Eur Arch Psychiatry Clin Neurosci (2002) 252 : 240 – 249 DOI 10.1007/s00406-002-0389-y

ORIGINAL PAPER

V. Henkel · P. Bussfeld · H.-J. Möller · U. Hegerl

Cognitive-behavioural theories of helplessness/hopelessness: Valid models of depression?

Received: 1 October 2002 / Accepted: 2 October 2002

Abstract Helplessness and hopelessness are central aspects of cognitive-behavioural explanations for the development and persistence of depression. In this article a general overview concerning the evolution of those approaches to depression is provided.

Included is a critical examination of the theories. The review of the literature suggests that those cognitive models describing helplessness/hopelessness as trait factors mediating depression do not really have a strong empirical base. The majority of those studies had been conducted in healthy or only mildly depressed subjects. Thus, there seems to be little justification for broad generalisations beyond the populations studied. It seems that some of the reported studies have not tested the underlying theories adequately (e. g. correlation had sometimes been interpreted as causation; adequate prospective longitudinal study designs had seldom been applied). Moreover, the theoretical models are not generally prepared to explain all depressive features (e. g. the possibility of a spontaneous shift in a manic episode).

Despite those limitations, there is a relevant impact of the learned helplessness paradigm on preclinical research in neurobiological correlates of depressive states. Last but not least, the models are of high interest with respect to the theoretical background of important modules of cognitive-behavioural therapy and its acute and prophylactic effects.

Key words depressive disorder · helplessness · hopelessness · cognitive models · neurobiological correlates

Verena Henkel, MD (✉) · P. Bussfeld · H.-J. Möller · U. Hegerl
Department of Psychiatry
Ludwig-Maximilians-University Munich
Nußbaumstr. 7
80336 Munich, Germany
Tel.: +49-89/51 60-55 58
Fax: +49-89/51 60-55 42
E-Mail: verena.henkel@psy.med.uni-muenchen.de

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Introduction

For psychiatric researchers and clinicians of a biological orientation helplessness and hopelessness are typical symptoms of depressive disorders among others such as anhedonia, feelings of guilt, loss of energy or sleep disturbances. These symptoms are described within the scope of a *symptom-oriented* approach to depression in the classification systems of DSM-IV (APA 1994) and ICD–10 (WHO 1992). According to cognitive-behavioural theories, helplessness and hopelessness are rather considered to be potential aetiological factors as antecendent causes for the onset or maintenance of depression. In this context, helplessness and hopelessness are considered as *trait* factors which enhance the vulnerability of a person to react to certain environmental stimuli with depression. In these concepts helplessness and hopelessness exist independently from depressive episodes as individual attitudes representing vulnerability and risk factors.

This review briefly describes and refers to the *major theoretical positions* most studies in this area are based on: those of Beck (1967), Beck et al. (1985), Overmier and Seligman (1967), Seligman (1974) as well as Abramson et al. (1989). Limitations concerning the empirical foundation of the concepts will be discussed. Studies evaluating the relevance of hopelessness and helplessness as trait characteristics, predictors and risk factors for the development of depression will be described. Preclinical and clinical research concerning neurobiological correlates will be presented. Finally, in a critical appraisal the relevance of the concepts for the treatment of depressive disorders and the prevention of relapse will be discussed.

The hypotheses and empirical data

The cognitive theory of depression by Beck et al.

In the 1960s, there was an awakening interest in the construction of psychological concepts of the origin and the nature of depression. In a number of these theoretical models, the dimensions helplessness and hopelessness are central features. One of the most influential of these theories was established by A. T. Beck. His cognitive theory of depression, although first proposed decades ago, is still an important element of the cognitive approach to depression in terms of both theory and therapy. Beck defines hopelessness as negative expectancies with respect to the future and helplessness as unrealistically low concepts of the own capabilities. Beck considers such a negative view of the self and the future as a central element of the "cognitive triad". The cognitive triad consists in negative attitudes to the self, the future and the environment. According to this theory the cognitive triad plays a specific aetiological role in depression (Beck et al. 1985). The basic hypothesis is that a cognitive disturbance precedes the affective change and is responsible for its maintenance. Before the onset of dysphoria and depression, the affected person is considered to misinterpret reality through a negative cognitive screen. Based on this theory, Beck and colleagues developed a structured and manualised treatment – *cognitive therapy*. We will refer to this extensively tested psychotherapeutic treatment for depression in the section Implications for treatment and prevention.

Hopelessness has been considered as the key variable linking depression to suicidal behaviour (Minkoff et al. 1973; Beck et al. 1975). The relation of hopelessness to levels of depression and suicidal intent has been explored both clinically and psychometrically. Beck and his staff have conducted a series of correlational and experimental studies of the construction of their model of depression. The majority of studies is based on questionnaires constructed to capture the proposed conceptions, e. g. the Beck Hopelessness Scale (BHS) (Beck et al. 1974). The psychometric properties of the scale – reliability and validity – were evaluated in 294 hospitalized suicide attempters (Beck et al. 1974). The reliability factor (Cronbach's alpha) was 0.93. For a cut-off score of nine, sensitivity was high with 94.1 %, but specificity was low with only 41 %. Several years later, Beck et al. (1985) stated that the BHS would not be a satisfactory instrument to *predict* future suicide in a sample of attempters. This finding was replicated by a Swedish research group in 1997 (Nimeus et al. 1997).

The model of "learned helplessness" by Seligman et al. and the reformulated theory by Abramson et al.

Overmier and Seligman (1967) reported a study, in which dogs that were given electrical shocks not contingent upon their behaviour, subsequently had difficulty learning to escape or avoid shocks. In this learned helplessness model, it is the experience of uncontrollable events and as a consequence the expectation that no action can control outcomes in the future which finally leads to the production of symptoms of helplessness. These symptoms are above all passivity, as well as cognitive and emotional deficits including anxiety and hostility. During inescapable exposure to electric shocks, dogs learned that shocks were independent of any responses. Those dogs showed striking deficits when placed later in a box in which a simple act of crossing a barrier would have terminated a new shock. Unlike dogs not previously exposed to uncontrollable shocks, these animals seemed to be helpless. They conducted only few attempts to escape the shock (motivational deficit). Moreover, they were not likely to follow an occasionally successful response (learning or cognitive deficit) and they did not show much overt emotionality while being shocked (emotional deficit) (Overmier and Seligman 1967) (Table 1).

This phenomenon was called "learned helplessness" and it was argued that it could serve as a model of depression in humans. However, the explanation of depression in man by use of this animal helplessness paradigm has been controversial. The rapid and spontaneous "remission" from the induced deficits is one of the main restrictions of this animal model. Moreover, only a minority of experimental animals actually develops the deficits, suggesting that individual factors, perhaps genetic ones exert influence over the phenomenon.

According to the limitations, the original helplessness model was revised (Abramson et al. 1978). Now, the focus was on the individual's personal causal *explanations* of negative events. It has been suggested that these explanations would play an important role for the mediation of symptoms of helplessness/hopelessness and furthermore for the development of depression. The theory was called the *explanatory or attributional style*. This model claimed that subjects tending to helplessness and depression, interpret bad events in internal, stable and global terms ("it is me; it is going to last for-

Table 1 Animal model of "learned helplessness"

Conditions	Experimental Group	Control Group
Step 1	**Uncontrollable** stress = learning of response-reinforcement independence	–
Step 2	**Controllable** stress = response and reinforcement are no longer independent	**Controllable** stress = response and reinforcement are never independent
Behaviour	Learning deficit	No learning deficit
Results	Step 1 results in a detrimental effect on subsequent learning = affected coping strategy = "learned helplessness"	Unaffected coping strategy

ever; it is going to affect everything I do"). This kind of negative explanation has been described as an important risk factor for the development and the maintenance of depression (Abramson et al. 1978). According to this theory, identification of individuals at risk for depression should be possible by the way they explain bad events. Specific psychometric instruments had been developed, e. g. the Attributional Style Questionnaire (ASQ) (Peterson et al. 1982). Peterson and Seligman (1984) presented eight studies in which explanatory style was measured by the ASQ. From a psychometric perspective it has to be noted that the reliability of the ASQ is less than optimal, with internal consistencies typically ranging between 0.40 and 0.70. Later, an extended version of the ASQ was developed with an internal consistency between 0.80 and 0.90. It was termed Expanded Attributional Style Questionnaire (EASQ) (Peterson and Villanova 1988).

In the 1980s, critics emerged stating that the learned helplessness model and its reformulation lack predictive power because they do not specify the conditions under which a certain causal attribution will be more probable than another. In addition, subjects tend to perceive noncontingent reinforcements as contingent ones, thus making the model experimentally unstable (Sauer and Müller 1980).

Metalsky and Joiner (1992) tested in a longitudinal study whether hopelessness and helplessness mediated by attributional style are vulnerability (trait) factors that cause depressive symptoms. They used a prospective methodology and included 172 students. Measures of depression, anxiety and of attributional style (Beck Depression Inventory (BDI) (Beck et al. 1961), EASQ (Peterson and Villanova 1988), Negative Life Events Questionnaire (Pitzner and Drummond 1997) and State Trait Anxiety Inventory (Spielberger et al. 1970)) were completed at two times with a time interval of 5 weeks. The authors concluded that hopelessness would be mediated by attributional style in the presence of negative life events and would *predict* the onset of depressive symptoms. The main limitation of this study lies in the student sample which is not a representative population. Moreover, the interval between baseline and the follow-up visit seems to be rather short.

Also Golin et al. (1981) as well as Firth and Brewin (1982) administered measures of both explanatory style and depressive symptoms in a test-retest design. The authors of both studies concluded that empirical data would support the theory of explanatory style. However, in the first study again students (180 volunteers) were examined, in the second study only 16 female depressive patients.

Review of the literature suggests that more cross-sectional than longitudinal studies have been conducted to examine the relationship between explanatory style and depression. Overall, results of these studies have shown that the cognitive tendency to attribute negative events to internal, stable and global causes is associated with severity of concurrent and future depression (compare Barnett and Gotlib 1988; Brewin 1985; Peterson and Seligman 1984; Sweeney et al. 1986 for *reviews*). Sweeney et al. (1986) and Robins (1988) conducted meta-analytic reviews of more than 100 explanatory style studies and reported evidence for the predicted relationship between explanatory style and depressive disorder. Moreover, they stated that severity of depression (measured by the BDI) is often correlated with the habitual use of internal, stable and global causes to explain bad events. However, since correlation is not causation, this seems to be only a rather weak argument in support of the theory.

The "hopelessness theory of depression" by Abramson et al.

Abramson and colleagues did not only propose a reformulation of the learned helplessness hypothesis in 1978 (see previous section), but extended and specified this reformulation eleven years later (Abramson et al. 1989). This revision was called the "hopelessness theory of depression". The negative attributional style has been postulated to be an important risk factor for a *specific* constellation of depressive symptoms, termed "hopelessness depression". In this theory, hopelessness was viewed as a subset of helplessness. This means that if hopelessness occurs then helplessness also occurs, but not vice versa. Furthermore, it was hypothesised that there would be an association of negative attributional style and depressive symptoms only in the presence, but not in the absence of negative life events. Some of the symptoms suggested to be part of the hopelessness depression are completely overlapping with symptoms that are part of the diagnostic criteria in DSM-IV (APA 1994) for major depression, e. g. sadness, suicidal ideation. Other symptoms only partly overlap with DSM-IV criteria for major depression, e. g. mood-exacerbated negative cognitions or motivational deficit. The hopelessness depression subtype was hypothesised to cut across currently diagnosed categories of clinical depression (e. g. major depression, dysthymic disorder) and to occur in a mild, subsyndromal form as well.

Two major research strategies have been used to examine the hopelessness theory. These strategies include the postmorbid (remitted depression) studies and premorbid case-control studies. In the first strategy mentioned, the explanatory styles of formerly depressed patients are compared to those of currently depressed and of nondepressed controls. In the second strategy, subjects who become depressed during the course of the study are compared to subjects who remain nondepressed throughout the study. Based on these kinds of studies, some researchers have concluded that the hopelessness theory is not well supported (Barnett and Gotlib 1988). Other investigators have argued that those studies have not tested the theory adequately as they suffer from methodological difficulties. Participants of those studies were recruited on the basis of the presence or absence of depression either in the past (remitted de-

pression studies) or future (premorbid case-control studies). According to Abramson et al. (1989) the more appropriate research strategy would be to select subjects on the basis of the presence or absence of the depressiogenic attributional style and to compare those groups. Using this strategy, several studies have examined the validity of the hopelessness theory (see Abramson et al. 1995, for a review). Alloy et al. (1992) found that currently non-depressed students with a depressive attributional style had higher rates of specific hopelessness depression symptoms in the past two years than did currently healthy students with a non-depressed attributional style. The limitation of this study consists above all in the use of a retrospective instead of a prospective design.

Another study (Whisman et al. 1995) explored the utility of categorizing depressed individuals based upon their level of hopelessness. It was examined if hopelessness would be related to treatment outcome arguing that the greatest worth of categorisation lies in its usefulness in predictions of therapy outcome. Compared to patients with a low degree of hopelessness, depressed inpatients high in hopelessness exhibited a poorer outcome to pharmacological and cognitive-behavioural treatment interventions.

A psychometric instrument was developed to measure the hopelessness depression as a specific subtype of depression. It is the Hopelessness Depression Symptom Questionnaire (HDSQ; Metalsky and Joiner 1997). This is a self-rating scale consisting of 32 items and it is proposed as a tool to test the hopelessness theory of depression with enhanced precision for research purposes. According to the authors its usefulness in a clinical setting to derive a profile depicting which particular symptoms are elevated for a given patient, and which are not, still has to be evaluated.

A recent publication by Joiner (2001) reported that he has tested with four studies the relation of negative attributional style to hopelessness depression symptoms versus major depression symptoms. First of all, there was a considerable *overlap* of hopelessness and major depression symptoms. Nevertheless, Joiner (2001) stressed the point that negative attributional style was more related to the former disease construct than the latter.

In summary, the hopelessness theory has received partial support in the empirical tests. Nevertheless, most of the studies used retrospective (e. g. Alloy et al. 1992) or cross-sectional (e. g. Spangler et al. 1993, Whisman and Pinto 1997) designs that do not allow for a test of the critical hypothesis that helplessness/hopelessness as one of the proposed causes of the hopelessness depression temporally *precedes* and *prospectively predicts* the onset of a *specific* hypothesised hopelessness depression symptom constellation.

Neurobiological substrates of the "learned helplessness" phenomenon

"Learned helplessness" animal model: advantages and limitations

Among the existing animal models of depressive disorders, the learned helplessness paradigm is still considered to be one of the better animal models of depression (Velbinger et al. 2000). It is used in preclinical pharmacological studies on the antidepressant effects of new compounds. Already registered antidepressant compounds as tricyclic antidepressants, MAO inhibitors or atypical antidepressants have been demonstrated to prevent and to reverse learned helplessness in animals during the preclinical phase of the development programs (see preclinical data in the product information of the corresponding manufacturers). The same had been shown for electric convulsive therapy (ECT) (Dorworthy et al. 1977). Important negative results have included the non-efficacy of neuroleptics, psychostimulants and anxiolytics (Sherman et al. 1982).

Genetic factors may exert critical influence on the learned helplessness phenomenon. It has been suggested that the regulation of NPY gene expression might be involved in the reduced vulnerability of NLH ("non-learned helpless") rats to develop learned helplessness (Lachman et al. 1992).

"Learned helplessness" and serotonergic mechanisms

Detailed study of the learned helplessness phenomenon has revealed that there are neurochemical changes with apparent specificity for the application of uncontrollable stress. Several research groups provided evidence for the involvement of *serotonergic pathways* (Amat et al. 1998, Edwards et al. 1992). The results of several animal studies suggested that the biological correlate of the behavioural deficit in animals caused by uncontrollable shocks implicates serotonergic mechanisms with a limbic-hypothalamic circuit serving as a center for adaptation to stress (Edwards et al. 1991, 1992). Interestingly, Sprague-Daweley rats can be separated into two different groups after exposure to uncontrollable shocks in terms of their performance in learning to escape from a subsequent controllable stress. Only a subgroup of rats ("Learned helpless" = "LH" rats) did not learn to escape a controllable shock after the previous experience of uncontrollable shocks. The other rats ("Non-learned helpless" = "NLH" rats) learned an adequate response as fast as naive control rats did. The neurobiological changes in "LH" rats were investigated and comprised *post*-synaptic 5-hydroxytryptamine (5-HT) receptor effects in the cortex, hippocampus, septum and hypothalamus. These included an up-regulation of 5-HT 1b receptors in the cortex, hippocampus, septum and simultaneously a down-regulation in the hypothalamus. In several in

vitro studies a significant increase of serotonin release in the hippocampal slices of LH rats had been found. In addition, changes of presynaptic serotonergic activity caused by uncontrollable shocks had been described in detail. The major findings concerned presynaptic serotonergic effects in the hippocampus and hypothalamus of LH rats. These included an increase in three presynaptic 5-HT mechanisms involved in signal transduction: release of serotonin, high affinity uptake of serotonin and maximum density of binding sites for uptake of 5-HT (Edwards et al. 1992). Also other authors suggested that one of the target regions might be located *pre*-synaptic at the 5-HT 1a receptor (Maier et al. 1995). Other research teams confirmed the meaning of the serotonergic system stressing the role of the 5-HT 2a receptor and this especially in the hippocampal subfield (Papolos et al. 1996). The density of 5-HT 1a and 5-HT 2a receptors was measured by quantitative autoradiography in "LH" rats and "NLH" rats. There were no changes in 5-HT 1a receptor density among the groups in any brain region investigated; however, in helpless rats, 5-HT 2a receptor density was found to be decreased, as compared to control rats (Wu et al. 1999).

The learned helplessness phenomenon and hypothalamo-pituitary-adrenal (HPA) activity

An activation of the pituitary-adrenal system is regarded as an important component in the stress response. Many of the effects of chronic stress (e. g. stress-induced gastric lesions) are thought to be mediated by stress-induced increases in circulating levels of glucocorticoids. Some investigators have used the learned helplessness model to probe the dynamics of the HPA system.

Central to Seligman's model of learned helplessness is the notion that one of the most important variables that determines whether an aversive stimulus will cause the helplessness syndrome is the degree to which an aversive situation can be controlled. This theoretical and empirical assumption could be endorsed in preclinical studies: rats which learned to avoid electric shocks by successful responses were compared to a control group of rats which had no opportunity for a control over the stressor. Those animals with a control over the aversive situation developed significantly fewer stomach ulcers in comparison to those which had no control over the shocks. The effect was not caused by the pain itself, because both groups of animals received exactly the same number of shocks (Weiss et al. 1970). The results may indicate that uncontrollable stress induces a stronger activation of the HPA axis than controllable stress. These effects of stress controllability on the HPA system could be confirmed several times (e. g. Weiss 1971, Weiss et al. 1975). However, Maier et al. (1995) could not replicate those findings. They found *no* significant differences between two groups of animals of which only one group could successfully respond to the aversive stimulus. Both groups exhibited the same level of adrenocortical activation. This may be explained by the intensity of a stimulus. It could be possible that in case of a very strong intensity of the stimulus, the influence of controllability and predictability becomes less important. Moreover, humans and laboratory animals show genetic differences in susceptibility to stressors, such as the tendency to develop peptic ulcers as an index of adrenal cortical activation (Glavin et al. 1991).

In an early study in 1964, Wolff and colleagues studied the parents of children dying of leukemia (Wolff et al. 1964). These study participants were examined while they were responding to exacerbations and remissions in their child's struggle with leukemia, which during this period of time usually ended in death. Some parents were extremely expressive in their discussion of their child's disease, while other individuals reacted more reclusive and in a passive way which could be interpreted as a condition similar to that of "learned helplessness". The more reclusive and passive reacting parents tended to have higher levels of adrenal cortical activation than those with a more overt, expressive emotional behaviour. Thus, a perceived state of helplessness might result in higher cortisol levels, whereas expressive or aggressive counteraction of an inevitable threatening situation might leave the individual with less increased cortisol levels. An issue which had not been addressed in this study was whether "baseline" urinary cortisol levels were different in the subjects before the onset of the stressful life event.

More recently conducted studies evaluating the psychoendocrinological response pattern in healthy humans have also indicated that low control as well as low predictability of an aversive situation significantly induce an activation of the HPA system (Kirschbaum et al. 1995). The role of stress in the origin of depression has often been investigated, producing evidence in support of this association. Also the acute depressed phase of primary affective disorder is frequently associated with sustained basal hypercortisolism. The characteristics of the adaptive response of the HPA axis seems to be determined by the attributes of the stressor itself, its appraisal and the coping strategies. However, individual differences in the diurnal cycle of cortisol and intra-individual instability across time aggravate the interpretations of changes in the HPA system. Furthermore, in recent years, a novel and paradox phenomenon has emerged from experimental studies on the effects of stress. In these studies, *hypo*cortisolism has been reported for healthy individuals living under conditions of *chronic* stress (Heim et al. 2000). Moreover, it has been shown that the function of the HPA axis in patients with *chronic* depression is normal (Watson et al. 2002). The authors have suggested that a *subgroup* of depressives with less HPA dysfunction may progress to chronicity.

Another key issue is whether elevated cortisol levels might induce acute or chronic changes in the brain tissue and thus might affect cognitive functioning. To

which extent increased cortisol levels have a direct effect on neuronal tissue and thus cognitive functioning is still unknown. Wellman (2001) reports that chronic glucocorticoid administration dramatically reorganises apical arbors in medial prefrontal cortex. Earlier studies described dendritic atrophy in hippocampal neurones (Woolley et al. 1990). These alterations in morphology might be reflected by functional changes. Newcomer et al. (1999) conducted a randomised, double-blind controlled study in healthy subjects comparing two fixed doses of cortisol. The authors concluded that several days of exposure to cortisol at doses and plasma concentrations associated with physical and psychological stress in humans can reversibly decrease specific elements of memory performance in otherwise healthy individuals. Belanoff et al. (2001) correlated character of depression (psychotic vs. non-psychotic) with cortisol levels and with the outcome of neuropsychological tests such as the Wallach Memory Recognition Test (Wallach et al. 1980). They found a positive correlation between high cortisol levels and a psychotic character of the depression and low neuropsychological cognitive performance (Belanoff et al. 2001).

In summary, there are numerous hints that an acute condition of learned helplessness is significantly associated with increased cortisol levels. Those increased cortisol levels might affect neuropsychological functioning and thus might even reinforce a vulnerability to depression.

Helplessness and hopelessness: state or trait?

Cognitive theories suggest that helplessness and hopelessness are rather trait than state factors, because these models consider helplessness and hopelessness rather as a cognitive entity *causally antecedent* to depression. Since this is a central issue of the cognitive paradigms, the validation of these concepts is important. The proof of efficacy of cognitive therapy in certain depressive patient groups suggests that the underlying theoretical concepts might be true. However, the efficacy of cognitive therapy is not a sufficient proof of the validity of the underlying models. In our view, it would be required that if helplessness and hopelessness are not only symptoms involved as state factors during a depressive episode, but persist beyond it as causal factors mediating the onset of a depressive episode, then it would be necessary to demonstrate that these features exist in the *same* subjects in clinically depressed states *as well as* in healthy conditions. One study which partly satisfies this requirement is the study of Young and colleagues (1996). They examined whether hopelessness is a stable trait component which influences the sensitivity to depression. The authors hypothesised that a person's hopelessness at a given time is a function of the severity of depression *and* two individual explanatory stable traits: baseline hopelessness (when not depressed) and hopelessness sensitivity. The authors interpreted the results of their longitudinal prospective study in 316 participants as a confirmation of this model.

An additional issue concerning the validation of the cognitive concepts of helplessness and hopelessness is the question whether helplessness and hopelessness can be influenced by therapeutic interventions not only during acute treatment, but in a way that the risk of relapse or recurrence of a depressive episode is reduced. Recently a couple of studies were conducted to address the issue whether cognitive therapy has a preventive effect and whether there are specific effects of cognitive therapy on helplessness/hopelessness features. These trials are referred to in the section Implications for treatment and prevention.

Overall, it has to be stated that the issue if helplessness and hopelessness are rather stable trait or rather state dependent variables in depressive disorder remains controversial. Thus, this matter of dispute could be an interesting topic for further research, because clinical implications are important: if helplessness and hopelessness exist as trait factors, they could be used to identify persons at risk for depression or might serve as potential predictors of treatment response or relapse in treatment regimes. If helplessness and hopelessness would above all be changeable characteristics, state-like features of a person, the change during therapy would be of interest as well as the question how these state forms correlate with other measures of depression. Finally, within an integrative approach it would be conceivable that both positions are right with respect to the above mentioned issues, so that relationships between depression and helplessness/hopelessness could exist in several ways (Fig. 1).

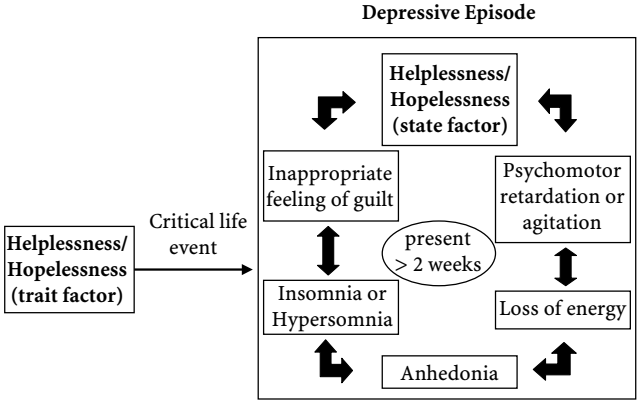

Fig. 1 *Possible relationships* between helplessness/hopelessness and depressive disorder. In cognitive theories helplessness/hopelessness play different roles: helplessness and hopelessness are considered as trait factors mediating the onset of a depression triggered by a life event and are regarded as a symptom factor responsible for the maintenance of a depressive episode. The biological perspective rather focuses on helplessness/hopelessness as symptoms caused by the depressive disorder.

Implications for treatment and prevention

The more clearly the psychopathology of a syndrome can be understood the more possible it becomes to design treatment appropriately. As pointed out earlier the attitudes helplessness and hopelessness may play an important role in the origin as well as in the symptomatology and the maintenance of depression. Thus, treatment has to focus on these aspects. First of all, it can be concluded that it is a matter of course that the evocation of hope and the increase of self-esteem are fundamental to *all* forms of treatment of depression.

The clinical problem of depression has been approached by several authors from a behavioural orientation. Already in the 1960s, it was hypothesised that depression "may be regarded as a function of inadequate reinforcers" (Lazarus 1968). This may exemplify the fact that cognitive theorists assign a primacy to cognitive factors in depression and *tend to treat depressive symptoms rather as secondary derivatives*. In this context, Beck suggested specific strategies. Beck saw successful treatments as changes of the negative cognitive set (e. g. "I am an ineffective and helpless person") to a more positive set (Beck 1974). He argued the primary task of the therapist is to change the negative expectational scheme of the depressed patient to a more optimistic one. To reach this goal, it has to be explained to the patient where he is beginning to misinterpret reality. In a second step, the patient's thinking, i. e. his conceptions, can be modified by learning alternative and more positive conceptions. Moreover, Beck stated that a prophylactic effect also in the absence of an ongoing therapy would distinguish cognitive-behavioural psychotherapy from pharmacotherapy. He postulated that cognitive therapy would reduce the likelihood of relapse and recurrence even after it is discontinued, if patients continue to use the cognitive tools of symptom reduction (Beck et al. 1979). This is in line with findings of several controlled clinical trials recently published (Scott et al. 2000, Jarrett et al. 2001, Paykel 2001) (for a description of these studies see below).

Seligman's argument is aimed at similar targets. In his helplessness view, the central theme in successful therapy should be having the patient find out and come to believe that his responses produce the gratifications he desires. Thus, the patient has to learn and to internalise that he is an effective being, capable to control and to influence various situations. Seligman himself stressed the overlaps to Beck's cognitive therapy for depression (Seligman 1974).

According to the *reformulated helplessness model* and the *subsequently emerging hopelessness theory*, a certain explanatory (attributional) style put people at risk for initial onset, relapse and recurrence of depressive symptoms. Insofar as negative events and situational information supporting depressiogenic inferences contribute to the maintenance of hopelessness, therapeutic interventions aimed at additionally modifying the hopelessness inducing environment are considered to be helpful. Thus, the hopelessness theory suggests *a greater additional focus on environmental modifications* which is practiced to a lesser extent in Beck's cognitive therapy. Several authors reported that cognitive intervention according to the hopelessness theory changes explanatory style measured by different cognitive measures (e. g. ASQ scores). Persons and Rao (1985) followed 32 depressed patients from hospital admission to discharge and found that ASQ scores changed significantly for bad events. This was confirmed by studies conducted by Seligman et al. (1988) and by DeRubeis et al. (1990): attributional style for bad events and mood have been found to change significantly during cognitive therapy for unipolar depression.

In recent years, *modern psychotherapy research* has begun to standardise and test the efficacy of cognitive behavioural therapy in a manner analogous to how antidepressant compounds are tested in clinical development programs. Thus, cognitive therapy has been tested by being administered according to standard study protocols by therapists receiving standardised training and using standardised manuals. Such use of cognitive therapy has been compared in clinical trials to placebo or antidepressants. In this context there have been many demonstrations of the effectiveness of cognitive therapy in the *acute* treatment of moderate and severe depression (e. g. Rush et al. 1977; Shaw 1977; Simons et al. 1984). Results of a study conducted by Rush et al. (1981) suggest *differential* effects of cognitive therapy and pharmacotherapy (imipramine) *on depressive symptoms*: during the cognitive therapy improvements in hopelessness, views of the self and mood generally preceded changes in vegetative and motivational symptoms. On the other hand, no consistent pattern of change was associated with pharmacotherapy. These results are compatible with the theoretical assumption that cognitive therapy initially alters negative thinking and mood, which secondarily leads to improvements in vegetative and motivational symptoms. Findings of a controlled trial including 158 patients showing only partial response to antidepressants also indicated that the addition of cognitive therapy produced statistically significant *differential* effects on the symptoms hopelessness, self-esteem and feelings of guilt (Scott et al. 2000).

Over the last decade it has been recognised that most patients with major depression will have either a relapsing or chronic course (Frank et al. 1990). Especially residual symptoms predict high relapse rates (Paykel 2001). Thus, it will be interesting to determine, whether cognitive therapy has effects on residual major depressive symptoms and has a preventive effect. Paykel (2001) conducted a clinical study of cognitive therapy in 158 patients and found that cognitive therapy combined with antidepressant significantly reduced relapse rates which occurred with moderately high dose continued antidepressants alone. However, the effect was mainly relapse reduction, with only weak effects on

residual symptom levels or achievement of complete remission.

Jarrett et al. (2001) developed a "continuation-phase" cognitive therapy to teach responders' skills to prevent relapse. Findings of this randomised clinical trial suggest that 8 months continuation-phase cognitive therapy significantly reduces relapse and recurrence in the patients with the highest risk to develop recurrent major depressive disorder. This was already suggested by the results of a pilot study (Jarrett et al. 1998).

If patients learn and use the associated skills, cognitive therapy may reduce depressive relapse and recurrence, thus producing clinically important benefit.

When Simons et al. (1984) looked for *differential effects* of cognitive therapy versus psychopharmacotherapy (nortriptyline) *on cognitive measures*, they found that cognitive change occurred as much in pharmacotherapy as in cognitive therapy. They concluded that "cognitive (phenomena) behave(d) more as symptoms (state factors) than as causes (trait factors)" and thus cannot be considered as mainly secondary derivatives. DeRubeis et al. (1990) argued that the equivalent symptom reduction for cognitive therapy and pharmacotherapy in the above mentioned study would be compatible with the proposition that the cognitive variables were mediators in one treatment and consequences of change in the other.

Research is accumulating that attests the efficacy of cognitive therapy in treating and even in preventing relapse in non-bipolar primary affective disorder. To our knowledge, no data is available for many other forms of depressive disorders, e. g. for major depression with mood-congruent and mood-incongruent *psychotic* features. However, it has to be concluded that there is no doubt that the cognitive approach to depression is useful in terms of acute treatment and prevention in *certain* depressive patient groups. However, the fact that cognitive therapy for depression may be as effective as antidepressants in certain patients does not necessarily support a categorical validity of the *underlying theoretical models*. Those studies investigating the validity of the *theoretical* concepts are partly not conducted according to standardised procedures and not in clinical populations. A large part of data is based on healthy volunteers and mildly depressed college students, but very rarely in patients suffering from major depression. Thus, it may be presumed that the cognitive *theories* of helplessness/hopelessness apply rather to sub-syndromal or minor levels of depression, but not really to severe depressive states, especially since all these theories are not prepared to explain important phenomena of a major depressive episode, e. g. the course of a bipolar disorder with the occurrence of manic episodes. They do not account for chronobiological changes or the antidepressant effect of sleep-deprivation. Nevertheless, in any case cognitive *therapy* can give certain depressive patients a valuable framework to cope with specific psychological conditions such as helplessness and hopelessness. Finally, it was Beck himself who stated that one of the most important aspects of his theory would be that the cognitive approach to depression is *therapeutically* useful (Beck 1974).

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