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Review

Improving prevention of depression and anxiety disorders: Repetitive negative thinking as a promising target

Maurice Topper*, Paul M.G. Emmelkamp, Thomas Ehring

University of Amsterdam, Faculty of Behavioral and Social Sciences, Department of Clinical Psychology, Amsterdam, The Netherlands

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ABSTRACT

Prevention of depression and anxiety disorders is widely acknowledged as an important health care investment. However, existing preventive interventions have only shown modest effects. In order to improve the efficacy of prevention of depression and anxiety disorders, a number of authors have suggested that it is promising to focus on selective prevention programs that are offered to individuals scoring high on clearly established risk factors, whereby the preventive intervention then specifically targets these risk variables. This review presents repetitive negative thinking (worry and rumination) as a promising target for the prevention of depression and anxiety disorders.

1. Introduction

Depression and anxiety disorders are highly prevalent (Kessler et al., 2005) and debilitating (Arikian & Gorman, 2001). Prevention has been suggested to be a promising undertaking that may help to reduce the individual and societal burden of these disorders. However, past prevention programs have only shown moderate effects. A number of researchers have therefore highlighted the need to develop innovative approaches to the prevention of anxiety disorders and depression (e.g., Horowitz & Garber, 2006; Muñoz, Cuijpers, Smit, Barrera, & Leykin, 2010; Zvolensky, Schmidt, Bernstein, & Keough, 2006). This review aims to explore the potential of repetitive negative thinking in the form of worry and rumination as a target for preventive interventions for depression and anxiety disorders.

In the first part of this article, the scope of the problem will be presented, followed by a summary and critical evaluation of existing research into the efficacy and effectiveness of preventive interventions for depression and anxiety disorders. Based on these findings, an alternative theory-based selective prevention approach will be sketched that has been suggested as a potential solution to a number of problems currently encountered in the field of prevention. In the second part of the review, we will explore the potential of repetitive negative thinking to be used as a target in a future prevention program using this approach.

2. The scope of the problem

Depression and anxiety disorders are among the most common psychological disorders worldwide. Retrospective surveys rank anxiety disorders as the most prevalent class of mental disorders, with an estimated 25.4–33.1% of the general population meeting criteria for an anxiety disorder at least once during their lifetime (Kessler et al., 2005; Oakley-Browne, Wells, Scott, McGee, & New Zealand Mental Hlth Survey, 2006). Depression is ranked as the most prevalent single mental disorder, with recent lifetime prevalence estimates ranging from 16.6% to 19% (Kessler et al., 2005). The true lifetime prevalence of anxiety disorders and depression may be even higher as recall failure in these retrospective surveys can be expected to lead to underestimation (Patten, 2003). For example, a prospective study following a 1972–1973 birth-cohort in New Zealand estimated a lifetime prevalence of 41.4% and 49.5% for depression and anxiety disorders respectively (Moffitt et al., 2010). Moreover, the prevalence of these disorders may even be increasing. Cross-temporal meta-analyses have shown that each successive generation since 1938 reports more depression and anxiety-related symptoms (Twenge et al., 2010).

On the societal level, depression and anxiety disorders impose enormous costs. For example, the medical care costs for participants meeting the criteria for a diagnosis of a depressive or an anxiety disorder are approximately twice as high as the costs for participants not meeting the criteria for a diagnosis (Simon, Ormel, VonKorff, & Barlow, 1995). Medical costs, however, only form a relatively small proportion of the costs involved as more than three quarters of the total cost burden can be attributed to indirect costs and encompass the effects of illness (morbidity), and early death (mortality) on earnings and productivity (Arikian & Gorman, 2001). A single episode of major depressive disorder is associated with

* Corresponding author at: University of Amsterdam, Faculty of Behavioral and Social Sciences, Department of Clinical Psychology, Weesperplein 4, 1018 XA Amsterdam, The Netherlands. Tel.: +31 20 5256651; fax: +31 20 6391396.
E-mail address: M.Topper@UvA.nl (M. Topper).

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an average of more than 5 weeks of lost productivity per worker (Kessler et al., 2006). Retrospective self-reports suggest that the comorbidity of a depressive disorder and an anxiety disorder results in 7.2 days of work cutback (number of days where usual activities are restricted), and 5.7 days of work loss (number of days unable to perform usual activities) per month (Lim, Sanderson, & Andrews, 2000).

On the individual level, depression and anxiety disorders are highly debilitating. The Global Burden of Disease study launched by the World Health Organization (WHO) in 2001 showed that depression is the leading cause of Years Lived with Disability (YLD), accounting for 12.1% of total YLDs, and the fourth leading cause of burden among all diseases, accounting for 4.46% of total Disability-Adjusted Life-Years (DALYs; Üstün, Ayuso-Mateos, Chatterji, Mathers, & Murray, 2004). Panic disorder, obsessive-compulsive disorder and posttraumatic stress disorder together account for 2.7% of total YLDs, and for .9% of total DALYs (World Health Organization, 2004). Similarly, depression and anxiety disorders are related to significantly reduced overall quality of life (Olatunji, Cisler, & Tolin, 2007; Strine et al., 2009).

In addition to the psychological symptoms that constitute depression and anxiety disorders, both types of disorders have also been associated with numerous negative outcomes including academic failure, lack of career advancement, unemployment, legal problems, physical health problems, migraine, smoking, drug use, poor social performance, impaired social relationships, social isolation, and high risk sexual behavior (e.g., Breslau & Davis, 1993; Klein, Torpey, Bufferd, & Dyson, 2008; Mazzaferro et al., 2006; McLoone, Hudson, & Rapee, 2006; Rohde, Lewinsohn, & Seeley, 1994). Moreover, there is a clear association of depression and anxiety disorders with an increased risk of suicide (Allgulander & Lavori, 1991; Davies, Naik, & Lee, 2001).

Reassuringly, evidence-based treatments for depression and anxiety disorders are available (e.g., Nathan & Gorman, 2007). However, a number of problems remain. First, estimates for a variety of populations show that a majority of people with a depression or an anxiety disorder do not seek treatment (Eisenberg, Golberstein, & Gollust, 2007; Eisenberg, Hunt, Speer, & Zivin, 2011; Wang et al., 2005). Second, a substantial number of those who do seek treatment do not respond (Barlow, 2004). As a consequence, the symptom reduction achieved in even exclusive and optimal use of evidence-based treatments for depression would only lead to a 36% (without placebo) to 67% (when including placebo) reduction of the total disease burden related to this disorder (Andrews, Sanderson, Corry, & Lapsley, 2000). Third, many clients terminate treatment prematurely, which leads to lower effects (Kazdin, 1996; Pinto-Meza et al., 2011). Fourth, even in those who do complete treatment and recover, 11% (Shapiro et al., 1995) to 83% (Jarrett et al., 2000) will experience a new episode of depression. Recurrence rates for anxiety disorders range from 39% for social phobia to 58% for panic disorder with agoraphobia (Bruce et al., 2005). In addition, a substantial number of individuals successfully treated for depression or an anxiety disorder continue to experience interpersonal problems (Garber, Kriss, Koch, & Lindholm, 1988) or physical health problems (Rohde et al., 1994). Finally, with the majority of treatments retaining the model of one-to-one treatment, the demand for mental health services far exceeds supply (Kazdin & Blase, 2011; Winnett, 1998).

In sum, although effective evidence-based treatments of depression and anxiety disorders are available, and although 5–13% of health budgets in established market economies are spent on mental health services (Andrews, Sanderson, Slade, & Issakidis, 2000), depression and anxiety disorders remain to present a high burden on societies, due to the problems presented above. A number of solutions to this serious problem have been suggested. These include efforts to disseminate evidence-based treatments in a more systematic way, so that they are available to a larger group of individuals (Clark et al., 2009; Shafran et al., 2009). However, it appears unlikely that *treatment* for already existing depression and anxiety disorders *alone* is sufficient to alleviate the burden of depression and anxiety disorders. Instead, the combination of high prevalence and high costs on the one hand, and low access of care, high risk of relapse, and the limited capacity of treatment resources on the other hand strongly argues for *prevention* as a complementary approach. Preventive interventions are usually provided in a group format and therefore demand relatively little investment from mental health resources compared to treatment. In addition, the wide-spread dissemination of effective prevention programs has the potential to diminish the prevalence of depression and anxiety disorders. Prevention has therefore been suggested to be the most promising solution to the problems around relapse, residual symptoms, and suboptimal treatment effects (National Research Council & Institute of Medicine, 2009). The following section will provide an overview of past research into the prevention of depression and anxiety disorders.

3. Past research into the prevention of depression and anxiety disorders[1]

3.1. Classification of preventive interventions

Preventive interventions can be subdivided into groups based on a number of different dimensions, including participant characteristics, setting, type of delivery, or techniques used. One of the most common distinctions made in the prevention literature concerns the risk status of participants that are offered the intervention. The original division into primary, secondary, and tertiary prevention (see e.g., Dozois & Dobson, 2004) has in recent years largely been replaced by a newer classification proposed by the National Research Council and the Institute of Medicine (IOM) (National Research Council & Institute of Medicine, 2009), which will also be used in this article. This classification distinguishes between universal, selective, and indicated prevention. *Universal prevention* includes all interventions targeting the general population without any pre-selection. *Selective prevention*, on the other hand, is offered to individuals who have an elevated risk of developing a disorder. Finally, *indicated prevention* is aimed at individuals showing early signs of symptoms of a disorder without meeting formal diagnostic criteria. Importantly, according to the IOM classification the term prevention is restricted to interventions offered to individuals who have not yet developed a particular disorder, whereby the interventions aim to prevent a future onset of the disorder (see also National Research Council & Institute of Medicine, 2009). Therefore, relapse or recurrence prevention in formerly diagnosed individuals, which was formerly subsumed under tertiary prevention, is explicitly excluded (see Muñoz et al., 2010; National Research Council & Institute of Medicine, 2009).

3.2. Target groups of preventive interventions

The typical target groups for preventive interventions are children, adolescents, or young adults (mostly undergraduate students) (for overviews see Horowitz & Garber, 2006; Merry et al., 2011; Muñoz et al., 2010; Teubert & Pinquart, 2011; Zalta, 2011). Targeting these groups appears advantageous from a developmental perspective as the typical onset of depression and anxiety disorders tends to be early in life. For example, in the 2007 National

[1] In preparation of this review of existing prevention programs, the literature was searched using the databases PsycInfo and Web of Science with the following search terms: (depress* OR anxiety) AND, (prevention OR preventive).

Survey of Mental Health and Wellbeing in Australia (McEvoy, Grove, & Slade, 2011) the median age of onset of anxiety disorders was 19 years. Longitudinal data indicate that the onset of depression starts to increase substantially at 15 years of age (Fergusson, Horwood, Ridder, & Beautrais, 2005). Early onset appears to be related with greater severity and comorbidity, and increases the likelihood of recurrence for depression and anxiety disorders during adulthood (Pine, Cohen, Gurley, Brook, & Ma, 1998; Ramsawh, Weisberg, Dyck, Stout, & Keller, 2011; Richards, 2011). Prevention during or prior to this period of heightened risk will therefore maximize the opportunity to alleviate individual and public health burden associated with depression and anxiety disorders. From a practical perspective, children, adolescent and young adult samples are particularly appropriate for prevention programs, as the school setting facilitates access to the relevant population for recruitment and intervention. Although community-based prevention programs exist (see Christensen, Pallister, Smale, Hickie, & Calear, 2010), most prevention programs have therefore been implemented in a school setting (Calear & Christensen, 2010).

3.3. Universal prevention of depression and anxiety disorders

3.3.1. Depression

The vast majority of universal prevention programs for depression follow a cognitive-behavioral orientation and include a set of basic Cognitive-Behavioral Therapy (CBT) strategies, most importantly psychoeducation, cognitive restructuring, and coping skills training. For example, the Problem Solving for Life (PSFL) program (Spence, Sheffield, & Donovan, 2003, 2005) is an 8-week intervention conducted by trained teachers, and offered to adolescents as part of the school curriculum. PSFL includes cognitive interventions teaching participants to identify and challenge dysfunctional thoughts as well as problems-solving skills. Similarly, the Penn Resiliency Program (Gillham et al., 2007; Jaycox, Reivich, Gillham, & Seligman, 1994b) consists of 12 sessions offered to preadolescent children in a group format. Interventions include psychoeducation about the effect of cognition on emotion and behavior, cognitive restructuring, and skills training for coping and problem-solving (e.g., relaxation, assertiveness, decision making). Only very few prevention programs have been developed that are not based on CBT. For example, Horowitz, Garber, Ciesla, Young, and Mufson (2007) used an 8-session interpersonal therapy (IPT) prevention group. Interventions include psychoeducation about the link between interpersonal relationships and mood, and learning coping skills to effectively deal with interpersonal problems.

In recent years, numerous narrative reviews of studies examining the efficacy of depression prevention programs have been published. Interestingly, the conclusions drawn in these reviews about the effects of universal prevention differ considerably, ranging from the very positive (e.g., Farrell & Barrett, 2007) to largely negative ones (e.g., Spence & Shortt, 2007). Results from meta-analyses on this issue therefore appear especially informative. Three recent meta-analyses have focused on the effects of preventive interventions on depressive symptom severities relative to a randomized control group (Horowitz & Garber, 2006; Merry et al., 2011; Stice, Shaw, Bohon, Marti, & Rohde, 2009b). Results showed only small effect sizes at post-intervention (Horowitz & Garber, 2006: $d = .12$; Merry et al., 2011: $g = -.10$; Stice, Shaw, Bohon, Marti, & Rohde, 2009a: $r = .04$)[2] as well as follow-up (Horowitz & Garber, 2006: $d = .02$; Merry et al., 2011: $g = -.09$; Stice et al., 2009a: $r = .06$). In addition, all three meta-analyses found significantly smaller effect sizes for universal prevention programs when compared to targeted (i.e., selected or indicated) programs.

It has been argued that the comparison between an intervention and a control group at post-intervention and follow-up as described above does not reflect true *prevention effects*; instead they parallel analyses conducted in the context of *treatment* efficacy studies (for a more extensive discussion of this issue see Cuijpers, van Straten, Smit, Mihalopoulos, & Beekman, 2008; Horowitz & Garber, 2006; Muñoz et al., 2010). Horowitz and Garber (2006) therefore proposed that two requirements are needed for a prevention effect in the strict sense to be present, namely (1) an increase in depressive symptoms in the control group and (2) no increase or a diminished increase of symptoms in the intervention group. When applying these criteria to the studies included in their meta-analysis, Horowitz and Garber found not a single prevention effect for any universal prevention study. Cuijpers, van Straten, Smit, et al. (2008) propose even stricter criteria suggesting that in order to be judged as effective, preventive interventions should be shown to prevent the onset of future disorders, as indicated by a lower incidence rate ratio than a control group. Only two studies evaluating the effects of universal prevention programs on the onset of depressive disorders could be identified in their meta-analysis, and the mean incidence rate ratio for these programs was non-significant. More recently, Merry et al. (2011) conducted a meta-analysis including data from a much larger group of studies, which painted a somewhat more positive picture. Universal prevention significantly reduced the incidence of depressive disorder post-treatment (RD = −.12)[3] and at 3–9 months follow-up (RD = −.19). Interestingly, the effect size was significantly larger compared to targeted programs immediately after treatment. However, the effect for universal programs disappeared at 1 year follow-up, whereas efficacy remained evident for targeted programs. Studies comparing preventive programs to a placebo control group showed no evidence of increased efficacy.

3.3.2. Anxiety disorders

As in the field of depression prevention, universal programs for the prevention of anxiety disorders also typically follow a basic CBT approach. In addition, they include more or less the same set of techniques already described in the context of depression prevention. The main strategies are psychoeducation, cognitive restructuring, and training to use functional coping skills, with the main difference being that techniques are focused on helping participants to deal with symptoms of anxiety rather than depression. The anxiety prevention program that has most extensively been studied to date was developed by Barrett and colleagues in Australia (FRIENDS program; e.g., Barrett & Turner, 2001; Barrett, Farrell, Ollendick, & Dadds, 2006). FRIENDS is offered in a group setting and focuses on psychoeducation about symptoms of anxiety, cognitive strategies aimed at identifying and challenging anxious thoughts, and the development of coping skills, including

[2] All effect sizes reported in this article are *between-group* effect sizes, i.e. they represent the effect of an intervention relative to a control group. In the meta-analysis by Horowitz and Garber (2006), Cohen's *d* was computed by dividing the difference between the post-treatment and follow-up scores, respectively, by the standard deviation of the control group. Cohen's (1988) criteria are used to classify effect sizes as small ($d = .20$–$.30$) medium ($d = .50$) or large ($d = .80$). In the meta-analysis by Stice et al. (2009b), the correlation coefficient *r* is used as the effect size. Criteria by Cohen (1988) are used to classify effect sizes as small ($r = .10$), medium ($r = .30$), and large ($r = .50$). In the meta-analysis by Merry et al. (2011), the particular definition of the standardized mean difference is Hedges' *g* which is the difference in means divided by the pooled standard deviation of participants' outcome across a trial. The effect size Hedge's *g* is derived from Cohen's *d*, but is regarded as less biased as they also take the sample size into account (Lipsey & Wilson, 2001).

[3] The risk difference (RD) is the difference between the observed risks (here: proportions of individuals with a diagnosis of depression) in the treatment group and the control group. It is calculated by subtracting the risk in the control group from the risk in the experimental condition. An outcome that is less than 0 indicates the experimental intervention lowers risk; an outcome greater than 0 means the intervention increases risk.

relaxation, problem-solving, and graduated exposure with self-reward for successful approach behavior. Other anxiety prevention programs include a similar set of strategies (e.g., Aune & Stiles, 2009; Hains & Ellmann, 1994; Roberts et al., 2010).

Three recent meta-analyses investigated the effects of anxiety prevention programs across studies (Fisak, Richard, & Mann, 2011; Teubert & Pinquart, 2011; Zalta, 2011). In all three meta-analyses, small but significant effect sizes were found when comparing universal interventions to a control group post-treatment (Fisak et al., 2011: $d = .17$; Teubert & Pinquart, 2011: $g = .12$; Zalta, 2011: $g = .29$) and at follow-up (Teubert & Pinquart, 2011: $g = .15$). In Fisak et al.'s (2011) meta-analysis, effects were found to remain stable at 6-month follow-up; however, no significant effect of the preventive intervention could be identified at 12-month follow-up any more. In one of the three meta-analyses, universal prevention programs showed significantly smaller effects than selected or indicated preventive interventions (Teubert & Pinquart, 2011); however, this was not replicated in the other two meta-analyses. None of the three studies analyzed prevention effects in a stricter sense as detailed by Horowitz and Garber (2006) or tested the effect of universal prevention trainings on the onset of future anxiety disorders.

3.3.3. Conclusions

In sum, universal prevention programs have been found to show significant *treatment effects* in that they lead to lower symptom severities of depression and anxiety at post-intervention and follow-up when compared to control groups. However, effect sizes are typically small, and tend to be smaller for universal prevention than selected or indicated prevention. Evidence for *preventive effects* according to strict criteria suggested by Horowitz and Garber (2006) and Cuijpers, van Straten, Smit, et al. (2008) is sparse. However, as described, Merry et al. (2011) come to a more positive conclusion regarding depression in their recent meta-analysis.

3.4. Selective and indicated prevention of depression and anxiety disorders

3.4.1. Depression

As in the case of universal prevention, the vast majority of selective and indicative prevention programs for depression are CBT-based. One of the most commonly used interventions in this area is the *Coping with Depression Course* (see Cuijpers, Munoz, Clarke, & Lewinsohn, 2009), a group intervention focusing on psychoeducation, social skills, cognitive restructuring, and behavioral activation. The program was also adapted for use with children and adolescents, the *Coping with Stress Course* (Clarke et al., 2001; Clarke et al., 1995). A small number of studies have evaluated interpersonal therapy as a preventive intervention for depression (e.g., Young, Mufson, & Davies, 2006; Zlotnick, Miller, Pearlstein, Howard, & Sweeney, 2006).

Studies evaluating *selective prevention* have focused on different high-risk groups including children of divorced parents (e.g., Gwynn & Brantley, 1987), children of parents suffering from psychiatric disorders (e.g., Beardslee et al., 1997; Roosa, Gensheimer, Short, Ayers, & Shell, 1989), children from low income ethnic minority families (e.g., Cardemil, Kim, Pinedo, & Miller, 2005), college students showing signs of cognitive vulnerability (Merry, McDowell, Wild, Bir, & Cunliffe, 2004) or pregnant women at risk of post-partum depression (Elliott et al., 2000; Zlotnick et al., 2006).

The crucial inclusion criterion for *indicated prevention* programs is the presence of elevated/subclinical levels of depressive symptoms without meeting full criteria for a depressive episode. Indicated prevention programs have been evaluated for different age groups, including children (e.g., Clarke et al., 1995; Jaycox et al., 1994b), adolescents (Arnarson & Craighead, 2011) adults (e.g., Allart-van Dam, Hosman, Hoogduin, & Schaap, 2007), and older adults (see Forsman, Schierenbeck, & Wahlbeck, 2011).

Results from the three meta-analyses presented earlier show small to moderate effect sizes for preventive interventions on symptom levels of depression at post-intervention (Horowitz & Garber, 2006, selected: $d = .30$, indicated: $d = .23$; Merry et al., 2011, selected and indicated: $g = -.31$; Stice et al., 2009b, selected and indicated: $r = .23$) as well as follow-up (Horowitz & Garber, 2006, selected: $d = .34$, indicated: $d = .31$; Merry et al., 2011, selected and indicated: $g = -.22$; Stice et al., 2009b, selected and indicated: $r = .14$). As described earlier, these meta-analyses found that selected and indicated prevention programs showed higher effect sizes than universal programs. When applying strict criteria, Horowitz and Garber (2006) found that only one selected prevention study (Quayle, Dzuirawiec, Roberts, Kane, & Ebsworthy, 2001), and three indicated prevention studies showed evidence for prevention (Freres, Gillham, Hamilton, & Patton, 2002; Jaycox, Reivich, Gillham, & Seligman, 1994a; Reivich, 1996), whereas the rest of the studies were indicative of treatment effects only. Results from the meta-analysis by Cuijpers, van Straten, Smit, et al. (2008) and Merry et al. (2011) testing the efficacy of prevention programs to prevent future onset of depression showed a somewhat more positive picture. Both studies demonstrated a significantly reduced risk for a future diagnosis of depression compared to control conditions (Cuijpers, van Straten, Smit, et al., 2008, selected: IRR = .72; indicated: IRR = .76; Merry et al., 2011, selected and targeted: RD = −.14). Although Merry (Merry et al., 2004) reported one study that demonstrated increased efficacy of targeted prevention programs compared to a placebo control group, meta-analytic results (Merry et al., 2011) did not confirm a significant difference.

3.4.2. Anxiety

In terms of the interventions used, selected and indicated preventive programs for anxiety are very similar to the universal programs described earlier, focusing on psychoeducation, cognitive restructuring, and learning coping skills (see Fisak et al., 2011; Teubert & Pinquart, 2011). Target groups of selected intervention programs include a variety of populations, for example adolescent migrants (Barrett, Sonderegger, & Xenos, 2003), children exposed to terrorism (Berger, Pat-Horenczyk, & Gelkopf, 2007), children from disadvantaged areas (e.g., Bonhauser et al., 2005; Roberts et al., 2010), smoking adolescents with high anxiety sensitivity (Feldner, Zvolensky, Babson, Leen-Feldner, & Schmidt, 2008), or children of parents with anxiety disorders (Ginsburg, 2009).

Meta-analytic studies show small to moderate effect sizes for selected and indicated prevention programs on anxiety symptoms at post-intervention (Fisak et al., 2011: $d = .26$; Teubert & Pinquart, 2011: $g = .32$; Zalta, 2011: $g = .21$) and follow-up (Teubert & Pinquart, 2011: $g = .23$). As described earlier, evidence from one meta-analysis suggests that selected and indicated prevention for anxiety is more effective than universal programs (Teubert & Pinquart, 2011). However, no information is available on preventive effects using strict criteria (Cuijpers, van Straten, Smit, et al., 2008; Horowitz & Garber, 2006).

3.4.3. Conclusions

Results from our review of the recent meta-analytic literature suggest that both universal and targeted intervention show significantly larger effects in reducing symptom severities of depression and anxiety than control groups. In addition, there is emerging evidence that preventive programs reduce the risk for future onset of depression. Overall, selected and indicated prevention appears to be more efficacious than universal prevention.

3.5. Limitations and possible future directions

In sum, past research has shown that preventive interventions for depression and anxiety can be effective. However, a number of limitations are noteworthy. First, there is increasing recognition in the field that the majority of past prevention studies suffer from serious methodological limitations. Most importantly, almost all studies evaluating universal prevention programs are seriously underpowered. To investigate true preventive effects, sample sizes well above $N = 10{,}000$ are needed (Muñoz et al., 2010). In addition, the majority of trials have compared prevention with no intervention. Only few studies compared intervention with placebo or attention control and results did not support a superiority of the preventive interventions. Although this finding may largely be the result of inadequate power, a larger number of trials comparing prevention programs to placebo or attention control conditions need to be conducted to rule out possible placebo effects. Finally, as pointed out by Horowitz and Garber (2006), most prevention trials to date have focused on *treatment* effects rather than *prevention*. Research using more rigorous methodology is needed before any firm conclusions regarding the efficacy of existing preventive interventions can be drawn.

Second, although results from recent meta-analyses show significant effects of preventive interventions, between group effect sizes in randomized controlled trials are typically only of small to moderate magnitude. Therefore, there certainly appears to be room for improvement in this area. In addition, the small magnitude of effects typically found raises the question as to whether preventive interventions for anxiety and depression are at all cost-effective, which can be assumed to be an important factor when it comes to the implementation of prevention into routine settings. Although targeted prevention includes the costs involved in screening to select individuals at risk, the efficiency of these interventions is relatively large as services are exclusively delivered to those in need. Universal prevention programs avoid the costs of screening but are related to high costs for the implementation of the program (e.g., training and possibly payment of facilitators) given the large scale on which universal programs operate. At the same time, the efficiency of universal interventions is lower as service is also delivered to individuals who have relatively little to gain. Universal prevention may still be cost-effective, however, if programs succeed to prevent the onset of depression in even a small number of cases at relatively low cost. Although there is some preliminary evidence for cost-effectiveness of indicated prevention programs (Mihalopoulos, Vos, Pirkis, Smit, & Carter, 2011), this area still needs further investigation.

Third, although evidence tends to show the highest effects for indicated prevention, a number of problems regarding this type of programs are noteworthy. Although clearly falling within the currently dominating definition of prevention suggested by the Institute of Medicine (National Research Council & Institute of Medicine, 2009), some authors have questioned whether it should not better be regarded as early intervention rather than prevention in its purest sense (e.g., Merry, 2007). In addition, there is increasing evidence that even in settings where indicated intervention is widely available, only a small minority of the target group is actually motivated to participate (Reynolds, 2009).

As described earlier, state-of-the-art reviews of prevention in the field of depression and/or anxiety disorders have come to differing conclusions, ranging from the enthusiastic to the very skeptical. However, regardless of the fact whether one regards the glass as half full or half empty, it is clear that future development in this area is needed, as the effects documented for existing programs fall short of the potential that is ascribed to prevention of emotional disorders (National Research Council & Institute of Medicine, 2009). So, how can we make prevention more efficacious, more cost-effective, and more acceptable to the target group?

A number of potential answers to this question have been put forward in the literature. First, based on emerging evidence that the effects of universal prevention falls behind that of selective and indicated prevention, it appears promising to mainly focus future prevention efforts on high risk individuals (see Bienvenu & Ginsburg, 2007; Craske & Zucker, 2001; Garber, 2006; Horowitz & Garber, 2006). A number of candidate variables have been suggested that could be used to identify individuals in need of preventive interventions. These include factors that are markers of increased risk, but are in themselves not modifiable, such as gender, family history of depression or genetic risk (see Garber, 2008; Reynolds, 2009). Muñoz et al. (2010) suggested a set of criteria that can be used to select risk factors for this purpose, including strength of the association between risk factor and disorder and health gain at population level. However, a different approach is also conceivable, in which theory-derived risk factors are not only used to identify at risk individuals, but in which these very risk factors are also the target of the following intervention (see also Craske & Zucker, 2001; Zvolensky et al., 2006). Whereas Garber (2006) suggested to develop broad-bent preventive interventions aiming at the modification of several theory-derived factors simultaneously before dismantling the resulting complex interventions in later research, a different approach would be to focus on one key risk factor instead. An advantage of this latter approach may be that interventions could be more focused and therefore potentially more effective.

Second, there is little variation in the content of past prevention programs that mostly consist of broadband CBT strategies. One intriguing idea on how to improve the efficacy of preventive interventions may be to individualize prevention programs to a greater degree (Vitiello, 2011). Individuals selected on the basis of cognitive vulnerability factors (see Bieling & Grant, 2007) could then receive a different program to those identified on the basis of a dysfunctional family environment (see Knappe, Beesdo-Baum, & Wittchen, 2010), or biological risk factors (see Reynolds, 2009). Individualizing prevention may also need to include the development of culturally adapted interventions (see Cardemil et al., 2005).

Third, past preventive interventions have specifically been developed for a particular disorder or group of disorders. However, there is increasing evidence that transdiagnostic risk factors are involved in a wide range of emotional disorders (Harvey, Watkins, Mansell, & Shafran, 2004; Nolen-Hoeksema & Watkins, 2011). The efficacy of prevention may possibly be increased by focusing more on risk factors across disorders and directly targeting these in the intervention (see Dozois, Seeds, & Collins, 2009; Nehmy, 2010).

Finally, whereas past prevention has mainly relied on group interventions carried out in schools or the community, researchers are increasingly testing whether the efficacy and cost-effectiveness of preventive interventions can be improved by using different settings and/or modes of delivery. In this context, the use of computerized and/or internet-based interventions appears especially promising (see Calear, Christensen, Mackinnon, Griffiths, & O'Kearney, 2009; Landback et al., 2009; Richardson, Stallard, & Velleman, 2010). The Internet is rapidly becoming an important tool for delivering psychological treatment (Cuijpers, van Straten, & Andersson, 2008). As Internet-based treatment will advance technologically and will become increasingly interactive, so will Internet-based intervention possibilities. Internet-based interventions have the potential to reach a large number of people and may boost cost-effectiveness through interventions requiring little or no therapeutic assistance (Kazdin & Blase, 2011).

In the remainder of this article, we will use repetitive negative thinking (worry and rumination) as an example to illustrate the theory-based selective prevention approach outlined above. We

will argue that repetitive negative thinking is a promising target for prevention as (1) there is ample evidence that this thought process is a transdiagnostic risk factor that can be used to identify individuals at risk of developing an emotional disorder, and (2) theoretically-derived evidence-based interventions are available that are effective in reducing levels of this risk factor, and could be adapted for use in preventive interventions.

4. Repetitive negative thinking as a promising target for preventive interventions

4.1. Repetitive negative thinking across disorders

Individuals suffering from different mental disorders report that they repeatedly think about their problems or negative experiences, and that they experience these thoughts as repetitive and difficult to control (for reviews see Ehring & Watkins, 2008; Ehring et al., 2011; Watkins, 2008). For example, depressed individuals often report thinking about their symptoms of depression, past failures, or problems they currently encounter (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Persistent and uncontrollable worrisome thoughts, on the other hand, are a key characteristic of generalized anxiety disorder (GAD) (Borkovec, Ray, & Stöber, 1998). However, repetitive negative thinking in the form of worry and/or rumination is also common in other anxiety disorders. Individuals suffering from social anxiety have been found to show heightened levels of post-event processing, i.e., repeated thinking about recent social situations, their own behavior within these situations and how they are evaluated by other people, as well as repetitive anticipatory processing related to future situations (e.g., Abbott & Rapee, 2004; McEvoy, Mahoney, Perini, & Kingsep, 2009). Similarly, heightened levels of rumination have been found to be present in posttraumatic stress disorder (PTSD) (Ehring, Frank, & Ehlers, 2008; Michael, Halligan, Clark, & Ehlers, 2007), and obsessive compulsive disorder (OCD) (Wahl et al., 2011). In sum, repetitive negative thinking has been shown to be characteristic of both depression and most anxiety disorders. It has therefore been suggested to be an important transdiagnostic process these disorders have in common (Ehring & Watkins, 2008; Harvey et al., 2004).

4.2. Repetitive negative thinking: one unitary process versus several related ones?

Research into the role of repetitive negative thinking in emotional disorders has typically distinguished between worry and rumination. Worry has been defined as "a chain of thoughts and images, negatively affect-laden and relatively uncontrollable" and as "an attempt to engage in mental problem-solving on an issue whose outcome is uncertain but contains the possibility of one or more negative outcomes" (Borkovec, Robinson, Pruzinsky, & DePree, 1983, p. 10). Worry has mainly been studied in the context of GAD, where it constitutes the core characteristic (APA, 2000). However, although the severity and intensity of worry is especially pronounced in GAD (Olatunji, Wolitzky-Taylor, Sawchuk, & Ciesielski, 2011), there is evidence showing that worry is similarly present across a range of Axis I disorders, including depression (Muris, Roelofs, Rassin, Franken, & Mayer, 2005; Starcevic, 1995; Stöber & Joormann, 2001) and other anxiety disorders (Chelminski & Zimmerman, 2003; Gladstone et al., 2005).

Rumination, on the other hand, has mainly been studied in the context of depression and is typically defined as "behaviors and thoughts that passively focus one's attention on one's depressive symptoms and on the implications of these symptoms" (Nolen-Hoeksema, 1998, p. 139). Importantly, however, rumination is not restricted to depression and has been found to be present in many other disorders, including anxiety disorders (e.g., Abbott & Rapee, 2004; Ehring et al., 2008; Wahl et al., 2011).

The finding that worry and rumination are present across different disorders has triggered an ongoing debate about the degree of overlap between these thought processes. Whereas some researchers argue that worry and rumination should be regarded as related, but distinct processes (e.g., Papageorgiou & Wells, 1999), others suggest that worry and rumination share the same process and only differ regarding disorder-related content (e.g., Segerstrom, Tsao, Alden, & Craske, 2000). Several studies using different methodological approaches have empirically examined the differences between worry and rumination. In sum, the findings reveal far more similarities than differences between worry and rumination (for an overview see Ehring & Watkins, 2008). Specifically, measures of worry and rumination are highly correlated and similarly related to symptom levels of anxiety and depression. Studies directly comparing characteristics of worry versus rumination have revealed some differences. Importantly, however, only one of these differences, namely temporal orientation, could be replicated across studies. Whereas worry was predominantly focused on the future, rumination was focused on the past and/or present. Finally, the experimental induction of worry versus rumination shows similar effects. In addition, worry and rumination appear to be generative of one another. A worry episode is likely to include a periodic recall of past loss or failure, whereas rumination about a past loss or failure also has implications for one's future, and will therefore generate worry (McLaughlin, Borkovec, & Sibrava, 2007).

The widespread presence of worry and rumination across disorders and the fact that the two processes show more similarities than differences have led a number of authors to conceptualize *repetitive negative thinking* as a transdiagnostic process that is characterized by similar or even identical process characteristics across disorders but is then applied to a different disorder-specific content (Ehring & Watkins, 2008; McEvoy, Mahoney, & Moulds, 2010; Segerstrom et al., 2000). Several generic disorder-independent definitions of this process have been suggested. For example, Martin and Tesser (1996) defined rumination as "a class of conscious thoughts that revolve around a common instrumental theme and that recur in the absence of immediate environmental demands requiring the thoughts" (p. 7). Similarly, Ehring et al. (2011) argued that the core of repetitive negative thinking is the fact that it is repetitive, intrusive, and difficult to disengage from, that it captures mental capacity and is perceived by individuals as unproductive.

4.3. The role of repetitive negative thinking in the development of depression and anxiety disorders

So far, the argument that a focus on repetitive negative thinking may be promising for preventive interventions was based on evidence showing that worry and rumination are present in depression and anxiety disorders. However, the mere presence of repetitive negative thinking in these disorders is only a prerequisite but not yet a persuasive argument for its usefulness as a target in preventive interventions. Some authors have suggested that worry may simply be an epiphenomenon or cognitive manifestation of emotion difficulties that only appears when the disorder has already developed (e.g., O'Neill, 1985). The use of repetitive negative thinking as a target for prevention appears only promising if it is shown to be a risk factor for psychopathology and causally involved in its onset and/or maintenance. In the following, emerging evidence supporting a causal relationship between repetitive negative thinking and depression and anxiety disorders will therefore be presented.

4.3.1. Rumination

A series of longitudinal studies have shown that elevated levels of rumination predict future depressive symptoms even after controlling for baseline levels of depression and anxiety (e.g., Abela, Brozina, & Haigh, 2002; Broderick & Korteland, 2004; Butler & Nolen-Hoeksema, 1994; Nolen-Hoeksema & Morrow, 1991). In addition, self-reported rumination has also been shown to predict the future onset, number and duration of major depressive episodes (Just & Alloy, 1997; Nolen-Hoeksema, 2000; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007; Robinson & Alloy, 2003) Similarly, naturally occurring rumination in caretakers of a deceased family member predicted depression six months after their relative had died (Nolen-Hoeksema, Parker, & Larson, 1994). Converging evidence comes from experimental studies showing that inducing rumination not only prolongs and exacerbates depressed mood, but also has a negative effect on a number of processes linked to the maintenance of depressive symptoms, including impaired concentration and problem solving, increased negative thinking, and poorer interpersonal coping (for reviews see Lyubomirsky & Tkach, 2004; Watkins, 2008).

Although the majority of research has focused on rumination in relation to depression, and worry in relation to anxiety, some studies have investigated the link between rumination and anxiety. Longitudinal studies have shown that individuals who have elevated levels of rumination report higher levels of anxiety symptoms over time even after controlling for baseline levels of anxiety symptoms (e.g., Ehring et al., 2008; Garnefski, Kraaij, & Spinhoven, 2001; Nolen-Hoeksema, 2000). Experimental studies inducing rumination additionally suggest that rumination prolongs anxious mood (e.g., Blagden & Craske, 1996; Ehring, Fuchs, & Kläsener, 2009; McLaughlin et al., 2007).

4.3.2. Worry

In comparison to the abundant evidence related to rumination as a risk factor for depression, considerably fewer longitudinal studies have tested worry as a predictor of anxiety. However, the studies that have been conducted clearly support the role of worry as a risk factor for anxiety. For example, two studies showed that worry predicts elevated levels of anxiety one month later (Hong, 2007), and anxious symptomatology 6–8 weeks later (Calmes & Roberts, 2007). In addition, worry prior to a presumably stressful exam period predicted higher anxiety after this period had ended (Segerstrom et al., 2000; Siddique, LaSalle-Ricci, Glass, Arnkoff, & Diaz, 2006). Finally, a series of prospective studies have shown that levels of worry and rumination assessed shortly after a traumatic event significantly and substantially predicted posttraumatic stress disorder some months later (e.g., Ehring et al., 2008; Mayou, Bryant, & Ehlers, 2001; Michael et al., 2007).

Importantly, extensive experimental research has shown negative effects of worry on a number of key processes. There is evidence that the induction of worry dampens the parasympathetic nervous system, leading to increased heart rate, decreased heart rate variability, and other symptoms of anxiety (e.g., Brosschot, Van Dijk, & Thayer, 2007). In addition, worry has been found to lead to increases in anxiety on the subjective and the physiological level (Lyonfields, Borkovec, & Thayer, 1995), as well as increases in threat expectancies (Stapinski, Abbott, & Rapee, 2010). On the physiological level, induced worry leads to a variety of responses reflecting anxiety, such as increased skin conductance and increased heart rate (Dua & King, 1987), decreased heart rate variability (e.g., Thayer, Friedman, & Borkovec, 1996), decreased respiratory sinus arrhythmia and increased left frontal activity (Hofmann et al., 2005).

Research investigating worry as a predictor of depressive symptoms has found that individuals who have elevated levels of worry report higher levels of depressive symptoms one month later (Hong, 2007). An experimental study inducing worry showed that worry not only evokes an anxious mood, but also contributes to a depressed mood (Andrews & Borkovec, 1988).

4.3.3. Conclusions

The studies discussed above suggest that repetitive negative thinking is not only present in depression and anxiety disorders, but also triggers symptoms of these disorders and predicts the development and maintenance of these disorders over time. This causal relationship is not only found between rumination and depression and between worry and anxiety, but also between worry and depression and between rumination and anxiety.

4.4. Summary

In this section we have reviewed evidence supporting repetitive negative thinking as a promising target for the prevention of depression and anxiety disorders. First, repetitive negative thinking is not only present in depression and anxiety disorders, but converging evidence points toward a causal role of this factor in the development of psychopathology. Second, there is evidence for repetitive negative thinking as a transdiagnostic process that shows similar characteristics across disorders and is similarly involved in depression as well as anxiety. Third, the two main forms of repetitive negative thinking, worry and rumination, are highly similar processes enabling intervention programs to directly target both processes using the same principles. Importantly, the proposition that worry and rumination require similar interventions is even shared by authors otherwise emphasizing differences between these processes (e.g., Papageorgiou & Wells, 1999).

A final requirement for repetitive negative thinking being a useful target for preventive interventions is of course noteworthy: It needs to be modifiable. To our knowledge, no *preventive* intervention targeting worry or rumination has been developed and evaluated to date. However, there are a number of different psychological *treatment* approaches for depression as well as generalized anxiety disorder that include techniques aiming to reduce levels of repetitive negative thinking. In the following chapter, we will therefore turn to the efficacy of interventions targeting worry and/or rumination. In a second step, we will discuss which of these approaches may be promising to be adapted for use in preventive interventions.

5. Efficacy of interventions targeting repetitive negative thinking

5.1. Interventions for depression that aim at targeting rumination

Rumination is regarded as a key maintaining factor of depression (e.g., Nolen-Hoeksema, 2000; Papageorgiou & Wells, 2004). Several treatments for depression therefore include strategies that are aimed at directly targeting rumination, including behavioral activation, rumination-focused cognitive therapy, metacognitive therapy, mindfulness-based techniques, and competitive memory training. These interventions will be described briefly and the effects of these treatments will be discussed.

5.1.1. Behavioral activation

In the seventies of the last century, Lewinsohn (1974) proposed that depression results from a lack of positively reinforced experiences in the environment and he developed a behavioral therapy for depression aimed at increasing positive events and reinforcers, as well as decreasing negative consequences. Behavioral activation is largely based on Lewinsohn's ideas and aims at keeping depressed clients active, rather than leading an avoidant lifestyle. Specifically, clients learn to monitor their mood and daily activities, and are guided to systematically increase the number of

pleasant as well as mastery activities and to increase positive interactions with their environment. In addition, recent extensions of behavioral activation treatment use functional analyses to identify depression-maintaining behavior, especially avoidance, and help clients to replace these with functional strategies. Within behavioral activation, rumination is conceptualized as a cognitive form of avoidance behavior, and functional analysis is used to replace this avoidance with more adequate approach behaviors (Kanter et al., 2010). Studies that have tested the effects of the behavioral components of CBT revealed that behavioral activation is as effective as cognitive therapy which is a combination of both cognitive and behavioral techniques (e.g., Dimidjian et al., 2006; Gortner, Gollan, Dobson, & Jacobson, 1998; Jacobson et al., 1996). However, to our knowledge no study to date has tested whether behavioral activation treatment is also effective in specifically reducing levels of rumination.

5.1.2. Rumination-focused CBT

Watkins (2008) suggested that rumination is a mediator of treatment effects for depression, and developed an adaptation of CBT (Rumination-Focused Cognitive-Behavioral Therapy – RFCBT), specifically targeting rumination (Watkins et al., 2007). On a theoretical as well as a technical level, RFCBT integrates two approaches. First, the basic behavior-analytic and contextual approach used in behavioral activation treatment is adapted to specifically focus on rumination as a dysfunctional coping and avoidance behavior respectively. Second, on the basis of experimental research (Watkins, 2008, 2011) a distinction is made between dysfunctional rumination characterized by an abstract-evaluative and conceptual style of processing on the one hand and a more functional concrete-experiential and non-evaluative mode of processing on the other hand. In practice, RFCBT uses functional behavior analysis to help clients to evaluate the (un)helpfulness of rumination about negative self-experience and teaches clients to shift into the most effective style of thinking. Concreteness training was developed to increase specificity of processing in clients with depression; experiential/imagery exercises and behavioral experiments are used in RFCBT to facilitate a more helpful concrete thinking style.

RFCBT was first investigated in a small case series of clients with residual depression (Watkins et al., 2007). Generally, treatment led to a substantial reduction in depressed mood and in self-reported rumination as assessed by the Ruminative Response Scale (RRS) (Nolen-Hoeksema & Morrow, 1991). A recent RCT investigated whether rumination-focused CBT provided added benefit to treatment as usual (TAU) in reducing residual symptoms of depression (Watkins et al., 2011). RFCBT plus TAU was more effective than TAU alone in reducing depressed mood and rumination. Moreover, change in rumination was found to be a mediator of the effects of treatment condition on reduction in depressive mood (Watkins et al., 2011).

5.1.3. Metacognitive therapy

According to the metacognitive model of emotional disorder, depression is maintained by engaging in dysfunctional coping strategies, including worry and rumination (Wells & Papageorgiou, 2004). Importantly, worry and rumination are thought to be maintained by positive as well as negative metacognitions. Positive metacognitions (e.g., "Rumination helps me to understand why I am feeling this way"; "Worry helps me to be prepared") are thought to initiate repetitive thinking. On the other hand, negative metacognitions (e.g., "Ruminating could make me go mad"; "Worrying puts my body under a lot of stress") are activated later in the process, which then leads to meta-worry/meta-rumination and engagement in dysfunctional coping strategies, such as thought suppression. In this way, repetitive thinking is maintained or even worsened.

Metacognitive therapy for depression aims to reduce the frequency of rumination and worry in response to negative thoughts, and more specifically focuses on (a) identifying rumination and threat monitoring, (b) challenging negative metacognitive beliefs about the uncontrollability and significance of depressive thoughts and feelings, and (c) challenging positive metacognitive beliefs about rumination and dysfunctional thought control strategies.

The effects of metacognitive therapy in depression were evaluated in a case series of four clients (Wells et al., 2009). Treatment was associated with clinically significant improvements in depressed mood, rumination and metacognitive beliefs. In a study by Nordahl (2009) clients of whom two third formally fulfilled ICD-10 criteria of depression were randomly assigned to either cognitive therapy or metacognitive therapy. Both treatments were found to be equally effective in terms of reduction of depressed mood, but metacognitive therapy resulted in greater improvement in meta-worry than cognitive therapy.

5.1.4. Mindfulness-based therapy

Mindfulness, a central concept in Buddhism, has been described as "bringing one's complete attention to the present experience" (Marlatt & Kristeller, 1999, p. 68), and as "paying attention in a particular way: on purpose, in the present moment and nonjudgmentally" (Kabat-Zinn, 1994, p. 4). The concept of mindfulness has been incorporated into therapeutic interventions to help clients adopt a more mindful approach to negative thoughts and feelings. Although different strategies exist to facilitate mindfulness, meditation is most commonly used in these interventions. The pure observant stance practiced in mindfulness therapy is supposed to discourage automatic, habitual thought patterns, including rumination (Teasdale et al., 2002). For instance, Mindfulness-Based Cognitive Therapy (MBCT) (Segal, Williams, & Teasdale, 2002), a treatment for chronic depression, promotes a decentered, nonjudgmental view of one's thoughts, emotions, and body sensations to prevent the deleterious loop of negative thoughts and moods characteristic for rumination.

The effect of mindfulness-based therapy has been investigated in several controlled studies on the occurrence of relapse in chronically depressed individuals. MBCT plus TAU significantly reduced relapse rates as compared to TAU (Godfrin & van Heeringen, 2010; Ma & Teasdale, 2004; Teasdale et al., 2000). Mindfulness-based training has also been shown to reduce depression in currently depressed individuals. In an uncontrolled study in a sample of 51 depressed patients, significant decreases in depression and anxiety were detected (Eisendrath et al., 2008). A controlled study conducted by Kingston, Dooley, Bates, Lawlor, and Malone (2007) showed that MBCT provided added benefit to treatment as usual in reducing depression and rumination. Finally, a recent meta-analysis found mindfulness based training an effective intervention in clients with depressed mood (Hofmann, Sawyer, Witt, & Oh, 2010).

5.1.5. Competitive Memory Training (COMET)

Competitive memory training (COMET) for depressive rumination focuses on the relative retrievability and activation of positive and functional personal experiences (Maarsingh, Korrelboom, & Huijbrechts, 2010). COMET targets the amount of involvement the patient has with negative thoughts and emotions, rather than on the content of dysfunctional cognitions. It uses counter memories to reduce the negative effects of rumination. An incompatible emotional network is installed by clients by teaching them to become indifferent or to adopt an attitude of acceptance. Through repetitive activation in the therapy sessions it is hoped that the incompatible emotional network inhibits the depressive ruminative network.

In two out-patient samples, one with mixed diagnoses (Olij et al., 2006) and one with major depression (Maarsingh et al., 2010),

COMET led to enhanced self-esteem and reduction in depressed mood, but these studies were uncontrolled. One controlled study with depressed elderly found COMET as add-on to care as usual more effective than care as usual alone in terms of reduction of depression and reduction of frequency of rumination (Ekkers et al., 2011).

5.2. Interventions for generalized anxiety disorder that aim at targeting worry

Worry is the key cognitive aspect of GAD. A number of primarily CBT interventions have been developed to target worry in GAD clients, including worry exposure, applied relaxation, cognitive and metacognitive therapy, and mindfulness training. Research into the effect of these treatments will be discussed below.

5.2.1. Overview of CBT treatments

Most evidence-based treatments for GAD include a complex package of treatment strategies aimed to target the cognitive, behavioral, and emotional processes thought to underlie pathological worry (Mitte, 2005). These treatments are based on theoretical models of GAD, such as Borkovec's cognitive avoidance model (Borkovec, Alcaine, & Behar, 2004; Borkovec et al., 1998) or the intolerance of uncertainty model by Dugas, Gagnon, Ladouceur, and Freeston (1998). Both models share an underlying commonality in their specific focus on worrying. Borkovec et al. (2004) suggest that worry serves as a cognitive avoidance strategy by suppressing anxious arousal. In this model, worry is primarily seen as a cognitive-verbal activity that inhibits emotional processing. In the multi-component CBT approach based on this model, clients are imaginally exposed to situations they are worrying about and to the associated physiological arousal which is then paired with a relaxation response (Borkovec, Newman, Pincus, & Lytle, 2002). This is combined with additional techniques, such as cognitive restructuring, applied relaxation, and stimulus control.

In the model developed by Dugas et al. (1998), intolerance of uncertainty is hold to result from a set of negative beliefs about uncertainty and its implications. In the CBT intervention developed on the basis of this model, clients are instructed to separate their worries into two categories, namely worries that are amenable to problem-solving and worries that are not. In the former case, clients are taught problem-solving strategies and in case that the problems cannot be changed, worry exposure is used (Dugas & Robichaud, 2006). In addition, cognitive restructuring is used to directly target intolerance of uncertainty. The goal of this approach is to help clients with GAD to develop beliefs about uncertainty that are less negative, rigid, and pervasive.

The CBT package of Borkovec and colleagues has been found effective in a number of controlled studies, in that it reduced symptoms of anxiety as well as levels of worry (Covin, Ouimet, Seeds, & Dozois, 2008; Fisher, 2006; Gould, Safren, Washington, & Otto, 2004; Mitte, 2005). The goal of a recent study by Newman et al. (2011) was to test whether the addition of techniques aimed at fostering emotional deepening and improving interpersonal functioning would increase the effect of the CBT package of Borkovec and colleagues. Results revealed that interpersonal emotion focused therapy did not enhance the effects of the standard CBT approach. Further, the CBT package of Borkovec and colleagues was significantly superior to psychodynamic therapy on worrying, trait-anxiety and depressed mood (Leichsenring et al., 2009).

The CBT package of Dugas and colleagues has been found to be more effective than a waiting list control condition (Dugas et al., 2010; Dugas et al., 2003; Ladouceur et al., 2000) and non-directive therapy (Gosselin, Ladouceur, Morin, Dugas, & Baillargeon, 2006). Interestingly, in the studies by Dugas et al. (2010, 2003), level of worry further decreased from posttreatment to 2-year follow-up.

In sum, both treatments described so far have been found to be effective. However, as these multi-component packages both include a number of different strategies, it remains unclear, which of these is responsible for the observed effects. In the following, studies focusing on one of the techniques only will be presented.

5.2.2. Worry exposure

To date, only one study investigated worry exposure as a stand-alone treatment for GAD (Hoyer et al., 2009). In this study, worry exposure led to clinically significant changes on anxiety, depression and worry. Worry exposure was more effective than a waiting list condition and as effective as applied relaxation.

5.2.3. Applied relaxation

There is substantial evidence that applied relaxation is an empirically supported treatment for GAD (Chambless & Ollendick, 2001). Results of studies reveal that applied relaxation results in reduced anxiety, worrying and depressed mood. In a study of Borkovec and Costello (1993), applied relaxation was as effective as the multi-component package based on the avoidance model of GAD. More recently, effects of applied relaxation were compared with the CBT package of Dugas and Robichaud (2006) described above. Results revealed that both treatments were more effective than no treatment, and that the multi-component CBT approach was slightly superior to applied relaxation (Dugas et al., 2010). Importantly, CBT did not lead to a statistically greater change in worry than applied relaxation. As described above, Hoyer et al. (2009) found applied relaxation to be as effective as worry exposure, not only on anxiety, but on severity of worry as well. Interestingly, although neither applied relaxation nor worry exposure involved cognitive restructuring, both treatments led to cognitive and metacognitive change.

5.2.4. Cognitive therapy

According to the cognitive model, anxiety is maintained by an individual's catastrophic misinterpretations of generally benign stimuli thus creating a self-perpetuating cycle of increasingly intensified anxiety (Beck, Emery, & Greenberg, 1985). As described above, cognitive restructuring techniques are part of most multi-component CBT packages for treating CBT. Siev and Chambless (2007) conducted a meta-analysis of studies that compared cognitive therapy and relaxation. Cognitive therapy proved equally efficacious in treating GAD in terms of anxiety, anxiety-related cognitions, and depression.

5.2.5. Metacognitive therapy

In contrast to the cognitive-behavioral theories where worrying has been proposed as a form of emotional avoidance (Borkovec et al., 2004) or as resulting from beliefs leading to an intolerance of uncertainty (Dugas, Gagnon, Ladouceur, & Freeston, 1998), the meta-cognitive model of Wells (2010) holds that pathological worry is the result of negative beliefs about worrying and counterproductive strategies of mental control. Central to the development of GAD is worry about worry or meta-worry. In Wells' view when negative beliefs about worrying are activated this will lead to negative appraisal of the worry process as uncontrollable and dangerous. Metacognitive therapy therefore focuses on challenging these meta-cognitive beliefs.

In an uncontrolled study (Wells & King, 2006), ten patients improved during treatment on measures of anxiety, worry and depressive symptoms and gains were maintained until one-year follow-up. A small RCT comparing metacognitive therapy to applied relaxation (Wells et al., 2010), showed that metacognitive therapy was superior to applied relaxation on most measures across 12-month follow-up. In a large study conducted in The Netherlands (van den Heiden, Muris & van der Molen, submitted for publication;

cited by Wells, 2010), metacognitive therapy was compared with a treatment based on the intolerance of uncertainty model described earlier. Although both treatments led to significant improvements on worry, anxiety and depressed mood, metacognitive therapy was significantly superior to intolerance of uncertainty treatment immediately after treatment at the posttest, but not at follow-up.

5.2.6. Mindfulness training

Research into mindfulness training in GAD is scarce. So far only a few uncontrolled studies have been reported. Although these studies found significant reductions in anxiety, depression and worry (Craigie, Rees, Marsh, & Nathan, 2008; Evans et al., 2008), generally results are weaker than those achieved with the multi-component packages of Borkovec and colleagues and Dugas and colleagues.

Mindfulness practices are also part of Acceptance and Commitment Therapy (ACT). In a study into the effects of ACT in GAD clients (Roemer, Orsillo, & Salters-Pedneault, 2008), clients were taught to become aware of the habitual nature of anxious responding, and the role of judgment and experiential avoidance in paradoxically worsening distress and interference. This treatment was more efficacious than a waiting list control condition.

5.3. Conclusions for the development of preventive interventions

In sum, past research has shown that there is a wide variety of approaches that can effectively be applied to targeting worry and rumination. All of these interventions have been shown to significantly reduce symptom severities of depression and anxiety disorders. However, although all treatments were intended to target worry and/or rumination, it has not consistently been studied whether repetitive negative thinking is indeed reduced alongside these reductions in symptoms of depression and anxiety. Even fewer studies have tested whether reductions of repetitive negative thinking mediate the treatment effects. Furthermore, it is important to note that the studies reported so far have almost exclusively been focused on targeting repetitive negative thinking in individuals who are currently suffering from depression or anxiety disorders, with the exception of studies testing the effects of Mindfulness-Based Cognitive Therapy in the context of relapse prevention. Nevertheless, evidence from these treatment trials can inform the development of preventive interventions in this area.

Although a number of different evidence-based strategies targeting rumination and/or worry exist, not all of these strategies appear equally promising to be adapted for use in preventive interventions. We suggest that the following criteria can help in the process of selecting appropriate interventions to be used in prevention. First, as described earlier a number of authors have convincingly argued that preventive interventions should be based on a clear theoretical model accounting for the role of the risk factor in the development of the disorders of interest, thereby guiding the selection of strategies to be used (e.g., Craske & Zucker, 2001; Zvolensky et al., 2006). In the context of repetitive negative thinking, one important issue theoretical models need to address is the question what exactly makes repetitive negative thinking dysfunctional and how this process can be distinguished from functional forms of dealing with problems or negative experiences.

Second, the strategies that are part of the preventive interventions should be applicable to worry as well as rumination, and should be expected to have a transdiagnostic preventive effect, i.e., reducing the risk for depression as well as anxiety disorders.

Third, a number of practical aspects appear crucial. In order to facilitate dissemination and cost-effectiveness, prevention is usually implemented in a group setting. Moreover, programs need to be relatively short, as motivation in prevention programs is typically low (Reynolds, 2009). An important criterion therefore concerns the question whether the intervention can be applied in a short intervention offered in a group setting. Alternatively, the degree to which a preventive program can be translated to an internet-based format may prove to be important.

Based on these general criteria, a number of options as to how to derive an effective prevention program from existing evidence-based strategies described above are conceivable. One approach would be to develop multi-component interventions by combining strategies coming from different treatment packages, or to adapt an existing multi-component treatment for the context of prevention. An advantage of such an approach may be that participants are presented with different strategies they can first experiment with in order to then select one or several strategies they have experienced as helpful. Such a program could initially focus on increasing awareness for repetitive negative thinking and then present several alternative strategies to adapt or replace this process. In this way, the intervention could be individualized and tailored to the needs of each individual. Importantly, however, based on the criteria described above the intervention should not be eclectic, but based on a clear theoretical rationale linking the different strategies to key ideas regarding the differences between functional and dysfunctional ways of coping with difficulties or negative experiences. One promising program to be adapted as a preventive intervention may be RFCBT (Watkins et al., 2007, 2011). This intervention is based on a clear theoretical model of repetitive negative thinking, distinguishing between different modes of processing (Watkins, 2008). Functional analysis, experiential exercises and general CBT strategies are then used to help participants switching from repetitive negative thinking to more functional modes of processing and active coping.

An alternative approach would be to adapt an intervention comprising a small set of techniques. The advantage may be that such an intervention would provide participants with an easy-to-comprehend rationale as well as a limited set of techniques that can conveniently be taught and practiced within a time-limited intervention. One promising intervention in this area may be the application of metacognitive therapy to be used in preventive interventions. This treatment is based on a clear theoretical model of repetitive negative thinking; in addition, it specifies a small set of techniques that can be used to reduce this process by targeting positive and negative metacognitions (Wells, 2000). In addition, it may be promising to develop a mindfulness-based training for the prevention of depression and anxiety disorders in individuals showing heightened levels of repetitive negative thinking.

6. Conclusion

Although prevention has been described as a key strategy to reduce the burden of depression and anxiety disorders at the level of the individual as well as society (National Research Council & Institute of Medicine, 2009), preventive interventions currently do not appear to live up to these high expectations. In order to improve the efficacy and effectiveness of prevention, a large number of experts agree that a focus on targeted prevention appears promising, whereby participants are selected who score high on a known risk factor, and whereby the intervention directly targets this risk factor using interventions deducted from clear theoretical models (see Bieling & Grant, 2007; Bienvenu & Ginsburg, 2007; Craske & Zucker, 2001; Dozois et al., 2009; Feldner, Zvolensky, & Schmidt, 2004; Garber, 2006; Horowitz & Garber, 2006; Nehmy, 2010; Reynolds, 2009; Vitiello, 2011; Zvolensky et al., 2006). Despite the apparent consensus in a large group of prevention researchers, these ideas have hardly been put into practice to date.

In this article, we have argued that repetitive negative thinking is a promising target for selective prevention of depression and anxiety disorders, for a number of reasons. First, it is a risk factor

that cannot only be used to select individuals, but that can also be modified and therefore used as a target for the intervention. Second, converging evidence from longitudinal and experimental studies points toward a causal role of repetitive negative thinking in the development of depression and anxiety disorders. Third, it is a transdiagnostic process that is similarly involved in depression and different anxiety disorders. Therefore, targeting this process has the potential to show transdiagnostic preventive effects (see also Dozois et al., 2009). Fourth, worry and rumination as the two main variants of repetitive negative thinking appear to be very similar, which means that both processes can be targeted using the same principles. Finally, a number of evidence-based treatment strategies are available that are effective in reducing repetitive negative thinking are available. The adaptation of these strategies for use in preventive interventions appears feasible.

Although repetitive negative thinking based on these arguments appears to be a promising target for intervention, some cautionary notes are warranted. First, repetitive negative thinking is clearly only one of a large number of risk factors for depression and anxiety disorders. It is therefore conceivable that preventive interventions targeting this process are not suitable for all individuals at risk for depression or anxiety disorders in the same way. For example, it has consistently been demonstrated that females report higher levels of RNT than males (e.g., Nolen-Hoeksema, Larson, & Grayson, 1999), suggesting that prevention programs targeting worry and rumination may be more relevant for women than for men. We therefore suggest that prevention programs targeting other risk factors could and should be developed in similar ways. Although most prevention programs developed in the literature to date, including the one suggested in this article, have mainly focused on cognitive and/or emotional factors, other prevention targets appear equally promising. For example, this may include interventions targeting family environment factors to prevent anxiety disorders in children of parents with a mental disorder (see Ginsburg, 2004; Knappe et al., 2010), prevention programs aiming at the reduction of substance misuse, physical health problems or interventions targeting socio-economic factors. Additionally, prevention programs could be offered to individuals who show a genetic and/or biological risk for emotional disorders, whereby the preventive interventions includes strategies to help participants compensate for these vulnerability factors (see Reynolds, 2009). In this context, the perspective presented in this article should be regarded as an example on how theory-based selective prevention can be conceptualized. The development of different prevention programs for individuals with different risk profiles may ultimately help to individualize prevention (Vitiello, 2011).

Second, whereas repetitive negative thinking has extensively been studied in the context of some disorders, such as depression and GAD, evidence regarding its role in other disorders, such as panic disorder, is still sparse. It is conceivable that targeting repetitive negative thinking may be more effective for the prevention of some anxiety disorders than others.

Third, most of the research investigating repetitive negative thinking as a risk factor for psychopathology has relied on a unimethod approach using self-report to assess this process. Therefore, it cannot be ruled out the results are biased in terms of method variance. Future research is needed to assess repetitive negative thinking in more objective ways. In addition, it appears promising to integrate research into worry and rumination with neurobiological research into risk for anxiety and depression.

Most importantly, however, the ideas presented in this article need to undergo thorough empirical testing. Adequately powered randomized controlled trials are needed to evaluate the efficacy of preventive interventions targeting repetitive negative thinking.

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