-
Maternal emotional support	.48	(1.29)	-.07	(.29)	.31	(1.53)	-	-
Maternal respect for autonomy	.05	(1.14)	-.08	(.23)	-1.10	(1.90)	-	-
Paternal psychopathology	2.56	(2.90)	.42	(.39)	-36.80	(20.78)	-	-
Paternal emotional support	-3.62**	(1.24)	.22	(.31)	-3.52	(1.86)	-	-
Paternal respect for autonomy	1.22	(1.25)	-.01	(.27)	-0.94	(1.46)	-	-
Maternal psychopathology × maternal emotional support					-.08	(2.79)	-	-
Maternal psychopathology × maternal respect for autonomy					2.31	(3.71)	-	-
Paternal psychopathology × paternal emotional support					-1.64	(4.14)	-	-
Paternal psychopathology × paternal respect for autonomy					11.95*	(5.38)	-	-

Note. Standard errors in parentheses * $p < .05$ ** $p < .01$

Table 4 Regression Estimates for Initial Level (Intercept) and Rate of Change (Slope) in Adolescents' Anxiety Symptoms on Main Effects of and Interactions Between Parental Psychopathology, Parental Emotional Support, and Parental Respect For Autonomy

	Model with main effects				Model with main and interaction effects			
Predictor	**Intercept**		**Slope**		**Intercept**		**Slope**	
Maternal psychopathology	-1.79	(4.22)	-.81	(.91)	-16.25	(16.67)	-2.30	(2.90)
Maternal emotional support	5.02	(2.68)	-.12	(.55)	3.48	(3.92)	.40	(.83)
Maternal respect for autonomy	-4.24	(2.63)	.78	(.47)	-4.95	(4.21)	.27	(.70)
Paternal psychopathology	3.93	(4.92)	-.54	(.89)	-42.51	(30.96)	6.65	(3.80)
Paternal emotional support	-5.22*	(2.20)	-.77	(.52)	-3.50	(3.74)	-1.09	(.81)
Paternal respect for autonomy	.70	(2.37)	.89*	(.45)	-3.22	(3.12)	1.50*	(.63)
Maternal psychopathology × maternal emotional support					3.64	(7.09)	-1.34	(1.57)
Maternal psychopathology × maternal respect for autonomy					.41	(8.09)	1.80	(1.74)
Paternal psychopathology × paternal emotional support					-7.54	(8.07)	1.01	(1.60)
Paternal psychopathology × paternal respect for autonomy					19.77	(10.19)	-2.91	(1.75)

Note. Standard errors in parentheses * $p < .05$

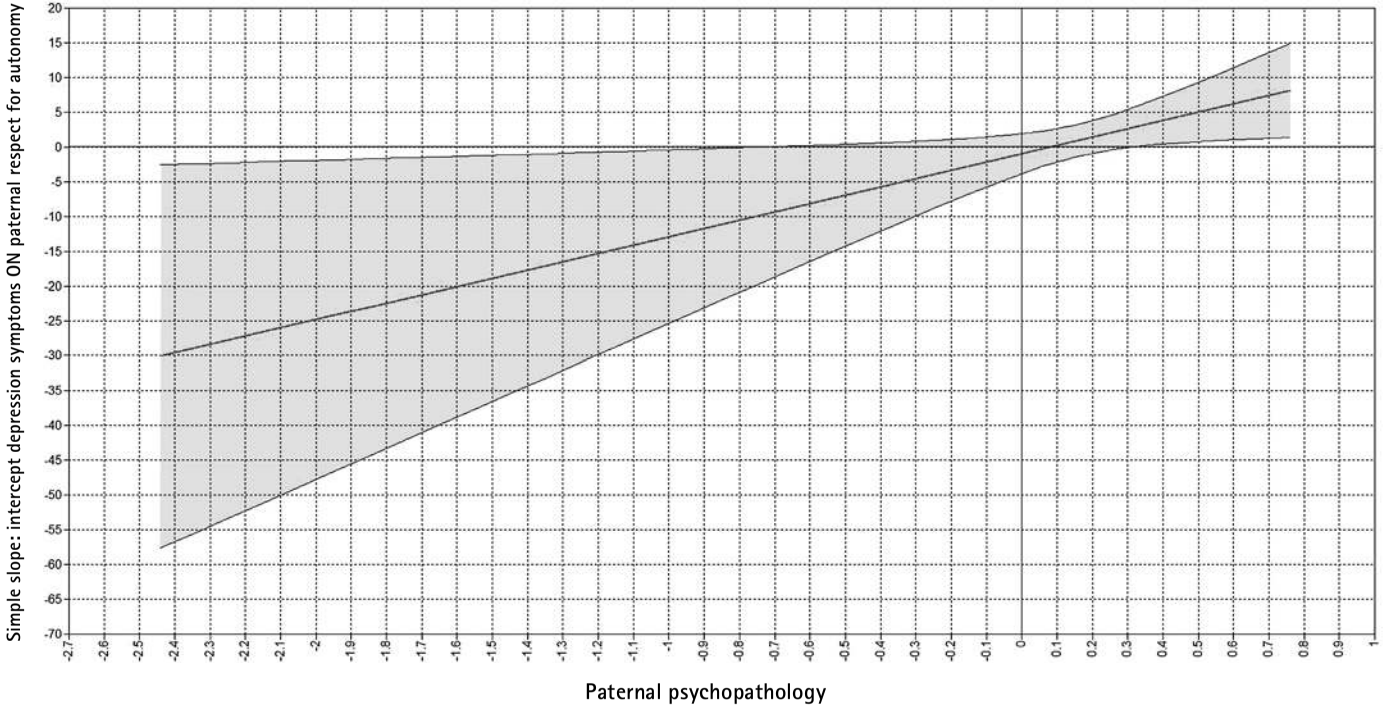


Figure 1 Johnson-Neyman plot of the simple slope of the intercept of development of depression symptoms on paternal respect for autonomy as a function of paternal psychopathology

psychopathology, and that at higher levels of parental psychopathology, paternal respect for autonomy was positively related to depression symptoms. This interaction is also presented in Figure 1, where a Johnson-Neyman plot shows the simple slope corresponding to the regression of the intercept (representing baseline levels of depression symptoms) on respect for autonomy across the range of parental psychopathology (Miller, Stromeyer, & Schwieterman, 2013). The interaction between paternal psychopathology and paternal respect for autonomy on anxiety symptoms at baseline (B = 19.77, p = .052) was not significant. However, it showed a trend towards a similar pattern as in depression symptoms. The results indicated that in lower levels of paternal psychopathology, paternal respect for autonomy is slightly more negatively related to anxiety symptoms, and in higher levels of paternal psychopathology, parental respect for autonomy is slightly positive associated with anxiety symptoms. Nonetheless, this interaction effect was not significant. Further, no interaction effects between parental psychopathology and parental emotional support on depression or anxiety symptoms were found.

Discussion

The aim of the present study was to investigate whether paternal and maternal psychopathology and parenting behavior were related to the level and development of depression and anxiety symptoms in female adolescents. In addition, we examined whether parental psychopathology and parenting behavior interacted with each other in affecting the development of depression and anxiety symptoms.

The findings of our study showed that adolescents who experienced lower paternal emotional support had higher levels of both depression and anxiety symptoms at baseline than adolescents who experienced higher paternal emotional support. Further, our findings showed that higher paternal respect for autonomy was related to an increase of anxiety symptoms over time. In addition, we found a significant interaction effect of paternal psychopathology and paternal respect for autonomy on depression symptoms in adolescents.

Parental psychopathology, parental emotional support and parental respect for autonomy

In the present study, no direct relationships were found between maternal and paternal psychopathology and the baseline level and development of depression and anxiety symptoms of the adolescent girls. This ran counter to our expectations and previous research on the impact of parental psychopathology, suggesting that parental disorders were found to be related to child depression and anxiety symptoms (Lieb et al., 2002; Micco et al., 2009). A possible explanation for not finding the expected relationships might be that the average level of parental psychopathology in our study was low. Therefore, the stressor of parental psychopathology may have been too mild compared to other studies that

examined the relationships between parental clinical depression or anxiety disorders and depression and anxiety symptoms in adolescents (Lieb et al., 2002). In addition, earlier studies found that children are influenced by their perceptions about parental attitudes and behavior, rather than by the actual parental behaviors or the behavior reported by parents (Demo, Small, & Savin-Williams, 1987; Lein, Roosa, & Michaels, 1994). In this study, parents reported about their own psychopathology, and that might have contributed to not finding a relationship, especially when levels of parental psychopathology were low.

Concerning parenting behavior, we found negative relations between paternal emotional support and depression and anxiety symptoms. This is partly in line with our expectations, namely that adolescents of mothers and fathers who provide low levels of support had the highest levels of depression and anxiety symptoms at baseline. Adolescents may develop negative schemas about themselves and the world when parents fail to provide adequate emotional support, which may in turn lead to an increased vulnerability for depression symptoms. Contrary to our expectations, none of the maternal parenting behavior aspects were related to depression and anxiety symptoms in high risk female adolescents. Previous research has been inconsistent about the different impact of maternal and paternal influences on the development of depression and anxiety symptoms. Recent research suggested that paternal influences might be more important during adolescence whereas maternal influences have the most important impact on younger children (Bögels & Phares, 2008; Verhoeven et al., 2012). This might explain why we found no relations between maternal psychopathology or maternal parenting behavior and depression and anxiety symptoms in adolescents.

Findings also showed a positive relationship between paternal respect for autonomy and the development of depression symptoms over time. Earlier studies have been inconsistent about the relationship between respect for autonomy and depression symptoms. Some found a relation whereas other studies reported no relation between parental respect for autonomy and depression symptoms (McLeod, Weisz, et al., 2007). Our study suggests that only paternal emotional support and paternal respect for autonomy were associated with female adolescents' depression and anxiety symptoms.

Interactions between parental psychopathology and parenting behavior

We found an interaction effect between paternal psychopathology and paternal respect for autonomy on depression symptoms in adolescents. The interaction effect indicated that at lower levels of paternal psychopathology, respect for autonomy was negatively related to depression symptoms and when fathers showed high levels of psychopathology, respect for autonomy was positively related to depression symptoms. This means that when fathers had average to lower levels of psychopathology, adolescent baseline symptom levels declined with increasing levels of paternal respect for autonomy. This part is in line with our expectations that a combination of lower parental psychopathology and lower negative parenting behavior would be related to lower depression symptoms in children (Knappe,

Lieb, et al., 2009). In addition we found that when fathers had very high levels of psychopathology, adolescent depressive symptoms increased with increasing levels of paternal respect for autonomy. This is not in line with our expectations. A possible explanation for this might be that adolescents interpret behaviors that are typically indicative of adaptive parenting behavior (respect for autonomy) as being maladaptive (i.e., signs of disinterest or low involvement) in the context of paternal psychopathology. Conversely, adolescents might interpret low respect for autonomy or high levels of paternal control as interest in the adolescent's behavior and establishing rules and consequences when paternal psychopathology is present. These parenting strategies are suggested to reduce the risk on depression and anxiety symptoms in adolescents (Yap, Pilkington, Ryan, Kelly, & Jorm, 2014). We also found an indication that the same relationships holds for anxiety symptoms, that is, that parenting behavior was more negatively related to anxiety symptoms when fathers showed lower parental psychopathology, and that when parental psychopathology was higher, higher respect for autonomy was related to more anxiety symptoms. However, results only showed a trend for these relationships and we have to be careful drawing conclusions.

Limitations

This study had important limitations that need to be taken into account when interpreting the results. First, the average decrease in depression and anxiety symptoms was low. Therefore, it was hard to predict a decrease or increase in depression or anxiety symptoms by using parental psychopathology and parenting behavior as predictors. Moreover, we measured the development of depression and anxiety symptoms for a period slightly over than a year. Future studies should consider examining the development of these symptoms during a longer period of time. In addition, the level of parental psychopathology was very low and therefore maybe too mild to detect an influence on child symptom development. Sharpening the inclusion criteria concerning parental psychopathology might improve results in the future. Finally, adolescents might be exposed to other stressors that are related to depression and anxiety symptoms, such as child abuse (Brown, Cohen, Johnson, & Smailes, 1999), marital divorce (Oldehinkel, Ormel, Veenstra, De Winter, & Verhulst, 2008) or other life events that we did not take into account in this study, but could have influenced the results. Future studies may consider taking these stressors into account.

Conclusion

Our results suggest paternal emotional support was negatively related to baseline level of depression and anxiety symptoms in female adolescents. In addition, we found an interaction effect of paternal respect for autonomy and paternal psychopathology on depression symptoms in adolescents. It indicated that when fathers had average to low levels of psychopathology, paternal respect for autonomy was negatively related to depression symptoms; and when fathers showed high levels of psychopathology, respect for autonomy was positively related to depression symptoms. Our results did not show

impact of maternal psychopathology and parenting behavior on depression or anxiety symptoms in adolescents. Since this study was one of the first studies to examine the influence of the interactions between parental psychopathology and parenting behavior on the development of depression and anxiety symptoms, replication of these findings is needed.

Part 2

Effects of prevention

4

Depression and Anxiety Prevention Based on Cognitive Behavioral Therapy for At-Risk Adolescents: A Meta-Analytic Review

Submitted for publication:

Rasing, S. P. A., Creemers, D. H. M, Janssens, J. M. A. M., & Scholte, R. H. J. Depression and anxiety prevention based on cognitive behavioral therapy for at-risk adolescents: A meta-analytic review.

Abstract

Depression and anxiety disorders are among the most common mental disorders during adolescence. During this life phase, the incidence of these clinical disorders rises dramatically, and even more adolescents suffer from symptoms of depression or anxiety that are just below the clinical threshold. Both clinical and subclinical levels of depression or anxiety symptoms are related to decreased functioning in various areas, such as social and academic functioning. Prevention of depression and anxiety in adolescents is therefore imperative. We conducted a meta-analytic review of the effects of school-based and community-based prevention programs that are based on cognitive behavioral therapy with the primary goal preventing depression, anxiety, or both in high risk adolescents. Articles were obtained by searching databases and hand searching reference lists of relevant articles and reviews. The selection process yielded 23 articles in the meta-analyses. One article reported on two studies and three articles reported on both depression and anxiety. This resulted in a total of 27 studies, 16 on depression and eleven on anxiety. For depression prevention aimed at high risk adolescents, meta-analysis showed a small effect of prevention programs directly after the intervention and at six months follow-up, but no effect at twelve months follow-up. For anxiety prevention aimed at high risk adolescents, no short-term effect was found, nor effects at six or twelve months follow-up. Although effect sizes on depression symptoms were small, current findings support the use of depression prevention programs because mental health of adolescents seems to improve. However, it also indicates that there is still much to be gained for prevention programs aimed at anxiety prevention. Current findings and possibilities for future research are discussed in order to further improve the effectiveness of targeted prevention on internalizing disorders.

Introduction

Depression and anxiety are among the most common mental disorders during adolescence (Kessler et al., 2001; Roza et al., 2003), with a prevalence of 5.6% for depression (Costello et al., 2006; Stallard et al., 2012) and a prevalence of 3% to 20% for anxiety (Albano et al., 2003). Research has shown that among 13 to 17 year old adolescents the lifetime prevalence is estimated to be 12.6% for depression and 32.4% for anxiety disorders (Kessler et al., 2012). Even more adolescents suffer from subclinical levels of depression or anxiety, with 21.4% of the adolescents estimated to suffer from subclinical depression symptoms (Smit, Bohlmeijer, & Cuijpers, 2003). Unfortunately, the number of adolescents suffering from subclinical anxiety is unknown.

Depression and anxiety during adolescence are associated with decreased psychosocial functioning (Birmaher et al., 1996), that is, malfunctioning in social relations (Strauss, Frame, & Forehand, 1987), poor academic performance or school drop-out (Birmaher et al., 1996; Strauss et al., 1987), and an increased risk for substance abuse, other mental health problems, and suicide (Birmaher et al., 1996). Further, adolescents with a depression or anxiety disorder are at considerable risk for developing recurrent depression and anxiety disorders later in life (Aalto-Setala et al., 2002; Copeland et al., 2014; Pine et al., 1999). These negative consequences are comparable between adolescents who meet the criteria for a depression or anxiety disorder and adolescents with subclinical depression and anxiety symptoms (Aalto-Setala et al., 2002; Beesdo et al., 2009; Lewinsohn et al., 2000). Therefore, it is imperative to reduce the incidence of depression and anxiety, but also to prevent further development of depression and anxiety symptoms. Because depression and anxiety symptoms rise dramatically during adolescence, this seems to be the appropriate age to implement prevention, because the risk for depression and anxiety rises during this phase. Further, adolescents are, better than younger children, able to understand the concepts that are being taught in the prevention programs due to their improved reasoning (Hankin et al., 1998; Stice et al., 2009).

Several prevention programs have been developed to prevent depression and anxiety during adolescence. These programs utilize different types of prevention strategies and focus on populations with different risks of developing depression or anxiety (Mrazek & Haggerty, 1994). First, universal prevention programs are intended for all individuals in a population, regardless of their level of risk. These programs have shown mixed results in reducing and preventing depression and anxiety symptoms (Fisak Jr et al., 2011; Horowitz & Garber, 2006; Merry et al., 2012; Sheffield et al., 2006; Teubert & Pinquart, 2011). Second, selective prevention programs are developed to target populations with risk factors, which are known to be related to the onset of depression and anxiety. Selective prevention programs can be aimed at children of parents with psychopathology or children from lower socio-economical environments (Garber et al., 2009; Hyun et al., 2005). Third, indicated prevention programs are developed to target adolescents who already have elevated

symptoms of depression or anxiety, but the symptoms do not qualify for a clinical diagnosis. Results of selective and indicated prevention programs, together also called targeted prevention, have shown to be more promising than universal prevention (Horowitz & Garber, 2006; Stice et al., 2009).

Selective and indicated prevention programs are both aimed at populations with risk factors for depression or anxiety. An important risk factor is parental psychopathology, as children are three times more likely to develop a major depressive disorder and two to seven times more likely to develop an anxiety disorder when their parents suffer from depression or anxiety, respectively (Beidel & Turner, 1997; Bijl et al., 2002; Birmaher et al., 1996; Kashani et al., 1990; Lieb et al., 2002; Merikangas et al., 1998; Micco et al., 2009; Van Dorsselaer et al., 2006).

Another risk factor for the development of adolescent depression and anxiety is the experience of stressful life events during adolescence (Auerbach, Richardt, Kertz, & Eberhart, 2012; Fox, Halpern, Ryan, & Lowe, 2010; Grant, Compas, Thurm, McMahon, & Gipson, 2004). Studies have shown that increased depressive and anxiety symptoms are often preceded by stress (Garber et al., 2002; Ge et al., 1994), and particularly in girls, stress and depression are closely associated during adolescence (Ge et al., 1994; Larson & Ham, 1993; Rudolph & Hammen, 1999). Further, the existence of subclinical symptoms of depression or anxiety, or undiagnosed clinical levels of these disorders, is a risk factor for the development of a clinical disorder (Clarke et al., 1995; Lowry-Webster, Barrett, & Dadds, 2001; Weissman, Wickramaratne, et al., 2006).

Whereas *prevention* in high risk populations aims to decrease the likelihood of the onset of a depressive or anxiety disorder or decrease in symptoms, *treatment* aims to reduce existing symptoms (Garber & Weersing, 2010). In targeting symptoms, prevention seems to parallel treatment in these goals. As we know from reviews of meta-analyses (Butler et al., 2006; Hofmann et al., 2012), cognitive behavioral therapy demonstrated to be an effective treatment for a wide range of psychological problems, including depression and anxiety. Based on the overlap in goals (i.e., decrease symptoms) between treatment and prevention, techniques of the cognitive behavioral approach seem to be suitable techniques to use in the prevention of depression and anxiety in high-risk adolescents. Several prevention programs for depression and anxiety are based on cognitive behavioral theories and in this meta-analysis we examined whether prevention programs based on the cognitive behavioral approach are effective in preventing depression and anxiety in high-risk adolescents.

Several reviews and meta-analyses have been conducted to evaluate the effectiveness of depression prevention programs and anxiety prevention programs for adolescents (Calear & Christensen, 2010; Christensen et al., 2010; Merry et al., 2012; Neil & Christensen, 2009; Teubert & Pinquart, 2011). These meta-analyses were focused on either depression or anxiety, and on prevention in general and not on high risk populations. In contrast to those, the present study focuses specifically on depression and anxiety prevention programs based on cognitive behavioral therapy approaches for adolescents with a high risk on developing depression or anxiety.

This review intended to identify and describe school-based and community-based prevention programs based on cognitive behavioral therapy with a primary goal of preventing depression, anxiety, or both in adolescents at risk for developing these disorders. Furthermore, we aimed to determine their effectiveness in reducing symptoms of depression and anxiety in the short-term and in the long-term.

Method

The study design will be reported in accordance with the PRISMA Statement for Reporting Systematic Reviews and Meta-Analyses of Studies That Evaluate Health Care Interventions (Liberati et al., 2009).

Search and screening

Databases Medline (from 1946 to July 2013), PsycInfo (from 1906 to July 2013), Embase (from 1974 to July 2013) and Eric (from 1965 to July 2013) were electronically searched using the key search terms "adolescen* OR teen-age* OR youth*", "prevent* OR early intervent*", "depress* OR anx* OR mood OR internali#ing" AND "at risk OR high risk".

Using these specified terms, we identified 1669 articles. After removing 482 duplicates, 1187 articles remained. First, titles and abstracts of the 1187 remaining articles were screened by the first author to determine their relevance to the review. This resulted in the exclusion of 1110 articles (913 articles included populations with other (mental) health problems than depression and/or anxiety; 26 articles were reviews, systematic reviews or meta-analyses; 10 articles reported on drug trials; 97 articles reported on other aspects than effectiveness; and 64 documents were not peer reviewed articles); thus 77 articles remained. Additionally, 27 articles were obtained by hand searching reference lists of relevant articles and reviews. Figure 1 demonstrates the flow chart of selected articles.

Second, of the 104 articles that were identified, the title, abstract and method section of each article were systematically reviewed and considered for inclusion. The inclusion criteria for the current review were that (1) the reported intervention aimed to prevent or reduce depression and/or anxiety symptoms, (2) study participants included adolescents aged 11-18, (3) the prevention program targeted a population at risk (i.e., the program was selective or indicated prevention), (4) the intervention was given in groups, (5) the intervention was delivered face to face, (6) the intervention was based on cognitive behavioral therapy, (7) the study design was a randomized controlled trial, and (8) the study was published in a peer-reviewed, English language journal. Rating the articles was done by four independent reviewers (postgraduate, post-doc and two full professors) in which each article was handled by two reviewers. Agreement between raters was between 79% and 91% (Cohen's Kappa = .58 - .80). Differences between reviewers were resolved by consensus. Main reasons for excluding articles can be found in Figure 1. Finally during data extraction,

we learned that six articles reported no measurements on depression or anxiety at baseline, 6-month or 12-month follow-up but only longer duration of follow-up and five articles showed a lack of data, and these were also excluded. This resulted in 23 articles included in the meta-analyses.

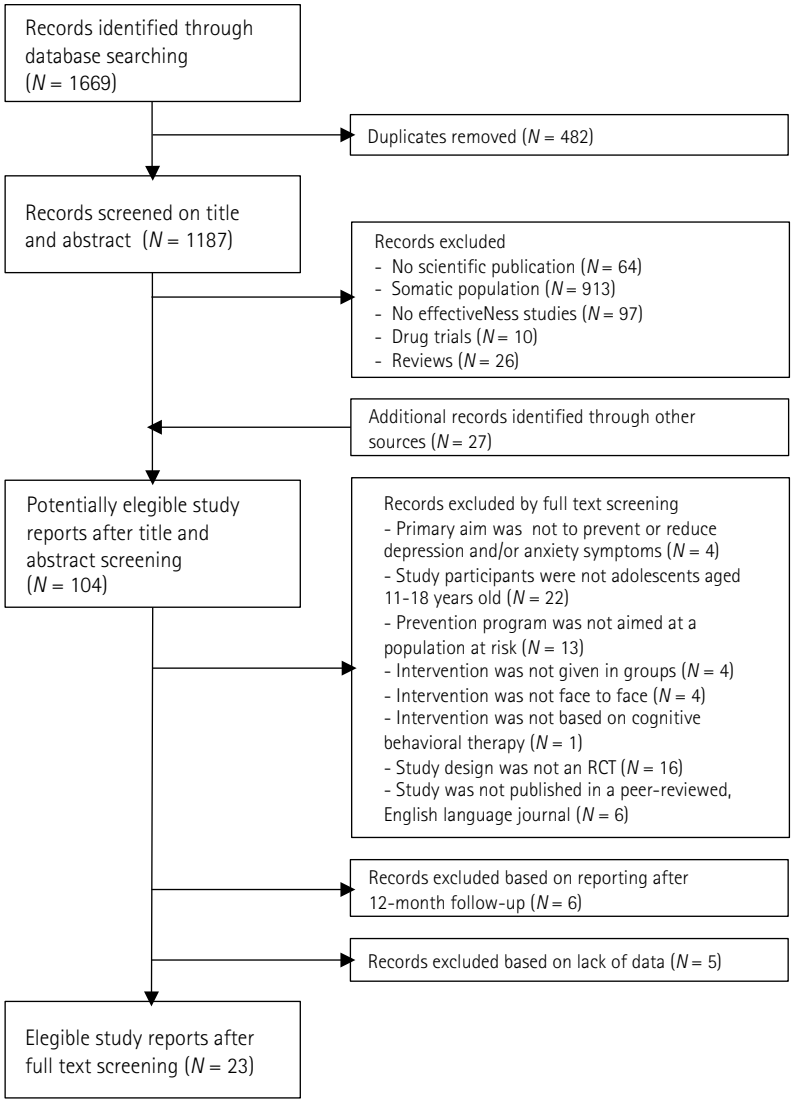


Figure 1 Flow chart

Coding of studies

All studies were coded for type of prevention, sample, number of participants, age of participants, percentage of females, group size, number and duration of sessions, characteristics of control condition, randomization, outcome measure, means and standard deviations at post-intervention, means and standard deviations at six months follow-up, and means and standard deviations at twelve months follow-up. Coding was done by two independent raters (both postgraduates). Overall agreement between raters was 95%. Differences between raters were resolved through discussion which led to total agreement.

Statistical analyses

We used the Cochrane Collaboration software Review Manager (RevMan version 5.2) to conduct meta-analyses. We analyzed the data with a random effects model, calculating the standardized mean differences (SMDs), also known as effect size Cohen's *d* (Cohen, 1977), and 95% confidence intervals (CIs). Cohen's effect sizes are generally categorized as small (0.2 – 0.5), moderate (0.5 – 0.8), or large (larger than 0.8). We based the effects sizes on the change in self-reported depression scores and anxiety scores. Heterogeneity between trials was assessed using Tau^2 (estimated standard deviation of underlying effects across studies), Chi^2 (whether observed differences in results are compatible with chance alone), and I^2 statistic (in which higher values indicate higher heterogeneity), which is defined by the percentage of total variation across trials due to heterogeneity rather than chance (Higgins & Thompson, 2002).

Results

Sample characteristics

The sample consisted of 23 articles. One article reported on two studies (Barrett et al., 2005), and three articles reported both on depression as well as anxiety (Gillham, Reivich, et al., 2006; Manassis et al., 2010). This resulted in 27 studies, 16 on depression and 11 on anxiety.

In the depression study sample (see Table 1), 14 studies used indicated prevention, one used selective prevention, and one used a combination of selective and indicated prevention. Twelve studies were conducted in schools, four were community-based. The size of the intervention groups varied from small groups of three to 13 adolescents in twelve studies, to large groups (school classes) in one study. Three studies did not report group size. Intervention intensity ranged from four sessions of 60 minutes (total of 240 minutes) to 12 sessions of 120 minutes (total of 1440 minutes). The control conditions were non-intervention groups (i.e., adolescents received no care or guidance at all) in four studies, usual care groups (i.e., adolescents received personal care or guidance if requested) in eight studies, waiting list groups (i.e., adolescents received the intervention after completion) in two

Table 1 Depression Prevention Programs

Study: country	Summary of intervention	Type of prevention; school– or community–based	Sample	Mean age; Age range
(Clarke et al., 1995): US	Coping with stress course teaches at-risk adolescents cognitive techniques to identify and challenge negative thoughts that may contribute to the development of affective disorders	Indicated; school-based	Children or adolescents with elevated but subdiagnostic depressive symptomatology (CES-D > 24)	15,3; 15-16
(Clarke et al., 2001): US	Cognitive behavioral program that teaches adolescents cognitive restructuring techniques to identify and challenge negative thoughts, with a focus on beliefs related to having a depressed parent	Indicated; community-based	Adolescents with elevated levels of depressive symptoms (CES-D > 24)	14,6; 13-18
(Dobson et al., 2010): Canada	Coping with stress program is based on CBT and teaches adolescents how to use cognitive restructuring techniques to identify and challenge negative thoughts	Indicated; school-based	Students with elevated depression symptoms (CES-D > 24), but no current or past clinical depression	15,3; 13-18
(Garber et al., 2009): US	Cognitive behavioral prevention program that teaches adolescents skills to identify and challenge negative thoughts and problem-solving skills	Selective and indicated; community-based	Adolescents with at least one parent with major depressive episode and with subsyndromal depressive symptoms	14,8; 13-17
(Gillham et al., 2012): US	Penn Resiliency Program is based on CBT and teaches participants the link between thoughts, feelings and behavior and skills for solving interpersonal problems and coping with stress	Indicated; school-based	Students with high levels of depression (on CDI and/ or RADS-2)	Unknown; 10-15
(Gillham, Hamilton, Freres, Patton, & Gallop, 2006): US	Penn Resiliency Program is based on CBT and teaches participants the link between thoughts, feelings and behavior and skills for solving interpersonal problems and coping with stress	Indicated; community-based	Adolescents with elevated depression scores (CDI > 50th percentile)	Unknown; 11-12
(Gillham, Reivich, et al., 2006): US	Penn Resiliency Program is based on CBT and teaches participants the link between thoughts, feelings and behavior and skills for solving interpersonal problems and coping with stress	Indicated; school-based	Students with the highest level of symptoms (on combined CDI and RCMAS Z-scores)	Unknown; 12-13

N per group	Characteristics of sessions	N (intervention/ control); % females; randomization	Control group	Outcome measure	Effect size post-intervention	Effect size 6 months follow-up	Effect size 12 months follow-up
6-11	15 sessions; 45 minutes; three times per week	150 (76/ 74); 70,0%; participant randomization	Usual care	CES-D	-.34	.07	.01
6-11	15 sessions; 60 minutes; unknown	94 (45/ 49); 59,6%; participant randomization	Usual care	CES-D	-.46	a	-.53
12-13	15 sessions; 45 minutes; unknown	46 (25/ 21); 69,6%; participant randomization	Active: Let's Talk	CDI	.13	.12	a
3-10	14 sessions; 90 minutes; eight weekly and six monthly	316 (159/ 157); 58,5%; participant randomization	Usual care	CES-D	-.30	-.31	a
Unknown	10 sessions; 90 minutes; weekly	266 (137/ 129); 48,0%; participant randomization	Non-intervention	CDI	-.26	-.15	a
Unknown	12 sessions; 90 minutes; weekly	271 (147/ 124); 53,1%; participant randomization	Usual care	CDI	.02	-.22	-.24
10-12	8 sessions; 90 minutes; weekly	44 (22/ 22); 70,5%; participant randomization	Non-intervention	CDI	-.09	-.63	-.45

Table 1 Continued

Study: country	Summary of intervention	Type of prevention; school- or community-based	Sample	Mean age; Age range
(Hyun et al., 2005): South Korea	Program bases on CBT teaches adolescents to alter their thought and interpretation of the situation and facilitates the development of the individual's adaptive behavior	Selective; community-based	Male runaway adolescents and residing in a shelter	15,2; Unknown
(Kowalenko, 2005): Australia	ACE program aims to build resilience and increase positive coping in young people using cognitive-behavioral and interpersonal techniques	Indicated; school-based	Students with elevated symptoms of depression (CDI ≥ 18)	14,6; 13-16
(Manassis et al., 2010): Canada	Feelings club is a CBT program focuses on recognizing and managing negative feelings and maladaptive thoughts using cognitive restructuring	Indicated; school-based	Children with elevated depressive symptoms (MASC or CDI t > 60)	Unknown; 8-12
(Puskar, Sereika, & Tusaie-Mumford, 2003): US	Teaching kids to cope aims to prevent depression and to maximize coping by focusing on self-esteem, stress and coping	Selective and indicated; school-based	Students from rural area with elevated symptoms of depression (RADS > 60)	16,0; 14-18
(Roberts, Kane, Thomson, Bishop, & Hart, 2003): Australia	Penn Prevention Program is CBT based and teaches children coping strategies to counteract cognitive distortions and deficiencies	Indicated; school-based	Children with elevated depression symptoms (highest scores on CDI per class)	11,9; 11-13
(Sheffield et al., 2006): Australia	The program for preventing depression integrated two major cognitive-behavioral components, namely cognitive restructuring and problem-solving skills training	Indicated; school-based	Students with elevated depressive symptoms (top 20% combined standardized CDI and CES-D scores)	14,3; 13-15
(Stallard et al., 2012): UK	The resourceful adolescent program is based on CBT and develops skills such as emotion-regulation, coping mechanisms, and thinking styles to protect against depression	Indicated; school-based	Students classified as at risk based on elevated levels of depression (SMFQ > 5)	14,1; 12-16
(Stice, Burton, Bearman, & Rohde, 2007): US	The Blues Group is a CBT based program and uses motivational enhancement exercises, strategic self-presentation, behavioral techniques, and group activities	Indicated; school-based	Students with elevated symptoms of depression (CES-D ≥ 20)	18,4; 15-22

N per group	Characteristics of sessions	N (intervention/ control); % females; randomization	Control group	Outcome measure	Effect size post-intervention	Effect size 6 months follow-up	Effect size 12 months follow-up
6-8	8 sessions; 90 minutes; weekly	32 (16/ 16); 0%; participant randomization	Non-intervention	BDI	-.70	a	a
8-10	8 sessions; 90 minutes; weekly	143 (87/ 56); 65,0%; School randomization	Waiting list	CDI	-.55	a	a
5-10	12 sessions; 60 minutes; weekly	148 (78/ 70); 43,2%; participant randomization	Active: Activity group	CDI	.01	a	-.19
Unknown	10 sessions; 45 minutes; weekly	89 (46/ 43); 82,0%; participant randomization within schools	Usual care	RADS	-.47	-.49	-.30
Small groups	12 sessions; 120 minutes; weekly	52 (25/ 27) ; 49,7%; School randomization	Usual care	CDI	.02	-.23	a
8-10	8 sessions; 90 minutes; weekly	283 (134/ 149); 69,0%; School randomization	Non-intervention	CDI	-.14	.04	.08
School class	9 sessions; 50-60 minutes; weekly	767 (393/ 374); 66,0%; Randomization per schoolyear	Usual care	SMFQ	a	a	.23
6-10	4 sessions; 60 minutes; weekly	117 (50/ 67); 70,0%; participant randomization	Waiting list	BDI	-.71	-.13	a

Table 1 Continued

Study: country	Summary of intervention	Type of prevention; school- or community-based	Sample	Mean age; Age range
(Stice, Rohde, Gau, & Wade, 2010): US	CBT based program for the prevention of depression uses motivational enhancement exercises, strategic self-presentation, behavioral techniques, and group activities	Indicated; school-based	Students with elevated depressive symptoms (CES-D ≥ 20)	15,6; 14-19

[a] no assessment at this time in the study

Table 2 Anxiety Prevention Programs

Study: country	Summary of intervention	Type of prevention; school- or community-based	Sample	Mean age; age range
(Balle & Tortella-Feliu, 2010): Spain	Educational program about anxiety, the basics of some emotional regulation techniques, and gradual exposure to feared situations	Indicated; school-based	Children with high anxiety sensitivity, but no current mental health disorders or treatment	13,6; 11-17
(Barrett et al., 2005) (group 1): Australia	FRIENDS program is based on CBT which assist children in learning important skills and techniques to cope with and manage anxiety and emotional distress	Indicated; school-based	High risk adolescents selected from a universal sample with elevated anxiety symptoms (SCAS > 32)	Unknown; 9-11
(Barrett et al., 2005) (group 2): Australia	FRIENDS program is based on CBT which assist children in learning important skills and techniques to cope with and manage anxiety and emotional distress	Indicated; school-based	High risk adolescents selected from a universal sample with elevated anxiety symptoms (SCAS > 32)	Unknown; 14-16
(Dadds, Spence, Holland, Barrett, & Laurens, 1997): Australia	Intervention, based on The Coping Koala, teaches children strategies for coping with anxiety and reinforces individual effort and change	Indicated; school-based	Adolescents identified by teachers as having anxiety disorders or showing elevated symptoms of anxiety (RCMAS ≥ 20)	9,4; 7-14

N per group	Characteristics of sessions	N (intervention/ control); % females; randomization	Control group	Outcome measure	Effect size post-intervention	Effect size 6 months follow-up	Effect size 12 months follow-up
3-10	6 sessions; 60 minutes; weekly	173 (89/ 84); 56,0%; participant randomization stratified for school and gender	Usual care	BDI	-.61	-.53	-.11

N per group	Characteristics of sessions	N (intervention group/ control group; % females; randomization	Control group	Outcome measure	Effect size post-intervention	Effect size 6 months follow-up	Effect size 12 months follow-up
10-12	6 sessions; 45 minutes; two times per week	92 (47/ 45); 61,0%; participant randomization	Waiting list	SCAS	.09	-.21	a
20-30	10 sessions (and two boosters); 45-60 minutes; weekly	47 (23/ 24); 76,6%; School randomization	Non-intervention	SCAS	-.05	a	-.56
20-30	10 sessions (and two boosters); 45-60 minutes; weekly	19 (12/ 7); 73,7%; School randomization	Non-intervention	SCAS	.24	a	-.13
5-12	10 sessions; 60-120 minutes; weekly	128 (61/ 67); 75,8%; School randomization	Non-intervention	RCMAS	.01	-.05	a

Table 2 Continued

Study: country	Summary of intervention	Type of prevention; school- or community-based	Sample	Mean age; age range
(Gillham et al., 2012): US	Penn Resiliency Program is based on CBT and teaches participants the link between thoughts, feelings and behavior and skills for solving interpersonal problems and coping with stress	Indicated; school-based	Students with high levels of depression (on CDI and/ or RADS-2)	Unknown; 10-15
(Gillham, Reivich, et al., 2006): US	Penn Resiliency Program is based on CBT and teaches participants the link between thoughts, feelings and behavior and skills for solving interpersonal problems and coping with stress	Indicated; school-based	Students with the highest level of symptoms (on combined CDI and RCMAS Z-scores)	Unknown; 12-13
(Kiselica, Baker, Thomas, & Reedy, 1994): US	The program based on Meichenbaum's stress inoculation training includes assertiveness training to provide participants with coping skills for dealing with external stressors	Indicated; school-based	Students with elevated anxiety symptoms (highest scores on STAI-A per class)	Unknown; 15
(Lock & Barrett, 2003): Australia	FRIENDS program is based on CBT which assist children in learning important skills and techniques to cope with and manage anxiety and emotional distress	Indicated; school-based	Adolescents with elevated anxiety symptoms (SCAS > 42)	Unknown; 9-16
(Lowry-Webster et al., 2001): Australia	The FRIENDS program is CBT based and teaches children strategies for coping with anxiety and challenge situations	Indicated; school-based	Students with elevated symptoms of anxiety (SCAS > 42)	Unknown; 10-13
(Manassis et al., 2010): Canada	Feelings club is a CBT program focuses on recognizing and managing negative feelings and maladaptive thoughts using cognitive restructuring	Indicated; school-based	Children with elevated internalizing symptoms (MASC or CDI t > 60)	Unknown; 8-12
(Simon, Bogels, & Voncken, 2011): Netherlands	The CBT based intervention teaches the children develop their own fear hierarchy, cognitive restructuring, task concentration training, and relaxation to decrease anxiety	Indicated; community-based	Children with elevated symptoms of anxiety (top 15% scores on SCARED)	10,1; 8-13

[a] no assessment at this time in the study

N per group	Characteristics of sessions	N (intervention group/ control group; % females; randomization	Control group	Outcome measure	Effect size post-intervention	Effect size 6 months follow-up	Effect size 12 months follow-up
Unknown	10 sessions; 90 minutes; weekly	266 (137/ 129); 48,0%; participant randomization	Non-intervention	RCMAS	-.17	-.10	a
10-12	8 sessions; 90 minutes; weekly	44 (22/ 22); 70,5%; participant randomization	Non-intervention	RCMAS	-.07	-.63	-.80
6	8 sessions; 60 minutes; weekly	48 (24/ 24); 45,8%; participant randomization	Active: guidance class	STAI	-.74	a	a
Unknown	10 sessions (and two boosters); 70 minutes; weekly	66 (35/ 31); 75,8%; School randomization	Non-intervention	SCAS	.20	a	.10
School class	10 sessions; 60 minutes; weekly	108 (77/ 31); 52,9%; Class randomization	Waiting list	SCAS	-.80	a	a
5-10	12 sessions; 60 minutes; weekly	148 (78/ 70); 43,2%; participant randomization	Active: activity class	MASC	-.06	a	-.06
6-8	8 sessions; 90 minutes; weekly	114 (58/ 56); 57,0%; participant randomization	Non-intervention	SCARED	a	a	-.13

studies, and active control conditions in two studies (Let's talk program and activity groups). In one study (Stallard et al., 2012), the intervention was given in school classes as universal prevention, and afterwards the data for high-risk adolescents were analyzed separately. In the other 15 studies, the intervention was given to selected groups based on features of the participants (i.e., elevated levels of depression).

In the anxiety study sample (see Table 2), eleven studies used indicated prevention. Ten studies were conducted in schools, one was community-based. Intervention group sizes varied from small groups of five to twelve adolescents in six studies to large groups (school classes of 30 students) in three studies. Two studies did not report group size. The length of the interventions varied from 6 sessions of 45 minutes (total of 270 minutes) to 10 sessions of 120 minutes (total of 1200 minutes). The control conditions were non-intervention groups in seven studies, waiting list groups in two studies, and active control conditions in two studies (guidance class and activity class). In three studies (Barrett et al., 2005; Lowry-Webster et al., 2001), school classes were given a universal preventive intervention and the data for at-risk adolescents were analyzed separately. In the other eight studies, the intervention was presented to selected groups (i.e., elevated levels of anxiety).

Outcome

Meta-analyses were conducted on the data presented in Tables 1 and 2. Means, standard deviations, number of participants, weight of the study and effect sizes are presented in Figure 2 for depression studies and Figure 3 for anxiety studies. Both are accompanied by a forest plot reporting effect sizes and confidence intervals per study (respectively square points and horizontal lines) and the pooled result for all studies (diamond). Measures of heterogeneity Tau^2, χ^2 and I^2 were calculated and presented in Figure 2 and 3.

Concerning depression prevention, the meta-analysis showed that there was a small effect in improving depression symptoms post-intervention (15 studies; d = -.29; 95% CI [-.42, -.16]). The meta-analytic Z-test showed a significant effect (Z = 4.42, p < .001). The heterogeneity test showed that the results of the various studies were moderately heterogeneous (Tau^2 = .03, χ^2 = 26.39, p = .02; I^2 = 47%).

The effect size of depression prevention six months after the intervention (11 studies) was also small (d = -.21; 95% CI [-.34, -.08]). The meta-analytic Z-test showed a significant effect (Z = 3.10, p < .01). The heterogeneity test revealed that the included studies were low heterogeneous (Tau^2 = .02, χ^2 = 15.69, p = .11; I^2 = 36%).

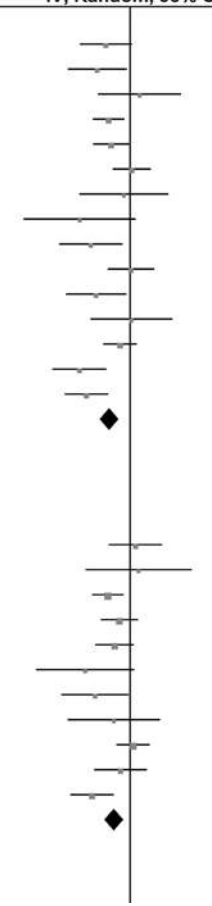
Depression prevention showed no effect (d = -.11; 95% CI [-.29, .07]) 12 months after the intervention (9 studies). The meta-analytic Z-test showed a non-significant effect (Z = 1.23, p = .22). The heterogeneity test showed that the various studies yielded moderate heterogeneous results (Tau^2 =.04, χ^2 = 22.61, p = .004; I^2 = 65%).

For anxiety prevention, the effect size (d = -.15; 95% CI [-.35, .05]) for the post-intervention effects (10 studies) showed there was no effect. The meta-analytic Z-test showed a non-significant effect (Z = 1.50, p = .13). The heterogeneity test showed that the results of the included studies were low heterogeneous (Tau^2 = .05, χ^2 = 17.49, p = .04; I^2 = 49%).

Anxiety prevention showed no effect (d = - .14; 95% CI [-.33, .04]) six months after the intervention (4 studies). The meta-analytic Z-test showed a non-significant effect (Z = 1.53, p = .13). The heterogeneity test revealed that the results of the included studies were homogeneous (Tau^2 = .00, χ^2 = 2.36, p = .50; I^2 = 0%).

Anxiety prevention 12 months after the intervention (6 studies) showed a small effect size (d = -.20; 95% CI [-.42, .02]). The meta-analytic Z-test showed a non-significant effect (Z = 1.78, p = .07). The heterogeneity test showed that the various studies yielded homogeneous results (Tau^2 = .01, χ^2 = 5.50, p = .36; I^2 = 9%).

Study or Subgroup	Intervention Mean	SD	Total	Control Mean	SD	Total	Weight	Std. Mean Difference IV, Random, 95% CI
Post-intervention								
Clarke et al. (1995)	17.88	9.3	52	21.67	12.3	68	6.9%	-0.34 [-0.70, 0.02]
Clarke et al. (2001)	17.8	8.7	45	22.5	11.3	49	6.0%	-0.46 [-0.87, -0.05]
Dobson et al (2010)	35.83	7.04	25	34.86	7.59	21	3.7%	0.13 [-0.45, 0.71]
Garber et al. (2009)	12.3	8.7	159	15.1	9.8	157	10.6%	-0.30 [-0.52, -0.08]
Gillham et al. (2012)	7.37	7.13	127	9.45	8.59	118	9.7%	-0.26 [-0.52, -0.01]
Gillham, Hamilton et al. (2006)	11.55	8.21	112	11.37	7.9	104	9.3%	0.02 [-0.24, 0.29]
Gillham, Reivich et al. (2006)	9.02	6.01	20	9.65	7.9	20	3.4%	-0.09 [-0.71, 0.53]
Hyun et al. (2005)	9.64	8.76	14	17.46	12.57	13	2.3%	-0.70 [-1.49, 0.08]
Kowalenko et al. (2005)	17.9	7.6	41	22.2	7.9	41	5.5%	-0.55 [-0.99, -0.11]
Manassis et al. (2010)	49.11	9	76	49	9.19	69	7.8%	0.01 [-0.31, 0.34]
Puskar et al. (2003)	63.85	13.48	46	69.68	10.6	43	5.8%	-0.47 [-0.90, -0.05]
Roberts et al (2003)	18.32	10.23	23	18.12	11.46	24	3.8%	0.02 [-0.55, 0.59]
Sheffield et al. (2006)	17.63	10.51	134	19.1	10.25	149	10.3%	-0.14 [-0.37, 0.09]
Stice et al. (2007)	13.14	8.14	50	18.51	7.02	67	6.6%	-0.71 [-1.09, -0.33]
Stice et al. (2010)	10.71	9.07	89	16.48	9.8	84	8.3%	-0.61 [-0.91, -0.30]
Subtotal (95% CI)			**1013**			**1027**	**100.0%**	**-0.29 [-0.42, -0.16]**
Heterogeneity: Tau² = 0.03; Chi² = 26.39, df = 14 (P = 0.02); I² = 47%								
Test for overall effect: Z = 4.42 (P < 0.00001)								
6-month follow-up								
Clarke et al. (1995)	19.35	10	52	18.55	11.2	60	8.6%	0.07 [-0.30, 0.45]
Dobson et al (2010)	34.46	5.58	14	33.71	6.72	14	2.8%	0.12 [-0.62, 0.86]
Garber et al. (2009)	10.9	8.4	159	13.5	8.3	157	15.4%	-0.31 [-0.53, -0.09]
Gillham et al. (2012)	6.97	6.98	114	8.18	8.88	113	13.2%	-0.15 [-0.41, 0.11]
Gillham, Hamilton et al. (2006)	9.87	7.79	111	11.53	7.49	101	12.7%	-0.22 [-0.49, 0.05]
Gillham, Reivich et al. (2006)	7.32	6.62	20	11.87	7.65	15	3.2%	-0.63 [-1.32, 0.06]
Puskar et al. (2003)	60.89	12.24	36	66.21	8.88	35	6.0%	-0.49 [-0.96, -0.02]
Roberts et al (2003)	11.27	8.48	18	13.34	9.24	19	3.6%	-0.23 [-0.88, 0.42]
Sheffield et al. (2006)	17.71	10.7	134	17.29	9.29	149	14.7%	0.04 [-0.19, 0.28]
Stice et al. (2007)	11.15	8.22	50	12.28	8.45	67	8.7%	-0.13 [-0.50, 0.23]
Stice et al. (2010)	11.87	10.06	89	17.48	10.98	84	11.1%	-0.53 [-0.83, -0.23]
Subtotal (95% CI)			**797**			**814**	**100.0%**	**-0.21 [-0.34, -0.08]**
Heterogeneity: Tau² = 0.02; Chi² = 15.69, df = 10 (P = 0.11); I² = 36%								
Test for overall effect: Z = 3.10 (P = 0.002)								



12-month follow-up								
Clarke et al. (1995)	18.4	9.3	52	18.34	11	58	10.3%	0.01 [-0.37, 0.38]
Clarke et al. (2001)	15.1	10	45	21.5	13.6	49	9.4%	-0.53 [-0.94, -0.12]
Gillham, Hamilton et al. (2006)	10.21	7.57	102	12.23	9.03	91	12.8%	-0.24 [-0.53, 0.04]
Gillham, Reivich et al. (2006)	8.31	9.81	17	14.21	15.41	14	4.6%	-0.45 [-1.17, 0.26]
Manassis et al. (2010)	47.04	9.35	76	48.74	8.66	69	11.6%	-0.19 [-0.51, 0.14]
Puskar et al. (2003)	61.11	12.72	36	64.97	12.51	35	8.2%	-0.30 [-0.77, 0.17]
Sheffield et al. (2006)	15.89	10.38	134	15.09	8.6	149	14.3%	0.08 [-0.15, 0.32]
Stallard et al. (2012)	8.22	6.45	296	6.81	5.7	308	16.5%	0.23 [0.07, 0.39]
Stice et al. (2010)	13.24	11.38	89	14.45	10.04	84	12.4%	-0.11 [-0.41, 0.19]
Subtotal (95% CI)			**847**			**857**	**100.0%**	**-0.11 [-0.29, 0.07]**

Heterogeneity: Tau² = 0.04; Chi² = 22.61, df = 8 (P = 0.004); I² = 65%
Test for overall effect: Z = 1.23 (P = 0.22)

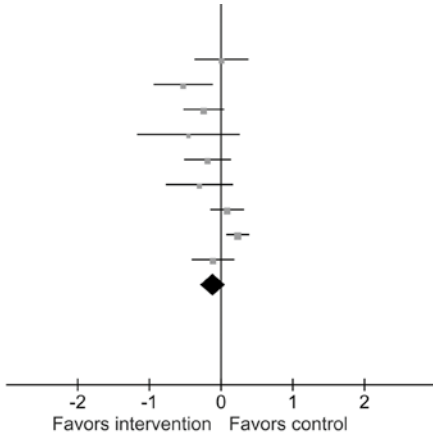


Figure 2 Forest plots of effects of depression prevention

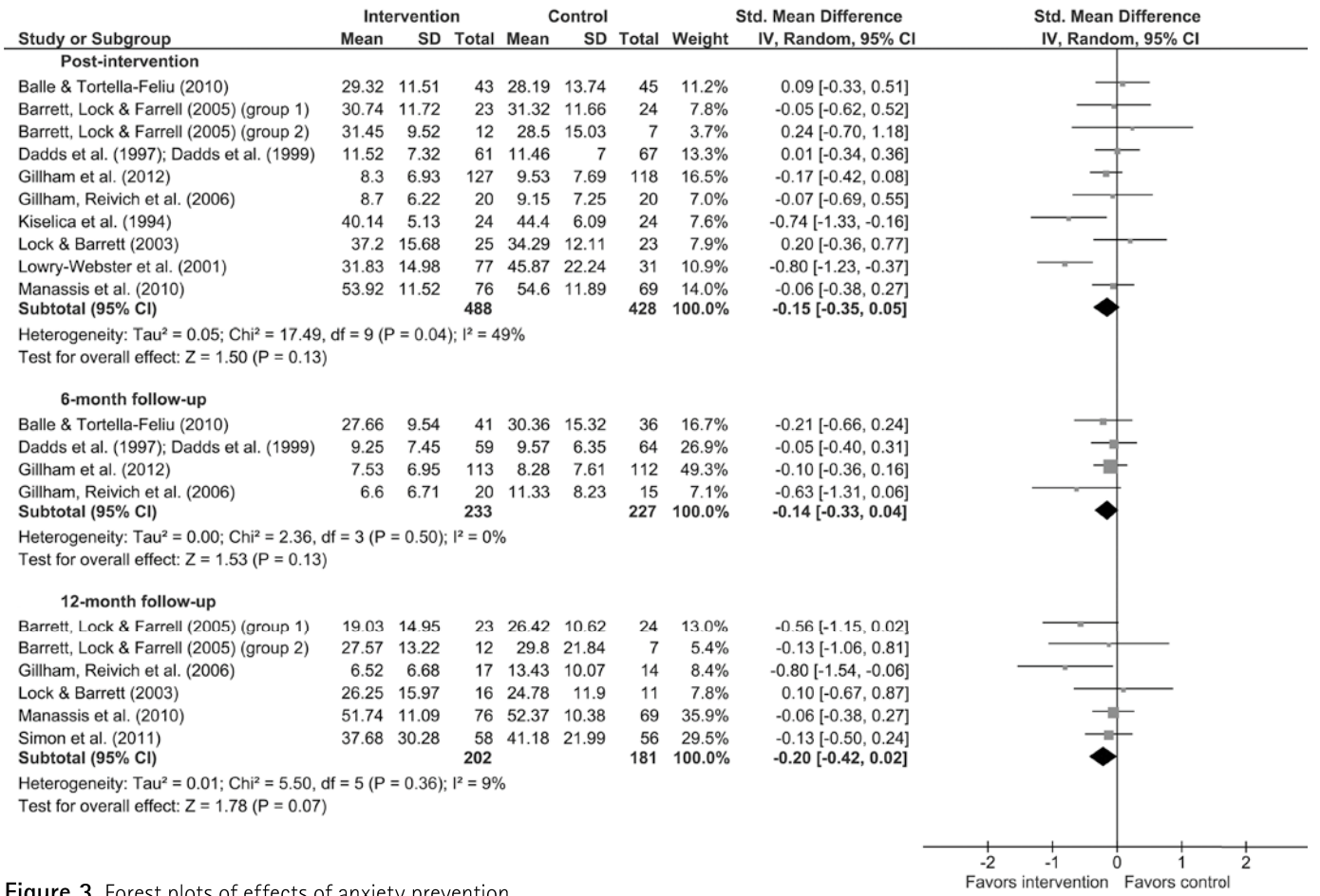


Figure 3 Forest plots of effects of anxiety prevention

Discussion

This review described school-based and community-based prevention programs based on cognitive behavioral therapy with the primary goal of preventing depression and anxiety symptoms in adolescents at risk for developing these disorders. Furthermore, we determined the effectiveness in reducing symptoms of depression and anxiety directly after the intervention and at six and twelve months after the intervention.

The findings of our study revealed that selective and indicated depression prevention programs using techniques of cognitive behavioral therapy not only decrease symptoms of depression immediately after the intervention, but also maintain the effect six months after the intervention. However, the effects did not seem to last to twelve months after the intervention. In other words, the depression prevention generally decreases the symptoms of depression up to six months follow-up and, therefore, possibly decreases the probability of the onset of a full-blown depressive disorder. For anxiety prevention, we did not find evidence for effects directly after the intervention, six months or twelve months after the intervention. Anxiety prevention did not seem to reduce anxiety symptoms.

The findings of our study partially reflect the findings from previous research. Previous reviews and meta-analyses also reported the effects of depression prevention directly after the intervention and after six months (Calear & Christensen, 2010; Horowitz & Garber, 2006; Merry et al., 2012), although they reported on a combination of several approaches of prevention in their studies, such as cognitive behavioral therapy, interpersonal therapy and mindfulness therapy. The studies that we included in the meta-analyses utilized a prevention program based on the principles of cognitive behavioral therapy. Cognitive behavioral therapy proved to be effective in treating depression in children and adolescents (Lewinsohn & Clarke, 1999; Michael & Crowley, 2002; Weisz, McCarty, & Valeri, 2006). It is likely that when these techniques are used in indicated and selective prevention of depression in adolescents, the results of these interventions are positive. Therefore, we can conclude that preventive interventions in high-risk populations, namely adolescents with substantial symptoms of depression, are meaningful for prevention of depression. In contrast to another review (Merry et al., 2012), we found no effects of depression prevention twelve months after the intervention. Our findings imply that depression prevention leads to short term symptoms reduction, and this reduction is still noted at six months but not at twelve months. The absence of an effect at twelve months after the intervention was also seen in the treatment of depression, where follow-ups of one year or more showed essentially no treatment effect (Weisz et al., 2006). There are several explanations for not finding an effect twelve months after the intervention. It is known that occasional long-term prevention sessions, so-called booster sessions, reduce the likelihood of relapse of depressive symptoms (Kroll, Harrington, Jayson, Fraser, & Gowers, 1996). Without these booster sessions, the effectiveness of a prevention program might diminish during the following period. Most studies included in our meta-analyses did not use booster sessions

following the preventive intervention, which might explain why depression prevention programs showed no effect twelve months after the intervention. Further, depression is known for its recurrent course and its fluctuations in level of depression symptoms (Judd et al., 1998; Kennedy & Paykel, 2004; Van Rijsbergen, 2014). This implies that in some adolescents depression symptoms might recur after some time, despite the preventive intervention they received. Especially when there is no change in the risk factors, the risk for depression remains high.

In contrast to other studies, we found that anxiety prevention programs did not show significant effects directly after the intervention, at six months or twelve months after the intervention. (Christensen et al., 2010; Neil & Christensen, 2009). It is noteworthy that the effect sizes in this study appear to be smaller compared to the effect sizes found in other reviews and meta-analyses. The main difference between our findings and those in other studies (Christensen et al., 2010; Neil & Christensen, 2009) concerns the inclusion criteria of the populations of the included studies. The previous reviews included studies on all types of anxiety prevention programs, namely studies with universal, selective, and indicated prevention programs. We, on the other hand, focused on adolescents at risk and included only studies with selective and indicated prevention programs using techniques of cognitive behavioral therapy. This means that the severity of anxiety symptoms in our meta-analytic review was higher compared to other reviews. To illustrate, adolescents in the studies we included reported elevated levels of anxiety, up to clinical levels. The samples and the severity of their symptoms might play a role in the difference in outcome of this study and other review studies. The content of the programs might explain the fact that we did not find any effects of prevention programs on anxiety on an at-risk population. Although the prevention programs that focused on anxiety used cognitive behavioral techniques, they were mainly based on social skills training (Dadds et al., 1997), relaxation exercises (Balle & Tortella-Feliu, 2010), and cognitive restructuring (Lock & Barrett, 2003). These techniques might be more effective in universal populations without symptoms of anxiety, as mentioned in earlier review studies (Christensen et al., 2010; Neil & Christensen, 2009), than in at-risk population, as in our study. The preventive interventions used in the studies that we reviewed may have lacked strong enough techniques for the prevention of anxiety in at-risk populations. The content of selective and indicated prevention programs for adolescents with elevated levels of anxiety that are almost reaching clinical levels of anxiety should presumably be more similar to the treatment of anxiety. Therefore, we suggest that preventive interventions for anxiety in at-risk adolescents should not only use cognitive restructuring techniques, but should include exposure techniques and cognitive behavioral therapy as is done in treatment of anxiety disorders (Cartwright-Hatton et al., 2004; Compton et al., 2004; Davis, May, & Whiting, 2011; Rapee, Schniering, & Hudson, 2009).

This meta-analytic review focused on the selective and indicated prevention for depression and anxiety using cognitive behavioral therapy, whereas other studies included also universal prevention and other prevention techniques. This allowed us to draw

conclusions about the cognitive behavioral therapy based prevention of depression and anxiety in adolescents with high risk for developing these disorders. We included only randomized controlled trials in our meta-analyses, which increased the internal validity of the studies in this meta-analysis. With this in mind, the results of the current studies can be interpreted with confidence. Limitations of the present study can be found in the studies included. The number of participants in some studies was low and differed largely in size across studies. We would like to indicate that the variations in sample size, in techniques used in the interventions, and in inclusion criteria resulted in lower heterogeneity between the anxiety prevention studies than between depression prevention studies. We think, however, that the similarities between the studies are larger than the methodological diversity. Furthermore, the outcomes might have been influenced by the bias in selective reporting, as only studies with positive results were likely to be published. A small number of studies was included in the meta-analyses; therefore, potential moderating variables were not tested. Consequently, we cannot draw any conclusions about the influence of, for example, the size of the intervention groups, duration and intensity of the prevention programs, and the selection of the participants in the prevention programs, neither about sociodemographic characteristics, family history of depression, and level of elevated symptoms.

Conclusions

This review presents evidence that cognitive behavioral therapy based prevention of depression in groups for high-risk adolescents is effective in the short term. These at-risk groups mostly have elevated levels of depression, and with these prevention programs their symptoms, and also the risk on a full-blown depressive disorder, reduce. For anxiety, cognitive behavioral therapy based prevention programs appear not to be effective. The findings of the current meta-analytic review support the implementation of depression prevention programs because mental health of adolescents seems to improve. But it also indicates that there is still much to be gained for prevention programs aimed at anxiety prevention. We focused on prevention of depression and anxiety in adolescents at risk using cognitive behavioral techniques. We, therefore, did not include universal prevention studies and studies using other techniques in our meta-analyses. Consequently, we could not compare universal prevention and targeted prevention, and we cannot conclude on about prevention programs based on for example interpersonal therapy or mindfulness therapy. Evaluations of the cost-effectiveness of depression and anxiety prevention should also be done, as they are currently lacking in the prevention literature. We suggest that, based on the similarities between treatment of depressive and anxiety disorder and the targeted prevention, the content of selective and indicated prevention programs for adolescents with subclinical depression and anxiety could profit from including techniques that have shown to be effective in the treatment of depression and anxiety.

5

Effectiveness of Depression and Anxiety Prevention in Adolescents with High Familial Risk: Study Protocol for a Randomized Controlled Trial

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Abstract

Depression and anxiety disorders during adolescence can have detrimental consequences. Both disorders are related to negative outcome in various areas during adolescence and are also predictive of depression and anxiety disorders later in life. Especially parental psychopathology and being female are risk factors that increase the probability of developing one of these disorders during adolescence. Research has shown that prevention programs have promising results, especially for adolescents who have these risk factors. Therefore, in this study, we will focus on the effectiveness of a prevention program 'Een Sprong Vooruit' [A Leap Forward] that has been developed for adolescent girls with a familial risk of depression and/or anxiety. We designed a randomized controlled trial to test the effectiveness of an indicated and selective prevention program aimed at depression and anxiety in adolescent girls. Adolescents aged between 11 and 15 years old with depressive and/or anxiety symptoms and with parents who show indicators of parental psychopathology will be randomly assigned to the experimental (N = 80) or control groups (N = 80). Participants in the experimental group will follow a preventive intervention, consisting of six sessions of 90 minutes each. All participants will complete baseline, intervention phase 1 (after session 2), intervention phase 2 (after session 4), post-intervention, 6 month follow-up, and 12 month follow-up assessments. Furthermore, parents will be asked to complete assessments at baseline, post-intervention, and 12-month follow-up. Primary outcome will be depressive symptoms. Secondary outcomes will be anxiety symptoms, suicidal ideation, response style, negative cognitive errors, parental emotional support and parental control, parental psychopathology, parenting stress and adolescents' depression and anxiety symptoms according to the parents. This paper described the study designed to evaluate a program for preventing depression and/or anxiety in high-risk adolescents over a 12-month follow-up period. If the program showed to be effective in reducing symptoms of depression and anxiety and preventing adolescents from developing clinical levels of these disorders, our results would be relevant to practice. Thus, the intervention could be used on a large scale. Moreover, this study aims to contribute to the evidence-based prevention of depression and anxiety of adolescents.

Introduction

Depression and anxiety disorders are a major public health concern. The prevalence of clinical depression in adolescents is approximately 5.6% (Costello et al., 2006). Regarding anxiety disorders, prevalence rates in children and adolescents vary from 3% to 20% (Albano et al., 2003). In addition, lifetime prevalence of depression in adolescents is suggested to be 28.8% and of anxiety disorders, lifetime prevalence is estimated at 16.6% (Kessler et al., 2005). These numbers do not even include children and adolescents with increased levels of depressive and anxiety symptoms, which are just below the diagnostic threshold (Kessler et al., 2005).

Depression and anxiety disorders during adolescence can have detrimental consequences. That is, both disorders at this age are related to negative outcomes in various areas, such as poor psychosocial functioning (Birmaher et al., 1996), impairment in social relations (Strauss et al., 1987), poor academic performance (Birmaher et al., 1996; Strauss et al., 1987), and an increased risk for substance abuse (Birmaher et al., 1996). Furthermore, adolescent depression and anxiety are both associated with depression, anxiety disorders, and even suicide later in life (Beesdo et al., 2009; Fergusson & Woodward, 2002). Not only clinical levels of depression or anxiety have negative outcomes, also subclinical levels of depression or anxiety are associated with significant distress and dysfunction, and they are also a risk for future disorders (Aalto-Setala et al., 2002; Beesdo et al., 2009; Lewinsohn et al., 2000).

It is important to examine the role of risk factors to understand the onset and maintenance of psychopathology during adolescence. With respect to depression and anxiety disorders, several factors increase the probability of developing one of these disorders. The first important risk factor is parental psychopathology, as children of parents with a depression are three times more likely to develop an episode of a major depressive disorder compared to children whose parents do not have this disorder (Bijl et al., 2002; Birmaher et al., 1996; Lieb et al., 2002). With regard to anxiety, 68% of children of parents with this disorder show symptoms of an actual anxiety disorder (Capps et al., 1996). Furthermore, these children are two to seven times more likely to develop an anxiety disorder compared to children of parents without an anxiety disorder (Beidel & Turner, 1997; Kashani et al., 1990; Merikangas et al., 1998; Micco et al., 2009; Van Dorsselaer et al., 2006). Moreover, children of parents with an anxiety disorder, a depression, or both, have significantly higher odds of developing depression or anxiety compared to children of parents without these disorders (Micco et al., 2009). The first explanation is that these children experience more stress at home and that their parents have less parenting skills. Nevertheless, the relations between particular patterns of depression and anxiety symptoms in adolescents on the one hand and parental psychopathology, parenting characteristics, parental emotional support, and parental control on the other hand remain to be further specified. The second explanation is that a genetic predisposition increases the probability

of developing mood and anxiety disorders. Theorists assume that depression and anxiety disorders are familial disorders and partly the result of a genetic heredity. However, the largest part of evidence suggests that depressive and anxiety symptoms rather than specific disorders are heritable. This genetic influence accounts for 30-50% of the variance in these symptoms (Eley, 2001; Kendler, Neale, Kessler, Heath, & Eaves, 1992; Merikangas et al., 2003; Ogliari et al., 2010; Rice, 2009; Sullivan et al., 2000). The second risk factor in the development of depression and anxiety during adolescence is gender. Females have been identified as being at a higher risk of developing depression and anxiety disorders (Chaplin et al., 2009; De Graaf et al., 2010; Kessler et al., 1994). Differences between boys and girls seem to arise during puberty, and particularly adolescent girls are vulnerable to develop a clinical depression (Nolen-Hoeksema & Girgus, 1994) and often show subclinical depressive and anxiety symptoms (Angold et al., 2002).

Given the high prevalence rates of depression and anxiety disorders in children and adolescents as well as the effect these disorders have on their current and future psychosocial functioning, many treatment programs have been developed. Most of these programs are based on cognitive behavioral therapy (CBT), which addresses, for example, dysfunctional emotions, maladaptive behaviors, and cognitive processes. That is, these negative cognitions generally result in higher levels of depression and anxiety. This implies that response style and cognitive errors are assumed to mediate the relation between prevention programs and symptoms of depression and anxiety. CBT is known to be effective in treating and preventing depression and anxiety in adolescents (Cartwright-Hatton et al., 2004; Essau, 2004; Wijnhoven et al., 2014) individually as well as in groups (David-Ferdon & Kaslow, 2008; Ishikawa, Okajima, Matsuoka, & Sakano, 2007). Importantly, it has been shown that the coping skills learned from a CBT intervention could mediate the relation between family stressors and the actual development of adolescent depression or anxiety disorders (Sander & McCarty, 2005).

Adolescents with symptoms of depression and/or anxiety who have parents with elevated levels of depression and/or anxiety have a relative high risk to develop depression or anxiety disorders later in life. Because children of parents with a mental illness often develop maladaptive self-schemas as a consequence of negative family-related events, CBT would be a preferred intervention (Sander & McCarty, 2005). Despite these facts, to date, only a limited number of studies have been conducted to examine the prevention of depression and/or anxiety disorders in adolescent offspring of depressed or anxious parents by means of CBT (Beardslee, Gladstone, Wright, & Cooper, 2003; Clarke et al., 2001; Garber et al., 2009; Ginsburg, 2009). The findings showed that preventive interventions had positive outcomes, such as decreased symptomatology. However, none of these preventive interventions targeted both depression and anxiety, although these disorders have a high comorbidity, simultaneous development (Axelson & Birmaher, 2001; Cole, Peeke, Martin, Truglio, & Seroczynski, 1998), and a considerable overlap in symptoms, especially during adolescence (Reardon & Williams, 2007). Since the etiology and pathogenesis of depression

and anxiety also overlap substantially, we argue that the prevention programs should focus on both disorders simultaneously, specifically during adolescence. Therefore, common factors can be prevented, and there might be greater potential to obtain benefits (Dozois et al., 2009; Hoek, 2012).

The current study combines indicated prevention and selective prevention (Mrazek & Haggerty, 1994), which respectively refer to the prevention provided to adolescents with elevated levels of depression or anxiety and to the prevention provided to targeted subgroups distinguished by specific traits, in our case children who have parents with elevated levels of depression or anxiety. As mentioned above, these adolescents have an extra high risk to develop a depression or anxiety disorder. The adolescents allocated to the experimental group will undergo a preventive intervention that consists of six weekly meetings of 90 minutes each. It will focus on acquiring knowledge of depression and anxiety, learning to recognize and distinguish emotions, dealing with difficult situations, solving social problems, and creating and using a supportive social network. Throughout the program, the adolescents will complete various exercises based on cognitive behavioral therapy, behavioral activation, and exposure.

The primary aim of this study is to test the effectiveness of the program 'Een Sprong Vooruit' [A Leap Forward] in preventing depressive and anxiety symptoms among adolescents with elevated symptoms of depression and anxiety who have parents with elevated symptoms of depression and/or anxiety. The secondary aim is to determine whether response style and cognitive errors mediate and whether parental psychopathology, parenting stress, parental emotional support and parental control moderate the effectiveness of the prevention program.

Methods

The study design will be reported in accordance with the CONSORT 2010 statement for reporting parallel group randomized trials (Schulz, Altman, Moher, & Consort Group, 2010). The medical ethics committee CMO Region Arnhem-Nijmegen, The Netherlands, has approved this study.

Design

The present study is designed as a non-blinded randomized controlled trial (RCT) with two conditions (intervention versus control). Adolescents will be screened for depression, anxiety, suicide ideation, and parental psychopathology in order to select high-risk adolescents. The program is designed for indicated and selective prevention (Mrazek & Haggerty, 1994); therefore, adolescents with elevated symptoms of depression and/or anxiety as well as with a parent who has elevated levels of depression or anxiety symptoms will be selected and recruited.

After the completion of screening and recruitment, participants will be randomized to either intervention or control group. The assessments will be conducted at baseline (T0), during the intervention phase after session 2 (T1), during the intervention phase after session 4 (T2), at post-intervention (T3), at 6-month months follow-up (T4), and at 12-month follow-up (T5). The overall study design is captured in Figure 1.

Participants' eligibility

Adolescents with depressive and/or anxiety symptoms who are willing to participate in a prevention program will be eligible for this study. Inclusion criteria are age between 11-15 years old; increased levels of depression and/or anxiety; at least one of the parents showing symptoms of a depression or anxiety disorder; and both adolescent and parents having sufficient knowledge of the Dutch language. Exclusion criteria are the absence of parental permission; adolescent already receiving treatment for mental health problems; and presence of prominent suicidal ideation (score 2 on CDI item: a desire to kill oneself, if given the chance).

Recruitment

Students in their first and second year of secondary school, from vocational training up to pre-university level, will receive written information about the screening and the study. After receiving passive consent, these students will be screened for depression, anxiety, and parental psychopathology. Students who meet the inclusion criteria will be randomly assigned to one of the two conditions. After that, the adolescents and their parents will receive verbal and written information about the study and written informed consent from adolescents and parents will be obtained

Sample size

The sample size is based on the expected difference (Cohen's d = 0.50) on the primary outcome variable between the experimental and control group at 12 months follow-up (based on a meta-analytic review (Horowitz & Garber, 2006)). With an alpha of 0.05 and a power of 0.80 of a two-tailed test, 64 participants will need to be included in each condition. The data will be analyzed according to the intent-to-treat principle. Multiple imputations will be used for missing observations at follow-ups. We intend to increase the sample size by 25% to compensate for dropout and potential loss of power due to clustering of data, resulting in 160 participants (80 in experimental condition and 80 in control condition).

The program

'Een Sprong Vooruit' [A Leap Forward], which consists of 6 sessions, each 90 minutes long. In the first session, the participants will learn about emotions, anxiety, and depression and their experiences with their parent's mental health problem. The adolescents learn about emotions they experience, and they will learn to recognize them. During this program, they

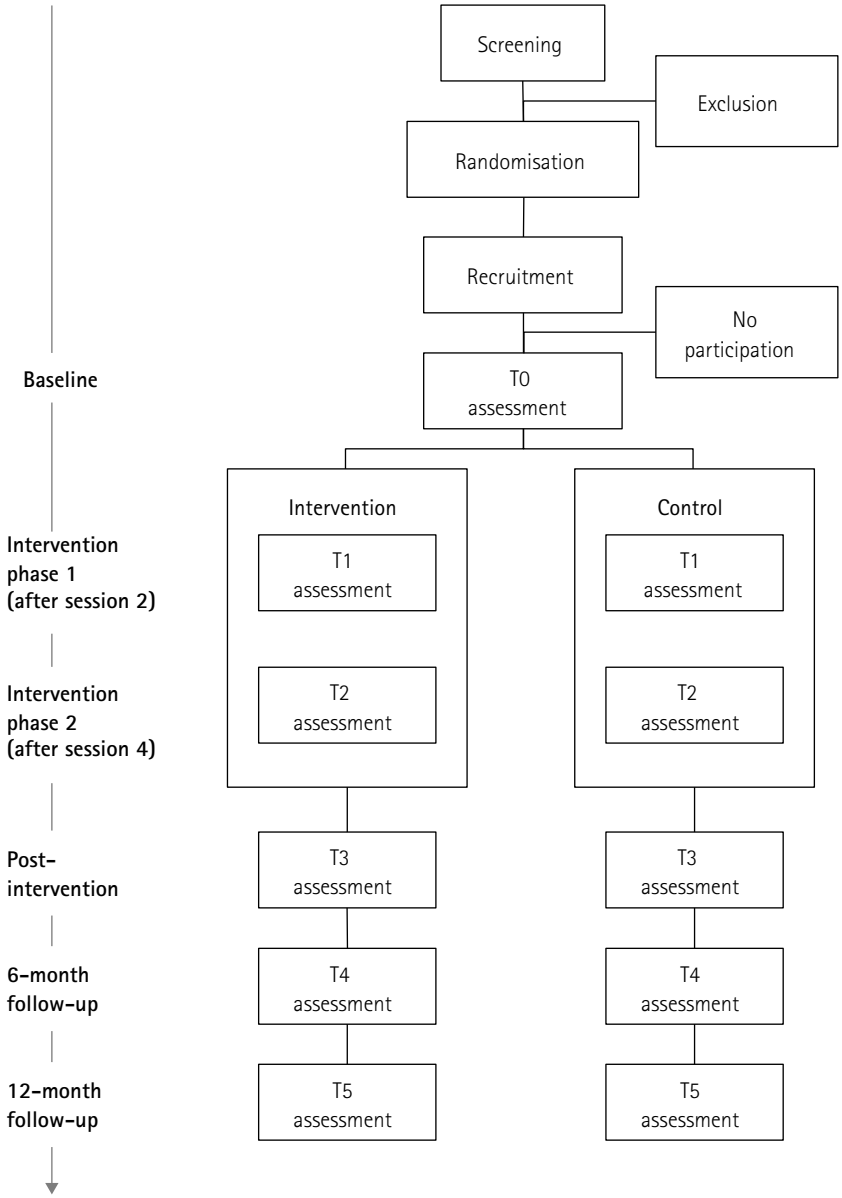

Figure 1 Overall study design

will use a schedule to examine the relations among activating events, beliefs, emotional consequences and behavioral consequences. In the second session, the adolescents will learn about the relationship between activating events, beliefs, and emotional consequences. Beliefs can be optimistic or pessimistic and play a major role in the emotional consequences. The adolescents in the study will learn to recognize pessimistic beliefs. In the third session, adolescents will be taught several strategies to replace the pessimistic beliefs with optimistic beliefs. They will learn to recognize the (negative) pattern of their beliefs, replace them, and prove that the alternative belief is true. In the fourth session, adolescents will learn that in some situations, the beliefs cannot be replaced or changed and that they have to change their behavior to influence the emotional consequences. Therefore, the assignments will comprise both pleasant and less pleasant activities. Furthermore, they will learn to organize and structure large tasks. In the fifth session, the adolescents will learn about anxiety and the development of fear over time. By means of exposure, they will learn to divide fearful activities into small steps, practice those steps, and experience the decrease in anxiety. In the sixth and last session, the fearful tasks will be evaluated and practiced again. Furthermore, the adolescents will learn that social support sometimes makes things easier. They will practice asking people in their social environment for help. Finally, the adolescents will take a look at the future and talk about how they can influence it.

Study outcome measures

For an elaborate overview of study outcome measures, see Table 1.

Screening measures

To assess the eligibility to participate, students will be screened for depressive and anxiety symptoms and suicidal ideation using the Children's Depression Inventory 2 (CDI 2) and Spence Children Anxiety Scale (SCAS). Furthermore, students will be asked to complete some questions about parental psychopathology.

Primary outcome measure

Depression in children and adolescents will be measured with the CDI 2 (Kovacs, 2012). The CDI 2 contains 28 items, each consisting of three statements graded in severity from 0 to 2. This instrument has good internal consistency and convergent validity (Bae, 2012).

Secondary outcome measures

Anxiety will be measured with the SCAS (Spence, 1997). This 44-item self-report questionnaire rates the frequency of symptoms on a 4-point scale ranging from "never" to "always". The scale has demonstrated high internal consistency, high concurrent validity, and adequate test-retest reliability across both child (Spence, 1998) and adolescent (Spence, Barrett, & Turner, 2003) samples.

Table 1 Overview of Assessments

	Screening	T0	T1	T2	T3	T4	T5
Adolescent							
Depression (CDI 2)	X	X	X	X	X	X	X
Anxiety (SCAS)	X	X	X	X	X	X	X
Suicidal ideation (CDI 2)	X	X	X	X	X	X	X
Response style (CRSQ)		X	X	X	X	X	X
Cognitive errors (CNCEQ-R)		X	X	X	X	X	X
Relational Support (RSI)		X			X		X
Parent[1]							
Parental psychopathology (BSI)		X					
Parenting stress (OBVL)		X			X		X
Child depression (CDI 2 P)		X			X		X
Child anxiety (SCAS-P)		X			X		X

[1] Both parents will complete the questionnaires. Only parents who have absolutely no contact with children will be excluded.

Suicidal ideation will be measured with the suicide item of the CDI 2 (Kovacs, 2012), with scores of 0 ("no suicidal ideation"), 1 ("thoughts of wanting to kill oneself, with no intent to do so"), and 2 ("a desire to kill oneself, if given the chance"). Participants with a score of 2 will be excluded from the study and referred to a therapist within an institute of mental health care or a general practitioner.

Response style will be measured with the Children's Response Style Questionnaire (CRSQ) (Abela, Brozina, & Haigh, 2002). This questionnaire consists of three subscales: ruminative response, distracting response, and problem solving. Items are rated on a 4-point scale ranging from "almost never" to "almost always". The scale has moderate levels of internal consistency.

Negative cognitive errors will be measured with the Children's Negative Cognitive Errors Questionnaire – Revised (CNCEQ-R) (Maric, Heyne, van Widenfelt, & Westenberg, 2011), which divides negative cognitive errors into five error categories, namely 'over-generalizing', 'personalizing', 'selective abstraction', 'threat conclusion', and 'underestimation of the ability to cope'. Items are rated on a 5-point scale from "not at all like I would think" to "almost exactly like I would think". The questionnaire is a revised version of the CNCEQ, which has a good internal consistency and test-retest reliability (Leitenberg, Yost, & Carroll-Wilson, 1986).

Parental emotional support and parental control will be measured respectively with 'Warmth versus Hostility' and 'Respect for Autonomy versus setting limits', which are two subscales of the Relational Support Inventory (RSI) (Scholte et al., 2001). Both subscales have six items rated on a 5-point scale ranging from "very untrue" to "very true". The reliability varies from low to moderate.

Parental psychopathology will be measured with the Brief Symptom Inventory (BSI) (Derogatis, 1975a). The BSI is a short version (53 items) of the Symptom Checklist-90 (SCL-90) (Derogatis, 1975b) divided into 9 symptom dimensions: Somatization, Obsessive-compulsive symptoms, Interpersonal sensitivity, Depression, Anxiety, Hostility, Phobic anxiety, Paranoid ideation, and Psychoticism. Items are rated on a 5-point scale from "not at all" to "extremely". Both international (Derogatis & Melisaratos, 1983) and national studies (De Beurs & Zitman, 2005) showed that the questionnaire has good psychometric properties.

Parenting stress will be measured with the Opvoeding Belasting Vragenlijst (OBVL) [Parenting Stress Questionnaire] (Vermulst, Kroes, De Meyer, Nguyen, & Veerman, 2012). It is used to determine how parents experience their child, how they interact with their child, and how they feel about their own health. The questionnaire consists of 34 items scored on a 4-point scale ranging from "not true" to "very true". This instrument has a good reliability and satisfactory validity (Vermulst et al., 2012).

Depression of the child according to the parent will be measured with the parent report of the CDI 2 (Kovacs, 2012). Same as the child report, this questionnaire consists of 28 items, and for each item, there are three possible answers; 0 indicating the absence of symptoms, 1 indicating mild symptoms, and 2 indicating definite symptoms. The validity of this instrument is qualified as good (Bae, 2012).

Anxiety of the child according to the parent will be measured using the Spence Children's Anxiety Scale-Parent Version (SCAS-P) (Spence, 1997). The items of the SCAS-P were formulated as closely as possible to the corresponding items of the child version of the SCAS. The parent version of the SCAS demonstrates sound psychometric properties (Nauta et al., 2004).

Data analyses/Statistical analyses

Data will be analyzed according to the intent-to-treat principle and will also be analyzed separately for the completers only. Multiple imputations will be used for missing observations at follow-ups. Regression analyses will be conducted to test differences in the development of depressive and anxiety symptoms between children in the intervention condition and children in the control condition. The results of the study will be reported in accordance with the CONSORT statement (Schulz et al., 2010). To investigate the mediating role of secondary outcome measures, such as response style and cognitive errors, mediation analyses will be performed in Mplus using bootstrap methods.

Possible baseline differences between the two conditions in demographic variables, depressive symptoms, and anxiety symptoms will be checked. Moreover, variables that show different distributions between the two groups will be entered as confounders in all models testing the effectiveness of the intervention.

Discussion

This study protocol presents the design of a randomized controlled trial, which will evaluate the effect of a prevention program for depression and anxiety for adolescents with a high familial risk. The primary aim of the study will be to evaluate the effectiveness of the prevention program for depression and/or anxiety among 11-15 year old female adolescents. Our secondary aim will be to examine theoretically meaningful parent and child factors that possibly moderate or mediate the effect of the prevention program. It is hypothesized that adolescents in the intervention condition will show lower levels of depressive and anxious symptoms during the follow-up compared to adolescents in the control group without a prevention program. Furthermore, it is hypothesized that response style and cognitive errors will mediate the effect of the program and that parental psychopathology, parenting stress, parental emotional support, and parental control will moderate the effect of the prevention program.

Strengths and limitations

The strength of the study is that it will conduct follow-up assessments at 12 months in addition to 6 months in both the intervention group and the control group. This will provide the opportunity to evaluate the long-term effects of this prevention program. Additional strength of the study is that in contrast to the most RCT studies, we will focus not only on the effectiveness of the program, but also on the mediators of change (i.e., how the intervention works). Finally, the prevention program will consist of only six sessions; therefore, we expect a low dropout rate.

This study also has limitations. We will not include a placebo intervention group, limiting the extent to which decreased depression and anxiety symptoms in the intervention group can be uniquely ascribed to the prevention program. Another study limitation is its selective design. It will enable a possible labeling or stigmatization effect, which can occur during the process of identification and participation of adolescents at risk in the prevention program.

Implications for practice

Depression and anxiety are the most common internalizing disorders among adolescents. Prolonged and elevated levels of depression and anxiety can develop into clinical disorders. They can also cause significant distress and dysfunction, even at subclinical levels

(Lewinsohn et al., 2000). Given the prevalence of depression and anxiety and their high rates of recurrence, there is a need for effective assessment, treatment, and prevention. Schools can play an important role in the identification of children and adolescents with elevated depressive and anxiety symptoms. If the prevention program shows to be effective in reducing elevated levels of depression and anxiety and preventing adolescents from developing a clinical mental health disorder, our results will be relevant to practice. Hence, the intervention will be able to be used on large scale.

Conclusion

This paper described the design of the study that will evaluate a prevention program for depression and anxiety in high-risk adolescents over a 12 months follow-up period. The targeted adolescents are at risk because both they and their parents have elevated levels of depression or anxiety. This study aims to contribute to the evidence-based prevention of depression and anxiety in adolescents.

6

Outcomes of a Randomized Controlled Trial on the Effectiveness of Depression and Anxiety Prevention for Adolescents with a High Familial Risk

Submitted for publication:

Rasing, S. P. A., Creemers, D. H. M., Vermulst, A. A., Janssens, J. M. A. M., Engels, R. C. M. E., & Scholte, R. H. J. Outcomes of a randomized controlled trial on the effectiveness of depression and anxiety prevention for adolescents with a high familial risk.

Abstract

A randomized controlled trail was conducted to examine the effectiveness of a depression and anxiety prevention program 'Een Sprong Vooruit' [A Leap Forward] among adolescent girls with a high familial risk (N = 142). The results showed no effects of the prevention program directly after the intervention nor at 6 or 12 months follow-up on either depression symptoms, or anxiety symptoms. Further, latent growth curve modeling (LGCM) was used to examine whether the growth functions for the intervention and the control condition were different. The slope representing the change in depression symptoms was not significantly different between the intervention and the control condition. For anxiety symptoms, the difference between slopes was also not significant. Based on these results, we suggested that these high risk adolescent girls might benefit more from a more intensive prevention program.

Introduction

Depression and anxiety are common mental health disorders and are among the most costly health problems (Mathers & Loncar, 2006; Olesen, Gustavsson, Svensson, Wittchen, & Jönsson, 2012; Üstün, Ayuso-Mateos, Chatterji, Mathers, & Murray, 2004). During adolescence, the rates of depression and anxiety rise dramatically and the presence of these disorders in young age are strongly associated with depression and anxiety in later life (Copeland, Shanahan, Costello, & Angold, 2009; Fergusson & Woodward, 2002; Reef, Diamantopoulou, Van Meurs, Verhulst, & Van Der Ende, 2009; Woodward & Fergusson, 2001). Suffering from depression and anxiety disorders during adolescence, but also having elevated levels of depression and anxiety symptoms, is negatively related to social and family functioning (Jaycox et al., 2009; Verboom et al., 2014), associated with poor academic performance (Balazs et al., 2013; Fergusson & Woodward, 2002; Verboom et al., 2014), related to depression and anxiety in later life (Roza et al., 2003; Seeley et al., 2009), and can even lead to suicide (Beesdo et al., 2009; Fletcher, 2008). Adolescence seems an important life phase to implement depression and anxiety prevention programs in order to reduce the risk on depression and anxiety disorders and to decrease depression and anxiety symptoms. Several meta-analyses showed that targeted prevention programs aimed at risk populations showed larger effects than universal prevention programs aimed at the total population (Horowitz & Garber, 2006; Stice et al., 2009). In order to be effective, prevention programs need to focus on specific risk factors that are related to the onset of anxiety and depression. One of the important risk factors in the development of depression or anxiety in children and adolescents is parental psychopathology (Beardslee et al., 2011; Garber & Cole, 2010; Weissman, Wickramaratne, et al., 2006). Despite the risk of intergenerational transmission of depression and anxiety disorders from parent to child, only a few studies on the effectiveness of prevention programs in this risk population have been conducted (Beardslee et al., 2003; Clarke et al., 2001; Garber et al., 2009; Ginsburg, 2009). These studies showed that symptoms decreased and the onset of clinical disorders reduced. None of these studies, however, targeted both depression and anxiety, although these disorders often co-occur and show a simultaneous development (Cummings et al., 2014; Reardon & Williams, 2007). The aim of the current study was to examine the effectiveness of a depression and anxiety prevention program in adolescents with a high familial risk when implemented in secondary schools.

Prevention can be divided in three levels of prevention that are appropriate for participants with different levels of risk factors (Mrazek & Haggerty, 1994). Universal prevention is aimed at all individuals in the eligible population, regardless of their risk level. Selective prevention is aimed at a subgroup of the population in which the individuals have an increased risk but do not (need to) show symptoms of the potential disorder to be included. Finally, indicated prevention is aimed at individuals who are already showing signs of a disorder and are identified as high risk individuals. In all three categories of prevention,

the goal is to reduce the number of new cases of the disorder. Recent review studies found that there is less evidence for the effectiveness of universally delivered depression prevention (Calear & Christensen, 2010; Horowitz & Garber, 2006; Stice et al., 2009). Further, the effects for universally delivered anxiety prevention are low and especially lower than the effects of selective and indicated prevention (Fisak Jr et al., 2011; Neil & Christensen, 2009). Prevention programs that are selective or indicated, and thus focus on adolescents at risk for developing depression or anxiety, have shown to reduce depression or anxiety symptoms or reduce the incidence of depression or anxiety disorders. The reason might be that high-risk individuals, participating in selective or indicated prevention programs, are more effectively engaged in the prevention program because they are motivated by their distress, which is not the case in non-risk individuals (Stice et al., 2009).

Prevention of depression and anxiety is especially important during adolescence, because these disorders rise dramatically during this life phase (Roza et al., 2003). Among 13 to 17-year old females, the lifetime prevalence of depression is estimated on 16.8% (Kessler et al., 2012). For anxiety disorders, lifetime prevalence in the same age group is estimated on 38.3% (Kessler et al., 2012). These numbers only account for clinical diagnoses of these disorders, and adolescents with elevated symptoms just below the clinical thresholds are not included. Children of parents with a depressive disorder are three times more likely to develop depression symptoms compared to children of parents without mental health problems (Bijl et al., 2002; Lieb et al., 2002). For children of parents with an anxiety disorder, it is up to seven times more likely that they develop anxiety symptoms (Micco et al., 2009; Van Dorsselaer et al., 2006). Besides the parental psychopathology itself, dysfunctional parenting behavior is found to be associated with an increased risk for depressive and anxiety disorders in offspring (Knappe, Lieb, et al., 2009; Needham, 2008). Parents with depression or anxiety were found to have a parenting style characterized by negativity (Berg-Nielsen, Vikan, & Dahl, 2002). Especially negative and affectionless behavior in parents is related to depression and anxiety symptoms in children (Berg-Nielsen et al., 2002). Because of the risk of parental psychopathology on development of depression and anxiety symptoms, it is important to aim prevention programs on children and adolescents with parents with mental health problems.

Besides familial risk, gender differences also play a role in the development of depression and anxiety disorders. Females are known to have a heightened risk on developing depression or anxiety symptoms (Kessler et al., 2012). Differences between males and females arise during adolescence and females show more often depression and anxiety symptoms (Kessler et al., 2012) and are more vulnerable to develop depression and anxiety disorders (Chaplin et al., 2009).

Because of detrimental consequences of depression and anxiety, reducing the risk and decreasing the elevated symptoms of depression and anxiety is important. Depression and anxiety disorders in adolescents are known to have a large comorbidity, shown in high diagnostic comorbidity rates (Sørensen et al., 2005; Weersing et al., 2008) as well as in

highly correlated symptoms (Cummings et al., 2014), and the co-occurrence of depression and anxiety symptoms is related to greater severity in symptoms (O'Neil et al., 2010). Further, research has shown that depression and anxiety are characterized by similar negative cognitions, negative affect and elevated distress (Dozois et al., 2009; Farchione et al., 2012). By focusing on these transdiagnostic underlying mechanisms of both depression and anxiety, and therefore providing intervention strategies for symptoms of both disorders, there is potentially a larger benefit of prevention programs (Chu et al., 2015; Dozois et al., 2009). Cognitive behavioral therapy has shown to be a highly efficacious treatment of depression and anxiety (Butler et al., 2006). The techniques used in cognitive behavioral therapy are known to target the underlying mechanisms of depression and anxiety, such as improve negative affect, decrease distress and increase of cognitive coping by experiencing more positive cognitions (Chu & Harrison, 2007). Prevention programs based on the cognitive behavioral therapy approach might therefore be appropriate for adolescents to decrease elevated depression and anxiety symptoms and to prevent the onset of both depression and anxiety disorders.

The present study evaluated the effects of a prevention program aimed at depression and anxiety prevention for adolescent girls with a high familial risk in a randomized controlled trial. To examine whether the program was effective, we tested effects directly after the preventive intervention and long-term effects after six and twelve months. The main hypothesis in the current study was that participants in the intervention condition would show a lower level of depression symptoms compared to participants in the control condition. Further, we hypothesized that the level of anxiety symptoms would be lower in participants in the intervention condition compared to participants in the control condition.

Method

Ethics

The study was approved by the medical ethics committee CMO Region Arnhem-Nijmegen, The Netherlands (NL41344.091.12). All participants provided written informed consent. The trial was registered in the Dutch Trial Register (NTR) as NTR3720. Results of this study were reported in accordance with the CONSORT 2010 statement for reporting parallel group randomized trials (Schulz et al., 2010).

Procedure

A preventive intervention aimed at preventing depression and anxiety in adolescent girls with high familial risk was examined with a non-blinded randomized controlled trial with two conditions (intervention versus control) (Rasing et al., 2013). A total of 862 female adolescent in the first and second year of five secondary schools were screened on depression symptoms using the Children's Depression Inventory 2 (CDI 2) (Kovacs, 2012),

anxiety symptoms using the Spence Children's Anxiety Scale (SCAS) (Spence, 1998), suicidal ideation using one item from the CDI 2, and perceived parental psychopathology using a self-developed instrument. Inclusion criteria were being aged between 11-15 years old; having increased levels of depression (CDI 2 ≥ 15) and/or anxiety (SCAS ≥ 39); having at least one of the parents with perceived parental psychopathology; and having sufficient knowledge of the Dutch language. Exclusion criteria were the absence of parental permission; adolescent already receiving treatment for mental health problems; and presence of prominent suicidal ideation (score 2 on CDI 2 item: a desire to kill oneself, if given the chance).

Of these 862 girls, 142 met the eligibility criteria and gave informed consent together with their parents. They were randomly assigned to either the intervention or the control condition. Participants in the intervention condition were assessed at baseline (T0), after two sessions (T1), after four sessions (T2), post-intervention (T3), at 6 months (T4) and 12 months (T5) follow-up. Participants in the control condition were assessed at the same time points. Adolescents in the control condition were offered to follow the preventive intervention as soon as the study ended. Participants in both conditions received rewards when they completed all assessments.

Sample size

A power calculation indicated that to detect a medium effect size (Cohen's d = 0.50) on depression symptoms at 12 months follow-up using a two-tailed test with α = .05 and power (1-β) = .80, 64 participants were needed per condition. We accounted for potential attrition and missing data, and therefore, we increased the sample size by 25%, resulting in 160 participants (80 in the intervention condition and 80 in the control condition).

Participants

One hundred forty-two female adolescents with elevated depression or anxiety symptoms participated in this effectiveness trial. The adolescents were aged 11-14 years (M = 12.87, SD = .69), and were in the first or second year of secondary education. Educational levels were: vocational training (18.3%), vocational training/ high school training (17.6%), high school training (15.5%), high school training/ pre-university training (30.3%) and pre-university training (18.3%). Most adolescents had the Dutch nationality (97.2%), and the remaining adolescents (2.8%) had a different European and non-European origin.

Randomization

Directly after screening, participants were randomly allocated to the conditions, stratified on school, grade and educational level (allocation ratio (1:1)). The randomization was done by an independent researcher using a computer-generated randomization procedure.

Intervention

The prevention program 'Een Sprong Vooruit' [A Leap Forward] was developed for adolescents with parents with mental health problems and focuses on depression and anxiety prevention. The program consists of six sessions, each 90 minutes and it mainly uses techniques based on cognitive behavioral therapy, behavioral activation and exposure. Adolescents will learn about emotions and how to recognize them; relationships between activating events, beliefs and consequences; strategies to replace pessimistic beliefs with optimistic beliefs; influence emotions with behavior; divide fearful activities into small steps and experience the decrease in anxiety; and how to get support from the social environment. The program 'Een Sprong Vooruit' uses components of the Dutch depression prevention program 'Op Volle Kracht' (Kindt, Kleinjan, Janssens, & Scholte, 2014; Tak, Lichtwarck-Aschoff, Gillham, Van Zundert, & Engels, 2015; Wijnhoven et al., 2014), which is an adapted version of the Penn Resiliency Program (Brunwasser et al., 2009), components of the Dutch anxiety treatment protocol 'Denken + Doen = Durven' (Bodden et al., 2008), and is inspired by the Friends program (Barrett et al., 2005). A detailed description of the preventive intervention can be found in the study protocol of this study (Rasing et al., 2013).

Group size of the intervention groups varied between 6 and 12 adolescents per group (group size $M = 8.63$, $SD = 1.85$), and the participants attended an average of 5.29 (1.34) sessions (median 6; range 0-6). Therapists were at least psychologists at master level. Further, treatment integrity was determined by assessing the percentage of the total program that was actually delivered, that is, how many instructions and exercises the program were actually given to and done by the participants. The prevention program was delivered with integrity in all groups ($M = 95\%$, $SD = 2.47$; range 91% - 98%).

Measures

The Children's Depression Inventory 2 (CDI 2) was used to measure depression symptoms (Kovacs, 2012). This questionnaire contained 28 items, each consisting of three statements graded in severity from 0 to 2. Sample statements included "Sometimes I feel sad", "Most of the times I feel sad" and "I always feel sad". Sum scores were computed by adding together scores of all 28 items. Cronbach's alpha was 0.71 at screening, 0.81 on T0, 0.83 at T1, 0.90 at T2, 0.89 at T3, 0.91 at T4 and 0.92 at T5. One of the items of this questionnaire was used to assess suicidal ideation (Kovacs, 2012). The item has score of 0 ("no suicidal ideation"), 1 ("thoughts of wanting to kill oneself, with no intent to do so"), and 2 ("a desire to kill oneself, if given the chance"). Participants with a score of 2 were directly referred to a therapist within an institute of mental health care.

The Spence Children's Anxiety Scale (SCAS) was used to measure anxiety symptoms (Spence, 1998). The 44 items of this self-report questionnaire are rated on a 4-point scale ranging from "never" to "always". Sample statements were "I worry about things" and "I am scared of the dark". Sum scores were computed by summing up scores of all items, excluding the filler 11, 17, 26, 31, 38 and 43 (e.g. "I am popular amongst other kids of my own age").

Cronbach's alpha was 0.81 at screening, 0.87 at T0, 0.91 at T1, 0.90 at T2, 0.92 at T3, 0.92 at T4, and 0.94 at T5.

Perceived parental psychopathology was used during the screening of the female adolescents. Students responded to seven statements about parental psychopathology for both mother and father, for example: "My parent received treatment from a psychologist or psychiatrist", "My parent had a decreased interest or pleasure in most or all activities", with wording of parent was replaced by either mother or father. Answers were rated as not present (0) or present (1). We counted presence of one items as presence of perceived parental psychopathology.

Statistical analyses

We conducted independent *t*-tests to analyze differences in the primary outcome measure depression symptoms at all time-points. Further, we did the same for the secondary outcome measure anxiety symptoms.

We applied latent growth curve modeling (LGCM) to examine the changes in the primary outcome measure depression symptoms and the secondary outcome measure anxiety symptoms over time, using Mplus version 6.11 (Muthén & Muthén, 2010). To estimate the parameters in the models, we applied the maximum likelihood estimator (ML). Using the full information maximum likelihood estimator has shown to be the preferred method for handling missing data (Johnson & Young, 2011). Model fit was assessed by χ^2, Comparative Fit Index (CFI: preferably .95 or higher), and Root Mean Square Error of Approximation (RMSEA: preferably .05 or lower, and satisfactory between .05 and .08) (Hu & Bentler, 1998).

To examine the changes in depression and anxiety symptoms, we first tested models, separately for depression and anxiety symptoms, without predictors. In these models, the mean of the intercept provides information about the average level of depression or anxiety symptoms and the mean of the slope represents the average change in depression or anxiety symptoms across the time-points. Second, we added condition (i.e., whether the adolescents participated in the intervention or control condition) to the models and tested separately for depression and anxiety symptoms whether the growth functions for the intervention and the control condition were different.

Results

Descriptive statistics

Means, standard deviations and *t*-values for differences on depression symptoms between the intervention and control condition were calculated (Table 1). As can be seen in Table 1, the level of depression symptoms was not significantly different at baseline between participants in the intervention condition compared to participants in the control condition.

Table 1 Means, Standard Deviations and t-values for Differences on Depression Symptoms Between the Intervention and Control Condition

	Total sample (N = 142)		Intervention condition (N = 69)		Control condition (N = 73)			
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*t*	*p*
Depression symptoms screening	16.08	5.46	15.93	4.91	16.23	5.97	-.33	.74
Depression symptoms T0	14.44	6.50	14.32	5.89	14.54	7.04	-.20	.85
Depression symptoms T1	14.49	6.92	14.60	6.53	14.39	7.29	-.17	.87
Depression symptoms T2	14.07	8.40	13.80	6.92	14.29	9.48	-.35	.73
Depression symptoms T3	13.66	8.06	13.36	7.65	13.91	8.45	-.39	.70
Depression symptoms T4	13.04	8.96	11.98	7.84	13.98	9.82	-1.25	.21
Depression symptoms T5	12.38	9.12	11.62	9.03	13.06	9.21	-.90	.37

This is also the case for the levels of depression symptoms during the intervention, after the intervention, and during follow-up. The within-group change of depression symptoms from baseline to 12-month follow-up showed a small effect size (Cohen's d = 0.35) in the intervention condition and no effect (Cohen's d = 0.18) in the control condition. Between-group difference in depression symptoms at 12-month follow-up showed no effect (Cohen's d = 0.16).

Further, means, standard deviations and t-values for anxiety symptoms were calculated between the intervention and control condition (Table 2). The level of anxiety symptoms was not significantly different between participants in the intervention condition and control condition at baseline, and neither during the intervention, after the intervention, and during the follow-up. The within-group change of anxiety symptoms from baseline to 12-month follow-up showed a medium effect size (Cohen's d = 0.58) in the intervention condition and a medium effect size (Cohen's d = 0.52) in the control condition. Between-group difference in anxiety symptoms at 12-month follow-up showed no effect (Cohen's d = 0.05).

The retention rates were high with 137 (96.5%) adolescents completing the baseline assessment, 131 (92.3%) completing the post-intervention assessment, 125 (88.0%) completing the 6-month follow-up assessment, and 130 (91.5%) completing the 12-month follow-up assessment. Figure 1 presents the flow diagram of participants.

Model findings

First, the linear growth model for depression symptoms was tested. All six measures of depression symptoms from baseline to 12-month follow-up were used to estimate a growth curve. The model showed an acceptable fit (χ^2 (15, N = 138) = 36.16, CFI = .97, and RMSEA

Table 2 Means, Standard Deviations and t-values for Differences on Anxiety Symptoms Between the Intervention and Control Condition

	Total sample (N = 142)		Intervention condition (N = 69)		Control condition (N = 73)			
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*t*	*p*
Anxiety symptoms screening	45.76	12.24	47.78	12.14	43.85	14.42	1.93	.06
Anxiety symptoms T0	37.77	13.58	38.97	13.51	36.69	13.62	.98	.33
Anxiety symptoms T1	37.29	15.78	37.52	16.52	37.10	15.23	.15	.88
Anxiety symptoms T2	35.69	15.11	36.57	15.37	34.99	14.98	.59	.55
Anxiety symptoms T3	33.19	16.03	33.92	16.41	32.57	15.79	.48	.64
Anxiety symptoms T4	32.81	16.18	32.72	16.19	32.89	16.23	-.06	.95
Anxiety symptoms T5	29.39	16.81	29.83	17.71	29.00	16.10	.28	.78

= .10; 90% CI [.05, .14]). Adding a quadratic slope did not improve the model fit, and therefore we decided to use the linear model. It is known that for small samples cut-off points for RMSEA are too restrictive (Chen, Curran, Bollen, Kirby, & Paxton, 2008) and acceptable models might be over rejected (Herzog & Boomsma, 2009). Unfortunately, Mplus does not provide individual fit indices. Therefore, the model was accepted. Next, we added condition as grouping variable to the model (χ^2 (32, N = 138) = 66.25, CFI = .95, and RMSEA = .13; 90% CI [.08, .17]). The intercept (B = 14.26, p < .001) and slope (B = -.21, p < .001) of the intervention condition were both significant, showing that the depression symptoms were significantly different from zero at baseline and decreased significantly over time. The intercept (B = 14.41, p < .001) of the control condition was significant, but the slope (B = -.11, p = .09) showed not to be significant, indicating that the depression symptoms were significantly different from zero at baseline and did not decrease over time. Finally, a Wald χ^2 test (χ^2 (1, N = 138) = .02, p = .89) showed that the intercepts of depression symptoms were not significantly different between the intervention and control condition. A Wald χ^2 test for the slopes (χ^2 (1, N = 138) = 1.46, p = .23) showed that the slopes of depression symptoms in the intervention condition and the control condition were not significantly different.

Second, the model for anxiety symptoms was tested. Again, all six measures of anxiety symptoms from baseline to 12-month follow-up were used in the estimation of the growth curve. The model showed an acceptable fit (χ^2 (16, N = 138) = 68.17, CFI = .92, and RMSEA = .15; 90% CI [.12, .19]). Here again, a quadratic slope did not improve the model fit. As mentioned before, RMSEA is known to be too restrictive in small samples, and, therefore, the model was accepted. In the next step, we added condition as a grouping variable to the model (χ^2 (10, N = 138) = 85.34, CFI = .92, and RMSEA = .16; 90% CI [.12, .20]). The intercept

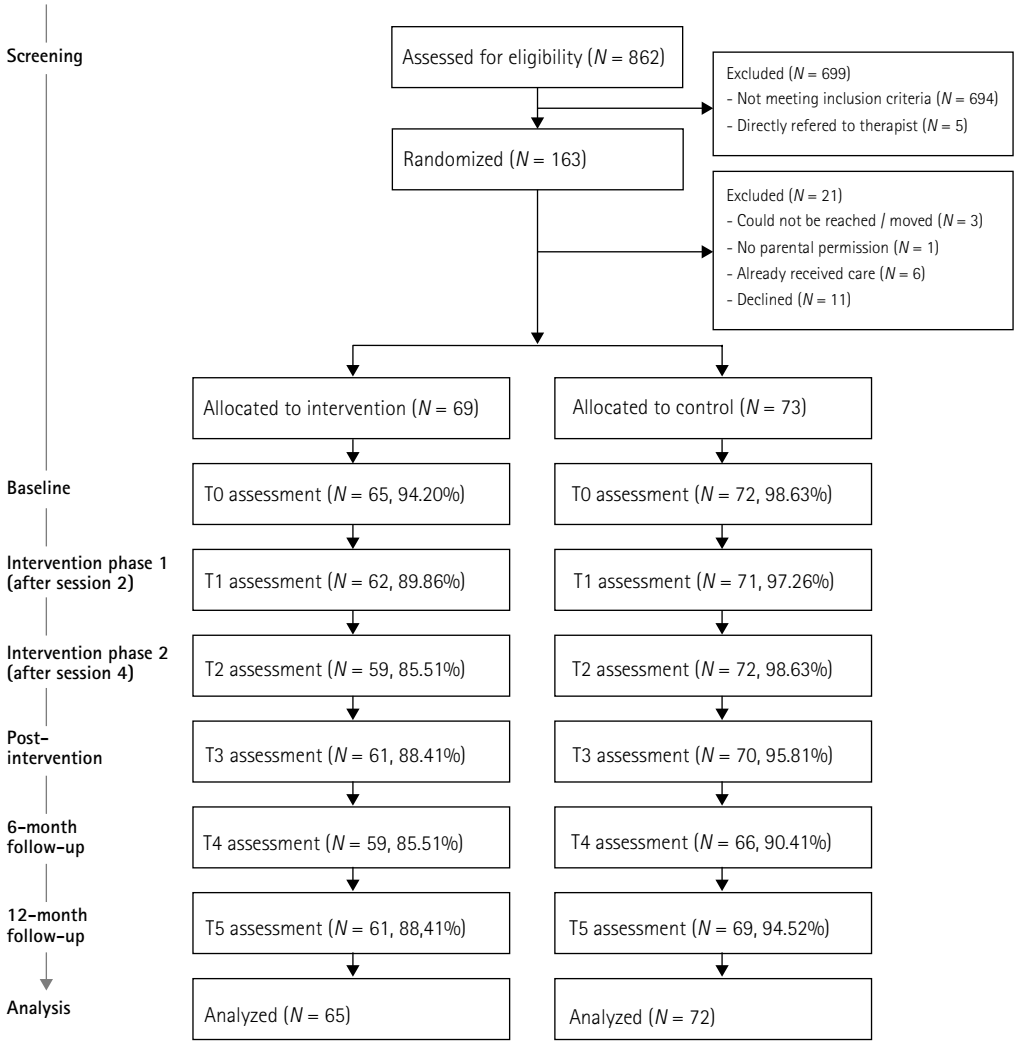

Figure 1 Flow diagram of participants

(B = 37.00, p < .001) and slope (B = -.54, p < .001) in the intervention condition were both significant, indicating that anxiety symptoms were significantly different from zero at baseline and decreased significantly over time. The intercept (B = 35.95, p < .001) and slope (B = -.53, p < .001) in the control condition were also both significant, showing that the anxiety symptoms at baseline were significantly different from zero and that they

significantly decreased over time. Finally, a Wald χ^2 test (χ^2 (1, N = 138) = .20, p = .66) for intercepts showed no significant difference between the intercepts of anxiety symptoms in the intervention condition and control condition. A Wald χ^2 test (χ^2 (1, N = 138) = .002, p = .96) showed that the slopes of anxiety symptoms were not significantly different between the intervention and control condition.

Discussion

In the present study, a randomized controlled trial was conducted to examine whether a novel depression and anxiety prevention program 'Een Sprong Vooruit' had an effect on depression and anxiety symptoms in adolescent girls with a high familial risk. In contrast to our hypotheses, participants in the intervention condition did not show a more favorable trajectory on continuous measures of depression and anxiety symptoms than adolescents in the control condition. The symptoms of depression decreased in participants in the intervention condition and symptoms of anxiety decreased in participants in both conditions. These results show that, in the present study, our prevention program did not seem to be effective in reducing symptoms or in preventing symptoms of depression and anxiety from developing.

Comparing the results of the current study to other studies on the effectiveness on indicated prevention, gives us interesting insights. Unlike findings of other studies on depression and anxiety in adolescents with elevated symptoms (Garber et al., 2009; Wijnhoven et al., 2014), we found no greater decrease in depression and anxiety symptoms in the intervention condition than in the control condition. Our findings are similar to studies focusing on prevention for adolescents with parents with mental health problems, which showed that among adolescents whose parents were depressed, the preventive intervention was not more effective than adolescents who did not receive the intervention (Garber et al., 2009). Our program was based on evidence-based treatment techniques for depression (Beck, 1967; Nolen-Hoeksema, 1991) as well as for anxiety disorders (Beck, 2005), and, therefore, we expected to find effects on both depression and anxiety symptoms. There are several possible reasons for the lack of effect of the intervention in the present study.

First, it can be the case that the program lacks focus on one technique, but covers several techniques in a short time (six sessions). That is, in the program adolescents were trained in psycho-education, cognitive restructuring, behavioral activation, exposure, and strengthening their social network. It could be that six sessions provide too little opportunity to become well trained and therefore these techniques and the program as such, failed to reduce symptoms of depression and anxiety. Research suggests that the ability to incorporate techniques and processes offered by the interventions is depending on the number of sessions of the intervention, and that eight or less sessions might be insufficient

(Jane-Llopis, Hosman, Jenkins, & Anderson, 2003). This suggests that providing several techniques in only six sessions by 'Een Sprong Vooruit' may not be sufficient to influence the development of depression or anxiety symptoms.

A second possible reason for a lack of intervention effects is the quality of the match between the participants and the prevention program. The program was developed as an efficient and short program. The participants in this intervention are high-risk adolescents, with elevated levels of depression and anxiety symptoms. They might benefit more from a more intensive and longer program, which allows participants to practice more with skills in more sessions. An intensive and short prevention program as 'Een Sprong Vooruit' might be more suitable for adolescents with lower levels of depression and anxiety symptoms. With regard to the match between participants and the prevention program, we are not sure whether adolescents with a high level of depression and anxiety symptoms can benefit from a prevention program that is focused on both depression and anxiety, whether or not with the same underlying mechanisms. The combination of an efficient and short program and targeting both depression and anxiety symptoms might have contributed to the limited effects of the prevention program.

Finally, the effects of the preventive intervention might have been more negatively influenced by parental mental health problems than we anticipated. According to recent studies, parents with mental health problems had a negative influence on the outcome of a prevention program for their children compared to healthy parents (Brent et al., 2015; Weersing et al., 2016). Studies on the effectiveness of prevention programs which included involvement of parents showed that outcome of these programs improved adolescents' symptomatology (Beardslee et al., 2013; Compas et al., 2009; Compas et al., 2011; Garber et al., 2009). The effects of the prevention program 'Een Sprong Vooruit' might have been limited by the negative influences of parental psychopathology on the adolescents.

Strengths of this study were the long-term follow-up measurements and the high retention rates. Further, the prevention program was strongly based on theories on treatment techniques for depression and anxiety. In addition, the group sessions of the prevention program showed high treatment integrity. Therefore, we are allowed to interpret our results unambiguously, and we can say that the results are related to the prevention program and not to the intentions of the program (Perepletchikova, 2011). However, some limitations should be mentioned. This study intended to measure changes in depression and anxiety symptoms during the intervention. Therefore, repeated assessments were necessary. We assessed depression and anxiety symptoms two times during the intervention, namely following the second session and following the fourth session. Yet, reporting too often on their own depression and anxiety symptoms might have led to awareness of their mental wellbeing and that in turn might have led to a reducing effect on symptoms. Further, data were collected only through self-report questionnaires, often mentioned to be subjective in measuring symptoms. Future studies should consider adding a more objective measure of depression and anxiety, for example using a clinical interview.

In conclusion, the present study showed no intervention effects of 'Een Sprong Vooruit' in reducing depression or anxiety symptoms compared to the control condition. We suggested that this short program might be more suitable for other populations than these high-risk adolescent girls and that this high-risk population needs a more focused intervention for either depression or anxiety prevention. Taking into account the limitations of the intervention and the targeted group of adolescents, an evaluation of a more suitable match between target groups and program is recommended.

7

Depression and Anxiety Prevention for Adolescents with High Familial Risk: Moderators of Effects and Underlying Mechanisms

In preparation:

Rasing, S. P. A., Creemers, D. H. M., Vermulst, A. A., Janssens, J. M. A. M., Engels, R. C. M. E., & Scholte, R. H. J. Outcomes of a randomized controlled trial on the effectiveness of depression and anxiety prevention for adolescents with a high familial risk.

Abstract

To gain more insight into potential moderating variables on the effectiveness of depression and anxiety prevention, we examined whether parental psychopathology, parenting behavior and parenting stress moderated the effect of the prevention program 'Een Sprong Vooruit' [A Leap Forward] on reducing depression and anxiety symptoms in female adolescents. We also explored the effects of the prevention program on underlying mechanisms, that is cognitive errors and response style, of depression and anxiety, and whether these effects were moderated by parental psychopathology, parenting behavior and parenting stress. A randomized controlled trial was performed on the effectiveness of 'Een Sprong Vooruit' among adolescent girls with a high familial risk ($N = 142$), their mothers ($N = 138$) and fathers ($N = 113$). Latent growth curve modeling was used to examine the moderating effects and to examine the effects of the prevention program on underlying mechanisms of depression and anxiety. We found no moderating effects of parental psychopathology, parenting behavior and parenting stress. Further, the study showed that the prevention program did not have an effect on underlying mechanisms of depression and anxiety.

Introduction

Depression and anxiety are the most common mental health disorders during adolescence and are among the largest contributors to costs in health care (Copeland et al., 2014; Copeland, Shanahan, Costello, & Angold, 2011; Maughan, Collishaw, & Stringaris, 2013; Patton et al., 2014). The rates of clinical, but also subclinical, depression and anxiety rise dramatically during adolescence (Kessler et al., 2012; Reef et al., 2009). Suffering from depression and anxiety disorders or from elevated symptoms of depression and anxiety, is related to dysfunctional family relations, poor academic performance, mental disorders during adulthood, and is one of the most important predictors for suicide (Balazs et al., 2013; Beesdo et al., 2009; Fletcher, 2008; Portzky, Audenaert, & Van Heeringen, 2005; Seeley et al., 2009; Verboom et al., 2014).

During adolescence, depression and anxiety prevention programs have the potential to reduce the societal burden of mental health problems, but can also reduce the serious individual consequences for adolescents (Cuijpers et al., 2012; Kessler & Greenberg, 2002; Muñoz, Beardslee, & Leykin, 2012). Indicated programs to prevent depression and anxiety are often based on cognitive behavioral therapy (Christensen et al., 2010; Fisak Jr et al., 2011; Merry et al., 2012), and show effectiveness in reducing the severity of depression and anxiety symptoms or reducing the prevalence of the disorders (McGorry et al., 2011). Nevertheless, the indicated depression and anxiety prevention program 'Een Sprong Vooruit' [A Leap Forward], that is based on CBT principles, was not effective in reducing the risk on depression and anxiety disorders and in decreasing depression and anxiety symptoms (Rasing et al., submitted).

The effects of depression and anxiety prevention programs are assumed to be moderated by various variables. Research has shown that effects of depression and anxiety prevention were moderated by mental health problems of the parents themselves (Garber et al., 2016; Ginsburg, Drake, Tein, Teetsel, & Riddle, 2015; Weersing et al., 2016), in that the effects were lower when parents had mental health problems compared to healthy parents. Besides parental psychopathology, parenting behavior and parenting stress are known to be important factors in the development of depression and anxiety symptoms in adolescents (Knappe, Lieb, et al., 2009; Needham, 2008). Less emotional support and less respect for autonomy are assumed to increase depression and anxiety symptoms (Pereira et al., 2014; Verhoeven et al., 2012). It is, however, unclear whether these factors moderate the effectiveness of depression and anxiety prevention.

Depression and anxiety prevention programs are suggested to not only target depression and anxiety symptoms, but also the mechanisms underlying depression and anxiety. The underlying mechanisms, that is cognitive errors and response style, are potentially targeted with the techniques that are used in cognitive behavioral therapy based prevention (Calvete et al., 2013; Cole et al., 2008). Cognitive errors have been defined as negative thinking that characterizes subclinical and clinical levels of depression and anxiety. Various types of

cognitive errors are described, including underestimation of the ability to cope (i.e., judging oneself as unable to cope with negative situations), personalizing without mind reading (i.e., believing negative outcomes have internal causes), selective abstraction (i.e., focusing only on negative outcomes), overgeneralizing (i.e., believing a single negative outcome is representative), and mind reading (i.e., predicting that others will react negatively) (Maric et al., 2011). According to Beck's cognitive theory are symptoms caused by cognitive errors, and not vice versa (Beck, Rush, Shaw, & Emery, 1979). Response style has been described as a reaction on depression and anxiety symptoms, and was divided in rumination, problem solving, and distraction (Abela et al., 2002). When these cognitive errors are stronger and the response style is more negative, levels of depression and anxiety are found to be higher (Brown et al., 1998; Johnson & Miller, 1990; LaGrange et al., 2011; Trosper et al., 2012). Cognitive behavioral therapy has shown to target negative cognitions in adults (Garratt, Ingram, Rand, & Sawalani, 2007). However, it is unknown whether this same effect accounts for adolescents receiving a preventive intervention for depression and anxiety.

To gain more insight into potential moderating variables on the effectiveness of depression and anxiety prevention, we examined whether parental psychopathology, parenting behavior and parenting stress moderated the effect of the preventive intervention on the primary outcome measure depression symptoms and the secondary outcome measure anxiety symptoms. Next, we explored whether the prevention program had an effect on the development and change over time in underlying mechanisms of depression and anxiety, in particular response style, consisting of distraction, problem solving and rumination, and cognitive errors, consisting of underestimation of the ability to cope, personalizing without mind reading, selective abstraction, overgeneralizing, and mind reading. Further, we explored whether the effects of the preventive intervention on these underlying mechanisms of depression and anxiety were moderated by parental psychopathology, parenting behavior and parenting stress.

Method

Ethics

The study was approved by the medical ethics committee CMO Region Arnhem-Nijmegen, The Netherlands, and the trial was registered in the Dutch Trial Register as NTR3720. Adolescents and parents provided written informed consent. Results of this study were reported in accordance with the CONSORT 2010 statement for reporting parallel group randomized trials (Schulz et al., 2010).

Procedure

A non-blinded randomized controlled trial with two conditions (intervention versus control) was performed to examine the effectiveness of a preventive intervention aimed at

preventing depression and anxiety in adolescent girls with high familial risk (Rasing et al., 2013). A total of 862 adolescent girls in the first and second year of five secondary schools were screened on depression symptoms using the Children's Depression Inventory 2 (CDI 2) (Kovacs, 2012), anxiety symptoms using the Spence Children's Anxiety Scale (SCAS) (Spence, 1998), suicidal ideation using one item from the CDI 2, and perceived parental psychopathology using a self-developed instrument.

Adolescents were included when they were aged between 11-15 years old, had increased levels of depression (CDI 2 ≥ 15) and/or anxiety (SCAS ≥ 39), had at least one parent with perceived parental psychopathology, and had sufficient knowledge of the Dutch language. They were excluded when their parents did not give permission to participate, when they already received treatment for mental health problems, and had prominent suicidal ideations (score 2 on CDI 2 item: a desire to kill oneself, if given the chance). A total of 142 girls met the eligibility criteria, gave informed consent together with their parents, and were randomly assigned to either the intervention or the control condition. Participants in the intervention condition were assessed at baseline, after two and four sessions, post-intervention, and at six and twelve months follow-up. Participants in the control condition were assessed at the same time points. Participants in both conditions received rewards when they completed all assessments.

Sample size

A power calculation indicated that to detect a medium effect size (Cohen's d = 0.50) on depression symptoms at 12 months follow-up using a two-tailed test with α = .05 and power (1-β) = .80, 64 participants were needed per condition. We increased the sample size by 25%, accounting for potential attrition and missing data. Therefore, 160 participants (80 in the intervention condition and 80 in the control condition) needed to be included.

Participants

One hundred forty-two female adolescents, aged 11-14 years (M = 12.87, SD = .69), with elevated depression or anxiety symptoms participated in this effectiveness trial. Most adolescents had the Dutch nationality (97.2%), others (2.8%) had different European and non-European nationalities. Educational levels were: vocational training (18.3%), vocational training/ high school training (17.6%), high school training (15.5%), high school training/ pre-university training (30.3%) and pre-university training (18.3%).

Besides the adolescents, 138 mothers and 113 fathers participated. The 138 mothers (biological mothers, stepmothers and foster mothers) were aged between 28 and 56 (M = 43.89, SD = 4.41). Most of the mothers had the Dutch nationality (93.5%), and the other mothers (6.5%) were of different European or non-European origin. The 113 fathers (biological fathers, stepfathers and foster fathers) were aged between 37 and 61 (M = 47.10, SD = 4.58). Most fathers had the Dutch nationality (92.0%), and the other fathers (8.0%) were of different European or non-European origin.

Randomization

Participants were randomly allocated to the conditions, stratified by school, grade and educational level. The randomization was done by an independent researcher using a computer-generated randomization procedure.

Intervention

The prevention program 'Een Sprong Vooruit' focused on depression and anxiety prevention and was developed for adolescents with parents with mental health problems. The program, consisting of six sessions of each 90 minutes, uses techniques based on cognitive behavioral therapy. Adolescents learn about emotions, about relationships between activating events, beliefs and consequences, strategies to replace pessimistic beliefs with optimistic beliefs, how to decrease anxiety by dividing fearful activities into small steps, and how to get support from their social environment. Especially the strategies to recognize their emotions, identify the cognitive interpretations of the events, and then restructure their cognitions, are suggested to target underlying mechanisms of depression and anxiety. Therapists who gave the program have to be at least psychologists at master level. 'Een Sprong Vooruit' uses components of the Dutch depression prevention program 'Op Volle Kracht' (Kindt et al., 2014; Tak et al., 2015; Wijnhoven et al., 2014), which is an adapted version of the Penn Resiliency Program (Brunwasser et al., 2009). It also uses components of the Dutch anxiety treatment protocol 'Denken + Doen = Durven' [Think + Do = Dare] (Bodden et al., 2008), and is inspired by the Friends program (Barrett et al., 2005).

Participants attended an average of 5.29 (1.34) sessions (median 6; range 0-6). Group size of the intervention groups varied between 6 and 12 adolescents per group (group size M = 8.63, SD = 1.85). The prevention program was delivered with integrity in all groups (M = 95%, SD = 2.47; range 91% - 98%), determined by assessing the percentage of delivered program components.

Measures

The Children's Depression Inventory 2 (CDI 2: Kovacs, 2012) was used to measure depression symptoms. This questionnaire contained 28 items, each consisting of three statements graded in severity from 0 to 2. Sample statements were "Sometimes I feel sad", "Most of the times I feel sad" and "I always feel sad". Sum scores were computed by adding together scores of all 28 items. Depression symptoms were measured at T0, T1, T2, T3, T4, and T5. Cronbach's alpha varied between 0.81 and 0.92.

The Spence Children's Anxiety Scale (SCAS) was used to measure anxiety symptoms (Spence, 1997). The 44 items of this self-report questionnaire were rated on a 4-point scale ranging from "never" to "always". Sample statements were "I worry about things" and "I am scared of the dark". Sum scores were computed by summing up all items, excluding the filler 11, 17, 26, 31, 38 and 43 (e.g. "I am popular amongst other kids my own age"). Anxiety symptoms were measured at T0, T1, T2, T3, T4, and T5. Cronbach's alpha varied between 0.87 and .94.

The Children's Depression Inventory 2 (CDI 2) was also used to assess suicidal ideation (Kovacs, 2012). The item had score of 0 ("no suicidal ideation"), 1 ("thoughts of wanting to kill oneself, with no intent to do so"), and 2 ("a desire to kill oneself, if given the chance"). Participants with a score of 2 were directly referred to a mental health care institute. Suicidal ideation was measured at T0, T1, T2, T3, T4, and T5.

The Brief Symptoms Inventory (BSI) was used to measure parental psychopathology (Derogatis, 1975a). The BSI, a short version of the Symptom Checklist-90 (SCL-90: Derogatis, 1975b), measures general symptoms of psychopathology. The 53 items were rated on a 5-point scale from "not at all" to "extremely". Sample statements were "Feeling no interest in things" and "Feeling tense or keyed up". Both mothers and fathers rated the questionnaire, resulting in scores on maternal and paternal psychopathology. Mean scores were computed by averaging all items. Parental psychopathology was measured at T0. Cronbach's alpha was 0.94 for maternal psychopathology and 0.93 for paternal psychopathology.

Subscales of the Relational Support Inventory (RSI) were used to measure parental emotional support and respect for autonomy, with the scales Warmth versus Hostility and Respect for autonomy versus Setting limits, respectively (Scholte, Van Lieshout, & Van Aken, 2001). Both subscales were rated by the adolescents for each parent, resulting in maternal emotional support, paternal emotional support, maternal respect for autonomy, and paternal respect for autonomy. Each subscale consisted of six items rated on a 5-point scale ranging from "very untrue" to "very true". Sample statements were "My mother/ father supports me in what I am doing" and "My mother/father sets strict rules, demands, and limits". Mean scores were computed by averaging all items. Emotional support and respect for autonomy were measured at T0. Cronbach's alpha was 0.86 for maternal emotional support, 0.73 for maternal respect for autonomy, 0.87 for paternal emotional support and 0.72 for paternal respect for autonomy.

The Parenting Stress Questionnaire (OBVL) was used to measure parenting stress (Vermulst, De Meyer, Nguyen, & Veerman, 2012). The questionnaire, to be filled in by both parents separately, consisted of 34 items scored on a 4-point scale ranging from "not true" to "very true". Sample statements were "Raising my child leaves me with too little personal time" and "I feel cheerful when my child is with me". Parenting stress was measured at T0. Cronbach's alpha was 0.72 for maternal parenting stress and 0.80 for paternal parenting stress.

The Children's Response Style Questionnaire (CRSQ) was used to measure response style (Abela, Brozina, & Haigh, 2002). The questionnaire consisted of 25 items, rated on a 4-point scale ranging from "almost never" to "almost always". Sample statements were "When I feel sad, I think about why I can't handle things better" and "When I feel sad, I help someone else with something so I don't think about my problem". Items were divided over the three subscales distraction, problem solving, and rumination. Sum scores of the three subscales were computed by summing up all item scores belonging to that subscale. Response style was measured at T0, T1, T2, T3, T4, and T5. Cronbach's alpha varied between

0.54 and 0.60 for distraction, between 0.69 and 0.75 for problem solving and between 0.88 and 0.93 for rumination.

The Children's Negative Cognitive Errors Questionnaire – Revised (CNCEQ-R) was used to measure negative cognitive errors (Maric, Heyne, Van Widenfelt, & Westenberg, 2011). The questionnaire contained 15 item rated on a 5-point scale from "not at all like I would think" to "almost exactly like I would think". A sample statement was "You are giving a talk in your class at school. You have just begun when some of your classmates suddenly start to laugh. You assume that they think you are not doing a good job". Items were divided into five subscales, namely underestimation of the ability to cope, personalizing without mind reading, selective abstraction, overgeneralizing, and mind reading. Negative cognitive errors were measured at T0, T1, T2, T3, T4, and T5. Cronbach's alpha varied between 0.53 and 0.76 for underestimation of the ability to cope, between 0.75 and 0.88 for personalizing without mind reading, between 0.35 and 0.68 for selective abstraction, between 0.61 and 0.86 for overgeneralizing, and between 0.71 and 0.81 for mind reading.

Statistical analyses

In the current study, moderating effects of parental emotional support, respect for autonomy and parenting stress on depression and anxiety symptoms were analyzed, in order to examine whether these parental variables moderated the relations between the program 'Een Sprong Vooruit' and depression and anxiety symptoms over the period from baseline to 12 months follow-up, using measurements at T0, T1, T2, T3, T4, and T5. Therefore, we first applied latent growth curve modeling using Mplus (version 6.11) to examine the development of depression symptoms at baseline (i.e., the intercept) and the change of symptoms over time (i.e., the slope), and tested whether there were effects of the moderating variables and condition on the parameters (intercept and slope) of the growth models. Parameters in the models were estimated by applying the maximum likelihood estimator (ML), which has shown to be the preferred method for handling missing data (Johnson & Young, 2011). Secondly, we performed the same models on the development of anxiety symptoms to test whether the parenting variables moderated the effect of the preventive intervention on anxiety symptoms. Model fit was assessed by χ^2, Comparative Fit Index (CFI: preferably .95 or higher), and Root Mean Square Error of Approximation (RMSEA: preferably .05 or lower, and satisfactory between .05 and .08) (Hu & Bentler, 1998).

Next, we applied latent growth curve modeling on underlying mechanisms of depression and anxiety, in particular response style, consisting of distraction, problem solving and rumination, and cognitive errors, consisting of underestimation of the ability to cope, personalizing without mind reading, selective abstraction, overgeneralizing, and mind reading to examine the development at baseline and the changes over time from baseline to 12 months follow-up, using measurements at T0, T1, T2, T3, T4, and T5, and to test whether there were differences between the intervention and control condition. The maximum likelihood estimator (ML) was applied to estimate the parameters in the models. Second, we

applied the same latent growth curve models and tested whether there were effects of the moderating variables parental psychopathology, emotional support, respect for autonomy, and parenting stress, and condition on the parameters (intercept and slope) of the growth models.

Results

Descriptive statistics

Means and standard deviations were calculated for the study variables at T0 and presented in Table 1. Further, *t*-values were calculated for differences between participants in the

Table 1 Means, Standard Deviations and T-Values for Differences on Outcome Variables at T0 Between the Intervention and Control Condition

	Total sample (N = 142)		Intervention condition (N = 69)		Control condition (N = 73)			
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*t*	*p*
Depression symptoms	14.44	6.50	14.32	5.89	14.54	7.04	-.20	.85
Anxiety symptoms	37.77	13.58	38.97	13.51	36.69	13.62	.98	.33
Maternal psychopathology	.33	.30	.33	.30	.32	.30	.14	.89
Maternal emotional support	4.18	.80	4.09	.92	4.27	.66	-1.27	.21
Maternal respect for autonomy	3.74	.73	3.67	.82	3.81	.64	-1.15	.25
Maternal parenting stress	78.25	6.62	76.58	6.98	79.80	5.91	-2.88	.01
Paternal psychopathology	.25	.25	.26	.24	.25	.26	.32	.75
Paternal emotional support	4.08	.88	3.94	.96	4.20	.79	-1.63	.11
Paternal respect for autonomy	3.83	.76	3.86	.78	3.81	.74	.37	.71
Paternal parenting stress	76.37	7.14	74.60	7.38	78.31	6.39	-2.79	.01
Distraction	7.33	3.70	7.22	3.68	7.43	3.75	-.34	.74
Problem solving	4.50	2.91	4.71	2.96	4.32	2.86	.78	.44
Rumination	14.97	8.18	14.69	8.68	15.22	7.75	-.38	.71
Underestimation of the ability to cope	8.95	2.91	9.35	3.01	8.58	2.78	1.56	.12
Personalizing without mind reading	10.00	3.15	9.52	3.07	10.43	3.18	-1.70	.09
Selective abstraction	10.71	2.58	10.54	2.75	10.86	2.42	-.73	.47
Overgeneralizing	9.82	3.02	9.83	2.96	9.82	3.10	.02	.98
Mind reading	11.99	3.99	11.89	4.12	12.07	3.90	-.26	.80

intervention and the control condition. With exception of maternal and paternal parenting stress, there were no differences in the study variables between the conditions. The retention rates were high with 137 (96.5%) adolescents completing the baseline assessment and 130 (91.5%) completing the 12-month follow-up assessment. Figure 1 presents the flow diagram of participants.

Model findings

First, the linear growth model for depression was fitted for six measures of depression symptoms from baseline to 12-month follow-up. The model showed an acceptable fit, taken into account that cut-off points for RMSEA are too restrictive for small samples (Chen et al., 2008; Herzog & Boomsma, 2009). Next, we added condition as predictor of intercept and slope of the model which also showed an acceptable fit. Finally, we added moderating variables and interaction terms (product terms of condition and moderating variables) as predictors of the growth parameters (intercept and slope) of the model. For each moderating variable, (i.e., maternal psychopathology, maternal emotional support, maternal respect for autonomy, maternal parenting stress, paternal psychopathology, paternal emotional support, paternal respect for autonomy, and paternal parenting stress) we ran a separate model. Intercepts, slopes, and fit indices for each interaction term are presented in the appendix. For all variables, models showed an acceptable fit. The analyses revealed that none of the potential moderating variables moderated the effect of 'Een Sprong Vooruit' on depression symptoms.

Second, a linear growth model was fitted for six measures from baseline to 12-month follow-up of anxiety symptoms. The model showed an acceptable fit. Next, condition was added to the model as predictor of intercept and slope. This showed not a perfect fit, but the fit was acceptable. Finally, moderating variables and interaction terms (product terms of condition and moderating variables) were added as predictors of the growth parameters (intercept and slope) of the model, separately for each moderating variable. Intercepts, slopes, and fit indices are presented in the appendix. Model fits were comparable to the model without interaction terms. The analyses revealed that none of the parental variables moderated the effect of 'Een Sprong Vooruit' on anxiety symptoms.

Latent growth curve findings for underlying mechanisms

To test effects of the intervention on underlying mechanisms, we ran separate models for all variables representing underlying mechanisms, that is, distraction, problem solving, rumination, underestimation of the ability to cope, personalization without mind reading, selective abstraction, overgeneralizing, and mind reading. In all models, we first ran the unconditional model, then we added condition as predictor of intercept and slope, and finally we added the moderating variable and interaction (product terms of condition and moderating variable) as predictors, separately for each moderating variable. The models for

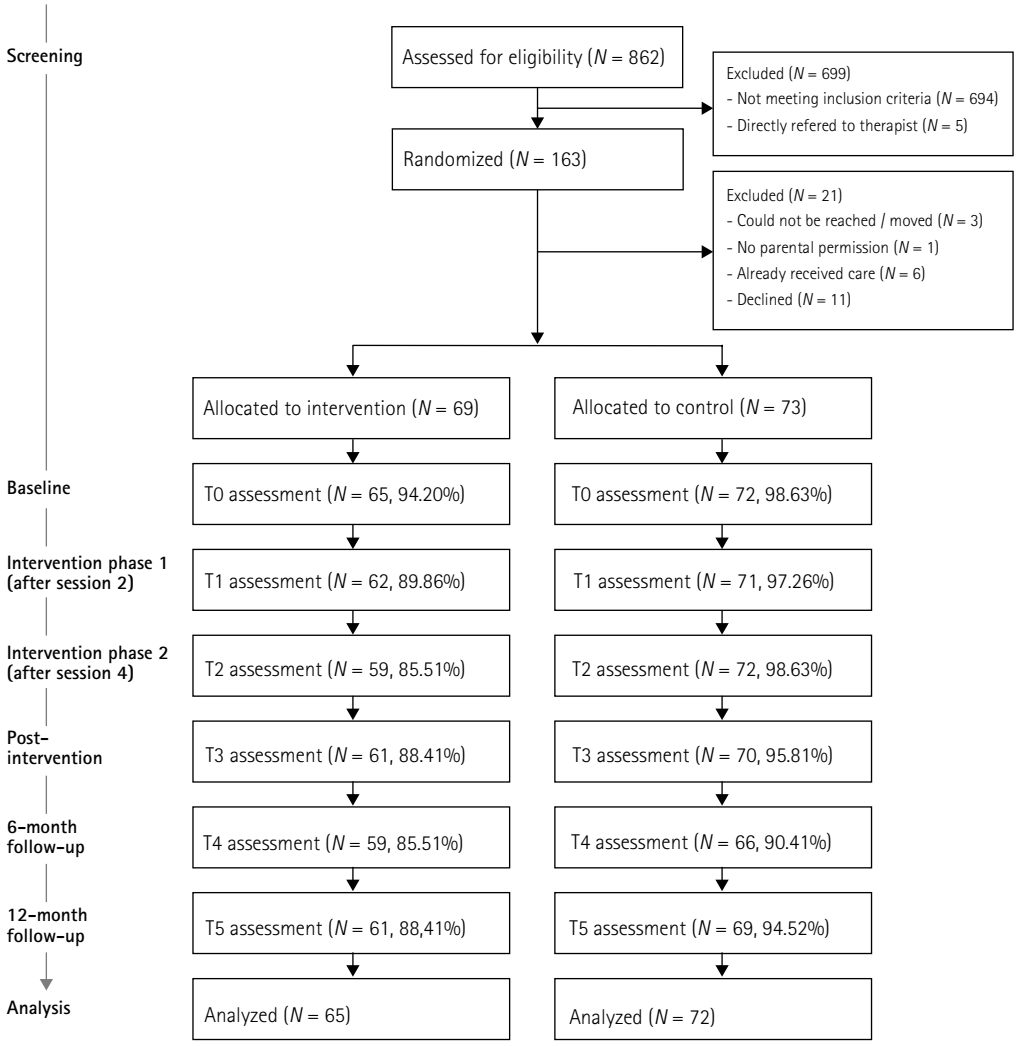

Figure 1 Flow diagram of participants

all outcome variables showed reasonable to good fit. Fit indices and results are presented in the appendix.

We found that all underlying mechanisms of depression and anxiety showed a significant intercept, which means that they were different from zero at baseline. Further we found that the slope of underestimation of the ability to cope was significant. A significant

decrease was found in participants in both conditions, but there was no significant effect of condition. Overgeneralizing and mind reading increased in participants in both conditions, but we found no significant effect of condition. Finally, no significant moderating effects were found.

Discussion

In the present study, we examined whether parental psychopathology, parenting behavior and parenting stress moderated the effect of the preventive intervention on the primary outcome measure depression symptoms and the secondary outcome measure anxiety symptoms. Next, we explored whether the prevention program had an effect on the development and change over time in underlying mechanisms of depression and anxiety, in particular response style, consisting of distraction, problem solving and rumination, and cognitive errors, consisting of underestimation of the ability to cope, personalizing without mind reading, selective abstraction, overgeneralizing, and mind reading. Further, we explored whether the effects of the preventive intervention on these underlying mechanisms of depression and anxiety were moderated by parental psychopathology, parenting behavior and parenting stress.

Overall, as presented in our previous study, it was found that the preventive intervention 'Een Sprong Vooruit' was not effective in reducing the risk on depression and anxiety disorders or in decreasing depression and anxiety symptoms (Rasing et al., submitted). In the present study, we found that parental psychopathology, parenting behavior, and parenting stress did not moderate the effectiveness of the intervention. Further, we found that the prevention program did not have an effect on underlying mechanisms of depression and anxiety, and that there were no moderating effects of parental psychopathology, emotional support, respect for autonomy, and parenting stress.

Unlike findings in other studies on effectiveness of depression or anxiety prevention, we did not find moderating effects of parental psychopathology. Earlier studies found that in children with parents with mental health problems, the effectiveness of depression prevention programs was lower than in children with healthy parents (Garber et al., 2016; Weersing et al., 2016). Based on these results, we expected that adolescents whose parents had lower levels of psychopathology would benefit from the prevention program. With regard to the known relation between parental psychopathology, parenting behavior, and parenting stress (Berg-Nielsen et al., 2002; Mowbray, Lewandowski, Bybee, & Oyserman, 2004), we expected equal moderating effects for parenting behavior and parenting stress on the effect of the prevention program. Nonetheless, findings showed that parental psychopathology, parenting behavior, and parenting stress, did not moderate the effect of prevention on reducing depression and anxiety symptoms. The absence of an effect of the preventive intervention in adolescence whose parents have low levels of parental psycho-

pathology, low levels of negative parenting behavior, and low levels of parenting stress, suggests that the prevention program does not effectuate a reduction of depression and anxiety symptoms at all. These findings indicate that the lack of an effect of the program is not a consequence of parental risk factors, but emphasizes that the prevention program might not be suitable for high risk female adolescents. In reporting on our main effects, we already suggested that the program lacks focus on one technique, and the six sessions of the prevention program were too few to incorporate the skills that are taught during the program. According to previous research, the number of sessions in the prevention program need to be eight or more in order to be effective (Jane-Llopis et al., 2003). Especially in a prevention program with several techniques, such as 'Een Sprong Vooruit', more sessions provide opportunity to benefit from the techniques. Our findings might suggest that in this group of adolescents mental health problems are more difficult to target, and therefore need a more intensive prevention program.

Further, we did not find any effects of the prevention program on the underlying mechanisms of depression and anxiety. This finding confirms our suggestion that the prevention program 'Een Sprong Vooruit' lacks a focus on techniques to target symptoms and underlying mechanisms in high risk adolescents (Rasing et al., submitted). Despite the fact that our program was based on evidence-based treatment techniques for depression (Beck, 1967; Nolen-Hoeksema, 1991) and for anxiety disorders (Beck, 2005), it is possible that the adolescents were not able to incorporate the techniques in the short time period of six sessions.

The main strengths of this study were its design, its long term follow-up and the low retention rates. Consequently, the results can be interpreted with some certainty. Further, we used not only adolescent-rated measures, but we also included parent-rated measures that were possibly of influence on the effects of the prevention program. Nonetheless, we should mention some limitations. In some variables (i.e., distraction, underestimation of the ability to cope, and selective abstraction), Cronbach's alpha was low and that resulted in a lower reliability of the analyses, and, therefore, we had to be cautious in interpreting the findings. Further, our sample size was large enough to test effectiveness of possible moderators, but was too small to test for mediating effects. Therefore, we could not test whether the underlying mechanisms influenced the presence of depression and anxiety symptoms. Furthermore, we only collected data through questionnaires, and not through, for example, clinical interviews. Therefore, our conclusions were only based on changes in symptoms. We cannot draw any conclusion about prevention of actual diagnoses of depression or anxiety, and whether parental factors are of influence in preventing depression or anxiety disorders.

In conclusion, the present study showed no moderating effects of parental psychopathology, parenting behavior and parenting stress on the effects of prevention of depression and anxiety in adolescents. Further, the study showed that the prevention program 'Een Sprong Vooruit' did not have an effect on underlying mechanisms of depression and anxiety.

Appendix

Table 2 Growth Models for Depression Symptoms with Moderating Effects from Baseline to 12-Month Follow-Up

	Intercept		Slope		X^2 *(df)*	CFI	RMSEA
	B	*p*	*B*	*p*			
Unconditional	14.37	< .001	-.16	< .001	36.16 (16)	.97	.10; 90% CI [.05, .14]
Condition	.07	.95	.12	.18	37.57 (20)	.97	.08; 90% CI [.04, .12]
Maternal psychopathology	-.03	1.00	.08	.87	57.16 (28)	.96	.09; 90% CI [.06, .12]
Condition × maternal psychopathology	-.54	.89	.05	.88	48.80 (28)		
Maternal emotional support	-1.77	.40	-.07	.66		.97	.07; 90% CI [.04, .11]
Condition × maternal emotional support	-.01	.99	.06	.62	43.59 (28)		
Maternal respect for autonomy	-.80	.73	.01	.96		.98	.06; 90% CI [.02, .10]
Condition × maternal respect for autonomy	-.22	.89	-.03	.79	52.15 (28)		
Maternal parenting stress	-.02	.96	-.02	.37		.96	.08; 90% CI [.05, .12]
Condition × maternal parenting stress	-.06	.73	.02	.27	50.04 (28)		
Paternal psychopathology	8.35	.33	1.09	.08		.96	.09; 90% CI [.05, .12]
Condition × paternal psychopathology	-2.87	.59	-.64	.09	47.91 (28)		
Paternal emotional support	-2.47	.23	-.24	.13		.97	.08; 90% CI [.04, .11]
Condition × paternal emotional support	.06	.97	.19	.06	49.86 (28)		
Paternal respect for autonomy	-1.77	.49	-.18	.34		.97	.08; 90% CI [.04, .12]
Condition × paternal respect for autonomy	.57	.73	.16	.20	49.98 (28)		
Paternal parenting stress	.32	.27	-.02	.31	36.16 (16)	.96	.09; 90% CI [.05, .12]
Condition × paternal parenting stress	-.28	.15	.02	.21			

Note. Bonferroni corrections were performed. Alphas were considered significant if smaller than .05 / 17 = .0029.

Table 3 Growth Models for Anxiety Symptoms with Moderating Effects from Baseline to 12-Month Follow-Up

	Intercept		Slope		χ^2 *(df)*	CFI	RMSEA
	B	*p*	*B*	*p*			
Unconditional	36.46	< .001	.76	< .001	68.17 (16)	.92	.15; 90% CI [.12, .19]
Condition	-1.45	.55	.06	.74	72.23 (20)	.92	.14; 90% CI [.10, .17]
Maternal psychopathology	-8.29	.52	1.07	.30	85.12 (28)	.91	.12; 90% CI [.10, .16]
Condition × maternal psychopathology	3.75	.64	-.53	.41			
Maternal emotional support	-.71	.86	-.26	.46	85.08 (28)	.91	.12; 90% CI [.09, .15]
Condition × maternal emotional support	-.84	.79	.23	.34			
Maternal respect for autonomy	1.26	.80	.28	.46	78.57 (28)	.92	.12; 90% CI [.09, .15]
Condition × maternal respect for autonomy	-2.15	.52	-.09	.72			
Maternal parenting stress	-.75	.20	-.02	.60	80.47 (28)	.92	.12; 90% CI [.09, .15]
Condition × maternal parenting stress	.41	.28	.02	.52			
Paternal psychopathology	12.13	.49	1.92	.17	60.99 (28)	.93	.10; 90% CI [.07, .14]
Condition × paternal psychopathology	-7.45	.50	-.90	.30			
Paternal emotional support	-3.08	.48	-.32	.34	69.75 (28)	.93	.11; 90% CI [.08, .14]
Condition × paternal emotional support	-.26	.93	.29	.20			
Paternal respect for autonomy	-.55	.92	.06	.89	69.72 (28)	.93	.11; 90% CI [.08, .14]
Condition × paternal respect for autonomy	-.27	.94	.14	.59			
Paternal parenting stress	.00	1.00	.05	.34	61.50 (28)	.93	.11; 90% CI [.07, .14]
Condition × paternal parenting stress	-.03	.95	-.03	.31			

Note. Bonferroni corrections were performed. Alphas were considered significant if smaller than .05 / 17 = .0029.

Table 4 Growth Models for Distraction with Moderating Effects from Baseline to 12-Month Follow-Up

	Intercept		Slope		χ^2 *(df)*	CFI	RMSEA
	B	*p*	*B*	*p*			
Unconditional	6.95	< .001	-.01	.55	38.40 (16)	.93	.10; 90% CI [.06, .14]
Condition	-.48	.35	-.01	.89	40.98 (20)	.94	.09; 90% CI [.05, .13]
Maternal psychopathology	-4.16	.13	.21	.36	57.57 (28)	.91	.09; 90% CI [.06, .12]
Condition × maternal psychopathology	2.62	.13	-.15	.28			
Maternal emotional support	.53	.59	-.12	.12	48.17 (28)	.94	.07; 90% CI [.04, .11]
Condition × maternal emotional support	-.18	.79	.09	.08			
Maternal respect for autonomy	-.18	.87	-.05	.58	52.37 (28)	.92	.08; 90% CI [.05, .11]
Condition × maternal respect for autonomy	-.01	.98	.06	.35			
Maternal parenting stress	-.17	.17	-.003	.78	53.85 (28)	.92	.08; 90% CI [.05, .12]
Condition × maternal parenting stress	.13	.10	.003	.62			
Paternal psychopathology	2.53	.47	-.10	.73	47.54 (28)	.92	.08; 90% CI [.04, .12]
Condition × paternal psychopathology	-3.22	.14	.06	.74			
Paternal emotional support	-.49	.60	-.03	.72	48.54 (28)	.93	.08; 90% CI [.04, .11]
Condition × paternal emotional support	.55	.37	.01	.80			
Paternal respect for autonomy	-.54	.62	-.08	.41	46.66 (28)	.93	.07; 90% CI [.03, .11]
Condition × paternal respect for autonomy	.49	.48	.03	.59			
Paternal parenting stress	.06	.59	.02	.04	40.69 (28)	.95	.07; 90% CI [.00, .11]
Condition × paternal parenting stress	.01	.91	-.01	.04			

Note. Bonferroni corrections were performed. Alphas were considered significant if smaller than .05 / 17 = .0029.

Table 5 Growth Models for Problem Solving with Moderating Effects from Baseline to 12-Month Follow-Up

	Intercept		Slope		χ^2 *(df)*	CFI	RMSEA
	B	*p*	*B*	*p*			
Unconditional	4.18	< .001	-.004	.83	23.84 (16)	.97	.06; 90% CI [.00, .11]
Condition	-.61	.12	-.01	.86	27.44 (20)	.97	.05; 90% CI [.00, .10]
Maternal psychopathology	-1.26	.55	.03	.86	44.87 (28)	.94	.07; 90% CI [.03, .10]
Condition × maternal psychopathology	0.54	.68	-.06	.61			
Maternal emotional support	.44	.56	.04	.61	35.02 (28)	.98	.04; 90% CI [.00, .08]
Condition × maternal emotional support	-.16	.76	.00	1.00			
Maternal respect for autonomy	.53	.51	.01	.89	35.36 (28)	.97	.04; 90% CI [.00, .08]
Condition × maternal respect for autonomy	-.47	.39	.001	.99			
Maternal parenting stress	.002	.98	.01	.54	35.47 (28)	.97	.05; 90% CI [.00, .09]
Condition × maternal parenting stress	.02	.74	-.003	.63			
Paternal psychopathology	.35	.90	.08	.76	30.53 (28)	.99	.03; 90% CI [.00, .08]
Condition × paternal psychopathology	-.64	.70	.01	.98			
Paternal emotional support	-.16	.82	.03	.61	23.86 (28)	1.00	.00; 90% CI [.00, .06]
Condition × paternal emotional support	.24	.60	-.02	.69			
Paternal respect for autonomy	.67	.42	.01	.88	27.70 (28)	1.00	.00; 90% CI [.00, .07]
Condition × paternal respect for autonomy	-.51	.34	-.01	.88			
Paternal parenting stress	.13	.16	.01	.36	25.82 (28)	1.00	.00; 90% CI [.00, .07]
Condition × paternal parenting stress	-.07	.26	-.01	.31			

Note. Bonferroni corrections were performed. Alphas were considered significant if smaller than .05 / 17 = .0029.

Table 6 Growth Models for Rumination with Moderating Effects from Baseline to 12-Month Follow-Up

	Intercept		Slope		χ^2 *(df)*	CFI	RMSEA
	B	*p*	*B*	*p*			
Unconditional	14.41	< .001	-.18	.03	55.26 (16)	.92	.13; 90% CI [.10, .17]
Condition	.03	.77	-.02	.89	57.75 (20)	.93	.12; 90% CI [.08, .15]
Maternal psychopathology	-2.25	.76	.16	.78	56.94 (28)	.94	.09; 90% CI [.06, .12]
Condition × maternal psychopathology	.74	.87	-.06	.86			
Maternal emotional support	-.66	.79	-.01	.98	60.77 (28)	.94	.09; 90% CI [.06, .12]
Condition × maternal emotional support	-.40	.82	.12	.36			
Maternal respect for autonomy	-2.54	.36	.35	.08	62.42 (28)	.93	.10; 90% CI [.06, .13]
Condition × maternal respect for autonomy	.51	.78	-.11	.42			
Maternal parenting stress	.08	.81	-.01	.66	60.33 (28)	.93	.09; 90% CI [.06, .13]
Condition × maternal parenting stress	-.05	.84	.01	.59			
Paternal psychopathology	4.17	.68	.99	.18	54.07 (28)	.94	.09; 90% CI [.06, .13]
Condition × paternal psychopathology	-.88	.89	-.57	.21			
Paternal emotional support	-2.24	.35	-.12	.50	62.55 (28)	.93	.10; 90% CI [.07, .13]
Condition × paternal emotional support	-.30	.85	.18	.14			
Paternal respect for autonomy	-2.97	.31	.10	.64	59.31 (28)	.93	.10; 90% CI [.06, .13]
Condition × paternal respect for autonomy	.72	.69	.05	.75			
Paternal parenting stress	.27	.42	.004	.87	52.76 (28)	.94	.09; 90% CI [.05, .13]
Condition × paternal parenting stress	-.26	.26	.001	.94			

Note. Bonferroni corrections were performed. Alphas were considered significant if smaller than .05 / 17 = .0029.

Table 7 Growth Models for Underestimation of The Ability to Cope With Moderating Effects from Baseline to 12-Month Follow-Up

	Intercept		Slope		χ^2 *(df)*	CFI	RMSEA
	B	*p*	*B*	*p*			
Unconditional	8.28	< .001	-.10	< .001	64.11 (16)	.86	.15; 90% CI [.11, .19]
Condition	-.02	.96	-.002	.96	71.36 (20)	.85	.14; 90% CI [.10, .17]
Maternal psychopathology	-.24	.92	.10	.64	70.77 (28)	.87	.11; 90% CI [.08, .14]
Condition × maternal psychopathology	.12	.94	-.09	.51			
Maternal emotional support	-.59	.49	-.08	.24	80.35 (28)	.84	.12; 90% CI [.09, .15]
Condition × maternal emotional support	.17	.77	.08	.12			
Maternal respect for autonomy	-.75	.42	.07	.41	90.38 (28)	.82	.13; 90% CI [.10, .16]
Condition × maternal respect for autonomy	.35	.57	-.03	.55			
Maternal parenting stress	-.18	.09	.003	.76	76.29 (28)	.85	.11; 90% CI [.08, .15]
Condition × maternal parenting stress	.12	.08	-.01	.44			
Paternal psychopathology	.24	.94	.55	.06	63.62 (28)	.89	.11; 90% CI [.07, .14]
Condition × paternal psychopathology	.40	.85	-.33	.06			
Paternal emotional support	-.92	.27	-.07	.32	70.76 (28)	.87	.11; 90% CI [.08, .14]
Condition × paternal emotional support	.40	.47	.05	.29			
Paternal respect for autonomy	-1.32	.19	.05	.55	71.52 (28)	.86	.11; 90% CI [.08, .14]
Condition × paternal respect for autonomy	.84	.18	-.04	.47			
Paternal parenting stress	-.05	.69	.002	.86	73.50 (28)	.86	.12; 90% CI [.09, .16]
Condition × paternal parenting stress	-.01	.94	-.001	.92			

Note. Bonferroni corrections were performed. Alphas were considered significant if smaller than .05 / 17 = .0029.

Table 8 Growth Models for Personalizing Without Mind Reading with Moderating Effects from Baseline to 12-Month Follow-Up

	Intercept		Slope		χ^2 *(df)*	CFI	RMSEA
	B	*p*	*B*	*p*			
Unconditional	10.00	< .001	.04	.03	24.69 (16)	.98	.06; 90% CI [.00, .11]
Condition	.54	.29	-.04	.33	28.82 (20)	.98	.06; 90% CI [.00, .10]
Maternal psychopathology	3.75	.17	-.10	.63	37.82 (28)	.98	.05; 90% CI [.00, .09]
Condition × maternal psychopathology	-2.45	.15	.04	.76			
Maternal emotional support	.05	.96	.06	.38	32.28 (28)	.99	.03; 90% CI [.00, .08]
Condition × maternal emotional support	.35	.60	-.05	.31			
Maternal respect for autonomy	-.91	.39	.04	.60	34.54 (28)	.99	.04; 90% CI [.00, .08]
Condition × maternal respect for autonomy	.76	.29	-.01	.85			
Maternal parenting stress	.23	.06	.003	.74	40.79 (28)	.97	.06; 90% CI [.00, .10]
Condition × maternal parenting stress	-.17	.04	-.001	.82			
Paternal psychopathology	-5.36	.17	-.13	.62	34.23 (28)	.99	.05; 90% CI [.00, .09]
Condition × paternal psychopathology	2.29	.35	.06	.72			
Paternal emotional support	.91	.34	.03	.68	31.70 (28)	.99	.03; 90% CI [.00, .08]
Condition × paternal emotional support	-.35	.58	-.01	.77			
Paternal respect for autonomy	.86	.45	-.06	.46	36.93 (28)	.98	.05; 90% CI [.00, .09]
Condition × paternal respect for autonomy	-.62	.39	.04	.46			
Paternal parenting stress	.00	.98	-.01	.37	29.61 (28)	1.00	.02; 90% CI [.00, .08]
Condition × paternal parenting stress	.04	.67	.01	.29			

Note. Bonferroni corrections were performed. Alphas were considered significant if smaller than .05 / 17 = .0029.

Table 9 Growth Models for Selective Abstraction with Moderating Effects from Baseline to 12-Month Follow-Up

	Intercept		Slope		χ^2 *(df)*	CFI	RMSEA
	B	*p*	*B*	*p*			
Unconditional	10.66	< .001	.03	.04	24.05 (16)	.98	.06; 90% CI [.00, .11]
Condition	.07	.86	-.03	.19	32.20 (20)	.97	.07; 90% CI [.01, .11]
Maternal psychopathology	3.56	.11	-.26	.13	46.62 (28)	.95	.07; 90% CI [.03, .11]
Condition × maternal psychopathology	-1.93	.16	.14	.20			
Maternal emotional support	1.36	.07	.01	.91	47.16 (28)	.95	.07; 90% CI [.03, .11]
Condition × maternal emotional support	-.67	.20	-.01	.81			
Maternal respect for autonomy	.50	.55	-.02	.78	58.91 (28)	.91	.09; 90% CI [.06, .12]
Condition × maternal respect for autonomy	-.07	.91	-.01	.81			
Maternal parenting stress	.25	.01	.001	.89	38.99 (28)	.97	.06; 90% CI [.00, .09]
Condition × maternal parenting stress	-.15	.02	.00	.98			
Paternal psychopathology	-.38	.91	-.56	.01	39.07 (28)	.97	.06; 90% CI [.00, .10]
Condition × paternal psychopathology	.64	.74	.25	.07			
Paternal emotional support	1.47	.05	.02	.70	48.31 (28)	.94	.08; 90% CI [.04, .11]
Condition × paternal emotional support	-.91	.07	-.01	.82			
Paternal respect for autonomy	2.01	.03	-.04	.56	52.52 (28)	.93	.08; 90% CI [.05, .12]
Condition × paternal respect for autonomy	-1.19	.04	.02	.71			
Paternal parenting stress	.16	.14	.004	.62	42.11 (28)	.96	.07; 90% CI [.01, .11]
Condition × paternal parenting stress	-.10	.17	-.002	.77			

Note. Bonferroni corrections were performed. Alphas were considered significant if smaller than .05 / 17 = .0029.

Table 10 Growth Models for Overgeneralizing with Moderating Effects from Baseline to 12-Month Follow-Up

	Intercept		Slope		χ^2 *(df)*	CFI	RMSEA
	B	*p*	*B*	*p*			
Unconditional	9.90	< .001	.08	<.001	29.96 (16)	.97	.08; 90% CI [.03, .12]
Condition	-.36	.49	.003	.94	36.15 (20)	.97	.08; 90% CI [.03, .12]
Maternal psychopathology	3.01	.27	-.17	0.38	39.74 (28)	.97	.06; 90% CI [.00, .09]
Condition × maternal psychopathology	-2.44	.15	.08	0.49			
Maternal emotional support	-.14	.89	.08	0.23	42.24 (28)	.97	.06; 90% CI [.01, .10]
Condition × maternal emotional support	.19	.78	-.05	0.24			
Maternal respect for autonomy	-1.09	.31	.07	0.34	44.37 (28)	.97	.07; 90% CI [.02, .10]
Condition × maternal respect for autonomy	.72	.32	-.04	0.40			
Maternal parenting stress	.33	.01	-.01	0.55	39.60 (28)	.97	.06; 90% CI [.00, .09]
Condition × maternal parenting stress	-.24	.003	.01	0.35			
Paternal psychopathology	-3.44	.39	-.32	0.20	48.49 (28)	.95	.08; 90% CI [.04, .12]
Condition × paternal psychopathology	1.70	.49	.14	0.36			
Paternal emotional support	.15	.88	.10	0.09	36.47 (28)	.98	.05; 90% CI [.00, .09]
Condition × paternal emotional support	-.02	.97	-.06	0.14			
Paternal respect for autonomy	.06	.96	.02	0.78	35.25 (28)	.98	.05; 90% CI [.00, .09]
Condition × paternal respect for autonomy	-.24	.74	-.01	0.76			
Paternal parenting stress	.14	.31	.003	0.95	47.96 (28)	.95	.08; 90% CI [.04, .12]
Condition × paternal parenting stress	-.05	.59	-.003	0.81			

Note. Bonferroni corrections were performed. Alphas were considered significant if smaller than .05 / 17 = .0029.

Table 11 Growth Models for Mind Reading with Moderating Effects from Baseline to 12-Month Follow-Up

	Intercept		Slope		χ^2 *(df)*	CFI	RMSEA
	B	*p*	*B*	*p*			
Unconditional	12.28	< .001	.09	< .001	20.48 (16)	.99	.05; 90% CI [.00, .10]
Condition	-.19	.77	.02	.69	23.25 (20)	.99	.03; 90% CI [.00, .08]
Maternal psychopathology	2.91	.39	-.04	.88	31.32 (28)	.99	.03; 90% CI [.00, .08]
Condition × maternal psychopathology	-1.95	.36	.03	.86			
Maternal emotional support	.97	.41	-.01	.92	29.72 (28)	1.00	.02; 90% CI [.00, .07]
Condition × maternal emotional support	-.23	.78	-.03	.67			
Maternal respect for autonomy	-.13	.92	.02	.86	32.62 (28)	.99	.04; 90% CI [.00, .08]
Condition × maternal respect for autonomy	.49	.57	-.03	.66			
Maternal parenting stress	.19	.20	.003	.82	30.04 (28)	1.00	.02; 90% CI [.00, .07]
Condition × maternal parenting stress	-.13	.19	-.002	.76			
Paternal psychopathology	-3.10	.53	-.14	.68	33.20 (28)	.99	.04; 90% CI [.00, .09]
Condition × paternal psychopathology	1.30	.68	.12	.56			
Paternal emotional support	1.48	.21	-.01	.95	35.00 (28)	.99	.05; 90% CI [.00, .09]
Condition × paternal emotional support	-.71	.36	-.01	.84			
Paternal respect for autonomy	1.79	.20	-.14	.19	33.32 (28)	.99	.04; 90% CI [.00, .08]
Condition × paternal respect for autonomy	-1.25	.16	.08	.22			
Paternal parenting stress	.04	.81	.003	.81	31.54 (28)	.99	.03; 90% CI [.00, .08]
Condition × paternal parenting stress	.03	.78	-.003	.73			

Note. Bonferroni corrections were performed. Alphas were considered significant if smaller than .05 / 17 = .0029.

8

General Discussion

Overall aim of current dissertation

The aim of this current dissertation was to contribute to the understanding of risk factors for the development of depression and anxiety symptoms and the effectiveness of prevention in adolescent girls with a high familial risk. In the first part, the effects of parent related risk factors on the development of depression and anxiety symptoms were assessed. The findings may contribute to improve identification of high risk adolescents for a preventive intervention. In the second part, we focused on effectiveness of depression and anxiety prevention and examined whether the prevention program 'Een Sprong Vooruit' [A Leap Forward] showed benefits for female adolescents who have a high risk for developing depression or anxiety disorders. In this final chapter (General Discussion), first, the main findings of this dissertation will be presented, followed by a reflection on these findings. Next, limitations of the studies of this dissertation will be described, followed by gaps in knowledge and suggestions for future research, and clinical implications.

Summary of main findings

In part 1, consisting of Chapter 2 and 3, parent-related risk factors for the development of depression and anxiety symptoms in female adolescents were described.

In Chapter 2, results showed that perceived maternal and paternal psychopathology were significant predictors of depression and anxiety symptoms in female adolescents, that is, higher perceived maternal and paternal psychopathology predicted higher levels of female adolescents' depression and anxiety symptoms. Further, the interaction between perceived maternal and paternal psychopathology was a significant predictor of depression and anxiety symptoms. Having two parents both with higher perceived psychopathology predicted stronger depression and anxiety symptoms in female adolescents compared to having one parent with high perceived psychopathology.

In Chapter 3, latent growth curve modeling showed that paternal emotional support was negatively related to baseline level, and not to the development of depression symptoms in female adolescents. This indicated that less paternal emotional support was related to a higher level of depression symptoms in adolescence. Paternal emotional support was also found to be negatively related to the baseline level of anxiety symptoms, and not to the development of anxiety symptoms in female adolescents, indicating that less paternal emotional support was related to a higher level of anxiety symptoms in female adolescents. Paternal respect for autonomy was found to be related to the development of adolescents' anxiety symptoms, that is, higher paternal respect for autonomy was related to an increase in anxiety symptoms in female adolescents. No significant relations were found between maternal emotional support and respect for autonomy on the one hand and depression and anxiety symptoms of their adolescent daughters on the other hand. Additionally, we found

a significant interaction effect of paternal psychopathology and paternal respect for autonomy on adolescents' depression symptoms at baseline, indicating that at lower levels of paternal psychopathology, respect for autonomy was negatively related to depression symptoms and that when fathers showed high levels of psychopathology, respect for autonomy was positively related to depression symptoms in their daughters.
In part 2, consisting of Chapter 4, 5, 6, and 7, the effectiveness of depression and anxiety prevention for high-risk adolescents was described. In Chapter 4, meta-analytic results of depression prevention programs in high-risk adolescents showed a small effect size at post-intervention. The effect size of depression prevention six months after the intervention was also small, and no effect was found 12 months after the intervention. For anxiety prevention in high-risk adolescents, meta-analytic results showed no effect at post-intervention and six months after the intervention. Results showed a small effect of anxiety prevention 12 months after the intervention.

In Chapter 5, we presented the study protocol of the randomized controlled trial to test the effectiveness of the prevention program 'Een Sprong Vooruit'. In Chapter 6, it was shown that the depression and anxiety prevention program 'Een Sprong Vooruit' had no effect on reducing depression or anxiety symptoms in high-risk female adolescents. Latent growth curve modeling showed that the development of depression and anxiety symptoms was not significantly different between female adolescents in the intervention condition and female adolescents in the control condition.

In chapter 7, results showed that the effects of the prevention program 'Een Sprong Vooruit' in reducing depression and anxiety symptoms in female adolescents were not moderated by parental psychopathology, parenting behavior and parenting stress. Further, latent growth curve modeling showed that the prevention program did not have effect on underlying mechanisms of depression and anxiety, that is, response style consisting of distraction, problem solving and rumination, and cognitive errors, consisting of under-estimation of the ability to cope, personalizing without mind reading, selective abstraction, overgeneralizing, and mind reading. Underestimation of the ability to cope decreased significantly, and overgeneralizing and mind reading increased significantly. However, no differences were found between female adolescents in the intervention condition and female adolescents in the control condition. Finally, no significant moderating effects were found.

Reflections on the main findings

Parental psychopathology

Earlier research has shown that exposure to parental depression and anxiety disorders heightens the risk for depression and anxiety symptoms as well as disorders in offspring (Bijl et al., 2002; Lieb et al., 2002; Mars et al., 2012; Weissman, Pilowsky, et al., 2006). From

early on, mothers were of primary interest in studies on the intergenerational transmission of mental health problems. In particular, maternal depression and anxiety are known to be predictors for depression and anxiety in their children (Singh et al., 2011; Van Loon, Van de Ven, Van Doesum, Witteman, & Hosman, 2014). Recently, the contribution of paternal psychopathology became of more interest to researchers and it has been shown that also paternal depression and anxiety are risk factors in the development of depression and anxiety in their offspring. More specifically, it was found to be of equal importance as maternal depression and anxiety (Connell & Goodman, 2002; Ramchandani et al., 2011). In our study in a general population, we found that both perceived maternal and paternal psychopathology predicted female adolescents' depression and anxiety symptoms. We also found that depression and anxiety symptoms in female adolescents were even higher when both parents had higher perceived psychopathology. Our findings were consistent with results of previous research, which also demonstrated that maternal and paternal psychopathology were both related to symptoms in female adolescents (Brennan et al., 2002; McClure et al., 2001). Further, Goodman and Gotlib (1999) described paternal mental health as a protective factor in their model of intergenerational transmission. When not only the mother, but also the father, suffers from mental health problems, there is no parent to serve as protective factor and to model healthy cognitions, behavior and affect (Connell & Goodman, 2002; Goodman & Gotlib, 1999; Ramchandani & Psychogiou, 2009). This might explain the additive effect of maternal and paternal psychopathology on depression and anxiety symptoms in female adolescents, which has also been found in earlier studies (Dierker et al., 1999; Goodman et al., 1993; Lieb et al., 2002; Mitchell, McCauley, Burke, Calderon, & Schloredt, 1989).

These results confirmed that not only mothers, but also fathers, are important for the mental health of adolescents. From early on, research on intergenerational transmission of mental health problems was exclusively focused on mothers (Phares, 1992; Phares, Fields, Kamboukos, & Lopez, 2005). This emanated both from the fact that women were long time seen as primary caregiver, and from their indisputable role in the emotional and behavioral development of their children. The role of fathers has gained increasing interest in recent years (Bögels & Phares, 2008; Connell & Goodman, 2002; Flouri, 2008; Lewis & Lamb, 2006; Ramchandani, Stein, Evans, O'Connor, & ALSPAC Study Team, 2005; Sarkadi, Kristiansson, Oberklaid, & Bremberg, 2008; Videon, 2005). From these studies, we may conclude that the role of paternal psychopathology is not inferior to maternal psychopathology, and, therefore, it is crucially important to target paternal risk factors in programs that aim to prevent the development of disorders in adolescents.

Parenting behavior

Along with parental psychopathology, parenting behavior has also been associated with symptoms of depression and anxiety in offspring (Knappe, Lieb, et al., 2009; Needham, 2008; Yap, Pilkington, Ryan, & Jorm, 2014). Parental emotional support and parental respect

for autonomy, also conceptualized as parental control, are of high interest in unraveling the link between parenting behavior and depression and anxiety in adolescents. Parental emotional support was found to be important in the development of depression symptoms, that is, a lack of emotional support predicted higher depression symptoms (McLeod, Weisz, et al., 2007; Needham, 2008; Stice, Ragan, & Randall, 2004; Van Roekel et al., 2011). Parental respect for autonomy was found to be negatively related to anxiety symptoms, namely that low parental respect for autonomy predicted more anxiety (Bögels & Perotti, 2011; McLeod, Wood, et al., 2007; Pereira et al., 2014). Similar to previous research, we found in a sample with high-risk female adolescents, that negative parenting behavior, that is, low emotional support and low respect for autonomy, predicted stronger symptoms of depression and anxiety in female adolescents.

There is growing evidence that lack of parental emotional support and lack of respect for autonomy are important factors in the development of depression and anxiety symptoms in adolescents. Our results confirmed the finding that paternal emotional support and respect for autonomy were negatively related to female adolescents' symptoms of depression and anxiety. We did not, however, find relationships between maternal emotional support or respect for autonomy and symptoms of depression and anxiety in female adolescents. Other studies showed that maternal influences might be more important during childhood whereas paternal influences have more impact on adolescents (Bögels & Phares, 2008; Verhoeven et al., 2012). The difference in relationships between mothers and fathers and their female offspring could be explained by the fact that our research focused on adolescents, and therefore influences of paternal parenting behavior were more eminent.

The importance of parental emotional support and respect for autonomy is emphasized by findings from a meta-analytic review, suggesting that prevention programs that increase emotional support from parents may reduce symptoms of depression and anxiety (Yap, Pilkington, Ryan, & Jorm, 2014; Yap, Pilkington, Ryan, Kelly, et al., 2014). Further, increasing parental warmth may contribute to reducing levels of depression and anxiety in young people (Zhou, Sandler, Millsap, Wolchik, & Dawson-McClure, 2008). These studies suggest that increasing parental emotional support and parental warmth should be elements in prevention programs to effectively reduce the risk on depression and anxiety disorders in children and adolescents.

Further, we found an interaction effect of paternal psychopathology and respect for autonomy on depression symptoms. This interaction effect revealed that when fathers had low to average levels of psychopathology, adolescent baseline symptom levels of depression declined with increasing levels of paternal respect for autonomy, in line with our expectations (Knappe, Lieb, et al., 2009). Additionally, against our expectations, when fathers had high levels of psychopathology, adolescent depressive symptoms increased with increasing levels of paternal respect for autonomy. A possible explanation for this latter finding might be that adolescents, in the context of paternal psychopathology, interpret behaviors that are typically indicative of adaptive parenting behavior (respect for autonomy) as being

maladaptive (i.e., signs of disinterest or low involvement). Conversely, adolescents might interpret low respect for autonomy or high levels of paternal control as interest in the adolescent's behavior and establishing rules and consequences when paternal psychopathology is present.

These results showed that especially in parents with mental health problems, parenting behavior could be crucial in relation to the emotional development in children and adolescents. Based on this knowledge, prevention programs aimed at increasing awareness in parents of the impact of stress and improving parenting skills seem to be suitable for children of parents with mental health problems. A family group intervention based on cognitive behavioral therapy and with elements of parenting skills training for depressed parents, showed positive results for their children up to two years after the intervention (Compas et al., 2009; Compas et al., 2011). For a high-risk population of children of parents with mental health problems, involvement of parents in prevention programs looks promising.

Effectiveness of depression and anxiety prevention programs based on cognitive behavioral therapy

Depression and anxiety prevention aims to decrease the likelihood of the onset of a depressive or anxiety disorder or to decrease subclinical symptoms, where treatment mainly aims to reduce clinical levels of symptoms (Garber & Weersing, 2010). Based on the overlap in goals (i.e., decrease symptoms), the cognitive behavioral therapy approach seems to be very suitable for indicated prevention of depression and anxiety (Butler et al., 2006; Hofmann et al., 2012). Several systematic reviews and meta-analyses have been conducted on the effectiveness of depression and anxiety prevention (Ahlen, Lenhard, & Ghaderi, 2015; Brunwasser et al., 2009; Calear & Christensen, 2010; Christensen et al., 2010; Corrieri et al., 2014; Fisak Jr et al., 2011; Hetrick, Cox, & Merry, 2015; Horowitz & Garber, 2006; Merry et al., 2012; Merry & Spence, 2007; Neil & Christensen, 2009; Stice et al., 2009; Stockings et al., 2016; Teubert & Pinquart, 2011). None of these studies, however, focused on prevention programs using cognitive behavioral therapy techniques for specifically high-risk adolescents, and therefore we aimed to examine their effectiveness in reducing symptoms of depression and anxiety in the short-term and in the long-term. Findings of our meta-analysis showed that depression prevention for at-risk adolescents has a small effect on the short term, but no effect on the longer term. Anxiety prevention, on the other hand, showed no significant effects on either the short term or on the longer term. Our results partially reflect other studies, although another meta-analysis did find effects 12 months after the preventive intervention for depression (Merry et al., 2012). Further, other studies on anxiety prevention found both short-term and long-term effects (Christensen et al., 2010; Neil & Christensen, 2009).

Effectiveness of depression prevention specifically in at-risk populations showed that the effect in at-risk adolescents is not enduring, and lasts no longer than 6 months. Noteworthy, it is known that effects of cognitive behavioral therapy in treatment of

depression also have no indefinite duration and the effects disappear after a year (Weisz et al., 2006). This similarity in stability of effects of indicated prevention and treatment of depression might be explained by the recurrent nature of depression. One could argue that indicated prevention and treatment of depression cure subclinical or clinical disorders significantly, but that recurrence of symptoms is unavoidable with current treatment strategies. Other researchers stated that if symptoms do return, then an indicated prevention or treatment still can be said to have an enduring effect and the symptoms return less intense than without prevention or treatment (Hollon et al., 2006). Additionally they state that effects of indicated prevention may not appear immediately, but may become beneficial over time. Our study does, however, not allow us to confirm or refute these statements, due to methodological limitations. Suggestions to improve the effectiveness are mostly focused on increasing the duration of the effects of the prevention programs. Results from a meta-analysis showed that the integration of occasional long-term sessions, so-called booster sessions, sustained the effects cognitive behavioral therapy based treatment of depression and anxiety disorders in youth (Gearing, Schwalbe, Lee, & Hoagwood, 2013). Implementing these booster sessions in prevention programs might result in similar sustaining effects in indicated depression and anxiety prevention for adolescents.

Anxiety prevention for at-risk populations showed no significant effects of prevention. This could suggest that the symptomatology in high-risk adolescents is actually too high to solve with prevention programs. To illustrate, adolescents in studies included in the meta-analysis showed clinical levels of anxiety symptoms. To effectively target these symptom levels, the interventions need more additional techniques than only social skills training or relaxation exercises, as some of the preventive interventions in our meta-analysis seem to contain (Balle & Tortella-Feliu, 2010; Dadds et al., 1997). This implies that anxiety prevention programs for at-risk adolescents should presumably be more similar to treatment techniques. It should use cognitive restructuring techniques, but also should include exposure techniques, which have shown to be effective in treating anxiety disorders (Cartwright-Hatton et al., 2004; Compton et al., 2004; Davis et al., 2011; Rapee et al., 2009).

The results of our meta-analysis gave insight to the shortcomings of depression and anxiety prevention for at-risk adolescents. Our suggestions to improve the effectiveness of depression and anxiety prevention are, firstly, to use techniques of cognitive behavioral therapy in prevention programs, and, secondly, to use booster sessions to improve the duration of effects. Consequently, more children and adolescents can benefit from effective indicated depression and anxiety prevention and potentially the number of youth needing treatment reduces.

Effectiveness of the prevention program 'Een Sprong Vooruit'

We examined the effectiveness of 'Een Sprong Vooruit' in preventing depression and anxiety among female adolescents with a high familial risk with a randomized controlled trial. 'Een Sprong Vooruit' is a program based on evidence-based techniques for the treatment of

depression (Beck, 1967) and anxiety disorders (Beck, 2005; Nolen-Hoeksema, 1991), given in groups on secondary schools. The program also includes psychoeducation about parental mental health problems and strengthening the social support network of the adolescents. Results showed that the decrease in depression and anxiety symptoms was not significantly different between adolescents in the intervention condition and adolescents in the control condition, and therefore we cannot conclude that 'Een Sprong Vooruit' was effective. Additional analyses showed that the development of depression and anxiety symptoms was not significantly different between adolescents who received the intervention and adolescents who did not receive a prevention program.

Based on earlier research, we expected that adolescents whose parents had low levels of parental psychopathology would benefit more from the prevention program than adolescents whose parents had higher levels of parental psychopathology (Garber et al., 2016; Weersing et al., 2016). With regard to the known relation between parental psychopathology, parenting behavior, and parenting stress (Berg-Nielsen et al., 2002; Mowbray et al., 2004), we expected the same for adolescents whose parents showed low levels of parental psychopathology, or low levels of negative parenting behavior. Nonetheless, findings showed that parental psychopathology, parenting behavior, and parenting stress, did not moderate the effect of prevention on reducing depression and anxiety symptoms.

Several factors related to this randomized controlled trial could have resulted in a lack of program effects in the present study. First, the number of sessions could have been too limited to effectively transfer the techniques to the adolescents. This is confirmed by meta-analytic results, which stated that the effectiveness of prevention programs was significantly lower in program with eight or fewer sessions (Jane-Llopis et al., 2003). Second, rather than focusing on one technique, we used several techniques, that is, psychoeducation, cognitive restructuring, behavioral activation, exposure, and strengthening adolescents' social network. Despite the fact that earlier research showed that prevention programs that included three or more different types of techniques were significantly better than those that included only one or two, we believe that this should be seen in perspective to the number of sessions (Jane-Llopis et al., 2003). Hence, when prevention programs are short and consist of a small number of sessions, in our case six sessions, the number of different techniques should be limited in order to give participants enough time to incorporate these techniques. Finally, the influence of parental mental health problems might have been of more influence in all female adolescents than we anticipated. Recent studies showed that parents with mental health problems had a negative influence on the outcome of a prevention program for their children (Brent et al., 2015; Weersing et al., 2016). Involving parents in a preventive intervention could possibly improve the outcome of preventive interventions for adolescents (Beardslee et al., 2013; Clarke et al., 2001; Collins & Dozois, 2008; Compas et al., 2009; Compas et al., 2011; Garber et al., 2009).

Limitations

Specific limitations are mentioned and discussed in the relevant chapters. We want to make some general remarks, however, regarding the limitations of the present dissertation.

First, although we assessed depression and anxiety symptoms with commonly used measures, no assessment of actual depression and anxiety disorders was performed by using a clinical interview. Using the Children's Depression Inventory 2 (CDI 2) to measure depression symptoms and Spence Children's Anxiety Scale (SCAS) to measure anxiety symptoms enables us to draw conclusions about the development of the severity of symptoms over time. Unfortunately, we cannot conclude whether the prevention program actually prevented diagnoses of depression and anxiety disorders. In order to do that, one should use a clinical interview to determine clinical diagnoses. The Schedule for Affective Disorders and Schizophrenia for School Aged Children (K-SADS) (Kaufman, Birmaher, Brent, Rao, & Ryan, 1996; Reichart, Wals, & Hillegers, 2000) and the Anxiety Disorders Interview Schedule for DSM-IV-Child version (ADIS-C) (Siebelinck, Treffers, & De Ryke, 2001; Silverman & Albano, 1996) are commonly used in the clinical practice in The Netherlands to diagnose depression and anxiety disorders in children and adolescents. Using these instruments in combination with severity questionnaires in prevention trails enables to draw conclusions on prevented diagnoses and not merely on development of severity of symptoms.

Another point of consideration is our targeted population. We aimed to focus on high-risk female adolescents with subclinical depression or anxiety symptoms and with parents with mental health problems. In order to select adolescents with these risk factors present, we assessed depression and anxiety symptoms, and screened their perception of parental psychopathology. This measure of perceived parental psychopathology is not valid enough to measure the actual psychopathological disorders in parents, but it is merely an indication of parental mental health problems. Parent of included adolescents were asked to report on their own psychopathology in order to verify the level of parental psychopathology. It appeared, however, that the level of parental psychopathology did not completely correspond to the perceived parental psychopathology that was rated by the adolescents. This discrepancy between perception of children of their parents' psychopathology and the level of general psychopathology rated by parents themselves could potentially be a result of a negative perceptual bias in adolescents caused by their own depression and anxiety symptoms. According to the distortion hypothesis, the informants' psychopathology influences the report of symptoms of a different person (Richters, 1992; Richters & Pellegrini, 1989). This might have caused that adolescents' perception of parental psychopathology was higher than the actual general psychopathology in parents. The measures of perceived parental psychopathology and general psychopathology did not allow to differentiate between healthy parents and parents with mental health problems, therefore, perceived parental psychopathology and general psychopathology in parents were used as continuous measures. To include participants for prevention program for children of parents

with a mental illness, we suggest that parental depression and anxiety should be measured using a disorder specific instrument and this may result in more valid and reliable measurements of specific types of parental psychopathology.

Further, participants in our study were all from a specific target group, namely female adolescents. Consequently, our findings cannot be easily generalized to other populations, such as males, younger children and young adults. As depression and anxiety prevention is a very important issue in these populations, replication studies are needed in order to generalize findings to these groups.

Finally, the length of the follow-up was relatively short for a prevention study. As we mentioned before, the aim of the described trial was to examine whether the prevention program was effective in reducing symptoms or in preventing symptoms of depression and anxiety from developing. A follow-up duration of twelve months enables to measure the development of depression and anxiety symptoms and allows to analyze whether the symptoms change as a results of the preventive intervention. It is known that having subclinical symptoms of depression and anxiety is a risk factor for developing a clinical disorder, and reducing the symptom level is actually also reducing the risk at a clinical disorder. Nonetheless, this relatively short follow-up does not allow to adequately analyze whether depression and anxiety disorders start developing over time and whether the intervention prevented the actual onset of the disorders. To accomplish that, we suggest to implement a larger follow-up period, which is not yet often done before (Brent et al., 2015).

Gaps in existing knowledge and suggestions for future research

There are a number of issues that need to be further investigated regarding depression and anxiety prevention in high-risk adolescents. Below, several potential avenues for future research will be described.

We studied several risk factors for the development of depression and anxiety symptoms. We know that mental health of parents is of major influence on the development of depression and anxiety symptoms. Additionally, it is known that mental disorders in parents have a negative influence on the outcome of a preventive intervention in their children. Research showed that adolescents whose parents had a diagnosis of depression during their children's intervention, experienced less benefits of the preventive intervention (Brent et al., 2015). This suggests that symptomatology of children of parents with mental health problems is more persistent and this could mean that this population of adolescents needs other forms of care. There are several interventions aimed at adolescents, their parents or both, that are focused on reducing stress that is associated with parental depression. The Coping With Depression program targets maladaptive coping strategies through cognitive behavioral therapy for children (Clarke et al., 2001). In the first study on

the effectiveness of the Coping With Depression program, it has been shown that the program could effectively reduce diagnoses of depression in children (Clarke et al., 2001). This result has also been replicated in a large randomized controlled trial (Beardslee et al., 2013; Garber et al., 2009). Despite these positive effects, it is known that involvement of parents, especially when parents suffer from mental health problems, improves the outcome of preventive interventions for adolescents (Collins & Dozois, 2008). The program Raising Healthy Children has combined cognitive behavioral therapy for adolescents with parenting skills training for parents with depression. This program also showed positive effects on reducing depression in children of parents with mental health problems (Compas et al., 2009; Compas et al., 2011). These programs are theoretically more adequate for adolescents with parents with mental health problems than depression and anxiety prevention programs for adolescents with elevated levels of depression and anxiety symptoms. Based on our findings that paternal psychopathology is an important risk factor in the development of depression and anxiety symptoms during adolescence, it is important that not only mother participate in these programs, but also fathers. The results of both programs are yet to be replicated when the programs are implemented in clinical practice.

Also, other risk factors could be important to keep in mind. We showed that parenting behavior is of influence on the development of depression and anxiety symptoms and needs to be taken into consideration when selecting adolescents for prevention programs. Previous research further showed that inter-parental communication, specifically during inter-parental conflict, is related to depression and anxiety symptoms in children and adolescents (Bögels & Brechman-Toussaint, 2006; Cummings, Davies, & Simpson, 1994). Specifically for these children and adolescents, prevention programs are developed (Wolchik et al., 2002; Wolchik et al., 2000). Nevertheless, translation of this evidence into prevention and intervention programs lags behind, and effects of programs remain to be identified.

When making suggestions for future research, we need to keep in mind the changes and trends in the way mental health care for youth is organized in The Netherlands. From January 2015, professionals in youth care are expected to work more integrated and combine forces from different clinical and practical backgrounds. This means that stronger connections need to be realized between specialized mental health care, community care, primary and secondary schools. Further, the focus of care should be empowering and strengthening the child as an individual in dialogue with parents and finding solutions for their questions. An important change is that youth policy should pay more attention to prevention and early support, all in perspective of youth's and parents' own capacities. Needless to say, prevention strategies need to be well adjusted to the changes in policy to be feasible for implementation. Important issues to work on in depression and anxiety prevention for adolescents in light of these changes in youth care, are not only to study the effectiveness of depression and anxiety prevention programs, but also study how to improve the effectiveness and how to increase the duration of the effects. But foremost, it needs to be clear how to effectively identify adolescents who can benefit from preventive

interventions for depression and anxiety, or who are possibly in need for actual treatment. Recent developments show prevention strategies where short school-based universal mental health programs for all students are combined with screening on depression symptoms by community care professionals (Wasserman et al., 2012; Wasserman et al., 2015). The integrated program showed reduced negative perceptions, improved coping skills in adverse life events, and increased mental health awareness in adolescents. Adolescents with subclinical symptoms will be offered an additional indicated depression prevention program. This strategy will make early identification possible and is fully consistent with the goals of the transformation in youth care in The Netherlands.

Clinical implications

Our findings provide suggestions for depression and anxiety prevention for adolescents. Our suggestions for clinical practice are twofold: early identification of at-risk adolescents is essential, and prevention of depression and anxiety needs to be more and effective with enduring effects.

Early identification of adolescents at risk for developing depression or anxiety is pivotal in the battle to reduce the prevalence of depression and anxiety. Given the individual consequences but also the societal burden of these disorders, particularly depression, it is necessary to execute an active screening strategy to identify adolescents who could benefit from prevention programs. Currently, regular mental health care is focused on adolescents with significant symptoms and a diagnosis of a clinical disorder, and these children and adolescents receive treatment for these disorders. Adolescents with subclinical symptoms but without a diagnosis of a clinical disorder fall outside the scope of the regular mental health care. Most adolescents themselves are not aware of their symptom severity, nor will they seek treatment if they do. Hence, adolescents with subclinical disorders are left unidentified and untreated. For this reason, we recommend that an early identification strategy is implemented in secondary schools and that every adolescent is screened on elevated symptoms. Our research revealed that many adolescents experienced symptoms of depression or anxiety which otherwise would not have been recognized.

In line with the recommendation of early identification, we suggest to subsequently provide a preventive intervention. Early identification and shortening the duration of untreated depression and anxiety symptoms may improve the prognosis and may diminish the risk on chronicity. Providing these prevention programs within the context of secondary schools gives an opportunity to improve mental health of adolescents within their own familiar environment. This also overcomes several barriers to seek help, such as stigmatization and negative perception of mental health care providers. Although depression and anxiety are strongly related, we suggest to focus on either one of them in one prevention program in order to successfully target symptoms. When promoting mental health in children, the

focus should be on preventing anxiety disorders, as this is the most prevalent mental disorders at this age (Merikangas et al., 2010). Focusing on mental health in adolescents, we advocate to focus on depression because especially this disorder seems specifically to rise during this life phase. Further, results of this dissertation showed that depression prevention for adolescents at risk is more effective and should therefore be the intervention of preference. Focusing on children of parents with mental health problems is more challenging because this group of children and adolescents is more difficult to define and select, and interventions for this group need more evidence before implementation in clinical practice, and therefore, this remains an area for future research.

Besides specific preventive interventions for adolescents, it is time to create awareness of the burden of depression and anxiety, to promote mental health and to support vulnerable adolescents effectively by organizing mental health care in a way that matches their convenience. Only when adolescents and their parents are aware of the detrimental consequences in physical and mental health and the societal burden of depression and anxiety, they are willing to invest in promotion of mental health, and reducing the prevalence of mental disorders on the longer term is possible.

Concluding remarks

Depression and anxiety are pervasive mental disorders that affect many young people in their daily lives and will continue to complicate their lives when it is not prevented or in worst case not treated effectively. In recent years, research has been done on the effectivity of depression and anxiety programs and on improving mental health in adolescents. The current dissertation contributed to the existing literature on risk factors for the development of depression and anxiety symptoms and on the effectiveness of prevention for adolescents with high-risk on developing depression or anxiety. Although the depression and anxiety prevention program was not effective in reducing depression or anxiety symptoms or reducing the onset of depression and anxiety symptoms on the longer term, we concluded from our meta-analysis that depression prevention is effective in high-risk adolescents. Forthcoming research should focus on early identification and effective prevention programs with longer duration of effects. It is time to create awareness of the burden of depression and anxiety, to promote mental health and to support vulnerable adolescents effectively.

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Nederlandse samenvatting (Dutch summary)

De adolescentie wordt gekenmerkt door het steeds meer zelfstandig worden van jongeren, en het steeds belangrijker worden van leeftijdsgenoten worden in verhouding tot ouders en leraren. Het is ook de periode waarin jongeren op sociaal en emotioneel gebied onafhankelijk worden. Daarnaast is deze ontwikkelingsfase van groot belang gezien de verhoogde kwetsbaarheid om psychische en gedragsproblemen te ontwikkelen. Depressie en angst zijn de meest voorkomende psychische stoornissen tijdens de adolescentie. De stoornissen komen vaak samen voor en symptomen zijn voor een deel gelijk. Maar ondanks de grote overeenkomsten zijn het weldegelijk verschillende stoornissen. Depressie wordt gekenmerkt door een sombere stemming en een verminderde interesse of verlies in plezier in activiteiten. Specifiek bij adolescenten worden irritatie en boosheid ook vaak waargenomen. Daarnaast is er vaak sprake van verstoring in slaap- en eetpatronen en fysieke klachten als buik- en hoofpijn. Angststoornissen worden gekenmerkt door buitensporige angst, fysiologische angstsymptomen, overmatig zorgen maken en rusteloosheid. Ondanks dat depressie en angst verschillende stoornissen zijn, kennen ze een grote overeenkomsten en hebben ze gelijke disfunctionele cognitieve processen, negatieve automatische gedachten en maladaptieve overtuigingen als latent onderliggende mechanismen.

Depressieve en angstsymptomen nemen toe gedurende de adolescentie. Er wordt geschat dat 12,6% van de adolescenten tussen 13 en 17 een depressieve stoornis heeft, en voor angststoornissen wordt dat geschat op 32,4%. De adolescenten die te maken hebben met depressieve of angstsymptomen zonder aan de kenmerken van een stoornis te voldoen zijn niet vertegenwoordigd in deze cijfers. Zowel depressie als angst komt vaker voor bij adolescente meiden dan bij adolescente jongens. De negatieve gevolgen van depressie en angst tijdens de adolescentie zijn groot. Beide stoornissen hebben nadelige consequenties voor individuen, zoals problemen op sociaal en familiair gebied, verminderde schoolprestaties en grotere kans op uitval of werkeloosheid. De meest schadelijke consequenties zijn de gevolgen op het gebied van gezondheid, zoals verhoogde kans op middelenmisbruik, depressie en angst op latere leeftijd en suïcide. Daarnaast zijn er ook maatschappelijke consequenties. Depressie zorgt wereldwijd namelijk voor de grootste ziektelast en ook angststoornissen leveren hierin een significante bijdrage. Ook in Nederland zorgen depressie en angst voor hoge directe en indirecte maatschappelijke kosten.

Er zijn verschillende factoren bekend die zorgen voor een verhoogd risico op de ontwikkeling van depressie en angstsymptomen. Wanneer ouders zelf een depressie of angststoornis hebben, neemt het risico voor kinderen toe. Dit kan deels worden verklaard door een genetische aanleg, maar deels ook door gedrag dat hoort bij deze psychische problemen, zoals weinig interesse tonen en vermijden. Daarnaast kan ook gedrag van ouders in de opvoeding een rol spelen, zoals emotionele steun van ouders aan hun kinderen en respect van ouders voor de autonomie van hun kinderen. Het is bekend dat weinig emotionele steun van ouders samenhangt met meer depressie en angstsymptomen bij

adolescenten. Weinig respect voor autonomie hangt daarentegen weer samen met hogere angstsymptomen bij adolescenten. De specifieke relaties tussen depressie en angstsymptomen enerzijds en psychische problemen, emotionele steun, respect voor autonomie en de interactie ertussen anderzijds moet nog verder onderzocht worden. Naast oudergerelateerde risicofactoren zijn er ook nog kenmerken van adolescenten die een rol kunnen spelen in de ontwikkeling van depressie en angststoornissen. Het hebben van een hoge mate van depressie en angstsymptomen is een risico om ook een depressie of angststoornis te ontwikkelen. Daarom is het vroeg herkennen van symptomen belangrijk in het voorkomen van die stoornissen. Daarnaast is bekend dat depressie en angststoornissen vaker voorkomen bij adolescente meiden en daarmee lopen zij dus een groter risico om een van deze stoornissen te ontwikkelen.

Onderzoek heeft aangetoond dat cognitieve gedragstherapie als behandeling effectief is voor het behandelen van depressie en angststoornissen. Het richt zich op de factoren die ten grondslag liggen aan depressie en angst, namelijk disfunctionele cognitieve processen, maladaptieve overtuigingen, respons stijl en cognitieve fouten. Tijdens de behandeling worden disfunctionele of negatieve gedachten geïdentificeerd en vervangen door meer positieve gedachten. Dit heeft als gevolg dat depressie en angstsymptomen afnemen. Waar behandeling als doel heeft om bestaande depressie en angststoornissen te behandelen heeft preventie als doel om bestaande symptomen te reduceren en te voorkomen. Aangezien de doelstelling van behandeling en preventie overeenkomst hebben en cognitieve gedragstherapie effectief is gebleken voor behandeling, lijkt het erop dit ook een effectieve strategie kan zijn voor depressie en angstpreventie.

Het doel van dit proefschrift was om een bijdrage te leveren aan de kennis over risicofactoren voor de ontwikkeling van depressie en angstsymptomen en de effectiviteit van preventie bij adolescente meiden met een verhoogd risico. Het onderzoeken van risicofactoren gerelateerd aan ouders zou kunnen bijdragen aan het herkennen en selecteren van adolescenten die al op jonge leeftijd baat kunnen hebben bij een preventieve interventie. Verder was het van belang om te bepalen wat de effectiviteit is van depressie en angstpreventie. Aanvullend onderzochten we de effectiviteit van het preventie programma 'Een Sprong Vooruit', en of het zou kunnen bijdragen aan verminderen van negatieve consequenties van depressie en angst bij adolescente meiden.

In het eerste deel, bestaande uit hoofdstuk 2 en 3, werd gekeken factoren die gerelateerd zijn aan ouders en een risico vormen voor de ontwikkeling van depressie en angst bij adolescente meiden. In hoofdstuk 2 keken we naar de perceptie van adolescenten over de psychische problemen van hun ouders, dat wil zeggen de adolescenten gaven aan in welke mate zij de kenmerken van depressie en angst bij hun ouders herkenden. De resultaten lieten zien dat dat de perceptie van psychische problemen bij ouders gerelateerd was aan depressie en angstsymptomen bij adolescente meiden. Hogere perceptie van psychische problemen bij moeder en vader was gerelateerd aan hogere depressie en angstsymptomen bij adolescenten.

Daarbij werd gevonden dat wanneer de perceptie van psychische problemen bij zowel moeder als vader hoog was, dat de symptomen bij adolescenten hoger was dan wanneer die slechts een van de ouders hoog was. In hoofdstuk 3 werden de factoren emotionele steun en respect voor autonomie van ouders bekeken in combinatie met psychische problemen bij ouders zoals ouders die zelf rapporteerden. Uit de resultaten bleek dat alleen emotionele steun en respect voor autonomie van vader een rol speelde, en niet van moeder. Weinig emotionele steun van vader was gerelateerd aan hogere depressie en angstsymptomen bij adolescente meiden. Hogere mate van respect voor autonomie van vader bleek gerelateerd aan de ontwikkeling angstsymptomen over tijd bij adolescenten. Aanvullend hier aan werd gevonden dat er een interactie effect bestond tussen psychische problemen en respect voor autonomie van vader op het niveau van depressie symptomen bij adolescenten meiden. Dit wil zeggen dat wanneer er geen sprake was van psychische problemen bij vader, een hoge mate van respect voor autonomie gerelateerd was aan weinig depressie symptomen. Wanneer er sprake was van hogere mate van psychische problemen bij vader draaide die relatie om en was een hoge mate van respect voor autonomie juist gerelateerd aan meer depressieve symptomen bij adolescente meiden.

In het tweede deel, bestaande uit hoofdstuk 4, 5, 6 en 7, werd gekeken naar de effectiviteit van preventie van depressie en angst bij adolescenten met een hoog risico. In hoofdstuk 4 werden de resultaten van een meta-analyse naar de effectiviteit van depressie en angst preventie voor adolescenten met een hoog risico gepresenteerd. Daaruit bleek dat depressiepreventie programma's een klein effect laten zien direct na de interventie en zes maanden na de interventie op de afname van depressie symptomen bij hoog-risico adolescenten. Twaalf maanden na de interventie was het effect achter verdwenen. Angstpreventie programma's bleken direct na de interventie en zes maanden na de interventie geen effect te hebben op afname van symptomen. Echter, 12 maanden na de interventie bleken de angstpreventie programma's een klein effect te hebben op de afname van angstsymptomen bij adolescenten met een hoog risico.

In hoofdstuk 5 presenteerden we het studieprotocol van de voorgenomen gerandomiseerde en gecontroleerde studie naar de effectiviteit van het depressie- en angstpreventie programma 'Een Sprong Vooruit'. Daarin wordt beschreven dat een grote groep adolescente meiden uit het eerste en tweede jaar van de middelbare school gaat deelnemen aan de studie. Adolescenten die meedoen aan de studie hebben verhoogde symptomen van depressie of angst en tenminste één van hun ouders hebben kenmerken van psychische problemen. Zij zullen op basis van toeval worden ingedeeld in de groep die het preventie programma gaat volgen en de groep die geen programma volgt. In hoofdstuk 6 werd getoond wat de resultaten waren van de studie naar deze effectiviteit. De preventieve interventie 'Een Sprong Vooruit' bleek geen effect te hebben op de afname van depressie en angstsymptomen bij adolescente meiden met verhoogde symptomen en met ouders met kenmerken van psychische problemen. De depressie symptomen van de adolescenten in de

groep die het preventie programma volgden en de adolescenten die geen programma volgden bleken evenveel af te nemen. Ook voor de angst symptomen gold dat er geen verschil bestond in afname tussen de adolescente meiden die het programma volgden en die geen programma volgden. In hoofdstuk 7 keken we nogmaals naar de effecten van het programma 'Een Sprong Vooruit'. Nu keken we naar de invloed van psychische problemen van ouders, emotionele steun en respect van autonomie van ouders en ouderlijke stress op de effecten van het preventie programma. Uit de resultaten bleek dat deze factoren van de zowel moeder als vader geen invloed had op de uitkomsten. Verder keken we naar het effect van het preventie programma op de eerder genoemde onderliggende mechanismen van depressie en angst, namelijk respons stijl, waaronder afleiding, probleem oplossen en rumineren, en cognitieve fouten, waaronder onderschatting van het vermogen om om te gaan met stressvolle situaties, personaliseren zonder gedachten lezen, selectieve abstractie, overgeneralisatie, en gedachten lezen. Ook op deze factoren bleek het preventie programma geen effect te hebben.

Concluderend kunnen we stellen dat de bevindingen in dit proefschrift hebben bijgedragen aan de kennis over risicofactoren voor de ontwikkeling van depressie en angst bij adolescente meiden. We hebben lieten zien dat psychische problemen bij ouders, minder emotionele steun en minder respect voor autonomie samenhangen met hogere symptomen van depressie en angst bij adolescenten. Daarnaast hebben we laten zien dat depressie en angstpreventie voor adolescenten met verhoogd risico effectief kan zijn. Het preventie-programma 'Een Sprong Vooruit' had echter geen effect op het verminderen van depressie en angstsymptomen bij adolescente meiden met een hoog risico.

Onze bevindingen bieden daarnaast ook suggesties voor depressie en angstpreventie in de klinische praktijk. Het vroegtijdig signaleren van adolescenten met een verhoogd risico is cruciaal. Gezien de individuelen negatieve consequenties en ook de maatschappelijke lasten, in het bijzonder van depressie, is het noodzakelijk om een actieve screeningsstrategie te hanteren om adolescenten die baat kunnen hebben bij een preventie programma vroegtijdig te herkennen. In lijn met deze vroegtijdige signalering moeten adolescenten de mogelijkheid krijgen deel te nemen aan een effectief preventie programma. Door die vroegtijdige signalering en directe interventie, en dus door de duur van onbehandelde depressie en angst zo kort mogelijk te laten zijn, verbetert de prognose voor de langere termijn. Deze strategie van vroegtijdige signalering en directe preventieve interventie voorkomt daarbij diverse problemen, zoals stigmatisering en moeite met het zoeken van hulp. Naast deze specifieke preventieve interventies voor adolescenten is het tijd om aandacht te creëren voor de last die depressie en angst met zich meebrengen, om de mentale gezondheid te bevorderen en om kwetsbare jongeren effectief te ondersteunen door het organiseren van de geestelijke gezondheidszorg op een manier die bij hun past. Alleen wanneer adolescenten en hun ouders zich bewust zijn van de schadelijke gevolgen in fysieke en mentale gezondheid en de maatschappelijke last van depressie en angst, zal de bereidheid om te investeren in mentale

gezondheid toenemen en zal de vermindering van psychische stoornissen op langere termijn mogelijk worden.

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Het zit erop! Graag wil ik de mensen bedanken die, direct of indirect, hebben bijgedragen aan dit fantastische resultaat!

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Curriculum Vitae

Sanne Rasing was born on February 12th 1983 in Arnhem, the Netherlands. After completing secondary education at Overbetuwe College in Bemmel, she started the bachelor study Psychology at the Radboud University in Nijmegen. In 2007, she graduated from the master study Neuro- and Rehabilitation Psychology at the Radboud University, and in 2009 from the master Mental Health Science at Maastricht University. After graduating, she spent one and a half year as a researcher at mental health care institution GGZ Oost Brabant working on several research projects, among which a longitudinal study on personality disorders in adult patients and a project on aggressive behavior of patients at the psychiatric emergency service.

In 2011 she started her PhD-project on the effectiveness of depression and anxiety prevention in high risk adolescents, which was a project of the academic workplace Inside-Out in Nijmegen and funded by ZonMw and GGZ Oost Brabant. In 2014, she received the National Youth Care Award with her research project, about which the jury stated: "This project assessed the need for care in a population which does not typically ask for care themselves. This makes this project one of the very few preventive initiatives." She spent the grant on a work visit to Vanderbilt University in the US to improve her statistical skills in structural equation modeling. Further, she presented her work at (inter)national conferences (e.g. European Association for Behavioral and Cognitive Therapies (EABCT) in Den Haag in 2014, ICPS in Amsterdam in 2015, Anxiety and Depression Association of America (ADAA) in Miami in 2015, EABCT in Stockholm in 2016), and participated in an international Summer School. She was involved in organizing an international expert meeting on depression prevention in adolescents attended by 16 international researchers and a national seminar on adolescent depression prevention attended by more than 200 professionals in health care, youth care and education. During her research project, she supervised students with their master thesis.

At this moment, she works as a senior researcher at the mental health care institution GGZ Oost Brabant. She is specializing in research on prevention and treatment of depression in children and adolescents and is supervising PhD students and health care professionals with their research. In October 2016, she also started working as a teacher at Utrecht University at the department of Youth & Family.

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