The Relationship between Cognitive Emotion Regulation Strategies and Emotional Problems: Comparison between a Clinical and a Non-Clinical Sample

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Abstract
This study focuses on the relationship between the use of specific cognitive emotion regulation strategies and emotional problems. Two samples were included: 99 adults from a clinical population and 99 matched non-clinical adults. Data was obtained in both groups on the use of nine cognitive emotion regulation strategies: self-blame, other-blame, rumination, catastrophizing, putting into perspective, positive refocusing, positive reappraisal, acceptance, and refocus on planning.

Logistic regression analyses show that self-blame, catastrophizing, and positive reappraisal were, relative to the other strategies, the most important variables for distinguishing between the two samples. While the first two strategies were reported significantly more often by the clinical than by the non-clinical sample, positive reappraisal was reported significantly more often by the non-clinical sample. The results suggest that cognitive emotion regulation strategies may be a useful target for prevention and intervention. Copyright © 2002 John Wiley & Sons, Ltd.

INTRODUCTION

Years of research have clearly demonstrated the important role emotions play in many aspects of daily life as well as their influence on adaptation to life stressors and transitions. Basically, emotions can be seen as the biological reactions that arise when a situation is appraised as presenting important opportunities or challenges and co-ordinate our responding to important environmental events (Gross & Munoz, 1995). Examples of human emotions are amusement, anger, disgust, fear, and anger (Gross & Munoz, 1995). Although emotions are biologically based, people are able to influence the emotions they have as well as the way these are expressed. This is called emotion regulation and refers to

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the broad category of ‘all the extrinsic and intrinsic processes responsible for monitoring, evaluating and modifying emotional reactions, especially their intensive and temporal features, to accomplish one’s goals’ (Thompson, 1994, p. 27). Generally speaking, emotion regulation is critical in initiating, motivating, and organizing adaptive behaviour, and in preventing stressful levels of negative emotions and maladaptive behaviour (Cicchetti, Ackerman, & Izard, 1995). It has been shown that there are important individual differences in the ways how people regulate their emotions and that some ways of regulating emotions may be more adaptive than others. Problems in emotion regulation are common among different forms of psychopathology (Gross & Muñoz, 1995; Kring, 2001). Although the process of emotion regulation in disordered and nondisordered individuals is essentially the same, the difference appears to be that individuals suffering from some form of psychopathology are impaired in one or more emotion regulation strategies (Kring, 2001). Therefore, as Bonanno (2001) suggests, an important direction for future empirical investigations pertains to the question of whether specific emotion regulation strategies can be considered adaptive or maladaptive.

According to Thompson’s definition, however, the concept of emotion regulation is a very broad conceptual rubric encompassing many biological, cognitive, and behavioural regulatory processes (Gross, 1998, 1999; Thompson & Calkins, 1996). In addition, it may refer equally well to how emotions regulate something else, such as thoughts or behaviour, or to how emotions themselves are regulated. Likewise, it may concern the regulation of emotions by oneself, or the regulation of emotions by others, or it may refer to conscious or unconscious forms of emotion regulation (Gross, 1998, 1999). It has been argued that although all kinds of emotion regulation are important and should be examined, the different forms should be clearly distinguished (Gross, 1999). It has been proposed to make a distinction between the regulation of the internal states themselves (and to call this emotion regulation) and the regulation of the behavioural reactions associated with these internal states (and to call this behavioural regulation; see Eisenberg, Fabes, Guthrie, & Reiser, 2000). The definition of emotion regulation could be narrowed down, then, to ‘the process of initiating, maintaining, modulating, or changing the occurrence, intensity, or duration of internal feeling states and emotion-related physiological processes, often in the service of accomplishing one’s goals’ (Eisenberg et al., 2000, p. 137). But still in this definition, emotion regulation remains to refer to a wide range of physiological, attentional, and cognitive processes.

For example, in a physiological way, emotions are self-regulated by a rapid pulse, increased breathing rate (or shortness of breath), perspiration or other concomitants of emotional arousal. Emotions can also be managed by a range of unconscious cognitive processes, such as selective attention processes, memory distortions, denial, or projection. In addition, emotions can be regulated by more conscious cognitive (coping) processes, such as cognitive restructuring, blaming oneself, ruminating, or catastrophizing. Although these aspects of emotion regulation all have received attention in recent years, most of the relevant work on emotion regulation is scattered through different bodies of literature and generally has not been integrated (Eisenberg, 2000; Gross, 1999). More and more it becomes clear that, although the concept is very useful as a theoretical description or explanation of the emotion system, the total process of emotion regulation is too complex and too broad to enable us to empirically focus on all aspects, mechanisms, and processes at once. As Cicchetti et al. (1995) state: although the construct of emotion regulation is useful as a metaphor of balance, it is too broad to be useful as an explanation of behaviour. Therefore, we believe theory and research would be facilitated by attempting to describe
aspects of the construct, and not trying to explain too many aspects of the construct at the same time.

The present study will focus on the conscious, cognitive part of emotion regulation, globally to be understood as the conscious, cognitive way of handling the intake of emotionally arousing information (see Thompson, 1991). This refers to the part of emotion regulation concerning the conscious, cognitive processes by which individuals regulate their own emotions. Obviously, the regulation of emotions through thoughts or cognitions is inextricably associated with human life and helps people to manage or regulate emotions or feelings, and to keep control over and/or not become overwhelmed by their emotions, for example during or after the experience of threatening or stressful events.

The concept of conscious, cognitive emotion regulation is narrowly related to the concept of cognitive coping. Current theory and research on coping rests on the notion that coping primarily involves conscious strategies of responding to stressful of negative events (Higgins & Endler, 1995). These strategies can refer either to cognitive or to behavioural strategies (thinking versus doing) (Lazarus, 1999). In general, two major functions of coping are distinguished: problem-focused coping and emotion-focused coping. Whereas problem-focused coping strategies refer to attempts to act on the stressor (comparable to emotion-related behaviour regulation), emotion-focused coping refers to attempts to manage the emotions associated with the stressor (comparable to emotion regulation, see Eisenberg et al., 2000; Compas, Orosan, & Grant, 1993; Lazarus, 1993).

Even though the operationalization of coping by the distinction between problem-focused and emotion-focused coping strategies is widely accepted and most coping measures are based on it, there is an important conceptual problem associated with it. There is another important dimension that crosses the boundaries of this division, i.e. the cognitive (what you think) versus the behavioural (what you do) strategies (see also Holahan, Moos, & Schaeffer, 1996). For example, thoughts about ‘planning’ are considered a problem-focused coping strategy, while an example of problem-focused coping in a behavioural way is ‘taking direct actions’. Examples of cognitive versus behavioural expressions of emotion-focused coping are respectively ‘positive reappraisal’ (a thought) and ‘social support seeking’ (an action). As Compas, Connor-Smith, Saltzman, Harding Thomsen, and Wadsworth (2001) have noted, the widely used problem- and emotion-focused dimensions are at best insufficient in explaining the diversity and complexity of the ways people cope with stress and in order to make a significant contribution to the coping field it is necessary to start focusing on specific coping subtypes.

In the present study cognitive coping or conscious emotion regulation strategies will therefore be studied in a conceptually pure way, separate from behavioural strategies.

Another related research area is the study of defence mechanisms and their function of protecting individuals from the emotional consequences of adversity (see for example Perry & Cooper, 1989; Vaillant, 1994). Generally speaking, both coping and defence processes have as a primary function the task of dealing with stress. They refer, however, to different processes: whereas coping mechanisms in general are assumed to involve a conscious, purposeful effort, defence mechanisms (such as projection, denial, distortion, displacement) in general are assumed to refer to processes that occur without conscious efforts (Cramer, 1998). It should, however, be acknowledged that the boundary between coping and defences may not always be so clear, as some defence processes may become conscious just as certain coping processes may become unconscious (see Vaillant, 1998, for a discussion on this topic). For the sake of clarity, however, defence mechanisms will not be included in the present study. The present study will exclusively focus on conscious...
cognitive coping or emotion regulation strategies. The term cognitive emotion regulation strategy can be used in two ways: (i) to indicate the conscious cognitive strategies used in a specific context or situation or in response to a specific stressor and (ii) to indicate the conscious thoughts or cognitions in stressful encounters that can be considered as a characteristic style of the individual and can be, empirically, defined by their stability or consistency over time and conditions (Lazarus, 1999). The present study will focus on the latter, i.e. conscious cognitive coping or emotion regulation styles.

Although the capability of advanced thinking and regulating emotions through thoughts or cognitions is universal, large individual differences exist in the amount of cognitive activity and in the content of thoughts of people by means of which they regulate their emotions in response to life experiences, events, and stressors. Large individual differences also exist in the extent that people develop symptoms of psychopathology in response to adverse experiences. This raises the important question of whether it might be true that by using certain cognitive styles, people are more vulnerable to developing psychopathology in response to negative life events or that by using other cognitive styles, people can more easily tolerate or master negative life experiences. If so, which aspects of cognitive emotion regulation are the most damaging or the most protective?

In a previous study, a new theoretically based instrument was developed including nine conceptually different conscious cognitive emotion regulation (or coping) strategies that people may use to regulate the emotions in response to life stress. The cognitive emotion regulation strategies that were distinguished were self-blame, other-blame, rumination, catastrophizing, putting into perspective, positive refocusing, positive reappraisal, acceptance, and refocus on planning (Garnefski, Kraaij, & Spinhoven, 2001). Some of these strategies, such as positive reappraisal or acceptance, have been called coping strategies in previous research. Others, such as rumination or catastrophizing, refer to separate literature fields. All separate concepts have been found to be related to mental ill-health in previous research in some way or another.

For example, self-blame, or a self-blaming attributional style, is assumed to refer to making internal, rather stable, and global causal attributions for the experience of negative events (McGee, Wolfe, & Olson, 2001). It has been shown that a cognitive style of self-blame is related to higher levels of depression (Anderson, Miller, Riger, Dill, & Sedikides, 1994; McGee et al., 2001; Kubany, Haynes, Abueg, Manke, Brennan, & Stahura, 1996), although it has also been suggested that certain forms of self-blame (‘behavioural self-blame’) may be associated with positive outcomes (Janoff-Bulman, 1992). Blaming others or other-blame refers to a style of putting the blame for what you have experienced on others. With regard to a cognitive style of blaming others, the literature suggests that other-blaming is mainly associated with behavioural problems (McGee et al., 2001). However, associations with poorer emotional well-being have also been found (Tennen & Affleck, 1990). Research on the role of other-blame in general has been sparse. Ruminative thought commonly refers to the experience of repetitive thoughts in the absence of immediate environmental cueing (see Koole, Smeets, van Knippenberg, & Dijksterhuis, 1999). Although certain forms of ruminative thinking may be helpful in coping with stressful life events (Janoff-Bulman, 1992; Tedeschi, 1999), a ruminative thinking style in general is found to be related to decreased psychological well-being and depression (Nolen-Hoeksema, 2000; Nolen-Hoeksema, McBride, & Larson, 1997; Nolen-Hoeksema, Parker, & Larson, 1994). Catastrophizing refers to thoughts of explicitly emphasizing the terror of an experience. In general, a catastrophizing style has been found to be related to maladaptation, emotional distress, and depression (Sullivan, Bishop, & Pivik, 1995).
**Putting into perspective** refers to thoughts of playing down the seriousness of the event or emphasizing its relativity when compared to other events. Also the concept of (social) comparison or putting into perspective has been found to be an important issue in relation to well-being (Allan & Gilbert, 1995; Janoff-Bulman, 1992). In addition, the coping literature has shown for positive refocusing, positive reappraisal, acceptance, and refocus on planning that these cognitive coping styles have moderately positive relationships with measures of optimism and self-esteem and negative relationships with measures of depression and anxiety (Carver, Scheier, & Weintraub, 1989; Janoff-Bulman, 1992). Other studies failed to find a significant relationship between these styles and psychopathology (Vollrath, Alnaes, & Torgersen, 1996). It may also be argued that there may be circumstances under which the use of these cognitive styles would not be adaptive at all.

Although it seems clear from the research evidence that each of the above mentioned cognitive emotion regulation strategies are important with regard to the understanding of mental ill-health, on the basis of these studies nothing can be concluded about the joint contributions of the separate cognitive strategies to psychopathology, as, generally speaking, the separate constructs refer to separate research traditions and a separate literature ranging from coping research to studies on mood regulation, mood repair, defence, and affect regulation (see Gross, 1998). Nevertheless, it seems reasonable to assume that the separate strategies rather refer to contributory or overlapping processes than to independent, disconnected processes. In order to fully understand the joint role of different cognitive emotion regulation strategies, a comprehensive, integrative study of the relationship between multiple cognitive emotion regulation strategies and mental health seems necessary.

A recent study in secondary school children studied the relative influence of the nine above mentioned cognitive emotion regulation strategies on the reporting of symptoms of depression and anxiety. It was found that whereas especially self-blame, rumination, and catastrophizing were related to the reporting of more symptomatology in adolescents, positive reappraisal was related to the reporting of fewer symptoms of depression and anxiety, showing that the study of the relative influence of the different strategies is an important research area (Garnefski et al., 2001). It also confirms that cognitive emotion regulation strategies may represent an important central theoretical issue in the explanation of symptomatology of mental disorders. Thus far, however, the research has been limited to ‘general population’ youngsters, which makes it unclear to what extent the findings of this study are applicable to other populations. Against this background, there is a need for more studies focusing on the question of what the relationship is between the use of specific cognitive emotion regulation strategies and psychopathology. On the basis of such studies, important clues may be found with regard to the identification of subgroups of people at risk for the development of psychopathology. If it turns out to be true that some people—by using certain cognitive styles—are more vulnerable, while others—by using other styles—are more resilient to the development of disturbances, this would carry important opportunities for a more targeted tailoring of treatment and preventive measures.

The aim of the present research was to study the relationship between the use of the nine specific cognitive emotion regulation strategies mentioned above and psychopathology in adults. More specifically, the cognitive emotion regulation strategies used in a clinical adult population with symptoms of depression and anxiety were compared to a group of non-clinical adults without symptoms of depression and anxiety in a cross-sectional design. The first goal was to focus on the extent to which differences existed in the
reporting of the specific emotion regulation strategies between members of the clinical and the non-clinical group. It was hypothesised that members of the clinical population would yield higher scores on self-blame, rumination, and catastrophizing as most of the previous research on these aspects showed relationships with symptoms of psychopathology (Anderson et al., 1994; McGee et al., 2001; Kubany et al., 1996; Nolen-Hoeksema, 2000; Sullivan et al., 1995). It was also expected that the non-clinical population would report more use of the cognitive strategies blaming others, putting into perspective, positive refocusing, positive reappraisal, acceptance, and refocus on planning, as most of the previous research had shown positive relationships of these strategies with well-being (Garnefski et al., 2001; Tedeschi, 1999).

The second goal was to study which of the specific cognitive emotion regulation strategies were relatively best able to distinguish between these two samples. As it was assumed that the separate strategies refer to overlapping processes, logistic regression analysis was performed to be able to study the unique ‘influence’ of the separate cognitive emotion regulation strategies, while controlling for the influence of the other strategies. It was expected that together the cognitive emotion regulation strategies would account for a considerable amount of the variance and that self-blame, rumination, and catastrophizing would be significantly related to clinical group membership, while the other cognitive strategies would be significantly related to non-clinical group membership (Garnefski et al., 2001).

**METHOD**

**Participants**

The total sample comprised 198 adults: 99 from a clinical sample and 99 from a non-clinical sample. The two groups were matched by age and gender. Each of the groups consisted of 47 males and 52 females, with ages ranging from 18 to 68 (mean age is 36). The two subsamples will be described in more detail below.

**Clinical group**

*Sample characteristics*

The subsample of clinical patients consisted of 99 adults ranging in age from 18 to 68 years (mean age 36). There were 47 males. As regards the other background variables, 56.3% were married or lived together with a partner, while 34.4% were unmarried, 7.3% was divorced, and 2.1% was widowed. As regards education level, 17.2% indicated to have lower education as the highest form of completed education (no secondary education at all), 16.1% had completed lower vocational education (three years of secondary education), 14.0% lower general secondary education (four years of secondary education), 12.9% intermediate vocational education (continuing education after finishing lower vocational or lower general secondary education), 20.4% higher general secondary or pre-university education (respectively five and six years of secondary education), and 19.4% higher vocational education or university.

*Procedure*

The 99 participants in the clinical group formed a subset of a larger sample of 169 persons referred for treatment at an outpatient psychiatric clinic in the Netherlands in the period 408

between January and October 2000. A self-report questionnaire was sent to the home address of all these 169 persons, which they were requested to fill in at home and bring along to their first interview on admission to outpatient psychiatric treatment. The subset of 99 patients was obtained in two steps. First, persons were excluded who had too many missing data, i.e. for whom it was not possible to calculate subscale scores. A subset of 120 participants remained. Second, to unambiguously define the clinical sample as consisting of persons with elevated depressive and anxiety symptoms, persons were excluded who showed only low to moderate levels of symptoms of depression and anxiety. To make this selection, scores on the SCL-90 subscales for depression and anxiety were used (see Instruments for a description of this questionnaire). In the final subset, only participants were included who had above average depression scores as well as above average anxiety scores according to the norm tables of the SCL-90 for the non-clinical population, for males and females separately (Arrindell & Ettema, 1986). Because there was a high rate of ‘comorbidity’ regarding the presence of anxiety and depressive symptoms in this sample (90%), it was decided that both depressive and anxiety symptoms had to be above average to be included in the sample. In total, a final subset of 99 participants was obtained. No significant differences were found between selected \(N = 99\) and nonselected participants \(N = 70\) regarding the background variables.

Non-clinical group

Sample characteristics

A matched non-clinical group of 99 adults was obtained with 47 males and ages ranging from 18 to 68 years (mean age 36). In this group 60.6% were married or lived together, 33.3% unmarried, 5.1% divorced, and 1.0% widowed. Education levels ranged from 3.0% with lower education as the highest form of completed education, 3.0% lower vocational education, 8.1% lower general secondary education, 18.2% intermediate vocational education, and 12.1% higher general secondary or pre-university education to 55.6% higher vocational education or university.

Procedure

The non-clinical group of 99 adults formed a subset of a total general population sample of 630 adults. This sample was obtained by approaching the population of a general practitioner’s office in the period between January and April 2000. In total 2029 questionnaires (one per household) were sent to the home addresses, of which 630 were returned and 22 could not be delivered to the correct address. Because of ethical issues, it was not possible to obtain information on eventual differences between the 630 people who filled out the questionnaire and the 1377 who did not. People who filled out the questionnaire were guaranteed anonymity.

The subset of 99 non-clinical adults was obtained in two steps. First, to unambiguously define the non-clinical group as consisting of persons without depressive and anxiety symptoms, persons were excluded who showed elevated levels of symptoms of depression and anxiety. To be able to define the non-clinical sample as participants without such symptoms, only participants were included who had depression and anxiety scores that were average or below average according to the same SCL-90 normscores (i.e. of the non-clinical population) that were used to determine the clinical sample (Arrindell & Ettema, 1986). On the basis of this criterion, a total of 393 persons was selected from the general population sample. Second, it was decided to obtain two equally sized and matched subgroups. As the smallest subgroup, i.e. the clinical sample, contained 99 participants, 99
participants had to be selected from the general population sample. Matching criteria were age and gender. Although the clinical and non-clinical sample obviously also differed in their level of education, it was not possible to use level of education as the third matching criterion. The size of the sample appeared too small to be able to find enough matches when including this criterion as well as gender and age.

Each person in the clinical group was matched with a person of the same sex and age in the general population group. No significant differences were found between selected ($N = 99$) and nonselected participants ($N = 531$) in their background variables except for mean age: the selected group had a mean age of 36, whereas in the non-selected group a mean age of 43 was observed ($t(611) = 5.22; p = 0.000$).

Instruments

Cognitive emotion regulation

The Cognitive Emotion Regulation Questionnaire (CERQ) (Garnefski et al., 2001) was used to assess what participants tend to think after the experiences of threatening or stressful life events. The instrument includes nine conceptually distinct scales. These scales all consist of four items referring to what people think after the experience of threatening or stressful life events, ranging from 1 ((almost) never) to 5 ((almost) always). A subscale score can be obtained by adding up the four items: the minimal score is 4 and the maximum score 20. The higher the subscale score, the more the specific cognitive strategy is used. The following cognitive emotion regulation strategies were measured: self-blame, referring to thoughts of putting the blame for what you have experienced on yourself (example item: ‘I feel that I am the one to blame for it’); other-blame, referring to thoughts of putting the blame for what you have experienced on the environment or another person (example item: ‘I feel that others are to blame for it’); rumination or focus on thought, referring to thinking about the feelings and thoughts associated with the negative event (example item: ‘I often think about how I feel about what I have experienced’); catastrophizing, referring to thoughts of explicitly emphasizing the terror of what you have experienced (example item: ‘I often think that what I have experienced is the worst that can happen to a person’); putting into perspective, referring to thoughts of brushing aside the seriousness of the event/emphasizing the relativity when comparing it with other events (example item: ‘I tell myself there are worse things in life’); positive refocusing, referring to thinking about joyful and pleasant issues instead of thinking about the actual event (example item: ‘I think of something nice instead of what has happened’); positive reappraisal, referring to thoughts of creating a positive meaning to the event in terms of personal growth (example item: ‘I think I can learn something from the situation’); acceptance, referring to thoughts of accepting what you have experienced and resigning yourself to what has happened (example item: ‘I think that I have to accept that this has happened’); and refocus on planning, referring to thinking about what steps to take and how to handle the negative event (example item: ‘I think about a plan of what I can do best’).

In a recent study the reliabilities of the scales of the CERQ were reported. The lowest alpha reliability was 0.68 (blaming others) and the highest 0.83 (rumination). Five of the alphas were above 0.80. The test–retest correlations after a period of five months were found to be acceptable to good with values ranging between 0.41 (acceptance) and 0.59 (refocus on planning) (Garnefski et al., 2001). In the clinical sample Cronbach’s alpha ranged from 0.72 (acceptance) to 0.85 (self-blame). In the non-clinical population reliabilities ranged from 0.76 (acceptance) to 0.86 (refocus on planning).
Depressive and anxiety symptomatology

Depressive and anxiety symptomatology was measured by two subscales of the SCL-90, namely Depression and Anxiety (Symptom Checklist: Derogatis, 1977; Dutch translation and adaptation by Arrindell & Ettema, 1986).

The main function of including this measure in the present study was to use its norm tables to determine whether participants have below average, average, or above average levels of symptoms of depression and/or anxiety. The subscale Depression includes 16 items, referring to symptoms of depression, for example low mood, incapability to enjoy, lowered self-esteem, loss of appetite, and lack of energy. The subscale Anxiety includes ten items, referring to symptoms of anxiety, such as heightened vegetative arousal, nervousness, tension, panic attacks, and restlessness. Each of the items is measured on a five-point Likert scale of distress, ranging from 1 (not at all) to 5 (very much). The minimum depression score is 16 and the maximum 64. The minimum anxiety score is 10, the maximum 40. The SCL-90 manual reports reliability coefficients ranging from 0.82 to 0.93 for the depression subscale and from 0.71 to 0.91 for the anxiety subscale and shows that test–retest reliabilities are good and that convergent validity with other conceptually related scales is strong, for both subscales (Arrindell & Ettema, 1986; Derogatis, 1977).

Life events

A checklist was used to collect data on the experience of negative life events. The main function of including this measure was to be able to control for the influence of number of negative life events in studying the relationships between cognitive emotion regulation strategies and symptomatology.

Life events that were measured were divorce of parents and/or self, long-lasting and/or severe physical or mental illness of self and/or significant others, death of a spouse and/or significant others, attempted suicide of self and/or significant others, violence, abuse of drugs and/or alcohol within family and/or relationship, unwanted pregnancy, having been victim of crime, accident, sexual abuse and/or physical abuse (self). These events were assessed for three different periods of life, before the age of 16, between the age of 16 and 1 year ago, and the last year, and in none of these periods. For the purpose of the present study only the total number of life events experienced throughout life was included as a variable.

RESULTS

Differences in reporting of cognitive emotion regulation strategies between clinical and non-clinical group

To study the extent to which the nine cognitive emotion regulation strategies were reported by members of the clinical sample in comparison with the non-clinical sample, means and standard deviations were calculated in both samples. The results are shown in Table 1.

To study whether an overall multivariate difference existed in the reporting of cognitive emotion regulation strategies between members of the clinical and the non-clinical group, multivariate analysis of variance (MANOVA) was performed. Gender was also included as independent variable in order to test for its possible main and interaction effects. The main effect for gender (Wilks $\lambda = 0.91; F(9, 185) = 1.92; p = 0.091$) as well as the interaction effect between gender and clinical versus non-clinical group membership (Wilks $\lambda = 0.98; F(9, 185) = 0.43; p = 0.917$) were not significant. The results showed that there was a significant difference between the clinical and non-clinical sample (Wilks $\lambda = 0.54$;
To study which of the nine specific cognitive emotion regulation strategies were at the basis of this overall significance, t-tests were used. Table 1 shows that significant differences between the clinical population and the non-clinical group were found for the reporting of the cognitive emotion regulation strategies catastrophizing, self-blame, rumination, other-blame, positive reappraisal, and acceptance. Of these strategies only the strategy positive reappraisal appeared to be reported significantly more often by the non-clinical group than by the clinical sample. All the other strategies were reported significantly more often by the clinical than by the non-clinical sample. The two samples did not show significant differences in the reporting of putting into perspective, refocus on planning, or positive refocusing.

Pearson intercorrelations between CERQ subscales among clinical and non-clinical sample

Correlations between subscales ranged between −0.05 (‘other-blame’ and ‘refocus on planning’) and 0.56 (‘positive reappraisal’ and ‘putting into perspective’) in the clinical sample and between 0.06 (‘positive refocusing’ and ‘self-blame’) and 0.62 (‘positive reappraisal and ‘refocus on planning’) in the non-clinical sample. This indicates moderate to strong correlations between the subscales (Table 2). This suggests that multivariate analyses should be performed to study the relationship between cognitive emotion regulation strategies and clinical versus non-clinical group membership in order to be able to determine unique relationships, while accounting for the mutual correlations.

Prediction of clinical and non-clinical group membership: logistic regression analysis

To identify which of the cognitive emotion regulation strategies, relative to the others, were the most important variables for distinguishing between the two samples, Logistic regression analysis was performed. Logistic regression is a statistical technique related to multiple regression analysis that can be used to predict a binary dependent variable from a set of independent variables. In the present analysis, the binary dependent variable referred to group membership (clinical versus non-clinical group), whereas the independent variable set referred to the cognitive emotion regulation strategies.
Table 2. Pearson intercorrelations between CERQ subscales for clinical sample ($n = 99$: below diagonal) and non-clinical sample ($n = 99$: above diagonal)

<table>
<thead>
<tr>
<th>Cognitive emotion regulation strategies</th>
<th>Self-blame</th>
<th>Other-blame</th>
<th>Rumination</th>
<th>Catastrophizing</th>
<th>Acceptance</th>
<th>Putting into perspective</th>
<th>Refocus positive</th>
<th>Positive reappraisal</th>
<th>Refocus on planning</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self-blame</td>
<td></td>
<td>0.29**</td>
<td>0.33**</td>
<td>0.28**</td>
<td>0.32**</td>
<td>0.28**</td>
<td>0.06</td>
<td>0.39***</td>
<td>0.49***</td>
</tr>
<tr>
<td>Other-blame</td>
<td>0.14</td>
<td></td>
<td>0.26*</td>
<td>0.45***</td>
<td>0.16</td>
<td>0.11</td>
<td>0.16</td>
<td>0.09</td>
<td>0.21*</td>
</tr>
<tr>
<td>Rumination</td>
<td>0.40***</td>
<td>0.28**</td>
<td></td>
<td>0.27**</td>
<td>0.31**</td>
<td>0.31**</td>
<td>0.24*</td>
<td>0.52***</td>
<td>0.33**</td>
</tr>
<tr>
<td>Catastrophizing</td>
<td>0.02</td>
<td>0.61***</td>
<td>0.45***</td>
<td></td>
<td>0.14</td>
<td>0.12</td>
<td>0.03</td>
<td>0.15</td>
<td>0.02</td>
</tr>
<tr>
<td>Acceptance</td>
<td>0.33**</td>
<td>-0.01</td>
<td>0.35***</td>
<td>0.29**</td>
<td></td>
<td>0.48***</td>
<td>0.39***</td>
<td>0.56***</td>
<td>0.51***</td>
</tr>
<tr>
<td>Putting into perspective</td>
<td>0.32**</td>
<td>0.07</td>
<td>0.17</td>
<td>0.18</td>
<td>0.37***</td>
<td></td>
<td>0.28**</td>
<td>0.56***</td>
<td>0.40***</td>
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<td>0.11</td>
<td>0.29**</td>
<td>0.54***</td>
<td></td>
<td>0.54***</td>
<td>0.41***</td>
</tr>
<tr>
<td>Positive reappraisal</td>
<td>0.21*</td>
<td>0.17</td>
<td>0.16</td>
<td>0.25*</td>
<td>0.38***</td>
<td>0.56***</td>
<td>0.41***</td>
<td></td>
<td>0.52***</td>
</tr>
<tr>
<td>Refocus on planning</td>
<td>0.27**</td>
<td>-0.05</td>
<td>0.61***</td>
<td>0.22*</td>
<td>0.35***</td>
<td>0.47***</td>
<td>0.43***</td>
<td>0.62***</td>
<td></td>
</tr>
</tbody>
</table>

*p < 0.05; **p < 0.01; ***p < 0.001.
Because the two groups appeared to differ significantly as regards the reporting of number of life events as well as level of education, it was decided to control for these variables in the logistic regression analysis. Therefore, first these variables were entered as independent variables in the logistic regression analysis, yielding a significant model ($\chi^2(2) = 66.05; p = 0.000$), explaining 29.2% of the variance ($\text{Cox & Snell } R^2$).

Subsequently, the nine cognitive emotion regulation strategies were added as independent variables, increasing the explained variance by 24%. Total variance explained was 53.2% ($\chi^2(11) = 145.08; p = 0.000$). Table 3 presents the results of this logistic regression analysis including level of education, total number of life events, and the nine cognitive emotion regulation strategies as independent variables. The Wald statistic was used to determine the significance of the contribution of the independent variables. The standardised logistic regression coefficient (standardised $B$) is used to determine the relative influence of the separate independent variables.

Table 3 shows that, after controlling for level of education, total number of life events, and the other cognitive emotion regulation strategies, three cognitive emotion regulation strategies appeared to have significant, independent contributions to the prediction of clinical group membership: self-blame, positive reappraisal, and catastrophizing. The cognitive emotion regulation strategy of self-blame appeared to be the best predictor of group membership, with a standardized logistic regression coefficient (standardized $B$) of $0.34$, showing that clinical group membership was associated with a higher reported use of this strategy. Also positive reappraisal appeared to be a strong predictor of group membership, with a standardized $B$ of $0.28$, indicating that clinical group membership also was associated with a lower reported use of this strategy. The third significant predictor was catastrophizing, with a standardized $B$ of $0.24$, showing that clinical group membership was related to a higher reported use of this particular strategy, as well.

As the model presented in Table 3 includes several strategies that are not significant contributors, they may have artificially increased the percentage of variance explained. Therefore, another logistic regression analysis was performed including the significant

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Table 3. Identification of cognitive emotion regulation strategies distinguishing clinical ($n = 99$) and non-clinical ($n = 99$) group membership: logistic regression analysis

<table>
<thead>
<tr>
<th>Predictors</th>
<th>$B$</th>
<th>SE $B$</th>
<th>Wald</th>
<th>Standardized $B$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of education</td>
<td>0.60</td>
<td>0.18</td>
<td>11.52</td>
<td>0.23</td>
<td>0.001</td>
</tr>
<tr>
<td>Total life events</td>
<td>$-0.21$</td>
<td>0.07</td>
<td>8.72</td>
<td>$-0.19$</td>
<td>0.003</td>
</tr>
<tr>
<td>Self-blame</td>
<td>$-0.38$</td>
<td>0.09</td>
<td>16.46</td>
<td>$-0.34$</td>
<td>0.000</td>
</tr>
<tr>
<td>Other-blame</td>
<td>0.15</td>
<td>0.12</td>
<td>1.73</td>
<td>0.11</td>
<td>0.188</td>
</tr>
<tr>
<td>Ruminating</td>
<td>$-0.13$</td>
<td>0.08</td>
<td>2.57</td>
<td>$-0.12$</td>
<td>0.109</td>
</tr>
<tr>
<td>Catastrophizing</td>
<td>$-0.28$</td>
<td>0.12</td>
<td>5.56</td>
<td>$-0.24$</td>
<td>0.018</td>
</tr>
<tr>
<td>Putting into perspective</td>
<td>$-0.04$</td>
<td>0.09</td>
<td>0.23</td>
<td>$-0.04$</td>
<td>0.631</td>
</tr>
<tr>
<td>Positive refocusing</td>
<td>0.01</td>
<td>0.08</td>
<td>0.01</td>
<td>0.01</td>
<td>0.927</td>
</tr>
<tr>
<td>Positive reappraisal</td>
<td>0.28</td>
<td>0.09</td>
<td>10.08</td>
<td>0.28</td>
<td>0.002</td>
</tr>
<tr>
<td>Acceptance</td>
<td>$-0.08$</td>
<td>0.09</td>
<td>0.91</td>
<td>$-0.07$</td>
<td>0.340</td>
</tr>
<tr>
<td>Refocus on planning</td>
<td>0.01</td>
<td>0.09</td>
<td>0.02</td>
<td>0.01</td>
<td>0.874</td>
</tr>
</tbody>
</table>

Total explained variance ($\text{Cox & Snell } R^2$): 53.2%.
Significance model: $\chi^2(11) = 145.08, p = 0.000$.

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1The clinical sample reported significantly more negative life events ($t(190) = 6.71; p = 0.000$) as well as a significantly lower level of education ($t(196) = -6.16; p = 0.000$).
The present study focused on the relationship between the use of specific conscious cognitive emotion regulation strategies and mental health. Although previous studies have clearly shown that cognitive emotion regulation strategies, such as self-blame and rumination, are related to poorer emotional well-being, conclusions about separate constructs in general appear to refer to separate research traditions (Gross, 1998). The present study adds to the existing literature by including the separate cognitive emotion regulation strategies in one and the same study in order to study their joint contributions to psychopathology.

As expected, members of the clinical population had significantly higher scores on self-blame, rumination, and catastrophizing and lower scores on positive reappraisal. These findings confirm previous research (e.g. McGee et al., 2001; Nolen-Hoeksema et al., 1997; Sullivan et al., 1995). The clinical population also showed higher scores on other-blame and acceptance, which was not in line with the expectations. The finding regarding other-blame does, however, concur with theories stating that both kinds of blame, i.e. a continuing focus on blaming oneself or another, may form an obstacle to adaptation to negative life events or trauma (Tedeschi, 1999). As regards acceptance, an explanation may be found in the theory that a distinction can be made between acceptance as an active process of self-affirmation and acceptance as a passive form of resignation to negative experiences (Wilson, 1996). It might be argued that the present study rather refers to the latter form of acceptance, which has typically been identified as a negative adjustment style associated with poor outcomes (Wilson, 1996). No significant differences were found between the clinical and non-clinical population in the reporting of thoughts of putting into perspective, positive refocusing, and refocus on planning. Although there were some exceptions, the bivariate results in general appeared to confirm the findings of previous studies showing a relationship between the separate cognitive emotion regulation strategies and psychopathology.

Table 4. Distinction between clinical (n = 99) and non-clinical (n = 99) group membership: final logistic regression model

<table>
<thead>
<tr>
<th>Predictors</th>
<th>B</th>
<th>SE B</th>
<th>Wald</th>
<th>Standardized B</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of education</td>
<td>0.57</td>
<td>0.16</td>
<td>13.67</td>
<td>0.24</td>
<td>0.000</td>
</tr>
<tr>
<td>Total life events</td>
<td>−0.24</td>
<td>0.07</td>
<td>10.62</td>
<td>−0.24</td>
<td>0.001</td>
</tr>
<tr>
<td>Self-blame</td>
<td>−0.41</td>
<td>0.08</td>
<td>24.35</td>
<td>0.40</td>
<td>0.000</td>
</tr>
<tr>
<td>Catastrophizing</td>
<td>−0.26</td>
<td>0.09</td>
<td>8.02</td>
<td>−0.24</td>
<td>0.005</td>
</tr>
<tr>
<td>Positive reappraisal</td>
<td>0.19</td>
<td>0.06</td>
<td>9.96</td>
<td>0.21</td>
<td>0.002</td>
</tr>
</tbody>
</table>

Total explained variance (Cox & Snell $R^2$): 51.9%.
Significance model: chi$^2$(5) = 140.43, $p = 0.000$.

DISCUSSION AND CONCLUSIONS

The present study focused on the relationship between the use of specific conscious cognitive emotion regulation strategies and mental health. Although previous studies have clearly shown that cognitive emotion regulation strategies, such as self-blame and rumination, are related to poorer emotional well-being, conclusions about separate constructs in general appear to refer to separate research traditions (Gross, 1998). The present study adds to the existing literature by including the separate cognitive emotion regulation strategies in one and the same study in order to study their joint contributions to psychopathology.

As expected, members of the clinical population had significantly higher scores on self-blame, rumination, and catastrophizing and lower scores on positive reappraisal. These findings confirm previous research (e.g. McGee et al., 2001; Nolen-Hoeksema et al., 1997; Sullivan et al., 1995). The clinical population also showed higher scores on other-blame and acceptance, which was not in line with the expectations. The finding regarding other-blame does, however, concur with theories stating that both kinds of blame, i.e. a continuing focus on blaming oneself or another, may form an obstacle to adaptation to negative life events or trauma (Tedeschi, 1999). As regards acceptance, an explanation may be found in the theory that a distinction can be made between acceptance as an active process of self-affirmation and acceptance as a passive form of resignation to negative experiences (Wilson, 1996). It might be argued that the present study rather refers to the latter form of acceptance, which has typically been identified as a negative adjustment style associated with poor outcomes (Wilson, 1996). No significant differences were found between the clinical and non-clinical population in the reporting of thoughts of putting into perspective, positive refocusing, and refocus on planning. Although there were some exceptions, the bivariate results in general appeared to confirm the findings of previous studies showing a relationship between the separate cognitive emotion regulation strategies and psychopathology.
In addition, the present study studied the joint contributions of the separate strategies to psychopathology, while controlling for their interrelations. It was shown that after controlling for level of education, number of life events, and the other strategies, only three cognitive emotion regulation strategies remained to have significant, independent contributions to the ‘prediction’ of clinical group membership: i.e. a higher reported use of the strategy self-blame, a higher reported use of catastrophizing, and a lower reported use of the strategy positive reappraisal. As expected, these cognitive emotion regulation strategies together explained a considerable amount of the variance. It seems clear from these results that although six of the nine specific cognitive emotion regulation strategies in some way or another show relationships with mental ill-health, the separate strategies rather refer to contributory or overlapping processes than to independent, disconnected processes. For example, our bivariate results clearly confirm the findings of previous studies showing that rumination as a cognitive emotion regulation style is related to psychopathology (Nolen-Hoeksema et al., 1994). On the basis of the present study, however, the conclusion can be added that after controlling for the influence of self-blame, catastrophizing, and positive reappraisal, an independent effect for rumination could not longer be established. Also the effects of other-blame and acceptance disappeared, after controlling for the other strategies.

Although the results should not lead to the conclusion that the strategies of rumination, other-blame, and acceptance are not important with regard to mental health, they do confirm our earlier statement that, in order to fully understand the role of separate cognitive emotion regulation strategies, comprehensive, integrative studies of the relationships between cognitive emotion regulation strategies and mental health are necessary. The results show, that psychopathology can not be traced to one specific emotion regulation strategy, but to a combined ‘play’ of various strategies. A theoretical implication is that studies on the relation between cognitive emotion regulation and dysfunction should not focus on a single cognitive strategy at the time, but on all cognitive strategies at the same time in order to be complete and of value. In addition, the results suggest the relevance of identifying (individual) symptom patterns crossing the boundaries of ‘adaptive’ and ‘less adaptive’ strategies. For example, there may exist large differences between adolescents who report different patterns of ‘cognitive coping strategies’, especially between adolescents who report predominantly ‘less adaptive’ strategies, adolescents who report predominantly ‘more adaptive’ strategies, adolescents who report both types of strategy, and those who report to make no or only scarce use of any of the cognitive coping strategies.

Another theoretical implication refers to the coping research tradition. Since about 1974, the year in which Lazarus et al. (1974) launched his cognitive appraisal model, the most widely used framework to classify coping responses has been the problem-focused versus emotion-focused model, despite some conceptual problems. One of the problems refers to the fact that both the problem and emotion-focused dimensions are made up of a mix of cognitive and behavioural coping strategies (i.e. thinking and acting), while it would be reasonable to assume that, theoretically, cognitive coping and taking actions can be considered as very different processes (Compas et al., 2001; Garnefski et al., 2001). Our results confirm the statement of Compas et al. (2001), suggesting that models that have included only the distinction between problem- and emotion-focused coping and those that have included only approach and avoidance coping do not reflect the actual structure of coping in individuals. Our results add the conclusion that theories about dysfunctioning should consider cognitive coping strategies and behavioural coping strategies in conceptually pure and distinct ways.
The relationship between the use of specific cognitive emotion regulation strategies and the reporting of symptoms of depression and anxiety suggests that the existence of depression and anxiety symptoms may form an indication for the existence of—possibly long-established—‘unadaptive’ strategies of cognitive emotion regulation. However, as the results of the present study are based on cross-sectional data, it is important to acknowledge that no conclusions can be drawn about directions of influence. Theoretically, it would be just as likely that certain cognitive coping strategies lead to emotional problems such as depression and anxiety, as the other way around. Circular causal mechanisms may also be at work, which would make both assumptions true at the same time. Or even a third variable may account for the relation between the reporting of specific cognitive emotion regulation strategies and the reporting of symptoms of depression and/or anxiety. The cross-sectional nature of the study will also not allow us to draw conclusions regarding the development, course, and changes of symptom patterns and patterns of cognitive emotion regulation in time. More prospective design studies should be set up in the future answering questions such as whether a temporal order can be found in the emergence of emotional and behavioural symptoms and the use of specific cognitive emotion regulation strategies or whether the individual’s use of a particular cognitive emotion regulation strategy in a certain stressful event or situation refers to a stable factor or is rather subject to change in the course of time.

Still, whatever the directions of influence may be, it is clearly shown that the use of certain cognitive coping strategies and serious disturbances are related issues. This suggests that cognitive strategies should play an important role in theoretical models and intervention strategies. It may, therefore, be worthwhile to aim intervention efforts simultaneously at psychopathology and cognitive emotion regulation strategies. The assumption that a patient’s symptoms will be relieved if irrational beliefs or dysfunctional thoughts are changed is not a new one. In fact, one of the basic premises of cognitive therapies is that things are inappropriately viewed by people suffering from depressive or anxiety symptoms and that therapy should bring about changes in those views (see for example Beck, 1976; Ellis, 1962). New is that our approach and results might give some clues for a more targeted tailoring of treatment, for example by challenging ‘unadaptive’ strategies such as self-blaming and catastrophizing and supplying more ‘adaptive’ strategies such as positive reappraisal, at the same time.

In the present study, cognitive emotion regulation strategies were studied, separate from other emotion regulation strategies. The conclusions of the present study therefore only refer to the conscious cognitive emotion regulation strategies people may use when handling a negative event. Nothing can be concluded about other, related emotion regulation strategies, such as defence mechanisms or behavioural coping strategies, or about the ways the different types of emotion regulation strategy co-occur or interact with each other. Although we believe that it is important to describe aspects of the construct, in conceptually pure ways, we also believe that it is only a first step. It should be acknowledged that boundaries between different emotion regulation strategies may not always be so clear and that apparently separate strategies may actually refer to related and/or overlapping emotion regulation processes. For example, Vaillant (1998) emphasized that overlap may exist between ‘unconscious’ defence and ‘conscious’ coping processes. Moreover, although cognitive and behavioural coping strategies refer to two different processes employed at different points of time, it could also not be denied that the processes are related and may influence each other (Garnefski et al., 2001). In the same way, overlap or relationships may be assumed between other emotion regulation
processes, for example between physiological, attentional, and cognitive processes (Gross, 1998). Future research should therefore focus on two directions: (i) to unravel the separate constructs referring to the broad concept of emotion regulation and (ii) to study the relations and possible interactions between those constructs.

A limitation of the design was that the detection of depression and anxiety symptoms as well as the assessment of cognitive emotion regulation strategies had to be made on the basis of self-reported evaluations, which may have caused some bias. The results of this study may be an under- or overestimation of the extent to which cognitive emotion regulation strategies are applied in reality. It should also be acknowledged that by using self-report measures the results may be biased by individual response styles. It is important for future studies to address research questions concerning cognitive emotion regulation by using both self-reported and other forms of data collection, such as interviews, expert judgements, or experimental research.

Our clinical sample comprised participants who were announced for treatment at an outpatient psychiatric clinic and were characterized by elevated symptoms of both depression and anxiety. Although by this selection homogeneity of the sample was obtained, it is unclear to what extent the findings are generalizable to persons with other types of disturbance. In addition, no information was available about the type of diagnosis the participants in the clinical sample had received. It should be acknowledged that the type of diagnosis, for example whether the participants were mostly diagnosed with anxiety disorder, mood disorder, or another or mixed type of disorder, may have influenced the results.

A strong point of the study, however, was that the clinical sample was compared with a ‘non-clinical’ sample without symptoms of depression and anxiety and that a matched control design was used to examine differences between the two groups. However, because of the sample sizes, we were not able to match the groups by educational level. Although the results of the logistic regression analyses were corrected for level of education, it could still be argued that some of the differences between clinical and non-clinical sample may reflect differences in intelligence.

To be able to further develop the concept of cognitive emotion regulation, it is important to answer questions such as whether strategies that are considered ‘adaptive’ in the present study are indeed adaptive in all circumstances (Gross, 1999). It may very well be true that a certain cognitive coping strategy that is highly adaptive in one situation is absolutely not in another situation. In the present study the concept of cognitive emotion regulation is considered from a trait or style perspective. According to Lazarus (1993) the approaches to coping both as a style and as a situation-specific process are essential in that they each address different aspects of the coping process. In our opinion, it is a challenge in the development of the concept of cognitive emotion regulation to address both the trait and the situation-specific aspects in future, or, as Lazarus very wisely states, ‘combining the approaches without sacrificing what is unique in each might be a worthwhile enterprise’ (Lazarus, 1993, p. 243). On the one hand, it is important to study cognitive emotion regulation over time and across diverse types of negative life event in the same persons. On the other hand, cognitive emotion regulation should be studied across individuals experiencing the same type of negative life event or trauma. Both types of study call for complex, long-term research designs.

Despite some limitations, the results clearly show that the use of certain cognitive emotion regulation strategies and the reporting of symptoms of depression and anxiety are closely related. The exploratory character of the results makes replication, thorough
testing and further development (e.g. inclusion of other factors), necessary. Prospective elements should be included in the model. However, if our results can be confirmed, they carry important implications for the focus and content of intervention and prevention of mental health problems.

REFERENCES


