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Cognitive Markers in Adolescent Major Depression

by

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M. A., Simon Fraser University 1984

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Cognitive Markers in Adolescent Major Depression

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ABSTRACT

Two studies were conducted to examine the role cognitions play in adolescent major depression. In the first study, the Cognitive Bias Questionnaire for Children (CBQC) was revised and validated on three groups of adolescents given diagnoses of endogenous major depression (n=23), nonendogenous major depression (n=25), and other nondepressed diagnoses (n=26). The results showed that there was an increase in internal consistency for the distortion and nondepressed-nondistorted scales from the revised CBQC when compared to a previous version. There were no significant differences between the endogenous and nonendogenous groups on any of the scales from the CBQC. As predicted, both groups of major depressive disorder evinced significantly more distortion and less positive thinking on the CBQC compared to the group of nondepressed psychiatric controls. Adolescents with major depression who experienced less stress showed more distortion than those who experienced high stress. These results validate the CBQC as a marker for major depression and support the validity of the distortion scale in assessing the tendency to negatively distort life events.

In the second study, adolescents with major depression were tested at two phases, once while actively depressed and

again when remitted from major depression. These adolescents (n=13) were compared to a group of adolescents with major depression who did not remit from depression (n=17) and to a group of psychiatric controls (n=14). While actively depressed, the remitted group did not differ significantly from the unremitted group in severity of depression, level of distortion and in thoughts of hopelessness or worthlessness. However, both depressed groups were significantly different from the psychiatric controls on these measures. As predicted, at the remission phase, the remitted group equalled the control group in severity of depression and in thoughts of hopelessness and worthlessness, but continued to display significantly more distortion and less positive thinking like the unremitted group. These results support the hypothesis that negative cognitions, such as hopelessness are state markers for major depression, whereas the tendency to distort and the lack of a self-serving bias are trait markers for major depression.

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TABLE OF CONTENTS

LIST OF TABLES

INTRODUCTION

The past decade has seen a significant shift towards the recognition that depressive disorders occur in prepubertal children and adolescents analogous to those which occur in adults. In the 1960s, depression in childhood was thought to be a qualitatively different phenomenon from depression which occurs in adulthood. Proponents of this view relied heavily on psychoanalytic developmental principles. For example, Rie (1966) argued that prepubertal children did not become depressed because the superego was not developed fully enough to produce the guilt and lowered self-esteem so characteristic of adult depression. Others, however, argued that depression was a basic psychobiological reaction to loss or deprivation (Joffe & Sandler, 1965) and could even occur in infants as Spitz putatively demonstrated in 1946. Finally, as if to resolve the varying viewpoints, depression in children was proposed to be existent, but only in a "masked" form (Toolen, 1962). Supporters of this view argued that not until the child reaches 16 years was depression expressed in the same form as in adults. Prior to this age, children were more inclined toward "doing" things than toward "thinking" about them (Weiner, 1980). The masked view was expressed this way: "The masked forms of depression seen in

early adolescence consist not so much of the toll this disorder is taking as of efforts to ward off depression and thereby avoid any toll....[they may] resort to temper tantrums, running away, stealing, truancy, and numerous other defiant rebellious antisocial acts" (Weiner, 1980, p. 456).

It was not until the 1975 National Institute of Mental Health conference on childhood depression, that depression began to be recognized as a clinical syndrome in children (Schulterbandt & Raskin, 1977). In a seminal paper from this conference, Kovacs and Beck (1977) successfully challenged the masked view of depression. They argued that since proof of depression is necessary in the diagnosis and that many of the masked symptoms (see Weiner above) are nothing more than presenting complaints, the term masked is unnecessary. Moreover, Kovacs and Beck after carefully reviewing symptom descriptions of childhood depression, concluded: "despite the insistence that childhood depressive disorders and the adult syndromes are dissimilar, we are struck by the similarities" (p. 11).

In the following year, while Welner (1978) was echoing the sentiments of Kovacs and Beck (1977), a review article published in the widely read journal, Psychological Bulletin, concluded that depression was far too frequent and

transient in children to be considered a clinical syndrome (Leftkowitz & Burton, 1978). Although subsequent research proved this view to be inadequate (see Kovacs et al., 1984), the transient view remained unconvincing even at the time (see Costello, 1980). Parallel to these developments and independent of each other, Puig-Antich et al., (1978; 1979) and Carlson and Cantwell (1979; 1980) were the first to demonstrate that the unmodified adult diagnostic criteria for major depression could be successfully applied to prepubertal children. A little later, Strober, Green and Carlson (1981a) demonstrated the identical phenomenon with adolescents. These developments opened the gate to a whole spectrum of validity studies aimed at demonstrating that childhood and adult depression were identical disorders (see Cantwell, 1985; Cantwell & Carlson, 1983; Finch & Saylor, 1984; Haley, 1984; Puig-Antich, 1985, 1986; Poznanski, 1985; Waters & Storm, 1985; Weller & Weller, 1983 for reviews). Briefly stated, the evidence indicated that the basic phenomenology of major depression was the same from 6 years to adulthood, that psychotic and endogenous forms of major depression were not rare in children or adolescents, that there was a high degree of family aggregation of major depression in first degree relatives of depressed children, and that affective disorders in children and adolescents demonstrated a chronic course consistent with the adult forms. Puig-Antich (1986) summed up the literature with

this comment: "The evidence reviewed so far suggests that we should start to think about affective illness as a unitary syndrome across the lifespan" (p. 348).

In reviewing these developments, Rutter (1986a) became concerned that child psychopathology had moved too far away from the developmental perspective. Rutter argued that the developmental perspective need not be equated with psychodynamic views of development, rather we should take into consideration empirical findings and theories from developmental psychology; we should be concerned with the discontinuities as well as the continuities of child psychopathology. To this end, Rutter gathered together the top researchers in the field of childhood depression to discuss developmental issues in childhood depression. The results of this endeavour were recently published in a book titled Depression in Young People (Rutter, Izard & Read, 1986). Rutter (1986b) noted that while it is now accepted that children and adolescents show major depressive disorder, there are developmental changes to be explained, including the findings that there appears to be a shift to more suicide, suicidal ideation, depressive feelings and depressed females at adolescence and the fact that depression in childhood is commonly associated with other psychiatric disorders such as conduct disorder.

In summary, the concept of depression in childhood has evolved from a psychoanalytic based view that the syndrome was structurally impossible, through the notion of depressive equivalents to the position that depression is an unitary entity throughout the lifespan. Recent modifications have restored a more modest role for the developmental discontinuities in expression of gender prevalence in the pre-adult syndrome.

This study focuses on major depression in adolescents. The diagnosis of major depression is made using Research Diagnostic Criteria (RDC; Sptizer, Endicott & Robins, 1978) or its near equivalent Diagnostic and Statistical Manual, III-Revised (DSM-III-R, American Psychiatric Association, 1987) criteria. The RDC define Major depression as a mood disorder characterized by depressed mood and at least five of the following symptoms: decreased or increased appetite or weight loss or gain, sleep disturbance, psychomotor retardation or agitation, guilt feelings, concentration difficulties, anhedonia, fatigue and suicidal ideation or suicide attempt. The symptoms must be present for a least 2 weeks and be of sufficient severity to interfere in daily activities. The prevalence of major depression in the normal population of adolescents is estimated to be 4.7% with a sex ratio of 5:1 in favour of girls (Kashani et al. 1987). This estimate compares with 1.8% found in

preadolescent children (Kashani et al., 1983) and with the 4.5% to 9.3 % range found in adult normal females and with the 2.3% to 3.2% range in adult normal males (American Psychiatric Association, 1987). The prevalence of major depression in inpatient adolescent clinics ranges from 17.8% (Strober, Green & Carlson, 1981b) to 55% (Haley, Fine & Marriage, 1988). The duration of an episode of major depression in adolescents can be lengthy with a mean duration of 29.4 weeks between onset and recovery (Strober, 1985). As noted earlier, empirical studies show that there is no difference in frequency of major depressive symptoms between children, adolescents and adults (Mitchell, McCauley, Burke,& Moss, 1988; Ryan et al., 1987; Strober, Green & Carlson, 1981a) with some minor exceptions. Adolescents exhibit more hallucinations relative to delusions, whereas adults show the converse with more delusions (Haley et al., 1988; Mitchell et al., 1988; Ryan et al., 1987). In this respect, adolescent depression is more akin to prepubertal major depression (Chambers, Puig-Antich, Tabrizi & Davies, 1982). As well, adolescents tend to report more suicide attempts, guilt, and somatic complaints than adults (Mitchell et al., 1988). The frequency of suicide attempts in adolescents with major depression ranges from 39% in outpatient samples (Mitchell et al., 1988; Ryan et al., 1987) to 76% for inpatient samples (Haley et al., 1988).

Clearly, major depression in adolescents represents a serious health problem, but little is known about its optimal treatment or aetiology. On the premise that identification of aetiology may inform the process of treatment selection and development, the present study attempts to explore one possible aetiological avenue, the role cognitions play in the development of adolescent major depressive disorder. As such, it essentially represents a downward extension of adult cognitive models for depression. In the following sections of this introduction, I will briefly review the adult cognitive models and in light of Rutter's comments (see above), I will endeavour to provide a developmental perspective on cognition and depression. Secondly, I will present the cognitive marker approach in conceptualizing the relationship between depression and cognition and I will review the empirical support for this approach in adult patients and in child and adolescent samples.

Cognitive Models of Depression

The two major theoretical positions on the role of cognition in depression are those of Beck and colleagues (Beck, 1967; Beck, Rush, Shaw, & Emery, 1979; Kovacs & Beck, 1978; 1979) and Seligman and colleagues (Abramson, Seligman

& Teasdale, 1978; Beach, Abramson & Levine, 1981; Seligman, 1975; Peterson & Seligman, 1984).

Beck's Cognitive Distortion Model

Beck (1967) proposed that the main component in depression was cognitive rather than affective. Beck focused on such symptoms as guilt, self-depreciation, hopelessness, commonly seen in depressed patients and concluded that it was the cognitive processes that were responsible for the dysphoria and anhedonia.

According to Beck, all depressive symptoms can be traced to three specific cognitive patterns, which he termed the cognitive triad. The first pattern involves a negative view of the self, the person views herself as defective, unworthy, and tends to attribute unpleasant experiences to physical, moral or mental defects in herself. The second pattern is a negative view of the world, in which the person interprets experiences and interactions in the world around her in a negative way when more plausible, alternative interpretations are available. The third pattern is a negative view of the future, the person believes her suffering will continue indefinitely into the future.

The second component of Beck's theory involves schemas. Schemas are stable cognitive patterns which readily interpret information related to specific sets of circumstance. The function of schemas is to filter out or screen stimuli for speed and ease of coding. When certain circumstances arise, a schema is activated to match them. In the depressed person, the orderly matching of schemas to environmental input is usurped by prepotent negative schemas which selectively attend to the negative elements of the stimuli while filtering out the positive. This filtered input reinforces the negative schema in so far as the process is matching negative input to negative schema as well as increasing the activity of a whole matrix of negative schemas, which in turn causes a cognitive distortion of reality. This phenomenon explains why depressed patients appear immune to positive feedback and appear to dwell on personal deficiency. As Beck et al., (1979) state: "the patient loses much of his voluntary control over his thinking processes and is unable to invoke other more appropriate schemas" (p. 13). Beck uses the term "systematic cognitive errors" to categorize the cognitive distortions observed in depressed patients. These errors includes 'selective abstraction', whereby negative events are removed from context, 'arbitrary inference', deduction unrelated to evidence, 'overgeneralization', arguing from one instance to a general rule,

'personalization', self reference without evidence and 'dichotomous thinking', thinking in extremes with the self at the negative extreme.

Another concept of the distortion theory is what Kovacs and Beck (1978) call silent assumptions or premises. These assumptions are termed silent because it is not readily observable, without some therapeutic probing, that the negative conclusions patients reach about themselves are based on any logic. The premises or assumptions are personal sets of contingency rules or formulas used to integrate experience. For example: "If I am not important to everyone, I can't go on living". This kind of premise will make a person particularly vulnerable to slights and snubs. "I have to be perfect at everything"; this premise would make a difficult task a devastating experience. The premises are faulty and rigid allowing no room for gray areas or gradations. As Kovacs and Beck note, the simplistic, rigid and childish nature of the premises and the contingency rules betrays their origin - in childhood

Beck's model may be summarized in the following manner:

1. The negative cognitive triad is the general thematic content of schemas which distinguishes the distortions of depressed persons from the distortions

of other disorders such as anxiety. The schemas, in addition, contain idiosyncratic content based on varying individual childhood experiences. The specific or individual content of the cognitive triad is expressed by the idiosyncratic silent assumptions or premises.

2. The schemas are latent but are activated by circumstances that resemble the original childhood events responsible for the formation of the negative schema. Once activated, these schemas distort reality in a way that is observable in the systematic errors of logic, characteristic of depressed patients.

Thus, by these two concepts, Beck explains why, in similar circumstances some individuals become depressed while others do not.

Seligman's Helplessness Model

In his original helplessness theory, Seligman (1975) proposed that the experience of bad events as uncontrollable leads to the expectation that no action will control outcomes in the future. This perception induces several helplessness and depressive symptoms including passivity, cognitive deficits, sadness, anxiety, decreased aggression,

decreased appetite, neurochemical changes and susceptibility to disease. However, this model could not explain why uncontrollable events would lead to long lasting sadness across situations from the original experience of uncontrollability. Furthermore, it could not account for the frequently seen low self-esteem and self-blame in depressed patients. How could one who has no control over the situation blame him or herself for the bad event?

To handle these difficulties, Abramson et al., (1978) reformulated the helplessness theory into a causal attributional frame work. In the reformulated model, the experience of uncontrollable events leads the person to ask why. The explanation people give can be conceptualized along three dimensions. First, the cause may be attributed to something in the person (internal) or to the situation (external). Second, the cause may be attributed to factors persisting across time (stable) or to transient factors (unstable). Third, the causal attribution can be made to factors affecting many situations or outcomes (global) or can be made to the particular situation or outcome (specific). Thus, these dimensions of causal attribution or as Seligman, now, wishes to refer to them, causal explanations (Peterson & Seligman, 1984), help explain the persistence of depressive symptoms across time (stable and factors) and across a range of situations (global factors)

and as well helps explain the low self-esteem (internal factors). The causal explanation style of the person at risk for depression is one in which the uncontrollable bad event is attributed to internal, stable and global factors while good events probably are attributed to external, unstable and specific factors (Abramson et al., 1978). These authors also explain how the reformulated theory is consistent with Beck's distortion theory as follows:

> Those people who typically tend to attribute failure to global, stable and internal factors should be most prone to general and chronic helplessness depressions with low self-esteem. By the reformulated hypothesis, such a style predisposes depression. Beck (1967) argued similarly that the premorbid depressive is an individual who makes logical errors in interpreting reality. For example, the depression prone individual overgeneralizes; a student regards his poor performance in a single class on one particular day as final proof of his stupidity. We believe that our framework provides a systematic framework for approaching such generalization: It is an attribution to a global, stable and internal factor. Our model predicts that attributional style will produce depression proneness, perhaps the depressive personality (p. 64).

Developmental Issues in Cognition and Depression

Beck and colleagues see no problems in extending the cognitive distortion model to children. Emery et al., (1983) argue that as long as the child is at the concrete operational level of development (around 7 years, see Flavell, 1985), he or she is capable of perceiving and inferring from reality as well as misinterpreting and distorting reality. The ability to perceive intentionality produces the cognitive capacity to experience guilt and misattribution of blame. However, because pre-operational children are tied to the here and now, they may have difficulty anticipating the future and inferring consequences that may occur later. Although these authors place the limits of the model at the operational level of development, it should be noted that pre-operational children have shown the ability to perceive intentionality in recent findings (Flavell, 1985). The important point for purposes of the present thesis is that according to Emery et al., adolescents who are in the concrete operational level of development will not show the hopelessness theme of the cognitive triad.

Seligman & Peterson, (1986) are more to the point concerning the application of the attributional model to children. They disagree with those who argue that adult

psychopathology models cannot in principle be applied to children. According to Seligman and Peterson, if the constructs can be evoked to explain the behaviour of both adults and children and research shows converging results, then the theory is general, the helplessness theory is such a general theory.

Working within a developmental framework, Cicchetti and Schneider-Rosen (1986) assert that prior to age 8, the child is protected from developing a depressive syndrome by his or her limited understanding of the self. Three developmental changes in cognitions about the self are necessary conditions for the production of depressive symptoms. First, the child needs to change from absolute to social comparisons. Second, there must be a change from describing oneself in terms of what activities one engages in, that is, from typical activity to competency based evaluations. Third, the child must change from physicallistic evaluations based on physical attributes and possessions to more psychological evaluations based on trait descriptions. If a child makes an attribution for a negative event while at the physicallistic stage, the causal explanation will be transient due to the changing nature of the physical self, possessions and the like. Thus, the attribution will be unstable, and external, whereas attributions made at the psychological stage are more likely to be internal, stable

and global. The change from absolute comparisons to social comparisons is also relevant, for it only when children are capable of discerning that they have failed where others have succeeded, are the conditions for helplessness set. If the attribution for failure is to lack of ability, the child will believe that there is no response in his or her repertoire that will change the situation and helplessness and depressive symptoms will persist. On the other hand, the child who is not at the stage of social comparisons will explain his failure to external factors; the child has no concept that he or she should do the same or better, it is an absolute comparison. The authors sum up their position this way:

> We contend that it is only following the transition in the nature of self-cognitions at about age 8 that it becomes possible for a child to experience a loss of self-esteem accompanying depressed affect that is associated with personal comparisons (based on global, psychological qualities) resulting in negative valuations of the self. This loss of self-esteem reflects an affective concomitant to the cognitive activity that may introduce a positive circular relationship between affect and cognition as they serve to maintain the depressed state (Cicchetti & Schneider-Rosen, 1986, p. 106).

Cicchetti and Schneider-Rosen (1986) propose that the negative self-schemas that ultimately give rise to depression are formed through the process of assimilation and accommodation (see Flavell, 1985 for a description of these concepts). Such negative experiences as early loss of a parent, maternal deprivation, inadequate environments all contribute to the formation of the negative schema. Through time, negative experiences are assimilated into the schema and, as well, the schemas may be accommodated or elaborated by prolonged affect or stress. The authors further contend that one need not understand the activation of schema in terms of psychic energy, rather activation can occur by means of affect which forces the structure to play a larger role in the processing of information. They also suggest that negative schemas can arise as consequence of the emotional language of the depressed parent. Noting studies that indicate that language referring to emotions helps facilitate control, the authors suggest that the emotional language of the depressed parent may serve as a means of transmission of depression via the assimilation by the child of poor coping skills to control depressed affect.

Garmezy (1986) citing studies on children's reaction to success and failure contends that even 4 year olds will lower expectations for success if the failure can be made more salient. Moreover, Garmezy argues that early failure

events may lead to the cognitive triad. The author cites a study in which motivational patterns of first graders shifted from confident of success to fear of failure. When those children who remained confident of success were compared to those who feared failure, there was no differences in IQ, however, parental style patterns showed that the children with fear of failure had parents who were neutral to the child's success, reacted negatively to the child's failure, blamed performance on lack of ability and used social comparison norms rather than individual judgements of achievements. It seems that negative evaluations of the self can occur earlier than 8 years especially if failure is a main theme in life circumstances and moreover, negative self-evaluations do not necessarily depend on cognitive maturity.

As Rutter (1986c) notes, negative cognitive sets may also play a role in changing the sex ratio of depression in adolescence. Although teachers tend to give more negative feedback to boys than girls, the pattern of feedback may contribute to the development of differential attributional patterns (Dweck, Davidson, Nelson & Enna, 1978). Dweck et al., (1978) reported that in one study, boys received negative feedback that tended to be diffuse whereas girls received negative feedback specifically for their intellectual failings. However, positive feedback tended to

be diffuse for girls and specific for boys. Such a pattern of feedback may contribute to the perception that there is no response in the girl's intellectual repertoire that could change the situation. Indeed, Dweck and Bush (1976) have found that girls tend to give up and attribute their failure to lack of ability when they receive negative feedback whereas boys tend to increase their efforts. This pattern of feedback may have its learning phase in middle childhood and become accommodated into a cognitive schema as the girl approaches adolescence.

Before concluding this section, I would like to briefly review the only empirical study that I know of that has directly addressed the issue cognitive development and depression from an empirical viewpoint. Kovacs and Paulauskas (1984) tested the assumption that depression in children covaries with cognitive stage of development. The authors employed 3 measures of cognition, a test of formal operations, a test of interpersonal reasoning and a test of self-understanding which included 3 levels, physicallistic evaluations, activity based evaluations, and internal, trait like attribution evaluations, (see Cicchetti & Rosen, above). The subjects were 53 children between the ages of 8 and 13 who met the DSM-III criteria for an affective disorder; 43 met the DSM-III criteria for major depression. Contrary to assertions by Cicchetti and Schneider-Rosen

(1986), only 50% of the depressed children were at the trait or psychological level of self-understanding but consistent with these authors, only 9% were at the physicallistic stage. These results do not support the notion that a high level of self-understanding is a necessary condition for depression.

Collapsing the cognitive measures into a 3 task index, primarily concrete, transitional and primarily abstract, Kovacs and Paulauskas examined whether depressive symptomatology varied as a function of cognitive maturity. They found no evidence that the symptoms of hopelessness, self-depreciation, depressed mood, guilt, suicidal ideation or any of the vegetative symptoms were dependent on cognitive stage of development. However, they did find that, those who were less cognitively mature were the most likely to have had a chronic disorder when in a major depressive episode and to have taken longer to recover than more cognitively mature children. The authors were somewhat at a loss to explain the results, as they stated: "Notwithstanding our emphasis on methodological rigor, we were unable to verify even the most common notions about the developmental-stage mediation of depressive disorders in children" (p.74). These results do not support Emery et al.'s position that children need to attain the abstract level of cognitive development before then can experience a

sense of hopelessness. These results also do not support the view that cognitive immaturity protects against depression.

In conclusion, the cognitive theories of Beck and Seligman are remarkably silent in explaining how negative schemas develop and how it is that some depressions manifest in childhood while others manifest in adulthood. Perhaps one of the most perplexing developmental changes for cognitive theories to explain is the dramatic rise in depressive disorders in adolescence with a corresponding preponderance of depressed females. Developmental psychologists are beginning to fill in the gaps left by the original authors of cognitive theories and are beginning to provide a few insights into the role that negative cognitions play in the development of depressive disorders in children.

Methodological Issues in Empirical Research

The research in support of these models has been controversial (Coyne & Gotlib, 1983 ;1986; Segal & Shaw, 1986). Coyne and Gotlib (1983) critically reviewed the evidence and were unconvinced that depressed persons displayed the kinds of distortions Beck (1967) postulated or the attributional style proposed by Abramson et al.,(1978). Several of the criticisms that apply to both models bear

repeating in that I believe more recent evidence can help clarify the criticisms and moreover, are equally likely to apply to the extension of these models to depressed adolescents. Briefly, some the main problems with the research, according to Coyne and Gotlib's review, are summarized as follows:

1. The bulk of the research has employed nonclinical samples with depression defined as a score on a single self-report measure, the Beck Depression Inventory (BDI; Beck, 1967). Not only does this limit generalizations to college student populations but there are serious questions about the appropriateness of using the BDI to assess depression in this population, a purpose to which the instrument was never intended.

2. Correlations between the BDI which is highly weighted with negative cognitive attributes and measures of cognitive distortion may represent a tautology, showing nothing more than negative cognitions correlate with negative cognitions. This phenomenon is also known as criterion contamination.

3. The evidence suggests that nondepressed persons show a positive bias relative to depressed persons, a

phenomenon know as a self-serving bias. Thus if it cannot be accepted that normal functioning is characterized by realistic appraisals, then differences between nondepressed and depressed persons on various cognitive measures do not point to distortion in the latter.

4. The cognitive models underestimate the role current life events play in depression and moreover distortion or bias may come from an accurate perception of the environment, a theme which has been more fully discussed by Krantz (1985).

Points 1 and 2 can easily be solved by employing multiple measures of depression by different methods especially if one insists on using nonclinical samples. Recent evidence has shown that the BDI, when used in college populations, correlates so highly with anxiety and social desirability measures that its accuracy in detecting depression is rendered suspect (Tanko-Matsumi & Kameoka, 1986). A better strategy is to use clinical population where depression is defined by diagnostic criteria for depression. Because the diagnostic criteria for depression are weighted on biological dysfunction, differences between depressed and nondepressed patients would solve the tautology problem. For example, Norman, Miller and Klee

(1983) found that the Cognitive Bias Questionnaire (Krantz & Hammen, 1979) could easily distinguish those with major depression from those with other diagnoses on the depressed-distortion scale. Moreover, they found that distortion correlated with the noncognitive components of the BDI as well as with the cognitive components and even with some of the noncognitive components of the independently assessed psychiatric ratings.

The issue of self-serving bias versus cognitive distortion (point 3 above) also seems dependent on the population studied. For example, Sackeim and Wegner (1986) examined how experiences with success and failure affected the self-evaluations of depressed college students, nondepressed college students, clinically depressed inpatients and schizophrenic patients. As expected both depressed students and patients rated themselves as deserving more blame for failure compared to nondepressed students and patients, whereas nondepressed students and psychiatric patients rated themselves as deserving more praise for success that either of the depressed groups. However, within group analysis showed that the depressed students were evenhanded in their assignment of blame and praise whereas the nondepressed students showed a clear preference for praise for positive outcomes over blame for negative outcomes (the self-serving bias). Thus, this

finding is in accord with the criticism that differences between groups do not demonstrate distortion in the depressed students. Unexpectedly, the clinically depressed patients showed an inverse bias, with a clear preference for blame for negative outcomes over praise for positive outcomes. The authors concluded that the study of cognitive bias in nonclinical samples may be misleading.

Finally, the last point can be clarified by reference to two studies on cognitive distortion and life events. Hammen (1978) reported that for depressed individuals (college sample), low life stress was associated with greater distortion than high life stress. Similarly, Michael and Funbiki (1985) found among the depressed individuals that both high and low life stress were associated with more cognitive distortion than moderate life stress. At least for some individuals, cognitive distortion plays the main role in the severity of depression while for others life stress may play a more direct role.

Cognitive State and Trait Markers for Major Depression

Analogous to biological markers for major depression (see Puig-Antich, 1986), cognitive attributes predicted from the distortion theory and the helplessness theory may be conceptualized as either state or trait markers. The term

'marker' indicates that the cognitive attribute is specifically associated with major depression. Cognitive attributes that arise with the onset of depression and normalize with symptom remission can be thought as state markers. Therefore to qualify as a state marker, the attribute must distinguish not only normals from major depression, but also other psychiatric disturbances from major depression. A trait marker not only demonstrates such specificity for major depression but should remain salient after recovery from an episode of depression. According to Puig-Antich, (1986) three conditions should be met for a marker to be identified as a marker of trait:

1. Persistently abnormal in fully recovered, drug free, patients.

2. Present at significantly higher rate in clinically normal (never mentally ill) subjects with a strong family history of major depressive disorder in first-and second-degree biological relatives.

3. Long-term follow-up studies of depression-vulnerable informative pedigrees should produce increasing concordance with time between presence of the marker and lifetime history of major depressive disorder (p. 344).

Therefore, cognitive trait markers should be present at a significantly higher rate in clinically normal subjects who are at high-risk for developing depression than in normals without any affected family member and predict the onset of an episode of depression. The cognitive trait marker reflects the psychopathogenesis of the depression and therefore, would be useful in identifying persons who are vulnerable to the disorder. Cognitive markers may also help identify subgroups of major depression that may be quite different from each other on a cognitive basis but not on a syndrome, clinical basis. Given that major depression reflects a heterogeneous group in terms of aetiology, it would be useful to identify those children who reflect an cognitive vulnerability in order to conduct more precise research.

Empirical Research on Cognitive Markers for Adult Major Depression

Several instruments have been designed to test the cognitive theories and can be categorized as either assessing state or trait cognitive markers. The Hopelessness Scale (HS, Beck et al., 1974) taps the negative view of the future pattern of the cognitive triad. Because the triad represents the general thematic content of the latent schemas, such observable characteristics would be

predicted to arise with activation of the schema and normalize with symptom reduction (see Beck above). Other tests which can be classified as potential cognitive state markers include the Automatic Thoughts Questionnaire (ATQ, Hollon & Kendall, 1980) and the Cognitive Bias Questionnaire (CBQ, Krantz & Hammen, 1979). The ATQ measures current negative cognitive ruminations and the CBQ measures the tendency to distort or make systematic errors, both of which are predicted to decrease with symptom remission. The Dysfunctional Attitude Scale (DAS, Weissman & Beck, 1978) assesses the idiosyncratic or specific content of the negative self-schemas and is predicted to reflect the stable aspects of the cognitive triad. The DAS taps the silent assumptions or premises and therefore has the potential to assess a cognitive trait marker. Finally, the Attribution Style Questionnaire (ASQ, Seligman et al., 1979) assesses the tendency to make the depressive attributional style (see Seligman above) predicted from the helplessness theory and therefore also has the potential to identify a cognitive trait marker.

In summary, there are currently two tests which purport to assess trait characteristics, one for each of the cognitive models and there are several more tests which purport to assess the state characteristics of depression. Recent studies show that these tests have the ability to

discriminate those with major depression from other psychiatric conditions; these tests include the Hopelessness Scale (Hamilton & Abramson, 1983), the ATQ (Hollon, Kendall & Lumry, 1986; Dobson & Shaw, 1986), the Dysfunctional Attitude Scale (Dobson & Shaw, 1986; Hamilton & Abramson, 1983; but also see Hollon, Kendall & Lumry, 1986), the Cognitive Bias Questionnaire (Norman, Miller & Klee, 1983), and the Attributional Style Questionnaire (Raps, Peterson, Reinhard, Abramson & Seligman, 1982; but also see Hamilton & Abramson, 1983).

While there is some evidence that the negative cognitions as assessed by the tests reviewed above qualify as markers, do they qualify as state or trait markers as predicted from the theories? Studies with remitted depressed patients and predictive studies may help answer this question (these studies are summarized in Tables 1 and 2). In selecting studies for this review, high priority went to studies using clinical samples of patients with major depression; the only exception to this rule will be noted. Studies which employed an unknown cognitive measure were excluded if the study showed that the measure did not distinguish major depression from normal controls (i.e Gotlib & Cane, 1987) or from other psychiatric groups (e.g. Fennell & Campbell, 1984). Studies were excluded from the review if they did not employ a nondepressed control group

in the longitudinal design even if a control group was included at pretest (e.g. Dobson & Shaw, 1986). The psychometric properties of the cognitive measures have not been so well established as to forego the need to control for psychometric artifacts introduced as a function of time.

As noted earlier, a trait marker should be detectable after recovery from an episode of depression. This can best be assessed in a longitudinal design from the actively depressed phase to the remitted phase. Hamilton and Abramson (1983) found no differences between psychiatric controls (n=20) and depressed patients (n=20) on the ATQ and the DAS upon symptom remission although there were clear differences at admission to hospital. These results were predicted for the ATQ (state marker) but not for the DAS (a trait marker). Eaves and Rush (1984), however, found that while the ATQ normalized following symptom remission, the endogenous (n=11) and nonendogenous (n=13) depressed patients still showed significantly elevated dysfunctional attitudes on the DAS and a significantly negative attributional style on the ASQ when compared to normal controls (n=17). These results were exactly as predicted with the DAS and ASQ showing trait characteristics. Reda et al., (1984) reported that while depressed, patients (n=60) showed significant elevations in dysfunctional attitudes (DAS) compared to normal controls (n=60). At remission, depressed patients

differed significantly from the control group on only 13 items from the DAS but not on the total score. At a one year follow-up, remitted depressed patients (n=30) continued to be distinguished from the normal controls (n=37) on the 13-items from the DAS but not on the total score.

Finally, Miller and Norman (1986) classified patients with major depression into high (n=13) and low distorters (n=7) based on their hospital admission CBQ scores. The authors contrasted these depressed groups with a nondepressed psychiatric control group (n=12) at 2 time periods. They found that although the proportion of high distorters decreased significantly from the symptomatic phase to the remission phase, the proportion of high distorters was still significantly greater in the high distortion group (54%) at symptom remission compared to nondepressed control group (8%) and compared to low distortion group (14%). The authors concluded that the distortion model applies to only 50% of those with major depression and these 50% may represent a distinct subtype. Although it was predicted that distortion scores would normalize following symptom remission, this appeared not to be the case. The authors suggested that the CBQ taps elements of the underlying self-schema and noted that the high correlation of the CBQ with the DAS supports this conclusion.

A number of studies have approached the same issue by studying remitted depressed patients and comparing this group to actively depressed patients in a cross-sectional research design. Wilkinson and Blackburn (1981) found that recovered depressed patients were indistinguishable from patients recovered from other nondepressed disorders and from normal controls on measures of hopelessness and cognitive distortion. In contrast, the currently depressed patients showed significantly elevated levels of hopelessness and distortion compared to the recovered and to the nondepressed subjects. While the authors concluded that these results dispute cognitive theory, they are nonetheless as expected with both hopelessness and distortion being state markers. However, this argument is weakened by the results from two cross-sectional studies which reported no significant differences between recovered depressed patients, psychiatric controls and normal controls on the Dysfunctional Attitude Scale (a trait marker) as well as on the Automatic Thoughts Questionnaire (Blackburn and Smyth 1985; Hollon et al., 1986). These results do not support dysfunctional attitudes as a vulnerability marker for depression.

Table 1
Studies on Negative Cognitions in Patients Remitted from Major Depression

Study	Time Lag	Measure	Results
Longitudinal			
Hamilton & Abramson, 1983	17 days	ASQ,DAS, ATQ	T1:CD > PC & NC on all T2:RD=PC=NC on all
Eaves & Rush 1983	73 days for MDD	ATQ,DAS ASQ	T1: CD>NC on ATQ,DAS & ASQ T2: RD>NC on DAS, & ASQ but RD=NC on ATQ
Reda et al. 1985	1 year	DAS	T1: CD>NC on DAS T2:RD=NC but RD>NC on 13 item subset from DAS
Miller & Norman, 1986	9 month	CBQ	T1: CD>PC in % high distorters on CBQ T2:RD=PC on CBQ but RD>PC for a subgroup of MDD positive on T1 CBQ
Cross-Sectional			
Wilkinson & Blackburn, 1981		HS, CST	RD=NC & PC but CD>RD,PC & NC on HS & CST-distortion
Blackburn & Smyth, 1985		ATQ,DAS, CST	RD=PC & NC
Hollen et al. 1986		DAS,ATQ	RD=PC & NC; CD>RD,PC on ATQ but CD=PC on DAS

Note. ATQ=Automatic Thoughts Questionnaire;CBQ=Cognitive Bias Questionnaire; CST=Cognitive Style Test; DAS=Dysfunctional Attitudes Scale; HS=Hopelessness Scale; MDD=Major Depressive Disorder; CD=Currently Depressed; RD=Remitted Depressives; PC=Psychiatric Controls; NC=Normal Controls; T1=Time one; T2=Time two.

As noted earlier, a cognitive trait marker should also predict risk for depression. A number of recent studies address this issue. Rush and Weissenberger (1986) tested a small group of patients (n=15) at three time periods, admission, remission and 6 months post remission. They reported that dysfunctional attitudes on the DAS but not automatic thoughts or attributional style for negative events assessed at remission predicted 6 month depression levels. Further analyses showed that remission DAS scores were a better predictor of subsequent depression than remission depression ratings. In a one year follow-up of depressed patients who had recovered from major depression following treatment with either cognitive therapy or antidepressant drug treatment, Simons, Murphy, Levine and Wetzel (1986) found that termination scores on the DAS but not on the hopelessness scale predicted those who relapsed. In this study, both termination depression scores and DAS scores improved prediction than either alone; other variables such as past episodes and age were not predictive. These results implicate dysfunctional attitudes as a possible trait marker as predicted from Beck's (1967) theory.

Although the subjects in the following study were not patients but a sample of community volunteers, it is included in this review because it is one of the only

studies which has assessed cognitive variables prior to the development of a depressive disorder. Lewinsohn and colleagues (Lewinsohn, Hoberman and Rosenbaum, 1988; Lewinsohn, Steinmetz, Larson and Franklin, 1981) measured depressive cognitions in a community sample (n=998) and followed them up over a one year period. The average length of time between pretest and follow-up was 8.3 months. In a preliminary report, Lewinsohn et al., 1981 reported that nondepressed persons at pretest who subsequently developed a RDC depressive disorder (n=85), between the pretest and the follow-up period, did not show any more negative attributions, irrational beliefs or negative expectations at pretest than those who did not develop depression (n=154). A second group who were depressed at pretest (n=63), however, did show significant differences on all the cognitive measures, with the exception of negative attributions, compared to nondepressed controls. On the basis on this preliminary analysis, the authors concluded that the results provide clear evidence that persons who become depressed in the future do not subscribe to negative cognitions or demonstrate any kind of cognitive vulnerability.

In a later analysis of this data, Lewinsohn, Hoberman and Rosenbaum (1988) modified their earlier conclusion. They reported that although negative cognitions did not

predict those who experienced an episode of depression as defined by a RDC diagnosis, negative cognitions did predict those who experienced an elevation of depressive symptoms at follow-up as assessed by a self-report measure of depression, the Centre for Epidemiological Studies Depression Scale (CES-D). Partial correlations, controlling for pretest CES-D scores, indicated that increases in depressive symptoms over time were significantly associated with a global dissatisfaction with the self (a variable not reported in the preliminary analysis, Lewinsohn et al., 1981), low self-esteem, low perception of control, expectation of negative outcomes and irrational beliefs. A regression analysis indicated those variables obtained at pretest that best predicted later depression (CES-D) included pretest depressive levels on the CES-D, low self-esteem and irrational personal beliefs (similar to the Dysfunctional Attitude Scale, a trait marker). They also found that dissatisfaction with the self as assessed at pretest was the only cognitive variable to significantly discriminate between subjects who developed an episode of depression (RDC) and the nondepressed controls. Subjects who subsequently developed a RDC depression were also characterized by having higher CES-D scores in the pretest period. The authors concluded that negative cognitions are related to the development of negative affect which in turn has a more direct effect on the development of a diagnosable

depressive disorder. While this may be a fair conclusion to make, it is not without problems. The authors did not report how many subjects in this study developed a major depressive episode rather than a minor depressive episode or a intermittent depression. These disorders may represent three different aetiologies and treating them as one group of depressive disorders may not be appropriate. All diagnostic information used in assigning a RDC depressive diagnosis was obtained at follow-up, whereas self-reported depression on the CES-D was obtained at both the pretest and the follow-up periods. It is therefore important to note that diagnoses assigned to the pretest period were based on the retrospective reports obtained in the follow-up period.

Table 2
Predictive Studies for Negative Cognitions in Adults

Study	Time Lag	Measure	Results
Lewinsohn et al. 1981	8 month	Own measures	Negative cognitions did not predict RDC diagnosis but predicted those who did not improve from a depressive episode
Lewinsohn et al. 1988	same	same	Negative self predicted RDC affective diagnosis. All cognitive measures predicted self-reported depression
Rush & Weissenberger 1986	6 month	ATQ,DAS ASQ	DAS but not ATQ or ASQ predicted relapse in RMDD
Simons et al. 1986	1 year	HS,DAS	DAS but not HS predicted relapse in RMDD

Note. ATQ=Automatic Thoughts Questionnaire; DAS=Dysfunctional Attitudes Scale; HS=Hopelessness Scale; RDC=Research Diagnostic Criteria;RMDD=Remitted Major Depressive Disorder.

In summary, the evidence from the longitudinal studies with remitted depressed patients is generally supportive of the trait concept of cognitive markers in that remitted patients demonstrate residual negative cognitions compared to controls. In contrast, the cross-sectional studies of remitted patients uniformly demonstrate that there is no difference between remitted patients and controls on

negative cognitions. The evidence from the predictive studies is consistent in demonstrating that negative cognitions predict later depression. It appears that dysfunctional attitudes have received more support than+ attributional style as a possible candidate for a trait marker for depression. However, the evidence suggests that there may be a subtype of major depression for whom cognitive distortion is a trait rather than a state phenomenon and the recognition of such a subgroup may help explain inconsistent findings in the literature. When this subgroup is pooled with other patients with major depression, differences between depressed and nondepressed patients are bound to be weaker.

In contrast to the above conclusions, Barnett and Gotlib (1988) reviewed the same studies with the exception of the Lewinsohn et al. (1988) study and concluded that there is little evidence that supports a stable cognitive vulnerability to depression. There are several problems with their review that weaken their conclusion. First, their conclusion is based almost entirely on studies conducted with students, pregnant women and community volunteers. For example, in their review of the attributional style literature only 3 out of the 8 studies involved psychiatric patients. In their review of the dysfunctional and distorted cognition literature, 12 out the

22 studies employed patient samples. Of these 12, 5 are supportive of cognitive theory. Interestingly, the authors failed to include in the review, the Simons et al., (1986) study (reviewed above) which showed that negative cognitions predicted the onset of major depression. Thus, approximately half the studies are supportive and half are not. Secondly, the authors reviewed a number of studies which examined the covariance of cognitions and depressive symptoms from the depressive phase to the remitted phase without employing a control group (e.g. Dobson & Shaw, 1986,1987; Silverman, Silverman & Eardly, 1984, and Simons, Garfield & Murphy, 1984). With one exception (Dobson & Shaw, 1986), these studies demonstrated that reductions in depressive symptoms were paralleled by reductions in negative cognitions. These results were interpreted by Barnett and Gotlib as indicating that cognitions are not stable and therefore provide evidence against a cognitive vulnerability model of depression. The problem is that in order to show support for the theory, the evidence has to prove the null hypothesis, a position that is not easily defensible. It is much more likely that there will be changes across time as a result of the rather poor psychometric properties of these measures. The trait marker theory argues that negative cognitions must be detectable in recovered patients. Without a control group, longitudinal studies of this type can neither support nor refute the

cognitive theory. Longitudinal studies which predict subsequent depression, however, are quite a different matter and need not employ a control group in order to support the theory. Thirdly, while Barnett and Gotlib acknowledged the supportive nature of the studies with children, they did not include these studies in their review because they felt that depression in children was qualitatively different from the adult disorder. This view, which as I have already pointed out, is not supported by empirical research.

In fairness, however, it should be pointed out that even if these studies were all supportive, it would not rule out the possibility that negative cognitions are sequela from the first depressive episode. Negative cognitions may not be true trait markers, but rather markers of past episode. None of the studies in Barnet and Gotlib's review employ subjects who have never been ill but who are nevertheless at risk for depression. At-risk subjects are the only nonclincal subjects which can support the cognitive theories. In also might be fair to state that, at best (considering only the clinical studies), the evidence is equivocal. However, as I shall soon review, studies from the child literature provide the only evidence that negative cognitions are not simply markers of past depressive states. Thus, final conclusions on whether or not negative

cognitions are trait or state markers should wait until the literature on childhood depression is reviewed.

Empirical Research on Cognitive Markers for Childhood Depression

Several studies with normal school age children have shown that depressive symptoms are correlated with measures of negative cognitions (Moyal, 1977), cognitive distortion (Leitenberg, Yorst & Carroll-Wilson, 1986; McCarthy, 1985) and negative attributions (Izard & Schwartz, 1986; Kaslow, Rehm, & Seigal, 1984; Leon, Kendall & Garber, 1980). Because these studies employ only a single self-report measure to define depression, they do not shed any light on whether negative cognitions constitute a marker for major depression.

Nevertheless, a number of longitudinal studies with nonclinical samples are worthy of some note because they provide insight into whether negative cognitions are simply a by-product of depressive symptoms or whether they act as independent phenomena (these studies are summarized in table 3). Seligman and colleagues (Seligman et al., 1984; Seligman & Peterson, 1986) designed a child's version of the Attributional Style Questionnaire to examine the negative attributional style in depressed children. The authors

tested 95 normal school children at two time periods separated by a six month interval. As predicted from the helplessness theory, the authors found that the tendency to attribute negative events to internal, stable and global causes (the negative attributional style) was significantly related to the child's depressive symptoms (r=.51), whereas the attribution for good events to internal, global and stable causes (the positive attributional style) was negatively related to depressive symptoms (r=-.53).

Although Seligman et al., (1984) reported that the negative attributional style was stable over the 6 month period (r=.66), they also reported that depressive symptoms as assessed on the CDI were even more stable (r=.80). In order to determine whether or not attributional style was a predictor of later depression, Seligman and Peterson (1986) examined the correlation between pretest attributional style and depressive symptoms 6 months later, while partially out pretest depressive symptoms. They found that the negative attributional style but not the positive attributional style significantly predicted subsequent depressive symptoms.

In a second longitudinal study, Seligman and his colleagues (Nalen-Haeksema, Girgus & Seligman, 1986) expanded both the sample size (n=164) and the time period of

study (1 year). School children were tested four times (every 3 months) throughout the year with the CDI, CASQ and a life events questionnaire. The negative attributional style reliably predicted future depressive symptoms on the CDI on the four successive pairs of administrations. The partial correlation coefficients between attributional style at pretest and 4 month depression level, with pretest depression partialled out, ranged from .29, $p<.009$, to .39, $p<.04$. Because depressive symptoms also predicted later attributional style, the authors attempted to determined whether or not depression caused the attributional style by examining the correlation between attributional style obtained at the second administration and the average of CDI scores obtained from the subsequent administrations while partialling CDI score from the first and second administrations. The resulting partial correlation (.37, $p<.05$) suggested that the power of the negative attributional style to predict subsequent depression was not due to the effects of prior or current depression. The authors also reported that while the interaction between life events and attributional style predicted future depression, this pattern could not be replicated for all 4 interval pairs. These results provide reasonable evidence that negative cognitions act independent of depressive symptoms and it might be fair to state that in some cases,

attributional distortions precede the onset of mild depression.

Jaenicke et al, (1987) employed a different methodology to determine the role negative cognitions play in the development of depression. They reasoned that the offspring of mothers with affective disorders would be at high risk for developing future depression and therefore, there should be some evidence of negative cognitions in these children compared to offspring of nondepressed mothers. The authors found that compared to offspring of medically ill mothers and normal mothers, the children (range 8-16 years) of mothers with depressive disorders showed a more negative attributional style on the CASQ, a lower self-concept and were able to recall less positive self-descriptions on a self-schema task. Differences between the groups could not be accounted for by socio-economic or stress factors in the families. Could these results simply have reflected the effects of concurrent depression? The authors reported that the majority of the offspring from the depressed mothers had no RDC diagnosable depression as assessed by the K-SADS interview and the prevalence of current depressive disorders did not differ from that of the controls. This evidence supports the concept that negative cognitions are true trait or vulnerability markers rather than state or episode markers for depression. In addition, these results

demonstrate that negative cognitions are not markers of past episodes of depression.

Analyses from a 6 month follow-up of the children (n=79) in the above study have provided some preliminary evidence on the trait versus state markers for negative cognitions. Hammen, Adrian and Hiroto (1988) reported that pretest attributional style was not a significant predictor of those children given a DSM-III diagnosis of major or dysthymic disorder whereas, negative cognitions about the self (derived from the Piers-Harris Scale) did predict follow-up affective diagnoses (Hammen, 1988). Of the 79 children, 10 were given a dysthymic diagnosis and 6 were given a diagnosis of major depression in the follow-up period. Further, stressful life events were predictive of affective diagnoses but there was no interaction between attributional style or negative self-cognitions and stressful life events. While these results appear to support the vulnerability model for depression, they are limited by the fact that the follow-up interviews were conducted by telephone and by the small number of children with a diagnosis of major depression.

Table 3
Predictive Studies for Negative Cognitions in Children

Study	Time Lag	Measure	Results
Seligman et al. 1984	6 month	CASQ	A negative AS but not a positive AS predicted future depression on CDI
Nalen-Hacksema et al 1986	1 year	CASQ	Positive AS minus negative AS score reliably predicted future CDI
Hammen et al. 1988	6 month	CASQ	CASQ did not predict affective diagnosis but Negative AS significantly correlated with T1 CDI
Hammen, 1988	same	P-H	Negative self cognitions predicted affective diagnoses

Note. AS=Attributional Style; CASQ= Children's Attributional Style Questionnaire;P-H=Piers-Harris Self Concept Scale;T1=Time one.

In nonclinical samples, cognitive distortions have been found to be infrequent in both small (McCarthy, 1985) and large samples of children (Leitenberg, Yorst & Carroll-Wilson, 1986). As one might expect most children do not appear to have a negatively biased interpretation of events. One would expect to see a high frequency of distortion among clinical samples of depressed patients as predicted from Beck's theory. However, there is evidence that negative cognitions may be infrequent even among clinical samples of

depressed children. Hurt, Freidman, Clarkin, Corn, and Aronoff (1982) compared adolescents (mean age=15.7) with major depression to young adults (mean age=25.7) with major depression to determine whether or not the groups differed in depressive symptomatology. The authors reported that while the two groups did not differ in endogenous and other depressive symptomatology, the adolescent patients demonstrated significantly lower scores on ratings of worthlessness, hopelessness and helplessness compared to the young adults. The authors concluded that the low prevalence of these cognitive features requires reconsideration of the importance of cognition for adolescent populations and furthermore, may represent an age-related difference in the expression of major depression. However, this study is limited by the rather small sample size of adolescents with major depression (n=9) and conclusions reached by the authors should be considered suggestive rather than conclusive.

Kazdin et al., (1983) adapted the Hopelessness Scale (Beck et al; 1974) for use with children and examined the scale's relationship to depression in inpatient psychiatric children (8-13 years). Although the authors reported that hopelessness was significantly related to depressive symptoms as assessed on the CDI, (r=.49) and negatively related to self-esteem (r=-.54), they did not find that

hopelessness distinguished those with a DSM-III diagnosis of major depression (n=12) from other psychiatric disorders. More recently, Kazdin, Rodgers and Colbus (1986) replicated their earlier findings on a larger sample (n=262) of inpatient children, but again the hopelessness scale failed to distinguish those with a diagnosis of major depression (n=46). However, a 6 week test-retest examination revealed that hopelessness was more stable in those with major depression (r=.63) than in nondepressed patients (r=.42). Nevertheless, these stability coefficients appear moderate at best. These results suggest that hopelessness is not a marker for major depression in children as it is for adults, but it may have specificity for suicidal ideation and suicide attempts in adolescents (see Kazdin et al., 1983; Spirito, Williams, Stark & Hart, 1988).

Benfield, Palmer, Pfefferbaum and Stowe (1988) contrasted a group of inpatient children given a DSM-III diagnosis of major depression (n=15) or dysthymic disorder (n=2) with children given other nondepressed diagnoses (n=20) on the CDI, the Hopelessness scale for Children, the Attributional Style Questionnaire and a life events questionnaire. All these measures failed to distinguish the groups with the exception of two subscales from the Attributional Style Questionnaire. The depressed group were significantly less likely to attribute good outcomes to

stable factors and to global factors. However, the helplessness theory predicts that negative attributional should be more characteristic of depression than a lack of a good attributional style. The authors concluded that there may not be a unique constellation of cognitive characteristics for clinical depression in children.

It should be noted that failure to find significant differences between clinically depressed and nondepressed groups in both the Kazdin et al. studies and in the Benfield et al. study could be due to the fact that psychiatric evaluations did not employ a structured interview (for further discussion on the importance of this point, see Haley, 1984; Kovacs, 1986; Puig-Antich. 1983). Support for this argument is evident in the following study which did employ a structured interview.

Employing a structured interview, the K-SADS-E, Asarnow, Carlson and Guthrie (1987) reported that inpatient children with a DSM-III diagnosis of depressive disorder (n=14) could be distinguished from nondepressed children (n=16) by demonstrating more hopelessness and lower self-worth. Although the depressed and nondepressed children did not differ in IQ or achievement, the depressed children perceived themselves as less academically competent. The authors noted that this finding supports the view that

negative self-perceptions of depressed children represent a negative bias or distortion rather than true differences. The authors also found that the depressed children did not perceive themselves as any different in social or athletic competence nor did they differ in their perception of their family milieu as more or less cohesive, conflicting, organized or controlling. Thus, while depressed children exhibit a negative view of the future and the self, their negative view of the world appears to less pervasive.

Haley, Fine, Marriage, Moretti and Freeman (1985) designed the Cognitive Bias Questionnaire for Children (CBQC) modelled after the adult version, the CBQ (Krantz & Hammen, 1979). The CBQC assesses the tendency to choose cognitively distorted responses over nondistorted responses to imaginary situations. In a sample of psychiatrically disturbed children (8-16 years), Haley et al., found that cognitive distortion responses on the CBQC were significantly related to depressive symptoms on the CDI (r=.64) and on independent psychiatric ratings (r=.45). Those with a DSM-III diagnosis of major depression (n=11) exhibited significantly more cognitive distortions on the CBQC than nondepressed psychiatric patients. These results parallel those found with adults using the CBQ (Norman, Miller & Klee, 1983) and supports the thesis that cognitive distortion is a marker for major depression even in

children. Although the interviewers employed no formal interview measure, the interviewer was required to rate the presence and absence of depressive symptoms for both major and dysthymic disorder. This procedure necessitated that the interviewer inquire into each depressive symptom. Thus some structure was imposed on the psychiatric interview and even this limited amount of structure in the interview may account for significant differences between the groups by more accurately diagnosing the groups.

Table 4
Studies on Cognitive Markers in Children with Major Depression

Study	Criteria	Measure	Results
Hurt et al. 1982	RDC	SADS ratings	Adolescent MDD<adult MDD on negative cognitions
Kazdin et al 1983	DSM-III	HSC	MDD=ND<on hopelessness
Kazdin et al. 1986	DSM-III	HSC	MDD=ND on hopelessness
Asarnow et al. 1987	DSM-III	3 item-HS;PCSC	MDD>ND on hopelessness low self-worth, low cognitive competency
Benfield et al 1988	DSM-III	CASQ;HSC	MDD=ND on negative AS & HSC but MDD>ND on Good/global & Good/Stable subscales of CASQ
Haley et al. 1985	DSM-III	CBQC	MDD>ND on distortion scale

Note. AS=Attributional Style; CASQ= Children's Attributional Style Questionnaire; HSC=Hopelessness Scale for Children; HS=Hopelessness Scale (adult version); PCSC=Perceived Competence Scale for Children;CBQC=Cognitive Bias Questionnaire for Children; MDD=Major Depressive Disorder;ND=Nondepressed patients.

In summary, the studies reviewed provide evidence that negative attributional style predicts future depressive symptoms but not depressive diagnoses, and that a negative view of the self predicts the development of a depressive

diagnosis. The evidence also indicates that negative thinking and attributional style are more frequent in healthy children at risk for depression than in children not at risk, and that children with a diagnosis of major depression show more cognitive distortions and negative bias than children given other diagnoses. While these results are not definitive in establishing negative cognitions as markers for major depression in children and adolescents, they are consistent with the cognitive theory. The studies reviewed are more consistent than the body of data reviewed for the adult literature. Moreover, they provide support for two of the three conditions of a trait marker, that it be present in healthy persons at risk for depression and that it predict the onset of depression. The other condition, that the marker be present in persons remitted from depression, has received little attention in the child literature whereas this condition has predominated the adult literature.

It should be noted that studies with clinical samples of children (see table 4) provide the weakest support for the cognitive theories of depression. Therefore, further research is needed with larger clinical samples of children and adolescents to determine whether negative cognitions are state or trait markers, that is, whether or not negative cognitions persist past the remission of the depressive

episode. There is some evidence that adolescents with major depression may not characterized by negative thinking. This finding highlights an often overlooked fact that negative thinking is not an essential prerequisite in the diagnosis of major depression and thus a theoretically meaningful question is whether or not cognitive distortions actually are found in the depressed adolescent when depressed. Clearly more research is needed to answer very basic questions including whether or not cognitive distortions even occur in adolescents with major depression.

The Present Study

The present study is divided into two substudies. In Study 1, the Cognitive Bias Questionnaire for Children (Haley et al., 1985) was revised and validated on a larger sample of adolescents with major depression. In Study 2, adolescents who had recovered from an episode of major depression were examined to determine whether negative cognitions normalized. Thus, in the first study, the methodological aspects of the revised CBQC were investigated and in the second study, the more substantive issues of trait versus state markers for major depression were examined.

STUDY 1: THE VALIDATION OF THE REVISED CBQC

Prior to undertaking this investigation, several inadequacies in the measure of cognitive distortion, the Cognitive Bias Questionnaire for Children (CBQC) needed to be addressed. The original CBQC was modelled after the adult version (Krantz & Hammen, 1979) and contained as did the adult version four response options to imaginary stories, a depressed-distorted, a depressed-nondistorted, a nondepressed-distorted and a nondepressed-nondistorted response. The problem was that the depressed-distorted option was always confounded or 'contaminated' by an affective self-reference. In a sample item, the subject was asked to imagine what a girl was thinking when she noticed a boy with a frown on his face. The four options were as follows: (a) Everyone should be happy all the time (nondepressed-distorted), (b) I feel bad because he must think I look pretty awful (depressed-distorted), (c) it doesn't bother me that he looked that way, some people have a lot on their minds (nondepressed-nondistorted), (d) I feel sad that some people aren't happy (depressed-nondistorted). In this example, the depressed-distorted item is confounded by the affective statement "I feel bad", which even though is supposedly controlled for by the depressed-nondistorted option "I feel sad", still represents the most extreme

affective option. It became clear that the affective statements in the depressed-distorted option had to be deleted to avoid the problem of criterion contamination. It does not further the investigations into cognitive bias and depression if all that can be stated from the evidence derived from the CBQC is that depressed patients are more likely to feel bad than nondepressed patients.

Although deleting actual depressive statements was a first step, it did not solve the problem of extremes. There is an implicit conundrum in attempting to make a distortion item appear less extreme in a set of response options when, by definition, distortion implies the most extreme position. I attempted to solve this problem by making the depressed-nondistorted items stronger in depressed affect than the depressed-distortion items. Thus the depressed-nondistorted items contained such statements as "I feel sad", "I feel lonely", I feel unhappy", "I feel down", "it makes me feel no good," while the distortion scale contained no such affective statements. The question to be answered was whether or not, with all these methodological revisions, the CBQC could perform as well as original version.

The specific hypotheses for study 1 are as follows:

1). Adolescents with major depression will demonstrate more distortions on the revised CBQC than adolescents given other diagnoses.

2). Adolescents with major depression will choose more distorted options relative to nondistorted-nondepressed options on the CBQC. This hypothesis addresses the issue that clinically depressed patients are not 'even handed' in their negative and positive thinking but tend to be negatively biased.

The relation between psychosocial stress and distortion will be given special attention as much of the criticism of cognitive models has centered on lack of consideration given to the environmental stressors (Coyne and Gotlib 1983; 1986; Krantz, 1985). Coyne and Gotlib (1986) argue that: "Depressed persons deal with distressing circumstances that often do not yield to their efforts, and they often do so in the face of overtly hostile, critical and rejecting significant others. It would seem that the negative verbalizations in such a context does not require the postulation of intractable cognitive processes " (p.703). This assertion however, remains to be tested empirically.

Method

Subjects

Subjects were 74 adolescents between the ages of 12.2 years and 18.6 years (mean age=15.1 years) who were referred to the Child and Adolescent Psychiatry outpatient department of Vancouver General Hospital. Subjects were recruited from the regular referrals to outpatient department as well as from announcements to various community agencies that the outpatient department was offering group therapy programs for depressed teenagers. Subjects were excluded if they were younger than 12.0 years and older than 19.0 years or if they could not read at a grade 1 level or there was evidence from psychological reports of an IQ below 70. There were 46 girls and 28 boys.

Diagnoses

Psychiatric diagnosis was based on direct interviews with the adolescent and the parent employing the Kiddie Schedule for Affective Disorders and Schizophrenia, present episode version (KSADS, Puig-Antich, Chambers & Ryan, 1986). The KSADS is a semi-structured interview of an ongoing psychiatric disorder in children 6 to 17 years of age. The

examiner's aim is to arrive at a clear judgement of the severity of each symptom at two time periods, when the symptom was at its worst during the current episode and in the last week. The last week ratings are used in assessing change over time. The child and the parent are interviewed separately and final summary ratings are based the clinician's integration of the two sets of ratings. The parent and child ratings are not independent assessments in that the child or the parent may be asked about disagreements that arise between the two and asked to clarify the disagreement. In general, parents are weighted for more observable behaviours such as conduct disorders and the chronology of the episode. Recent research has supported this as a sensible "rule of thumb" (Edelbrock et al., 1985; 1986; Haley, 1984).

Each symptom rating has explicit criteria for grading severity. For a symptom to be positive for a diagnosis of depression a score of 3 or above is useful, this usually connotes that the symptom is present at least 50% of awake time, including weekends.

Interrater reliability as assessed by the mean intraclass correlation coefficient for symptoms of the depressive syndrome, in the joint interview design, is .86 for the interview with the parents and .89 for the interview

with the child. Test-retest (conducted over 72 hour time period) coefficients for the summary ratings range from .72 for the sum of depressed mood and anhedonia to .81 for the 17-item sum of depressive symptoms. The test-retest Kappa coefficient for interrater reliability on the diagnosis of major depression is .54 (Chambers et al., 1985).

In the present study, all diagnoses were based on summary ratings for the worst period for the current episode in the last year. If no worst period was identified in the last year or if the episode was greater than one year in length, the interview ratings were based on the total year period. An episode was defined as the onset of symptoms preceded by a two month symptom free period. If however,. the adolescent had a long standing disorder, such as chronic mild depression, the onset the current episode was taken from the change from mild to major depression. Thus, durations of current episodes of major depression were calculated from the onset as defined above to the time of assessment or to episode remission, defined as a two month symptom-free period prior to assessment. The diagnosis of major depression was based on Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978). All other disorders were diagnosed using DSM-III-R criteria (American Psychiatric Association, 1987). For a diagnosis of major depression, present or current episode ratings were

dichotomized between present-absent at the point between KSADS rating points 2 (slight) and 3 (mild) and then the RDC criteria were used, unmodified, to diagnose major depression (definite only) and its endogenous subtype (definite only).

All interviews were conducted by this author with the exception of 12 cases in which this author either served as the initial rater or as a second rater as part of a validity study not reported here. Final diagnoses of all cases were decided by this author and the chief child psychiatrist of the outpatient department by consensus agreement.

Measures

Depression Measures

The KSADS Depression Scale. The KSADS depression scale consists of 12 items assessing the Research Diagnostic Criteria (RDC; Spitzer, Endicott & Robins, 1978) for major depression. These items include depressed mood, guilt, anhedonia or loss of interest, fatigue, concentration difficulty, psychomotor retardation, psychomotor agitation, insomnia, hypersomnia, decreased appetite, increased appetite, and suicidal ideation. The KSADS depression scale is used to assess severity of the worst period of the current episode as well as the severity of the last week

prior to assessment. The internal consistency estimate for this 12-item scale is adequate (alpha=.72) and the test-retest coefficient is also adequate (r=.72; Chambers et al., 1985). A total score is formed by summing all 12 items. The mean score for adolescents with major depression is reported to be 38.40 (sd=7.2) based on worst period ratings (Ryan et al., 1987). There are no significant differences between adolescent and prepubertal patients on the 12-item scale (Ryan et al., 1987).

The Children's Depression Inventory. The Children's Depression Inventory (CDI; Kovacs, 1980) is a 27-item self-report measure of depression based on the adult version, the Beck Depression Inventory. This is the most widely used self-report measure of children's depression and has shown a high degree of reliability. Internal consistency estimates are in the range of .80 to .94 (Saylor, Finch, Spiritot & Bennet, 1984). CDI scores have been found to discriminate clinically depressed children (Fine, Moretti, Haley & Marriage, 1985; Rotundo & Hensley, 1985) and to correlate with clinical interview ratings of depression (Haley et al., 1985). However, the CDI tends to overlap with several other nondepressed measures, enough to regard the CDI as insufficient as a criterion for childhood depression (Asarnow & Carlson, 1985; Saylor et al., 1984).

Studies with both normal and clinical samples of children have consistently shown that there is no significant difference between prepubertal and adolescent children on the CDI (Garber, 1984; Kovacs, 1980; Nelson, Politano, Finch, Wendel & Mayall, 1987; Smucker, Craighead, Craighead & Green, 1986). In normal samples of adolescents, the mean CDI score has been reported to be 9.59 (sd=6.57) (Smucker et al.,1986) and for clinical samples of adolescents the mean has been reported as 12.95 (Sd=8.81) (Nelson et al., 1987).

Cognitive Measures

The Cognitive Bias Questionnaire for Children. The Cognitive Bias Questionnaire for Children (CBQC; Haley, 1985; see appendix A) will test Beck's (1967) model of depression. Some of the problems with the original version of the CBQC have already been discussed. The revisions will now be discussed in some detail. The first version consisted of ten hypothetical events followed by 4 response options, depressed-distorted (DD), nondepressed-nondistorted (NN), nondepressed-distorted (ND) and depressed-nondistorted (DN). The first major change was to delete the ND scale because it received so few responses as to make it totally unreliable and moreover, I was not sure whether it made sense to control for distortion. Second, the number of

vignettes was reduced from 10 to 8 while the number of sets of questions was increased from 10 to 20 to increase the reliability. Third, as previously noted, all DD items were pruned of affectively toned words such as "down", "sad" and "bad" but such words remained in the DN scale. Thus the respondent now has to choose between a strongly affective response option such as "I feel down" on the DN scale and "I begin to wonder what I have done wrong" on the DD scale. This would provide a stronger and more conservative test for the cognitive over the affective components of depression.

The vignettes were completely rewritten to have a stronger pull for distortion; now, for example, when Dan fails the test he might think other won't like him (negative view of the world), but what if others fail the test also, will he still feel his answer was the worst in the class (negative view of the self) and go on generalize this to future classes (negative view of the future)? Of the 20 items, 8 reflected a negative view of the self, 8 reflected a negative view of the world and 4 reflected a negative view of the future. Finally, separate male and female versions were made by introducing proper names but the third person format was maintained. All of the other measures of cognition reviewed in the preceding sections use the first person.

To determine whether or not depressed subjects choose more distortions than nondistorted options on the CBQC, a difference score was formed by subtracting the DD scale from the NN scale. High scores on the CBQC difference scale reflect a self-serving bias whereas negative scores reflect a self-derogating bias.

To estimate the reliability of the revised CBQC, 10 adolescent females attending a private high school and enrolled in an learning assistance program were administered the CBQC and the CDI at two time periods separated by an 10 day interval. The subjects were volunteers who had permission to participate in the study from their parents. The mean age of the subjects was 14.6 years. The tests were administered by their learning assistance teacher who was conducting the study as part of lab requirements in course on psychological assessment. The test-retest coefficients for the DD, the NN and the DN scales were .81, $p<.01$, .88, $p<.01$, .60, n.s., respectively. The CBQC difference score demonstrated a test-retest coefficient of .92, $p<.01$. These reliability coefficients were comparable to those obtained for the CDI, $r=.84$, $p<.01$.

The Hopelessness-Worthlessness Scale. The Hopelessness-Worthlessness Scale (HW scale) consists of two rating scales from the KSADS that assess the symptoms of

worthlessness and hopelessness. Each symptom is rated on a 6 point scale with well specified criteria reflecting increasing severity. For example on the worthlessness scale, a rating of 3 is defined as:"often feels like a failure, or would like to change his looks or his brains or his personality", a rating of 6 is defined as:"Pervasive feelings of being worthless or a failure. Often says he hates himself". On the hopelessness scale, a rating of 3 is defined as:"Often discouraged; doubts he will get better", a rating of 5 is defined as:"Pervasive feelings of intense pessimism; has given up; helpless." These two scales are summed to form the HW scale.

The HW scale was based on ratings obtained from the interview with the adolescent only and did not reflect an integration of parental and child ratings. It should be noted that both worthlessness and hopelessness ratings were not used in the KSADS depression scale as both of these symptoms are not part of the RDC definition of major depression.

Psychosocial Stressors

As part of the initial interview with the KSADS, each adolescent and his or her parent was asked about the frequency and kind of psychosocial stressors occurring

within the last year. Following DSM-III-R Axis IV criteria, the worst stressor in the last year was assigned a rating of 1 for none or minimal, 3 for mild, 4 for moderate, 5 for severe and 6 for extreme stress. To aid in assignment of stressor ratings, the RAU scale for adolescents was employed (Plapp, Rey, Stewart, Bashir, & Richards, 1987). The RAU consists of 77 stressors common to adolescent psychiatric outpatients. Each of the 77 stressors is presented with an axis IV severity rating ranging from 1.7 to 5.7. The severity ratings are the mean ratings of 54 adolescent clinicians on the severity of each of the 77 stressors using DSM-III criteria.

The reliability of this procedure was investigated by examining interrater reliability. This author first reviewed all cases and assigned an axis IV rating and then a second rater, a psychology graduate student reviewed the same material and assigned an axis IV rating. Both raters employed the RAU scale as a guide. The correlation coefficient between the two sets of ratings was .723, $p<.001$. The percent exact agreement was 53% and the percent close agreement was 87%. All disagreements were resolved in favour of the second rater in order to minimize any bias that might have occurred by having the same rater for both diagnoses and stressors (for comparable results see Rey, Plapp, Stewart, Richards & Bashir, 1987).

In addition to the severity rating, a second stressor score was calculated by adding up the number of psychosocial stressors occurring over the last year. Only those stressors that could be classified by the RAU scale were counted. Again the RAU provided only a guide because the stressors occurring in the present sample only approximated the items on the RAU scale. Interrater reliability was found to be acceptable for the number of psychosocial stressors, $r(72)=.79$, $p<.001$.

Procedure

Informed consent was obtained from the adolescent and from at least one parent. Consent information differed for subjects who were not interested in group therapy. These subjects were simply informed that they would undergo two and half hours of assessment. Subjects interested in group therapy were informed of all the details of the therapy program as well as all the information involving assessment. They were also informed that similar follow up assessments would be conducted. If the patient was not referred from a psychiatrist, then the patient was required to undergo a brief interview with the chief psychiatrist at the outpatient department. After obtaining the consent, I interviewed the parent with the KSADS while the adolescent completed the CDI, the CBQC, the Shipley-Hartfort vocabulary

test and other self-report measures not related to the present study in a separate room. A research assistant administered and scored these self-report measures and was instructed not to reveal the scores to either this author or the chief psychiatrist until completion of this study. Following the interview with the parent, I then interviewed the adolescent with the KSADS.

Results

Preliminary Analyses

Internal Consistency of the CBQC

The internal consistency of the responses from all 74 patients was investigated using the KR-20 formula. The internal consistency of the distortion (DD) scale was acceptable (alpha=.82). The internal consistency for the depressed-nondistorted (DN) scale was unacceptably low (alpha=.42) and for the nondepressed-nondistorted (NN) scale, was acceptable (alpha=.80). These results show a substantial increase in reliability for the distortion and NN scales from the previous version of the CBQC (see Haley et al., 1985).

To determine the response pattern to the three scales of the CBQC, Pearson correlations were computed between the scales. Those who chose more distorted responses did not necessarily choose any more DN responses ($r(72) = -.06$, ns) but did choose significantly less NN responses ($r(72) = -.81$, $p = .000$). Those who chose more NN responses were significantly less likely to chose a depressed response ($r(72) = -.53$, $p=000$). Thus, adolescents who were less

inclined to chose a N[illegible] response were more likely to chose a distorted or a DN response but not necessarily both.

Internal Consistency of KSADS Depression Scale

Internal consistency estimates were calculated for the 12 symptom ratings of the KSADS major depression syndrome using all 74 adolescents. These calculations were done because the version of the KSADS, used in this study, was the 1986 version and no data had been reported for this version. For the current episode ratings, the alpha coefficient was .80, and for the last-week ratings, the alpha coefficient was .79. These estimates are slightly higher than those reported by authors of the KSADS for the 12 item depression scale but are nevertheless comparable to the 1978 version, (alpha=.72; Chambers et al., 1985).

Diagnoses

Twenty-three adolescents met the RDC criteria for a definite diagnosis of endogenous (ED) major depression and 25 adolescents met the RDC criteria for definite diagnosis of nonendogenous (NE) major depression. Psychotic features were present in 22% of the ED group and in (20%) of the NE group. Two subjects in the NE group (8%) and one subject in the ED group (4%) had a history of mania.

The Axis I DSM-III-R diagnoses of the remaining 26 subjects were as follows: conduct disorder (n=8), dysthymic disorder (n=6), separation anxiety (n=3), oppositional defiant disorder (n=2), adjustment disorder with depressed mood (n=3), Tic disorder, attention deficit disorder, overanxious disorder and no axis I disorder, each n=1. These subjects formed the psychiatric control group.

The distribution of concurrent diagnoses was examined across the three groups. Eight percent of the ED group and 4% of the NE had a DSM-III-R concurrent diagnosis of conduct disorder. These percentages are lower than those reported for inpatient samples of adolescents with major depression (Haley, Fine & Marriage, 1988). Substance abuse was diagnosed in 22% of the ED group, in 20% of NE group and in 8% of the control group.

Statistical Analyses

Oneway analyses of variance were conducted on all continuous variables. In all analyses, if the overall F-test was not significant, group differences were not examined. The $Tukey_a$ or the Honestly Significant Difference (HSD) test at the .05 level was used to determine group differences. The hypotheses were examined by the Bonferroni t-test procedure regardless of the significance of the overall F.

For the categorical variables, chi-square analyses were performed. If the overall chi-square was not significant at the .05 level, further testing was halted. To control for type I errors, the alpha level was set at .01 for multiple group comparisons using the chi-square.

Patient Characteristics

Demographics. Table 5 presents the sample characteristics related to nonclinical individual differences for the three groups. As can be seen, there was good comparability across groups on most of these demographics. The Blishen Socioeconomic Status index (SES; Blishen & McRoberts, 1976) indicated that the average adolescent in each group was from a middle class family. Analyses of variance conducted on the continuous variables revealed no significant overall differences on age, ses, grade level or verbal IQ. Chi-Square analyses indicated no significant overall differences on race or living status. However, there was a significant overall difference for sex, $X^2(2, N=74)=14.76$, $p=.0006$. Multiple comparisons indicated there was no significant difference in sex ratio between the ED and NE groups, $X^2(1, N=48)=2.44$, ns. Compared to the control group, the NE group tended to have a different sex ratio, but this difference was not significant at the preset alpha level, $X^2(1, N=51)=5.68$, $p=.02$. The difference in

sex ratio, however, was accounted for by the ED group, which had significantly more girls than the control group, $X^2(1, N=49)=13.84$, $p=.0002$.

Table 5
Demographic Characteristics of the Diagnostic Groups

Measure	ED MDD n=23	NE MDD n=25	Psychiatric Controls n=26
Age			
M	15.3	15.1	14.8
SD	01.5	01.5	01.4
Sex			
Females	20	17	09
Males	03	08	17
Race			
Caucasian	21	18	21
Oriental	01	05	01
Native	01	01	03
Other	00	01	01
SES			
M	42.7	49.3	41.6
SD	22.4	16.9	20.7
Shipley-Hartford Verbal IQ			
M	14.5	15.7	14.9
SD	02.2	02.1	02.1
Grade			
M	09.5	09.4	09.0
SD	01.5	01.3	01.4
Living Status			
2 Bio Parents	10	11	03
1 Bio Parent	11	10	14
Adopted	00	01	00
Foster Home	01	00	04
Group Home	01	03	05

Note. ED=endogenous; NE=nonendogenous; MDD=major depressive disorder; SES=socioeconomic status.

Clinical Characteristics. Table 6 presents the characteristics of the groups on a number of clinical variables. Analyses of variance on the continuous variables revealed no group effects for the severity of psychosocial stress as rated on the DSM-III-R Axis IV, the number of psychosocial stressors present in the last year, the age at which the first signs of the disorder occurred, and the age of onset for the current disorder. There was a group effect for episode duration, $F(2, 71)=8.39$, $p=.0005$. Multiple comparison tests indicated that the control group had significantly longer episodes than either the ED group or the NE group (Tukey-HSD tests, $ps<.05$). This finding is not surprising given the fact that those with major depression, by definition, had a definite onset to the episode in the last year whereas those in control group had disorders for which there was no worsening of symptoms or no two month symptom-free period in the last year. Chi-Square analyses on the categorical variables showed a significant group effect for suicide attempt within the current episode, $X^2(2, N=74)=13.95$, $p=.0009$. Group comparison analyses revealed that 61% of the ED group attempted suicide within the current episode compared to 15% of the control group, $X^2(1, N=49)=10.86$, $p=.001$ and compared to 23% of the NE group, $X^2(1, N=48)=8.36$, $p=.004$. The NE group and the control group did not differ significantly in the number with a suicide attempt, $X^2(1, N=51)=.66$, ns.

Table 6
Clinical Characteristics of the Diagnostic Groups

Measure	ED MDD n=23	NE MDD n=25	Psychiatric Controls n=26
DSM-III-R			
Stressor rating			
M	3.96	3.84	3.74
SD	.53	.78	.71
Number of Stressors			
M	3.35	3.80	3.24
SD	1.11	1.65	1.64
Age of Onset of Current Episode			
M	14.6	14.5	13.6
SD	1.6	1.9	1.4
Duration of Current Episode in weeks			
M	14.4	17.5	33.9
SD	8.0	11.8	27.2
Age of 1st Signs of Disorder			
M	12.3	12.5	11.7
SD	2.6	3.3	3.0
Suicide Attempt	14	5	4
Patient Status[a]			
Inpatient	2	5	3
Outpatient	25	21	18
Hospitalization in current Episode	7	10	4
No previous Psych Contact	17	12	10
Medications			
Antidepressants	5	3	2
Antipsychotic	1	2	1
Other	2	2	2
Family History			
Depression	9	9	5
Bipolar	1	4	0
Psychotic	0	4	0

Note. ED=endogenous; NED=nonendogenous; MDD=major depressive disorder.
[a] Patient status at the time of assessment.

Depression Measures. Table 7 presents the means and standard deviations for the depression measures. There was a significant group effect for the KSADS depression scale for the current episode. This result is as expected because group classification was based on current episode ratings. Group contrasts revealed that the ED group was significantly more depressed on KSADS-CE depression scale than the NE group, Tukey-HSD, $p<.05$. Compared to the psychiatric controls, both the ED and NE groups were significantly more depressed on the KSADS-CE depression scale, (Tukey-HSD tests, $ps<.05$). The means for the ED and NE groups on the 12-item KSADS-CE depression scale are very similar to those reported for outpatient adolescents employing the same diagnostic grouping (see Goetz et al., 1987). At the time of assessment, however, the depressed groups did not differ significantly in severity of depression as indicated by the KSADS-Last Week depression scores (Tukey-HSD, $p=ns$). Both depressed groups were significantly more depressed at the time of assessment on the KSADS-LW depression scale than the control group (Tukey-HSD tests, $ps<.05$).

Although there was a tendency for the ED group to be more depressed on the CDI compared to the NE group, this difference did not meet Tukey-HSD criteria for significance. Compared to the control group, both the ED and NE groups

were significantly more depressed on the CDI, (Tukey-HSD tests, ps<.05).

Table 7
Group Means for the Depression Measures

Measure	ED MDD	NED MDD	Psychiatric Controls	F-Value
KSADS depression				
Current Episode				
M	45.04	39.76	23.96	76.45*
SD	5.46	7.08	6.09	
Last Week				
M	37.68	34.28	21.62	55.48*
SD	6.96	4.06	5.71	
CDI				
M	21.65	18.04	8.00	23.34*
SD	6.95	8.36	6.48	

Note. ED=endogenous, NED=nonendogenous, MDD=major depressive disorder, KSADS=Kiddie Schedule for Affective Disorders and Schizophrenia, CDI=Children's Depression Inventory.
*P=.0000.

Hypotheses Testing

Cognitive Distortion

Table 8 presents the means for the distortion scale from the revised CBQC. Because the distortion scale violated the homogeniety of variance assumption (Cochranes C=.53, p=.02 and Bartlett-Box F=13.56, p=.000), group comparisons

were analyzed using separate variance estimates. As predicted from hypothesis one, both the ED group and the NE group evinced significantly more distortions on the CBQC compared to the control group, Bonferroni tests, $t(27.2) = -6.15$, $p<.001$ and $t(28.8)=4.45$, $p<.001$, respectively. The ED and NE depressed groups did not differ significantly in terms of distortion, $t(45.9)=-.96$, p=ns. A square-root transform of the distortion scale was performed and this transformation was successful in equalizing the variances between the groups as indicated by tests for homogeniety of variance (Cochranes $C=.46$, p=ns and Bartlett-Box $F=1.56$, p=ns). Bonferroni tests performed on the transformed distortion scores produced similar results to those using the untransformed scores with ED and NE groups scoring significantly higher than the control group ($t(71)=-5.89$, $p<.001$ and $t(71)=4.58$, $p<.001$, respectively).

Table 8
Group Means for the Cognitive Measures

Measure	ED MDD	NED MDD	Psychiatric Controls	F-Value (DF=2,71)
CBQC Scales				
Distortion				
M	5.83	4.80	1.11	16.01**
SD	3.47	3.95	1.27	
Nondepressed-Nondistorted				
M	6.96	7.84	13.23	25.36**
SD	3.86	3.85	2.77	
Difference Score				
M	1.13	3.04	12.12	18.87**
SD	7.10	6.76	3.74	
Depressed-Nondistorted				
M	7.21	7.32	5.69	3.42*
SD	1.85	3.17	2.18	
KSADS				
Hopelessness/Worthlessness				
M	6.91	6.70	3.71	14.50**
SD	2.47	2.28	1.82	

Note. ED=endogenous, NED=nonendogenous, MDD=major depressive disorder, KSADS=Kiddie Schedule for Affective Disorders and Schizophrenia, CBQC=Cognitive Bias Questionnaire for Children.
*P=.038; **P=.0000.

Self-derogatory vs Self-serving Bias

The second hypothesis predicted that depressed adolescents would show more distortions relative to positive

thinking on the CBQC. This hypothesis predicted that the depressed groups would show a mean negative score on the CBQC difference score. Contrary to expectation, the means of the depressed groups on the CBQC difference score tended to be on the positive side (see table 4). The control group, however, demonstrated a clear positive or self-serving bias on the CBQC that was significantly greater that the ED group, (Tukey-HSD, $p<.05$) and the NE group, (Tukey-HSD, $P<.05$). However, homogeniety of variance tests indicated that the difference score violated the homogeniety of variance assumption (Bartlett Box $F=5.13$, $p=.006$). Attempts at normalizing this scale by various transforms were unsuccessful. Therefore a categorical analysis was performed on the number of cases in each group who obtained a negative value (≤ -1) on the CBQC difference score. This analysis revealed that 48% of the ED group, 32% of the NE group and 0% of the control group exhibited a self-derogatory or 'inverse' bias. Chi-Square analysis indicated a significant group effect for these ratios, $X^2(2, N=74)=15.79$, $P=.0004$. The ED group tended to have more cases exhibiting the self-derogatory bias than the NE group, $X^2(1, n=48)=2.97$, $p=.08$, and to have significantly more cases than the controls, $X^2(1, n=49)=16.03$, $p=.0001$. The difference between the NE group and the control group was also significant, $X^2(1, n=51)=7.07$, $p=.008$. Thus, the

prediction was supported only for a subgroup of those with major depression.

Other Cognitive Scales

A similar pattern of results was found for the NN scale of the CBQC and the HW scale of the SADS as was found for the distortion scale. The control group was significantly more positive in their thinking style, as evidenced by their higher NN scores, compared to the ED group (Tukey-HSD, $p<.05$) and the NE group (Tukey-HSD, $p<.05$). The depressed groups were not significantly different in positive thinking. Compared to the control group, both the ED and NE groups evinced significantly more hopelessness and worthlessness on the HW scale, (Tukey-HSD tests, $p<.05$). The depressed groups were not significantly different on the HW scale. Although the depressed groups tended to exhibit more depressed-nondistorted scores on the CBQC than the control group, the Tukey-HSD procedure indicated that these differences were nonsignificant (see table 8).

Supplementary Analyses

Separate Sex Analyses

Because the groups differed with respect to the sex distribution, separate analyses were calculated for females and for males. In the first set of analyses, the 9 females from the control group were compared to the females from the depressed groups. Preliminary analyses indicated that there were no significant differences among the groups for any demographic variable, including age, verbal IQ, stress rating, SES or grade level. Nevertheless, compared to the control group, both the ED and the NE groups were significantly more depressed as measured by the CDI and the KSADS depression scale, exhibited significantly more distortions and less positive thinking on the CBQC, and experienced significantly more hopelessness and worthlessness as measured by the HW scale, (Tukey-HSD tests, $ps<.05$). This pattern of results is identical to that with the full sample (see table 9).

To obtain a large enough sample of depressed boys, the ED and NE groups were collapsed into one group of boys with major depression ($n=11$) and compared to the control group of boys ($n=17$). There was no significant differences on any demographic variable including stress, SES, Verbal IQ and

age. From table 10, it can be seen that the same pattern of results emerges as that for the full sample although at a much reduced significance level.

Table 9
Group Means for the Depression and Cognitive Measures for Females

Measure	ED MDD (n=20)	NED MDD (n=17)	Psychiatric Controls (n=9)	F-Value (DF=2,43)
KSADS depression-LW				
M	36.50	34.29	21.33	19.63**
SD	8.02	4.92	4.92	
CDI				
M	21.85	19.00	10.00	8.03**
SD	7.45	8.34	5.00	
CBQC Scales Distortion[a]				
M	5.95	6.05	1.89	5.29*
SD	3.44	3.44	1.45	
Nondepressed-Nondistorted				
M	6.40	6.29	12.00	11.71**
SD	3.33	2.57	3.67	
Difference Score				
M	.45	.23	10.11	9.75**
SD	6.62	5.56	4.91	
Depressed-Nondistorted				
M	7.65	7.59	6.22	.93
SD	4.42	3.79	2.72	
KSADS Hopelessness/ Worthlessness				
M	6.70	7.82	4.67	6.28*
SD	2.45	1.59	2.40	

Note. CDI=Children's Depression Inventory, ED=endogenous, NED=nonendogenous, MDD=major depressive disorder, KSADS=Kiddie Schedule for Affective Disorders and Schizophrenia, CBQC=Cognitive Bias Questionnaire for Children. *P<.01; **P<.001. [a]p-value for transform, p=.004.

Table 10
Group Means for the Depression and Cognitive Measures for Males

Measure	Major Depression (n=11)	Psychiatric Controls (n=17)	(DF)	T-value
KSADS-LW Depression				
M	35.45	21.76	26	5.61***
SD	6.42	6.23		
CDI				
M	17.18	6.91	26	3.66***
SD	7.52	7.04		
CBQC Scales				
Distortion[a]				
M	3.00	.71	11.37	2.32*
SD	3.00	.99		
Nondepressed-Nondistorted				
M	11.00	13.88	14.08	-2.44*
SD	3.57	2.00		
Difference Score				
M	8.09	13.18	12.03	-2.50*
SD	6.42	2.53		
Depressed-Nondistorted				
M	6.09	5.41	26	.97
SD	1.70	1.87		
KSADS				
Hopelessness/ Worthlessness				
M	6.73	3.29	11.61	4.07**
SD	2.45	1.02		

Note. CDI=Children's Depression Inventory, KSADS=Kiddie Schedule for Affective Disorders and Schizophrenia, CBQC=Cognitive Bias Questionnaire for Children. *P<.05; **P<.01;***p<.001.[a] for square root transform, t(26)=2.62, p=.01.

Stress and Cognitive Distortion

The relation between stress and distortion was further examined by dividing all subjects with a diagnosis of major depression into high and low stress groups. The impetus for this approach comes from the two studies mentioned in the introduction which found that in mildly depressed college students, low stress was associated with high distortion (Hammen, 1978; Michael & Funbiki, 1985). Depressed subjects in the present study were divided into two groups based on the mean split of 4 psychosocial stressors. Those who had 3 or less stressors, formed the low stress group and those that had 4 or more stressors formed the high stress group. This cut-off divided the depressed subjects equally. Six demographic variables and 6 clinical variables including the cognitive distortion scale were examined. To control for type I errors, the Bonferroni correction was applied to each of these families, (.05/6) which indicated that an alpha level of .008 was acceptable.

The means of the demographic and clinical variables are presented in table 11. T-test analyses revealed that the stress groups did not differ significantly at the .05 level on age, sex, SES, grade, IQ but the high stress subjects obtained a significantly higher severity of stress rating than the low stress subjects. In terms of clinical

variables, the stress groups did not differ significantly on any of the depression measures nor on the HW scale. However, the low stress group evinced significantly more cognitive distortions than the high stress group ($p=.003$). These results provide clear evidence that the distortion scale is not measuring life stress and even suggests that it is measuring true distortion of life events.

Depressed Control Subjects

Due to the overlap in criteria between major depression and dysthymia, fluctuations in one or two symptoms could shift the diagnosis from major to minor or dysthymic depression or vice versa. It also could be argued that because dysthymic disorder subjects are at risk for major depression (see Kovacs et al., 1984), at least some of these subjects should exhibit distortions. Could the inclusion of depressed subjects in the control group have overestimated the differences between the depressed subjects and the control subjects on the distortion scale? At issue is whether or not the control group contains non-distorting depressed subjects who by their absence in the NE group bias the difference in favour of the NE group? To explore this possibility, the 6 subjects with a diagnosis of dysthymia plus 2 of the 3 subjects with adjustment disorder who also met the RDC criteria for minor depression were combined with

the nonendogenous group. It was expected that if the differences were inflated, the addition of these 8 subjects to the NE group would result in a substantial decrease in the t-values and significance levels. Analysis of variance conducted on the square-root transformed scores of the DD scale indicated a significant effect for group, $F(2, 71)=19.77$, $p=.0000$. Multiple contrasts showed that the combined NE group ($n=33$) endorsed significantly more distortions on the CBQC than the control group ($n=18$), means=4.12 and .722 respectively, $t(71)=-4.72$, $p=.000$. This t-value is slightly higher than the original t-value ($t=4.58$) but the significance level remains unchanged. The possibility that the significant differences between control and depressed subjects on the distortion scale are overestimated by biased group assignment is rejected.

Table 11
Group Means for High and Low Stress Subjects with Major Depression

Measure	High Stress (n=24)	Low Stress (n=24)	(DF)	T-value
Age				
M	15.20	15.25	46	-.49
SD	1.62	1.32		
SES				
M	42.99	49.66	46	1.17
SD	21.77	17.18		
Verbal IQ				
M	14.71	15.58	46	1.25
SD	1.94	2.47		
Grade				
M	9.37	9.50	46	.31
SD	1.34	1.44		
DSM-III Stress rating				
M	4.23	3.56	46	-4.05***
SD	.55	.61		
KSADS-CE				
M	43.83	40.75	46	-1.59
SD	1.87	2.59		
KSADS-LW				
M	36.08	34.87	46	-.65
SD	6.52	6.26		
Hopeless/worthless				
M	6.87	7.29	46	.64
SD	1.87	2.59		
CDI				
M	17.92	21.62	46	1.67
SD	7.51	7.51		
Distortion				
M	3.75	6.83	46	3.12***
SD	2.75	3.96		

Note. CDI=Children's Depression Inventory, KSADS=Kiddie Schedule for Affective Disorders and Schizophrenia, CBQC=Cognitive Bias Questionnaire for Children.
*P≤.05; **P≤.01;***p<.008

Discussion

The results of study 1 support the hypothesis that cognitive distortion is a marker for major depression. As predicted, the distortion scale from the CBQC reliably distinguished those adolescents with major depression from those with other psychiatric diagnoses. These results are consistent with those reported for the first version of the CBQC (Haley et al., 1985). The present study extends previous work by Haley et al. by demonstrating that the differences between major depression and others were not due to the presence of criterion contamination or other methodological problems as outlined in the introduction. Furthermore, differences between groups could not be accounted for by such variables as age, sex, IQ, stress and other demographic variables.

The second hypothesis received partial support. For a minority (40%) of adolescents with major depression, distorted responses were more prevalent than positive responses. Although lacking in sensitivity in describing most of those with major depression, a negative difference score is highly specific to the disorder. A negative difference score was not found in any of the psychiatric controls which is all the more significant when one considers that 35% of the subjects in the control group had

a non-major depressive disorder. Thus, a negative difference score appears to be a rather specific marker for major depression. A more sensitive marker, appears to be the degree to which the adolescent is positively biased. Thus a competing hypothesis to the negative bias theory may be that the depressed children are less positively biased in their thinking style compared to nondepressed children.

The finding that depressed adolescents also chose more nondepressed-nondistorted options on the CBQC deserves some comment. This result may have been due to the inclusion the depressed-nondistorted (DN) option on the CBQC. Recall that the DN scale was primarily designed to control for negative affective. Depressed adolescents tended to choose this option if they did not choose the distorted option whereas the nondepressed adolescents generally chose the positive options on the NN scale. A low NN score can be interpreted to mean a general negative bias or alternatively, a negative response style. The most useful scores derived from the CBQC appear to be the distortion scale and the difference score because these scales a more easily interpreted as reflected negative thinking rather than response style.

With regard to hopelessness and worthlessness, the depressed patients were rated at interview as experiencing more of these negative cognitions than controls. This

finding is consistent with Beck's cognitive theory which posits that when depressed, patients show a global negative thinking style that includes, a negative view of the self and the future. Recall that group assignment in the present study was made without regard to the presence or absence of these two symptoms. This procedure demonstrates that differences between the groups on measures of negative thinking were not due in any way to the inclusion of negative thinking as part of the initial diagnostic assignment. Despite this methodological clarification, the issue remains as to whether, negative thinking is simply a associated feature of major depression or exhibits more trait-like properties. This issue will be examined in the second study.

No significant differences in distortion, negative bias, hopelessness and worthlessness were found between the endogenous and nonendogenous groups. This finding is consistent with a number of studies with adult patients which have reported no significant differences between endogenous and nonendogenous patients on measures of dysfunctional thinking, negative attributions and negative thinking (Eaves & Rush, 1984; Giles & Rush, 1982; Zimmerman & Coryell, 1986). Because there is evidence that endogenous depression can be discriminated by a biological marker (Carroll et al., 1981), researchers had hoped that negative

thinking would predominate in the nonendogenous depression, thereby supporting the traditional dualist position that depressions can be divided into those that are psychologically based or into those that are constitutionally based (Zimmerman & Coryell, 1986). However, it appears from other evidence that depressed patients with elevated cognitive distortions form a separate subgroup independent of the endogenous or nonendogenous subtyping (Norman, Miller & Dow, 1988).

It is noteworthy that the present data do not support the hypothesis that cognitive distortion is a reflection of actual life stress (Coyne & Gotlib, 1986; Krantz, 1985). Neither the DSM-III ratings of psychosocial stress nor the number of psychosocial stressors could distinguish those with and without major depression. All three groups of adolescents experienced moderate levels of stress over the last year, yet only the depressed groups exhibited distortions. Furthermore, depressed adolescents who had low levels of stress actually demonstrated significantly higher distortions than depressed adolescents who had high levels of stress. This finding suggests that these depressed children were truly distorting life events. As previously noted, these findings are consistent with studies employing the Cognitive Bias Questionnaire in adult samples of depressed college students (Hammen, 1978; Michael & Funbiki,

1985). The present findings extend previous studies two fold, first, it is the first demonstration of a negative relationship between distortion and stress in a clinical sample of depressed patients and second, it is the first demonstration of this phenomenon with depressed adolescents. The evidence from this study places Coyne and Gotlib's (1986) view on the importance of stress in accounting for depression in serious doubt. The evidence clearly shows that distortion not stress distinguishes depressed from nondepressed individuals. In addition, Coyne and Gotlib (1986) and Barnett and Gotlib (1988) have assumed that a negative relationship between cognition and stress is impossible because, as they argue, this interaction would mask a main effect for cognitions in a sufficiently large sample. Clearly this assumption also is not supported. It might be asked how the present results accord with the diathesis-stress model proposed by Beck (1987). This question cannot be addressed fully by the present data, other than to state that all subjects had stress present and thus a necessary condition was fulfilled in the model. However, the evidence suggests that the relationship is not a straight forward one and that the presence of a negative relationship between cognition and stress cannot ipso facto be ruled out.

Finally, because the sample was a select one, it might be prudent to examine how comparable this sample is with other outpatient samples. Fortunately, two studies are available which not only concern outpatient adolescent subjects but employ the KSADS as a diagnostic instrument (Mitchell et al., 1988; Ryan et al., 1988). These two studies allow comparisons of the MDD group only and basically shed light on how generalizable the results are to other MDD samples or whether the present sample of MDD subjects are in some way an anomaly. The present study found that 48% of those with major depression were positive for endogenous subtype. This rate is consistent with the 50% rate reported for outpatient prepubertal children (Chambers et al., 1982) and outpatient adolescents (Mitchell et al., 1988; Ryan et al., 1988). Endogenous patients were also significantly more depressed, significantly more likely to have made a suicide attempt but not any more likely to differ significantly on the frequency of psychotic features or conduct symptoms. Similarly, both Mitchell et al. and Ryan et al. have reported significantly higher KSADS ratings in the endogenous group and Ryan et al. have noted more suicide lethality for the endogenous group. The 21% rate found in this study for the presence of psychotic symptoms in MDD is also comparable to the 27% rate reported by Mitchell et al. and the 18% reported by Ryan et al. In the present study, it was found that psychotic features were no

more likely to be associated with endogenous than nonendogenous depression. While this finding might seem unusual, it is not so with child and adolescent samples. Chambers et al., reported that 28% of the nonendogenous subjects exhibited psychotic features. In a component analysis of KSADS symptoms, Ryan et al., reported that psychotic features were more likely to be associated with anxiety symptoms than endogenous symptoms. The 39% rate found for suicide attempts in the MDD subjects is identical to that reported by Mitchell et al. for their adolescent MDD group and close the 34% rate reported by Ryan et al. The only obvious way in which the present sample of adolescent MDD differs from the other samples is in the high female to male ratio (37:10) which is considerably higher than that reported by Ryan et al. (30:32) and Mitchell et al. (29:21). This preponderance of females probably accounts for low prevalence of associated conduct disorder, 6% in the present study compared to the 11% reported in Ryan et al.,'s study and 14% in Mitchell et al.'s study. Overall, the sample of MDD subjects in the present study is remarkably consistent with other samples suggesting that the diagnosis of MDD and depressive subtypes is accurate.

In summary, the revised CBQC was found to be a reliable and valid instrument for assessing cognitive markers in adolescent major depression. The CBQC can now be used to

explore whether cognitive distortion is a state or trait marker for major depression.

STUDY 2: TRAIT VS. STATE MARKERS

Hypotheses

The second aspect of the present study examined the trait versus state issue of cognitive markers for major depression. Depressed adolescents were tested at two time periods, while actively depressed and when remitted from depression. Because the CBQC is a mix of trait and state elements of negative schemas, it was predicted that even though depressed patients would show a decrease in distortion following remission of depressive symptoms, they would continue to show more distortion compared to nondepressed controls. It was further hypothesized that measures of the state aspects of depression including hopelessness and worthlessness will distinguish the depressed from the control subjects during the depressed phase, but will normalize at the remission phase. These predictions are in accordance with the cognitive model of depression (Beck, 1967) which posits that measures which tap into the silent assumptions or the negative schemas are more resistant to change (see Miller & Norman, 1986) than measures which tap into the automatic negative thoughts of depressed patients (see Eaves & Rush, 1984).

Method

Subjects

From the original sample of 74 subjects used in study 1, 56 subjects (36 with a diagnosis of major depression and 20 with nondepressed diagnoses) had elected to attend a group therapy program offered by the outpatient department. Subjects in study 2 were a subsample of these 56 adolescents who met the following criteria: (1) all depressed subjects were required to met the RDC criteria for major depression on the KSADS-LW as well as on the KSADS-CE depression ratings, (2) all subjects were required to attend at least one group therapy session and (3) all subjects were required to complete a second assessment. There were 44 subjects who met these criteria, including 15 boys and 28 girls (mean age=15.25). Thirty were diagnosed with major depression and 14 were diagnosed with other nondepressed disorders.

Remission Criteria

Measurements were obtained on all depressed subjects at two time periods, when symptomatic, Time 1 (T1) and later when remitted, Time 2 (T2). A patient was considered

remitted from major depression if he or she obtained a rating of 2 or less on the KSADS depressed mood item and the anhedonia item. A score of 3 on the depressed mood item ("mild depression, e.g., often experiences dysphoric mood at least 3 times a week for more than 3 hours") or greater or on the anhedonia item ("several activities less interesting or pleasurable or boredom over 50% of the time") was defined as unremitted from major depression. These criteria for remission are identical to the criteria used by researchers to define responders versus nonresponders to imipramine treatment in studies with prepubertal major depression (Puig-Antich et al., 1987) and adolescent major depression (Ryan et al., 1986). In addition, a remitted patient from major depression could not have a CDI score in excess of 16. This score represents 1 standard deviation from the mean of normal Canadian children and adolescents (Kovacs, 1981). At time 2, thirteen depressed patients met the remission criteria while 17 depressed patients remained unremitted. These unremitted depressed subjects were included as an additional control group. The psychiatric controls included the following DSM-III-R diagnoses: dysthymic/avoidant disorder, $\underline{n}$=4, conduct disorder, $\underline{n}$=3, adjustment disorder with depressed mood, $\underline{n}$=2, overanxious disorder, tic disorder, separation anxiety disorder, oppositional defiant disorder, and no axis I disorder, each $\underline{n}$=1.

Measures and Procedure

Time 1 data included all the data from the measures completed at the pretest assessment prior to group therapy treatment. Time 2 data included data obtained from either the posttest at 15 weeks or the 1 year follow-up assessment whichever was the most recent assessment. At the time of this writing, 15 subjects had completed the follow-up, including 9 depressed and 6 control subjects. At posttest, only 9 of 30 subjects with major depression met the remission criteria with 21 remaining unremitted. Of the 9 depressed subjects who completed the follow-up assessment, 4 met the remission criteria who had being unremitted at posttest period, 4 remained unremitted from the initial episode and one remained remitted from the posttest period. Thus, the total number of remitted patients totalled 13. To control for time, only the most recent test scores were used in the T2 data including all the test data from the 15 subjects who had completed the follow-up. The average time between T1 and T2 for all subjects was 29.80 weeks.

In the pretest, subjects were assessed as described in study 1. Upon completion of their group therapy treatment, subjects and at least one parent were interviewed a second time with the KSADS-LW depression scale to determine changes in depression level. They were not interviewed with the

full KSADS interview. This interview also included ratings on the KSADS hopelessness and worthlessness scales which were again combined to form the HW scale as described in study 1. Subjects who had dropped out of the group were also requested to complete a second assessment at same time as those who completed treatment. All subjects completed at posttest, the Children's Depression Inventory (CDI) and the Cognitive Bias Questionnaire for Children (CBQC). Interviewers were unaware of the results of the CDI and the CBQC. As part of an ongoing study, follow-up assessment data, including the full KSADS interview, the CDI and the CBQC, were obtained from the first 15 of 44 subjects who had complete an 1 year follow-up.

Results

Preliminary Analyses

Patient Characteristics

Table 12 presents a selection of demographic and clinical characteristics of the unremitted depressed group, the remitted depressed group and psychiatric control group. Oneway analyses of variance revealed that the three groups did not differ in age, socioeconomic status, IQ, and grade level or time between testing sessions (T1 to T2). There

was a significant overall effect for sex, X^2 (2, N=44)=9.19, p=.01. Multiple comparisons showed no significant differences in sex ratio between the remitted group and the control group, X^2(1, N=27)=1.80. p=ns., and between the remitted group and the unremitted group, X^2(1, N=30)=2.03, p=.09. However, there was a significance difference in sex ratio between the unremitted group and the control group, X^2(1, N=31)=5.82, p<.02. Overall, the remitted group is fairly well matched with the nondepressed control group on all of the initial assessment demographic characteristics.

In terms of clinical characteristics, 53% (n=9) of the unremitted group were diagnosed with a endogenous subtype compared to 23% (n=3) of the remitted group, but this difference was not significant, X^2(1, N=30)=2.73, p=.09. Psychotic features were present in 29% (n=5) of the unremitted and in 15% (n=2) of the remitted depressed subjects. Of the subjects in the remitted group who presented with psychotic features, one was treated with an neuroleptic and the other was treated with a combination of lithium and a tricyclic. Of the subjects with psychotic features from the unremitted group, one was treated with a tricyclic and the rest were not treated with any medication. Antidepressant medication was relatively rare in both depressed groups. One subject in the control group received an neuroleptic for a tic disorder. The depressed groups did

not differ significantly in episode duration, in depression severity for the current episode or in the number with a suicide attempt within the current episode. No subject received cognitive therapy between T1 and T2.

Table 12
Sample Characteristics of Subject Groups

Measure	Unremitted (n=17)	Remitted (n=13)	Psychiatric Controls (n=14)
Age			
M	15.08	15.35	15.38
SD	1.12	1.69	1.45
Sex			
Females	15	8	5
Males	2	5	9
SES			
M	43.98	50.27	44.92
SD	21.94	15.27	17.8
Shipley-Hartford Verbal IQ			
M	15.32	15.61	15.07
SD	2.22	2.11	2.13
Grade			
M	9.41	9.69	9.43
SD	1.37	1.25	1.28
DSM-III-R Stressor rating			
M	3.71	3.98	3.58
SD	.73	.82	.81
Number of Stressors			
M	3.41	3.62	3.71
SD	1.32	.87	1.86
Duration of Current Episode in weeks			
M	15.7	20.1	
SD	9.4	11.7	
Time between T1 and T2 in weeks			
M	24.59	29.31	36.57
SD	20.64	18.61	22.18
Suicide Attempt	7	4	1
KSADS-CE			
M	41.59	39.84	23.21
SD	5.89	4.96	4.59

Note. SES=Socioeconomic Status.

Statistical Analyses

An analysis of variance (ANOVA) with groups as a between-subjects factor and Time as a within-subjects factor was performed on each of the CBQC scales and the HW scale and on the depression measures. Whenever the ANOVA yielded a significant group x time interaction, I followed the recommended formulas by Howell, 1982 to compute the degrees of freedom for the simple main effect of Group. The degrees of freedom will vary as a function of the values of the $MS_{\text{subjects within groups}}$ and of the $MS_{\text{time X subjects within groups}}$ (see appendices A to F). Following a significant simple main effect for T1 or T2, Tukey_a (Honestly Significant Difference) tests were performed to determine which comparison was significant. If the interaction effect was not significant, significant main effects were examined by the Tukey_a procedure on the average of T1 and T2 data. Only two pairwise comparisons were of interest, the remitted group versus the control group and the remitted group versus the unremitted group.

The hypothesis for the HW scale asserts that there will be no significant differences between remitted and control subjects at T2. However, this constitutes predicting the null hypothesis which is not statistically defensible. Therefore, the analysis for the HW scale needed a different

approach involving the possibility of rejecting a false null hypothesis. If the probability of detecting a significant difference between the means on the HW scale by a t-test is set to 80% and the test still fails to reject the null hypothesis, then one can be reasonably sure that a false null hypothesis is not present. From study one, it was observed that the depressed subjects differed from the control subjects by slightly more than a standard deviation on the HW scale. A meaningful difference between the remitted and the control subjects at T2 might be set at .50 of a standard deviation. However, to detect this difference with power set to .80, and the alpha level set to .05, requires a sample size of 126 subjects (Howell, 1982). In order to estimate the possibility of overlooking true differences in this study's small sample size, a t-test was calculated between the remitted and control groups and evaluated on an alpha level set to .20. If the difference are significant at this level, then there may be grounds for rejecting a false null hypothesis.

Table 13
Group Means for Depression Measures at Time 1 and Time 2

Measure	Unremitted (n=17)	Remitted (n=13)	Psychiatric Controls (n=14)
KSADS-D:T1			
M	36.17	35.69	22.07
SD	5.70	4.78	4.73
KSADS-D:T2			
M	29.52	17.46	17.14
SD	5.30	3.80	4.91
CDI:T1			
M	21.29	19.92	8.35
SD	7.02	7.11	5.54
CDI:T2			
M	17.76	9.30	5.35
SD	7.41	3.86	4.04

Note. KSADS-D=Depression scale from the Kiddie Schedule for Affective Disorders and Schizophrenia; CDI=Children's depression Inventory.

KSADS Depression Scale

A group x time Anova on the last-week KSADS depression scale, revealed a significant main effect for group, $F(2,41)=48.10$, $p=.000$, and time, $F(1,41)=100.52$, $p=000$, and a significant group x time interaction effect, $F(2,41)=16.66$, $p=.000$. Simple main effects for groups revealed that the three groups differed significantly at T1, $F(2,81)=37.66$, $p<.001$ and at T2, $F(2,81)=31.64$, $p<.001$. At T1, the remitted and unremitted groups were equivalent in depressive symptomatology ($Tukey_a$, p=ns) and as expected,

the remitted group was significantly more depressed on the KSADS compared to the control group ($Tukey_a$, $p<.05$). At T2, however, the means of KSADS depression scale, for the remitted and the control subjects, were virtually identical (see Table 13). As expected, the unremitted group exhibited significantly more depression on the KSADS compared to the remitted group ($Tukey_a$, $p<.05$).

As the above analyses suggest, the remitted group's depression level reduced substantially from T1 to T2, $F(1,41)=101.28$, $P<.001$. The unremitted group also improved significantly from T1 to T2 in depression level on the KSADS, $F(1,41)=17.61$, $p<.001$ and an significant reduction in depression level was also evident for the control group, $F(1,41)=7.97$, $p<.01$). Thus a more accurate label for the unremitted group should actually be - the improved group.

Children's Depression Inventory

A similar pattern of results was found for the CDI. A group x time ANOVA on subject's CDI scores yielded a significant main effect for group, $F(2,41)=23.36$, $p=.000$, and for time, $F(1,41)=32.58$, $p=.000$ and a significant group x time interaction, $F(2,41)=5.67$, $p=.007$. Simple main effects for group revealed that the groups differed significantly at T1, $F(2,70)=19.63$, $P<.001$ and at T2,

$F(2,70)=16.80$, $P<001$. At T1, the depressed groups did not differ significantly on the CDI but the remitted subjects were significantly more depressed on the CDI compared to controls ($Tukey_a$, $P<.05$). At T2, the remitted group was not significantly different on the CDI compared to controls ($Tukey_a$, $p=ns$) although they did evince higher scores. The unremitted group demonstrated significantly higher scores on the CDI compared to the remitted group, ($Tukey_a$, $P<.05$).

The CDI appeared more resistant to change than the KSADs depression scale. The remitted group again showed a great reduction in depression as measured by the CDI from T1 to T2, $F(1,41)=33.64$), $P<.001$ but the control subjects did not show a significant change on the CDI, $F(1,41)=2.89$, $p=ns$. The unremitted subjects also showed a significant change on the CDI from T1 to T2, $F(1,41)=4.86$, $p<.05$.

Hypothesis Testing

The Distortion Scale

Because the distortion scale violated the homogeniety of variance assumption, analyses were performed on the square-root transformed scores. The means and standard deviations for both transformed and untransformed scores are presented in Table 14. The transformation of the distortion

scale satisfied the tests for homogeniety of dispersion matrices (Box M, $F(6,30791)=1.57$; p=ns, $X^2(6)=9.436$, p=ns). A group x time ANOVA yielded a significant main effect for group, $F(2,41)=18.45$, $P<.000$, and for time, $F(1,41)=18.24$, $p<.000$ and the group X time interaction effect approached significance, $F(2,41)=2.46$, $p=.09$. As predicted, the $Tukey_a$ procedure conducted on the average of T1 plus T2 scores, revealed that the remitted group had significantly higher distortions scores than the controls but not significantly higher than the unremitted group.

Although the interaction effect was not significant, simple main effects were calculated for the distortion scale at T1 and at T2 to insure that all the variance in the main effect was not due to T1 scores. Moreover, the a priori hypothesis concerned separate T1 and T2 comparisons. A simple main effect was found for T1, $F(1,54)=11.58$, $P<.001$ and for T2, $F(2,54)=17.14$, $p<.001$. At T1, the remitted and the unremitted subjects did not differ significantly, but both groups were significantly more distorted than the controls ($Tukey_a$ Tests, ps<.05). At T2, the remitted subjects were somewhere between the unremitted subjects and the control subjects displaying significantly less distortion than the unremitted group but displaying significantly more distortion than the control group ($Tukey_a$ Tests, ps<.05).

Hopelessness-Worthlessness

A group x time Anova on the HW scale revealed a significant main effect for group, $F(2,41)=50.81$, $P<.000$, and for time, $F(1,41)=98.06$, $p<.000$, and a significant group x time interaction effect, $F(2,41)=3.46$, $p=.04$. Simple main effects for group indicated the three groups were significantly different at T1, $F(2,80)=6.64$, $P<.01$ and at T2, $F(2,80)=6.95$, $p<.01$. At T1, the remitted and the unremitted groups did not differ significantly on the HW scale but the remitted subjects were significantly more hopeless and worthless compared to the control subjects. At T2, the remitted and the control subjects did not differ significantly on the HW scale ($Tukey_a$, $p=ns$). The unremitted subjects continued to evince more hopelessness compared to the remitted subjects at T2 ($Tukey_a$, $P<.05$). The t-test analysis calculated between the remitted and the control group resulted in a t-value of .703, $p=.49$ which failed to meet the .20 level of significance. Thus it seems unlikely that true differences exist on the HW scale.

As the above analysis suggest, the remitted group showed significant change from T1 to T2, $F(1,41)=22.52$, $P<.001$ on the HW scale. Both the unremitted and the control subjects showed no significant changes on the HW scale from T1 to T2, $F(1,41)=2.86$, $p=ns$ and $F(1,41)=3.48$, $p=ns$,

respectively. Thus, while the unremitted subjects experienced a substantial reduction in depressive symptomatology on the KSADS, this was not accompanied a corresponding reduction in thoughts of hopelessness and worthlessness on the KSADS.

Table 14
Group Means for Distortion and Hopelessness-Worthlessness Measures at Time 1 and Time 2

Measure	Unremitted (n=17)	Remitted (n=13)	Psychiatric Controls (n=14)
CBQC			
Distortion: T1			
M	6.71	5.39	1.36
SD	4.37	3.69	1.50
Distortion: T2			
M	5.94	2.00	.29
SD	4.11	2.52	.47
Transform-D:T1[a]			
M	2.39	2.13	.89
SD	1.02	.97	.77
Transform-D:T2[a]			
M	2.18	1.17	.29
SD	1.11	.82	.50
KSADS			
Hopelessness-Worthlessness:T1			
M	6.94	7.23	4.50
SD	2.22	2.89	1.91
Hopelessness-Worthlessness:T2			
M	5.76	3.46	3.07
SD	2.66	1.39	1.49

Note. CBQC= Cognitive Bias Questionnaire for Children. KSADS=Kiddie Schedule for Affective Disorders and Schizophrenia
[a] Square root transform of the distortion scale

Ancillary Findings

Table 15 presents the means and standard deviations for the NN, the DN and the CBQC difference score at T1 and T2.

However, only the analysis with the CBQC difference score is presented here as this score is interpretable in terms of the self-derogatory versus the self-serving bias.

Self-derogatory vs Self-serving Bias

Because the CBQC difference score seriously violated the homogeniety of variance assumption, it was unsuitable for a repeated measures analysis of variance where the number of subjects also differed substantially in each group. Therefore, this score was evaluated by chi-square analyses. A second reason for choosing a categorical analysis was to examine the CBQC difference score from two approaches using two different cut-off scores. These cut-off scores were made on the basis of logic and did not depend on the inspection of the data. Recall from study 1, that a score of less than or equal to -1 means that the subject chose more distorted than positive options on the CBQC, the self-derogatory bias. In the first analysis then, the scores were dichotomized at less than or equal to -1. A score of 11 means that at least 55% of the responses on the CBQC remain positive after the distorted responses are subtracted out. This was defined as a self-serving bias. In the second analysis, therefore, the scores were dichotomized at a score of greater than or equal to 11. The minimum level for significance for multiple contrasts was

.05/4=.012, for the two contrasts at T1 and the two contrast at T2.

At T1, 59% of the unremitted, 31% of the remitted and 0% of the control subjects exhibited a self-derogatory bias, $X^2(2, N=44)=12.26$, $p=.002$. Group comparisons revealed that there was a trend for the remitted group to be more self-derogatory than the control group, but this trend was not significant, $X^2(1, n=27)=5.06$, $p=.02$. The remitted and unremitted groups also did not differ significantly, $X^2(1, n=30)=2.33$, $p=$ns. At T2, there was again an overall effect for the difference score with 59% of the unremitted, 8% of the remitted and 0% of the control subjects exhibiting the self-derogatory bias, X^2 $(N=44)=17.12$, $p=.0002$. At T2, the differences were more clear, the unremitted patients were significantly different from the remitted patients $X^2(1,n=30)=8.29$, $p=.004$, while the remitted and the control patient did not differ significantly $X^2(1, 27)=1.11$. $p=$ns. This analysis suggests that a self-derogatory bias is associated with continued persistence of depression and the presence of such a bias predicts a poor outcome.

At T1, 6% of the unremitted, 15% of the remitted group and 71% of the control group evinced a self-serving bias, a significant difference, X^2 $(2, N=44)=17.62$, $p=.0001$. At T1, the remitted group had significantly less cases with a self-

serving bias compared to the control group, $X^2(1, n=27)=8.57$, $p=.003$, but did not differ significantly from the unremitted group, $X^2(1, n=30)=.73$, p=ns. At T2, 17% of the unremitted, 38% of the remitted compared to 100% of the control subjects exhibited a self-serving bias, $X^2(2, N=44)=21.81$, $p=.0000$. The remitted subjects had significantly less cases with a self-serving bias than the control subjects, $X^2(1, n=27)=12.24$, $p=.0005$, but again did not differ significantly from the unremitted group, $X^2(1, n=30)=1.63$, p=ns. This analysis is clear in implicating a lack of a self-serving bias as trait marker for depression.

Table 15
Other CBQC Scales at T1 and T2

Measure	Unremitted (n=17)	Remitted (n=13)	Psychiatric Controls (n=14)
CBQC Scales			
Nondepressed-Nondistorted:T1			
M	5.94	7.77	13.00
SD	3.89	3.30	2.88
Nondepressed-Nondistorted:T2			
M	6.70	10.85	15.07
SD	4.07	4.10	1.49
Depressed-Nondistorted:T1			
M	7.29	6.85	5.71
SD	2.17	1.86	1.90
Depressed-Nondistorted:T2			
M	7.35	7.15	4.64
SD	2.26	3.67	1.21
Difference Score:T1			
M	-.76	2.39	11.64
SD	8.02	6.74	4.23
Difference Score:T2			
M	.76	8.85	14.79
SD	7.87	5.72	1.85

Note. CBQC= Cognitive Bias Questionnaire for Children.

Residual Depression

Although the remitted and the control subjects did not differ significantly on the CDI at T2, CDI scores were higher in the remitted group. To determine whether or not

differences on the distortion scale at T2 were due in part to residual depression on the CDI, an analysis of covariance was conducted between the remitted and the control group covarying T2 CDI scores. In this analysis, only the square-root transform scores of the distortion scale were used. The results of this analysis revealed that the distortion scale still distinguished the groups after the effects for CDI were partialled out, $F(2,24)=6.00$, $p=.02$. An analysis of covariance on the CBQC difference score at T2 also yielded a significant effect after the CDI scores were partialled out, $F(2,24)=5.67$, $p=.025$. Therefore, the differences between the remitted and the control subjects can not be accounted for by residual depression on the CDI.

Discussion

The hypotheses were confirmed. As predicted, the tendency to distort events was found to be hypervalent in the symptomatic phase and to be less hypervalent but still salient in the remission phase. In contrast, thoughts of hopelessness and worthlessness, which were prevalent in the symptomatic phase, normalized in the remission phase. These findings suggest that the tendency to distort events is a trait marker for major depression whereas thoughts of hopelessness and worthlessness are state markers.

These results are most comparable to four studies mentioned in the introduction (Eaves & Rush, 1984; Hamilton & Abramson, 1983; Norman and Miller, 1986; Reda, Carpinello, Secchiarole & Blanco, 1985). These four studies all share the same methodology, that is they all assessed the patients during the depressed phase and in the remitted phase and importantly, they all employed a nondepressed control group which was also assessed at 2 time periods. As noted previously, three of the studies found that cognitive distortion (Norman & Miller, 1986) and dysfunctional attitudes (Eaves & Rush, 1984; Reda et al., 1985) remain elevated in the remitted group compared to the nondepressed control group. The present findings are consistent with these three studies. In the fourth study, Hamilton and

Abramson (1983) failed to find evidence that the remitted depressives had elevated negative attributions or dysfunctional attitudes. When the present findings are considered with these studies and with the more favourable studies from the child literature, the evidence is tipped in favour of the hypothesis that certain ways of thinking are trait rather than state dependent cognitions at least in child and adolescent populations.

Although the unremitted depressed patients, in the present study, significantly improved in depressive symptomatology as assessed by both the KSADS and the CDI, these improvements were not paralleled by reductions in hopelessness and worthlessness. However, hopelessness and worthlessness were normalized in the remitted group with accompanying remission of depressive symptoms. These findings suggest that the cognitive aspects of depression are more resilient to change or may take longer to normalize than other depressive symptoms. These results also have an important methodological implication. The differential behaviour of the HW scale and the distortion scale cannot be attributed to differences in the assessment methods (i.e interview ratings vs self-report). The unremitted subjects showed significant changes on measures of the same construct rather than on measures by the same methodology. More specifically, they showed a significant reduction in

depression as assessed by both the CDI and the KSADS but did not show a significant reduction on measures of cognition, the CBQC and the HW scale. These findings suggest that hopelessness and worthlessness are state markers of major depression which only normalize when complete recovery from depression is achieved.

The evidence in this study indicates that the remitted depressives are not any more self-derogating than the controls when remitted, but rather are clearly less self-serving or positively biased. In contrast, the majority of unremitted patients evinced the self-derogatory bias both at T1 and T2. This evidence implicates the self-derogatory bias in the maintenance of depression. This finding is consistent with a recent longitudinal study which showed that women who exhibited much "globally self-devaluative thinking" recovered more slowly than equally depressed women who showed low levels of such thinking (Dent & Teasdale, 1988). It should be noted, however, that the percentage of depressed patients who fail to manifest a self-serving bias is considerably greater than the percentage of those who manifest a self-derogating bias in either the remitted or the unremitted patients. This evidence suggests that when remitted, depressed adolescents are not as self-derogating as they were when in the depressed phase, but remain less positive in thinking compared to nondepressed adolescents.

This failure to be more self-serving in thinking may lead to increased vulnerability to depression. It should be pointed out that the term "bias" in the self-serving sense means that positive thinking predominates over negative thinking on the CBQC. It also should be noted that the failure of the remitted subjects to show a more self-serving bias is due in part to the presence of cognitive distortions at remission.

One possible explanation of the present results is that cognitive markers take longer to normalize than depressive symptoms. The patients are too close in time to the last episode of depression. However, this seems unlikely to be completely explanatory because research with adult depressives shows that, in general, cognitions and depressive symptoms covary quite closely and that those studies that have a longer follow-up period tend to show residual distortion in remitted depressives (e.g. Reda et al., 1985; see table 1). There has been no evidence that the time interval between recovery and testing for negative thinking is related to the outcome of these studies.

Another possibility is that the remitted depressed adolescents are not really remitted but are actually exhibiting subacute or latent depression. Several factors make this an unlikely explanation for the results. First,

the means of remitted and the control subjects are practically identical on the KSADS depression scale. Second, none of the remitted subjects could even meet the RDC criteria for minor depression or the DSM-III-R criteria for an atypical or an adjustment disorder with depressed mood. Thirdly, the groups remain significantly distinguished on the basis of distortion even when the effects of depression are controlled by statistical means.

Another possible problem is the inclusion of dysthymic subjects in the control group. Sixty-two percent of the remitted subjects failed to show a self-serving bias compared to zero percent of the controls. If the four dysthymic subjects are included in the remitted group on the basis that they constitute an at-risk group, then the percent of those who fail to exhibit an self-serving bias is reduced to 48 percent. There are several problems with this logic. First, the dysthymic subjects were placed in the control group prior to the inspection of the data on the basis that they had not ever exhibited a major depressive episode in the past and were not currently expressing a major depressive episode. There may be completely different reasons why these dysthymics have failed to exhibit major depression or exhibit a different course than those who are currently in a major depressive episode. In other words, there are different aetiologies to these two disorders. In

fact, as already noted, the distortion model in the present sample accounts for only 62% of those with major depression. Thus, the distortion model may identify a subgroup for whom negatively biased thinking is a trait marker. Therefore, it might be more useful to examine the course of those who exhibit high levels of distortion as compared to those who show low levels of distortion. Do these two types of major depression offer a better classification than the endogenous subtyping in terms of predicting treatment and course? Future research can begin to focus on these issues.

The main limitation in this study is the small number of remitted depressed adolescents. Only 43% of the depressed subjects met the remission criteria. Perhaps these criteria were too strict. If the criteria were made less stringent, the findings would not change and in fact there would be even higher distortion scores at T2 for the remitted group. The evidence for this assertion is based on the fact that the unremitted group significantly improved in depressive symptoms yet displayed significantly higher distortion scores at T2 than the remitted group. The remission criteria were specific in ensuring that no depressed mood or anhedonia remained at T2.

The evidence from the present study does not show that cognitions cause depression nor does it show the existence

of cognitive schemas. My intention in this study was to demonstrate that certain ways of thinking may predispose adolescents to experiences of depression. One step in support of the concept of cognitive vulnerability markers is to show that negative cognitions persist in the remitted phase of depressive illness. The evidence from the present study is clearly supportive of this concept. The present evidence must be considered in context of other studies which have shown that healthy children of depressed mothers not only exhibit negative thinking in a healthy state but are more likely to develop a depressive disorder than children without negative self cognitions (i.e Hammen, 1988; Hammen, Adrian & Hiroto, 1988; Jaenicke et al., 1987). These studies together with the present study have fulfilled all three conditions, as outlined in the introduction, that constitute a true trait marker. To fully accept the notion of trait markers, these studies including the present one need to be replicated and expanded to larger samples of patients and subjects.

What are the implications of these findings for therapy? Cognitive therapy which aims at altering cognitive distortion may have a beneficial effect in reducing depressive symptomatology by its ability to break the hopelessness and the self-derogatory bias. These findings support the strategy of therapists who focus on the

cognitive elements in depression in obtaining significant change in depression (Beck, Rush, Shaw & Emery 1979). If it is to protect against future episodes of depression, the therapy should aim to increase the accessibility to more positive thinking. More importantly, the evidence from this study suggests that cognitive therapies might be adapted for use with depressed adolescent patients.

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APPENDICES

Appendix A: The Cognitive Bias Questionnaire for Children

Scoring Key

Dan/Danielle

	DD	DN	NN
1.	a	b	c
2.	b	a	b
3.	c	a	b

John/Jane

1.	a	c	b
2.	c	b	a

Michael/Michelle

1.	b	a	c
2.	a	b	c

Carl/Carla

1.	c	b	c
2.	b	c	a
3.	a	b	c

Julian/Julia

	DD	DN	NN
1.	c	a	b
2.	b	a	c

Don/Donna

1.	a	b	c
2.	a	b	c
3.	c	b	a

Paul/Paula

1.	b	a	c
2.	b	c	a
3.	a	b	c

Martin/Mary

1.	a	c	b
2.	c	a	b

C B Q C

Instructions

These pages contain some short stories about kids in situations that might occur. Please read each story and put yourself in the girl's place and try to imagine as clearly as possible how the girl might think and feel.

Each story ends with 2 or 3 questions. The answer is up to you, there are no right or wrong answers. Choose one sentence that best describes how you might respond if you were the person in the story.

Please circle the letter beside the sentence that you choose.
Be sure you answer parts 1, 2 and 3.

Glenn Haley, 1981
Simon Fraser University
Revised October, 1985

Danielle is in a new class and she does not know any of the other kids. During the first week of class the teacher asks her a question on history. Danielle thinks hard and gives her best answer. The teacher says that Danielle is not quite right.

1. What does she say to herself?
   a. The kids in this class must think I'm dumb and won't like me.
   b. It upsets me and makes me feel no good.
   c. It doesn't bother me because one mistake is not that important.

2. When other kids in the class give wrong answers to the question, Danielle thinks:
   a. I still feel bad .
   b. My answer was the worst in the class.
   c. I don't feel so bad anymore.

3. Imagining what will happen in other classes, Danielle thinks:
   a. I started out bad, but other classes should be better.
   b. Just because of this history class, doesn't mean all my oher classes will be bad.
   c. I started out bad in this class, the other classes will be the same.

Jane is walking by herself from school one day and she is thinking about her school work. She suddenly notices a boy with a frown on his face walking towards her.

1. When the boy passes her by, what does Jane think?
   a. He must think I look pretty awful.
   b. It doesn't bother me that he looks that way, Some people have a lot on their minds.
   c. I feel sad that some people are not happy.

2. Later, a friend stops to tell her that she looks good today. Jane thinks:
   a. It was nice of her to tell me that.
   b. When people tell me that, I feel a little sad.
   c. Most other days my friend probably thinks I look terrible.

Michelle has a younger sister and an older brother. One Staturday afternoon she finds out that her brother and sister have been invited to a birthday party. Michelle has not been invited and has to stay home alone.

1.What does she say to herself?
   a. I feel lonely but now I can do all those things I have put off.
   b. I begin to wonder if any one really likes me.
   c. It's OK 'cause I get invited to parties without my brother and sister.

2.When Michelle is alone that day, She:
   a. Begins to imagine endless weekends of being alone.
   b. Begins to feel sad.
   c. Starts to think that everyone is probably alone once in a while.

Carla and Phillis are best friends. They always go downtown on saturday. One saturday afternoon phillis phones to say to Carla that she is going downtown with a new friend that she has just met.

1.What does Carla think?
   a. It doesn't bother me that Phillis has another friend. It will be nice to meet to meet a new friend.
   b. It makes me feel sad that she did not ask me to go with them.
   c. It upsets me because she does not want me for a best friend anymore.

2.Remembering the telephone conversation, Carla thinks:
   a. She sounded friendly, Phillis must still like me.
   b. Maybe the way I talked wasn't that good.
   c. I feel unhappy, but at least our conversation was OK.

3.Thinking about, making new friends, Carla imagines:
   a. I am not likeable person, nobody will want me as a friend.
   b. It's sometimes hard to make friends, but worth it.
   c. Everyone makes friends sooner or later, so will I.

Julia is the youngest in a family of three kids. Her parents fight with one another now and then. Her older brother and sister tell her that mom and dad never fought when they were growing up.

1. She begins to think:
   a. It makes me feel bad that they fight, parents should try to get along better.
   b. I guess most parents fight sooner or later.
   c. If it were not for me, things would be better around the house, like they were for my brother and sister.

2. One night her parents have a little fight, Julia immediately thinks:
   a. I feel down and hope things will turn out ok for them.
   b. I begin to wonder what I have done wrong.
   c. It doesn't bother me, it's only an little fight.

Donna was listening to her records after school one day. Her mom came into her room and started yelling at her to clean up her room and put her clothes away.

1. Her first thought is:
   a. It upsets me and makes me feel like a bad person.
   b. It makes me feel sad, but even moms can get upset once in awhile.
   c. I wonder what is bothering my mother.

2. When mom was gone, what did she feel?
   a. When mom yells at me, I think she doesn't love me anymore.
   b. I feel bad but I know she still likes me.
   c. I suppose I could try harder to be more neat.

3. Thinking about what will happen with mom, Donna imagines:
   a. Just because she yelled at me, doesn't mean she won't like me tommorrow.
   b. I feel bad now, but things will be ok later.
   c. I will never get along with mom, now.

Paula is in an english class. Everyone in the class has to read aloud an english story. Finally, it came time for Paula to read her story. While she was reading, she heard a giggle from the back of the class.

1. She immediately thinks:
   a. I feel bad that some kids can be rude.
   b. Everyone here must think I am doing badly.
   c. I'll just ignore the giggle and continue my reading.

2. Later, Paula's classmates said she had read well. What does Paula think?
   a. My classmates like my story and I feel good about that.
   b. They are just trying to make me feel good about my awful reading.
   c. I still feel bad but my classmates help me feel better.

3. Remembering what happened that day in class, Paula thinks:
   a. I probably didn't do well because I heard giggles.
   b. I always feel bad when I have to speak in front of the class.
   c. I probably did as well as anyone else.

Mary was looking forward to going to a party with her friends. They had made plans for a long time. But, a few days before the party, Mary caught the flu and became really sick and could not go to the party.

1. Her first thought is:
   a. This always happens to me. I probably made myself sick.
   b. My friends will understand that everyone gets sick now and then.
   c. It's too bad that I will miss the party.

2. When the flu had gone, what did Mary think?
   a. Now I will be able to go to any party that comes up.
   b. I feel bad about missing the party, but things like that just can't be helped.
   c. Nobody will invite a sick person like me to any parties now.

Appendix B: Repeated Measures ANOVA for KSADS Depression Scale

Source	df	SS	MS	F
Between Subj				
A(Groups)	2	2695.61	1347.80	48.10*
Ss within groups	41	1148.79	28.02	
Within Subj				
B (Time)	1	2144.13	2144.13	100.51*
AB	2	710.92	355.46	16.67*
BxSs Within Groups	41	874.56	21.33	

*p<.001

Multivariate test for Homogeniety of Dispersion matrices

Boxs M=6.60

F with (6,30791) DF=1.02, p=.409

ChiSquare with 6 DF= 6.13, p=.409

Appendix C: Repeated Measures ANOVA for CDI

Source	df	SS	MS	F
Between Subj				
A (Groups)	2	2477.76	1238.88	23.36*
Ss within groups	41	2174.05	53.02	
Within Subj				
B (Time)	1	709.41	709.41	32.58*
AB	2	246.79	123.40	5.67*
BxSs Within Groups	41	892.65	21.77	

*p<.01

Multivariate test for Homogeniety of Dispersion matrices

Boxs M=11.49

F with (6,30791) DF=1.78, p=.099

Chi-Square with 6 DF= 10.67, p=.099

Appendix D: Repeated Measures ANOVA for HW Scale

Source	df	SS	MS	F
Between Subj				
A(Groups)	2	101.63	50.81	9.31*
Ss within groups	41	223.86	5.46	
Within Subj				
B (Time)	1	98.06	98.06	23.92*
AB	2	28.39	14.19	3.46*
BxSs Within Groups	41	168.10	4.10	

*p<.05

Multivariate test for Homogeniety of Dispersion matrices

Boxs M=11.49

F with (6,30791) DF=1.78, p=.099

ChiSquare with 6 DF= 10.67, p=.099

Appendix E: Repeated Measures ANOVA for Distortion Scale

nontransformed.

Source	df	SS	MS	F
Between Subj				
A(Groups)	2	465.23	232.62	15.41*
Ss within groups	41	619.09	15.10	
Within Subj				
B (Time)	1	65.78	65.78	11.80*
AB	2	28.56	14.28	2.56
BxSs Within Groups	41	228.53	5.57	

*p<.01

Multivariate test for Homogeniety of Dispersion matrices

Boxs M=53.69

F with (6,30791) DF=8.31, p=.000

Chi-Square with 6 DF= 49.85, p=.000

Appendix F: Repeated Measures ANOVA for the Square-root transformed Distortion Scale

Source	df	SS	MS	F
Between Subj				
A(Groups)	2	44.56	22.28	18.45*
Ss within groups	41	49.51	1.21	
Within Subj				
B (Time)	1	7.56	7.56	18.24*
AB	2	2.04	1.02	2.46
BxSs Within Groups	41	16.99	.41	

*p<.01

Multivariate test for Homogeniety of Dispersion matrices

Boxs M=10.16

F with (6,30791) DF=1.57, p=.150

Chi-Square with 6 DF= 9.43, p=.150

Appendix G: Repeated Measures ANOVA for CBQC Difference Score

Source	df	SS	MS	F
Between Subj				
A(Groups)	2	2683.50	1341.75	23.69*
Ss within groups	41	2321.87	56.63	
Within Subj				
B (Time)	1	299.17	299.17	13.44*
AB	2	90.91	45.45	2.04
BxSs Within Groups	41	912.59	22.26	

*p<.01

Multivariate test for Homogeniety of Dispersion matrices

Boxs M=25.43

F with (6,30791) DF=3.93, p=.000

Chi-Square with 6 DF= 23.61, p=.000