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INVITED ESSAY

PREVENTION OF DEPRESSIVE SYMPTOMS IN SCHOOL CHILDREN

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Summary—This paper describes the development and preliminary efficacy of a program designed to prevent depressive symptoms in at-risk 10–13 year-olds, and relates the findings to the current understanding of childhood depression. The treatment targets depressive symptoms and related difficulties such as conduct problems, low academic achievement, low social competence, and poor peer relations, by proactively teaching cognitive techniques. Children were identified as 'at-risk' based on depressive symptoms and their reports of parental conflict. Sixty-nine children participated in treatment groups and were compared to 73 children in control groups. Depressive symptoms were significantly reduced and classroom behavior was significantly improved in the treatment group as compared to controls at post-test. Six-month follow-up showed continued reduction in depressive symptoms, as well as significantly fewer externalizing conduct problems, as compared to controls. The reduction in symptoms was most pronounced in the children who were most at risk.

INTRODUCTION

We developed a new program (The Penn Prevention Program) to prevent depressive symptoms among at-risk 10–13 year-old children. There is a growing body of research which points to the role of cognitive distortions and deficiencies in the etiology and maintenance of childhood depression. Based on this research, we sought to target these cognitive variables in children who were at-risk for future depression, and thus prevent depression from occurring. This strategy for prevention is consistent with recent recommendations concerning public health policy addressing depression (Munoz, Hollon, McGrath, Rehm & VandenBos, 1994). The Penn Prevention Program (PPP) uses cognitive-behavioral techniques proactively to teach children coping strategies to use in the face of negative life events and to enhance their sense of mastery and competence across a variety of situations.

In addition to preventing depressive symptoms, the program was designed to combat the deficits associated with depression in children, such as lowered academic achievement, poor peer relations, lowered self-esteem, and in particular, behavioral problems. As many as one third of children with a depressive disorder also develop a comorbid conduct disorder (Kovacs, Paulauskas, Gatsonis & Richards, 1988). In addition, a mixture of depressive symptoms, low self-esteem, and conduct problems are usually associated with stressors in childhood, including the relevant stressor in this study: exposure to marital or family conflict (Emery, 1982; Jaycox & Repetti, 1993; Grych & Fincham, 1990). For these reasons, the approach of the program was broad. Our attention to associated symptoms adheres to Kendall and colleagues' recommendation that manualized treatment programs address comorbid conditions (Kendall, Kortlander, Chansky & Brady, 1992).

There are several lines of research which have identified cognitive distortions and deficiencies in children who are diagnosed with depression or behavioral problems. For instance, children with depressive symptoms tend to have a more pessimistic explanatory style. That is, they tend to

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attribute internal, global, and stable causes to negative events (Cole & Turner, 1993; Nolen–Hoeksema, Girgus & Seligman, 1992; Quiggle, Garber, Panak & Dodge, 1992). In the realm of social cognition, researchers have demonstrated that in ambiguous social situations, depressed children tend to attribute hostile intentions to others. These children are also less likely to generate assertive solutions to interpersonal problems (Quiggle, Garber, Panak & Dodge, 1992). In experiments measuring performance on cognitive tasks, children who are depressed have lower expectations for their own performance and more stringent criteria for failure (Kaslow, Rehm & Siegel, 1984). These expectations may lead to self-fulfilling prophecies which ultimately serve to reinforce their depressogenic cognitions and maintain the depression. In addition, research on depressed adolescents indicates that they show more negative self-evaluation, dysfunctional attitudes, hopelessness, and pessimistic explanatory styles than their non-depressed counterparts (Garber, Weiss, & Shanley, 1993).

There have also been cognitive factors identified in children with behavioral problems, particularly for those children who act out with aggression. They show impaired social problem-solving ability, with selective attention to hostile cues and a hostile attributional bias (Dodge, 1986; Dodge & Frame, 1982). These children tend to produce fewer assertive responses to interpersonal problems, and produce more direct action or aggressive responses (Lochman & Lampron, 1986; Shure & Spivack, 1972; Vitaro & Pelletier, 1991; Waas, 1988). Thus it appears that not only is there an overlap in the expression of depressive symptoms and behavioral problems in children, but there are also some similarities in the proposed cognitive underpinnings for these two problems.

The development of this prevention program was based on the efficacy of cognitive–behavioral techniques in the treatment of depression among adults, as well as the prevention of relapse (Dobson, 1989; Evans, Hollon, DeRubeis, Piasecki, Grove, Garvey & Tuason, 1992). An attempt to teach cognitive techniques to a sample of 10–13 year-old children raises an important question: Do pre-adolescent children have the cognitive capacity to benefit from such training? Children in this age group are entering the formal operations phase of cognitive development (Piaget, 1977), and thus should have the cognitive maturity necessary to understand and apply the skills taught. In addition, empirical data suggest these skills can be successfully applied to this age group. Several group interventions using similar strategies have been shown to reduce depression in moderately depressed adolescents and children (Butler, Mietzitis, Friedman & Cole, 1980; Kahn, Kehle, Jenson, & Clarke, 1990; Lewinsohn, Clarke, Hops & Andrews, 1990; Reynolds & Coats, 1986; Stark, Rouse & Livingston, 1991; Stark, Reynolds & Kaslow, 1987). The PPP is also based on previous work on reducing aggressive or antisocial behavior and raising self-esteem (Camp, Blom, Hebert & van Doorinck, 1977; Lochman & Curry, 1986; Lochman, Burch, Curry & Lampron, 1984; Kazdin, Siegel & Bass, 1992), and on preventive social problem solving programs designed to reduce subsequent stressors and improve adjustment in children (Elias, Gara, Ubriaco, Rothbaum, Clabby & Schuyler, 1986; Weissberg, Gesten, Rapkin, Cowen, Davidson, Flores de Apodaca & McKim, 1981).

The program described in this paper is importantly different from those cited above. Our goal was to *prevent* significant future depressive symptoms in children at risk. The term prevention warrants some discussion from the outset, since it has been used in many different ways. Ideally, prevention should be proactive and occur before the onset of a disorder, to prevent problems in groups of unaffected people. However, when the onset of a disorder is unclear, as in the case of depression or the accretion of depressive symptoms, the distinction between primary prevention and early intervention becomes blurred (Albee & Gullotta, 1986). For instance, mild depressive symptoms put adults at risk for a later depressive disorder. Depressive symptoms, therefore, are simultaneously a risk factor and part of an unclearly defined onset of depression (Lewinsohn, Hoberman & Rosenbaum, 1988). In this paper, the term 'relief' means reduction of symptoms at the end of treatment. The term 'prevention' means that depressive symptoms are reduced long after the treatment is over. These definitions overlap with the concepts of intervention and maintenance of gains, and the results will be discussed in relation to such findings at the end of this paper.

In order to identify those children who might be 'at-risk' for depression from a normal school population, two separate indices were used. First, depressive symptoms were measured in order to select children who were beginning to show some degree of dysphoria. Second, in line with evidence that marital conflict and low family cohesion are associated with increased depressive symptoms

in children, children rated the degree of parental conflict in the home (Fendrich, Warner & Weissman, 1990; Johnston, Gonzalez & Campbell, 1987; Peterson & Zill, 1986; Stark, Humphrey, Crook & Lewis, 1990). With these two factors, a group of children with high base-rate risk for future depression was identified. While there may be other more reliable risk factors for childhood depression, such as parental depression, these would not occur with enough frequency in a normal school population to use them as selection criteria. This sample therefore represents a population which is probably only mildly at risk for future depression.

We know of only one other study in which prevention of depressive symptoms was attempted. Clark and colleagues showed a reduction in the incidence of Major Depression or Dysthymia among at-risk adolescents who were assigned to a cognitive-behavioral group treatment as compared to a "usual care" control condition (Clark, Hawkins, Murphy, Sheeber, Lewinsohn & Seeley, 1993).

Hypotheses

In this paper five main hypotheses are examined. First, a larger decrease in depressive symptoms was expected at the end of the treatment period in the treatment group than in the control group (Hypothesis 1: Relief of Depressive Symptoms). Second, after 6 months, the treatment group was expected to have fewer depressive symptoms than the control group, indicating prevention in the treatment group (Hypothesis 2: Prevention of Depressive Symptoms). Third, a larger decrease in behavior problems at home was expected during the treatment period in the treatment group than in the control group, as measured immediately following the treatment period (Hypothesis 3: Relief of Conduct Problems at Home). After 6 months, the treatment group was expected to show fewer behavior problems at home than the control group, indicating prevention in the treatment group (Hypothesis 4: Prevention of Conduct Problems at Home). Finally, children in the treatment group were expected to show improved classroom behavior as compared to controls at post-test (Hypothesis 5: Relief of Classroom Behavioral Problems). We tested this hypothesis separately from the hypothesis predicting relief of behavior problems at home because of previous findings of low correspondence in behavioral ratings by teachers and parents, which has been attributed to the situational specificity of children's behavior (Achenbach, McConaughy, & Howell, 1987; Hinshaw, Han, Erhardt, & Huber, 1992). Since teacher reports of classroom behavior were not available at follow-up, the prevention of classroom behavioral problems could not be tested.

Several secondary hypotheses were also explored. First, explanatory style for negative events was expected to become more optimistic in the treatment group compared to the control group. Based on cognitive theory, we thought that the change in explanatory style might mediate the treatment effects on depressive symptoms. If found, this would extend the findings regarding the importance of explanatory style change in the treatment of adult depression to children (DeRubeis, Evans, Hollon, Garvey, Grove & Tuason, 1990; Seligman, Castellon, Cacciola, Schulman, Luborsky, Ollove & Downing, 1988). We did not expect explanatory style change to mediate treatment effects on behavioral problems. Second, we expected that relief and prevention would be especially strong for the children who reported a higher degree of parental conflict, since the program directly taught techniques for coping with conflict. Finally, changes were examined in two subsets of children: those who entered the project with few depressive symptoms (referred to as the low-symptom group), and those who entered with relatively many symptoms (referred to as the high-symptom group). Different treatment effects were expected in the two groups: immediate and abiding relief of symptoms in the high symptom group, and nonoccurrence of symptoms in the low-symptom group.

METHOD

Design

In this five-year prospective study, the effectiveness of three versions of the program were compared to a combined control group consisting of a wait-list group and a no-participation control group. This paper describes the first phase of the project. Phase one includes relief (the immediate effects), and prevention (the 6-month follow-up) of the treatment group ($N = 69$) as compared to the control group ($N = 24$ wait-list; $N = 50$ no-participation). Three different active

treatments were conducted: a cognitive training component, a social problem-solving component, and a combined treatment, which included both components.

Treatment groups met once a week after school for approximately 1 and 1/2 hr. Three doctoral students in clinical psychology conducted the treatment for groups of 10–12 children, using a detailed training manual. The manual precisely spells out the didactic material, with examples, games, and group exercises. To insure consistency across groups the program was first pilot-tested by teams of two leaders who were and supervised by a licensed clinical psychologist using videotapes of the group sessions.

Subject recruitment

Letters and consent forms describing the screening phase and the prevention program were sent to all parents of 5th and 6th grade children in a school district outside of Philadelphia, Pennsylvania. From an initial pool of approximately 900 children, the parents of 174 children (19%) returned consent forms. Each of the 174 children filled out two screening questionnaires. The screening was conducted in groups at each of seven elementary schools.

The same selection procedure was used to recruit children for a no-participation control group in a second suburban school district. The letters and consent forms were identical to those used to recruit participants in the treatment group, except no mention was made of the treatment groups, and a $5 donation per child per year to a school activity fund was included as an incentive for participation. Of an initial pool of approximately 700 5th and 6th grade children, parental consent was returned for 88 children (13%).

The low response rates in both school districts reflect a self-selection bias. Although self-selection is a problem in most treatment or intervention studies, it is important to note that the subjects in this study were children and parents who volunteered to participate in a long and time-demanding research study, and thus may not be a truly representative sample of children at-risk for depression.

Screening measures and selection criterion

From the pool of 262 children given the screening measures, 149 children were identified as being at-risk for depression based on two criteria: current level of depressive symptoms and perception of parental conflict. Together, these two measures comprised a *risk score*.

The *Children's Depression Inventory*, a 27-item self-report measure of severity of depressive symptoms (Kovacs, 1985), assesses symptoms such as low mood, somatic symptoms, behavioral problems, low self-worth, and anhedonia. This scale is a childhood extension of the adult Beck Depression Inventory (Beck, Ward, Mendelson, Mock & Erbaugh, 1961), and has been shown to be reliable and valid in measuring severity of depression (Reynolds, 1992).

The *Child's Perception Questionnaire*, a 19-item self-report questionnaire (Emery & O'Leary, 1982) assesses the degree to which the child perceives marital conflict. Twelve of the items are indicative of interparental conflict ("My parents often yell and scream with each other when I'm around"), while the other 7 items indicate parental acceptance. The children endorse a 3-point scale to indicate whether each statement is 'not true', 'sort of true', or 'true' of their parents. Although this scale has not been widely used, the marital conflict items have been shown to have adequate internal consistency, with a Cronbach's α of 0.90 (Emery & O'Leary, 1982) and 0.86 (Kurdek & Sinclair, 1988).

To create a single selection criterion of risk status, the two scores were converted to z-scores and summed. All children who had a risk score above a cut-off (0.50) were invited to participate. Some children with scores below the cut-off were also invited to participate. This was done in descending order as space in the groups permitted. Fifty-three percent of the children screened were selected to be in the program. In addition, we attempted to recruit an equal numbers of boys and girls in each group.

Group assignment

Six groups of 10–12 children were formed at 6 small schools, and 2 groups at one much larger school, for a total of 8 groups. Of the 99 children offered the chance to participate in this project, the parents of 6 children declined to have their child participate.

Because this was a district wide school-based program, random assignment of children to conditions was not possible. The experimental conditions were assigned without bias to schools

rather than to individual children. Pre-randomization significance tests indicated that one school had a significantly higher SES (as measured by average income and average level of parental education) than the other 7 groups. Since there tend to be fewer depressive symptoms among children from wealthy families (Lefkowitz, Tesiny & Gordon, 1980), we biased the groups against our hypotheses and assigned the higher income group to the wait-list condition.

In addition, examination of risk scores at each school revealed between-group differences in the initial selection criterion. Five groups had a better mean risk score (range 0.70–0.90) while 3 groups had a worse mean score (range 1.31–1.46). Even though these differences were not significant, high and low risk groups were paired to each other prior to assignment to treatment or control condition in order to minimize group differences. Experimental conditions were then randomized to provide unbiased assignment of condition to pairs. As a result, the 3 treatment variations (combined, cognitive, social problem-solving), and the wait-list group each contained 2 groups of 11–12 children, for a total of 22–24 children per experimental condition.

The no-participation control group was selected using the same selection criterion. The 50 children who scored highest on this risk score served as the no-participation control group, resulting in 57% of the screened population being chosen as at-risk. This group did not differ significantly from the treatment or wait-list groups on the initial level of distress.

It is important to note that throughout this paper the hypotheses concern changes in individual children, not groups of children. Thus, the assumption that each observation is independent may not be met because of group effects. In all major analyses, differences between groups will be controlled by examining the group factors nested within the treatment condition. In addition, the 3 versions of the treatment will be looked at together in this paper and compared to controls, since there were no major differences between the 3 active treatment groups at post-test.

Sample characteristics

The final sample consisted of 143 children: 69 children (34 girls, 35 boys) in the treatment conditions; and 74 children (32 girls, 42 boys) in the combined control group. Ages ranged from 10 to 13 years (mean age = 11.4; SD = 0.67). As seen in Table 1, most of the children in the study were Caucasian (83%) or African-American (11%). Children came from families with an average yearly income of $37,500 in 1991. There were no significant differences among any of the groups

Table 1. Demographic variables by group

	Treatment	Control	Statistic
Age of child in years			
Mean	11.36	11.52	$t = -1.43$
SD	(0.70)	(0.63)	
Sex of child:			
Male	50.7%	56.8%	
Female	49.3%	43.2%	$\chi^2 = 0.52$
Race of child:			
Caucasian	79.7%	85.3%	
African American	17.2%	5.9%	
Other	3.1%	8.8%	$\chi^2 = 5.60$
Total family income:			
Less than $20,000	16.4%	12.1%	
$20,001–$40,000	44.3%	21.2%	
$40,001–$60,000	26.2%	24.2%	
$60,001–$80,000	6.6%	13.6%	
More than $80,000	6.6%	28.8%	$\chi^2 = 15.88$**
Education of father:			
Some high school	8.1%	4.2%	
High school graduate	35.5%	26.4%	
Some college	19.4%	16.7%	
College graduate	22.6%	26.4%	
More than college	14.5%	26.4%	$\chi^2 = 4.33$
Education of mother:			
Some high school	3.0%	4.1%	
High school graduate	47.0%	26.0%	
Some college	28.8%	21.9%	
College graduate	13.6%	27.4%	
More than college	7.6%	20.5%	$\chi^2 = 12.19$*

Note. Percentages do not always add up to 100% due to rounding.
*$P \leq 0.05$. **$P \leq 0.01$.

Table 2. Means and group differences in symptomatology

Measure / Group	Pre-test Mean (SD)	Post-test Mean (SD)	Follow-up Mean (SD)
Child Depression Inventory Scores			
Treatment	9.08 (6.7)	7.65 (6.0)	7.76 (6.7)
	($N = 67$)	($N = 61$)	($N = 55$)
Controls	9.94 (6.5)	9.47 (7.3)	10.22 (6.8)
	($N = 70$)	($N = 60$)	($N = 64$)
Reynolds Children's Depression Scale Scores			
Treatment	51.31 (11.2)	46.35 (9.5)	48.18 (10.6)
	($N = 67$)	($N = 61$)	($N = 55$)
Controls	52.59 (11.2)	50.27 (11.1)	50.96 (11.2)
	($N = 70$)	($N = 60$)	($N = 64$)
Explanatory Style (CP CN)			
Treatment	5.55 (3.9)	6.02 (5.3)	6.14 (4.0)
	($N = 67$)	($N = 61$)	($N = 57$)
Controls	4.93 (3.7)	5.66 (4.1)	5.46 (4.8)
	($N = 71$)	($N = 65$)	($N = 65$)
Internalizing Behavior Problems at Home			
Treatment	55.44 (13.4)	50.58 (12.4)	50.32 (10.4)
	($N = 31$)	($N = 31$)	($N = 31$)
Controls	56.21 (11.1)	54.16 (10.7)	52.36 (10.2)
	($N = 38$)	($N = 38$)	($N = 38$)
Externalizing Behavior Problems at Home			
Treatment	53.31 (13.3)	50.65 (11.5)	48.44 (11.7)
	($N = 31$)	($N = 31$)	($N = 31$)
Controls	53.13 (12.9)	51.34 (12.7)	52.09 (11.9)
	($N = 38$)	($N = 38$)	($N = 38$)
Classroom Behavior			
Treatment	4.19 (0.80)	4.38 (0.89)	
	($N = 66$)	($N = 66$)	
Controls	4.38 (0.89)	4.31 (1.07)	N A
	($N = 24$)	($N = 24$)	

Table 3. Topics of prevention program by session

Session	Topic in combined program
1	Pre-test measures
2	Introduction: feelings and thoughts
3	Link between thoughts and feelings
4	Labelling thoughts, considering causes
5	Considering consequences, marital conflict
6	Assertiveness and negotiation
7	Coping strategies
8	Emotion control, scheduling, and decision-making
9	Social problem-solving: 5-step approach
10	Problem-solving review
11	Post-test measures
12	Review, diplomas

on pre-test dependent measures, age, sex, or race (see Tables 1 and 2). However, there were group differences on two indices of socioeconomic status, namely the mother's level of education and the total family income. The control group had significantly higher levels of education and family income than the treatment group.

The Penn Prevention Program

The Penn Prevention Program contains two components: a cognitive component and a social problem-solving component. The central elements of the cognitive component were drawn from traditional cognitive therapy. In particular, this component was based on Ellis' ABC model, which emphasizes that it is beliefs about events rather than the events themselves that generate feelings (Ellis, 1962); and Beck's model which emphasizes challenging negative beliefs about the self, present circumstances, and the future (Beck, 1967; Beck, 1976). This component focuses on instilling a flexible thinking style and on learning to evaluate the accuracy of beliefs.

The cognitive component also contains explanatory style training to help children make more accurate, less pessimistic attributions. Explanatory style is an active ingredient in successful outcome of cognitive therapy with depressed adults (DeRubeis, Evans, Hollon, Garvey, Grove & Tuason, 1990; Seligman, Castellon, Cacciola, Schulman, Luborsky, Ollove & Downing, 1988). Children learned to identify and label causal attributions and to challenge inaccurate, pessimistic explanations. For those situations in which the accurate interpretation of an event was, in fact, a pessimistic one, children are taught to look towards solutions for the problem or ways to cope with the emotions.

The social problem-solving component teaches children goal-setting, perspective-taking, information gathering, generating alternatives for action, decision-making, and self-instruction.

Finally, the program provides direct training on coping with family conflict and other stressors. These coping techniques include de-catastrophizing about potential outcomes of a problem, ways to distance oneself from highly stressful situations, distraction techniques, relaxation training, and ways to seek social support.

The Penn Prevention Program includes in-session instruction with weekly homework assignments which target how children think, feel and react when faced with problems. The group format consisted of in-session activities such as skits and stories to demonstrate the concepts, and group activities are used to practice and reinforce the skills. Participants are also encouraged to complete structured assignments between the sessions. A list of topics by session for the combined program is provided in Table 3.

Distinct and common elements

Both components of the Penn Prevention Program used cognitive techniques. The cognitive training section, however, focused on children's interpretations about problems, while the social problem-solving and coping component focused on the child's actions to solve problems, rather than interpretations.

The distinction between the two parts roughly follows Kendall's (1993) distinction between deficiency and distortion in cognitive processes. While the cognitive training attempts to correct distorted depressogenic cognitions, the social problem-solving and coping training helps build in thought processes to counteract deficient cognitive processing in children who act out.

The two components contain many common elements. For example, both components include assertiveness and negotiation, as well as the building blocks to any cognitively-based program, such as identifying thoughts and understanding the link between cognition and affect or behavior. The two components are conceptually linked and together form the combined prevention program. Because the two parts did not produce major differences in the groups at post-test, they were collapsed and are considered together in evaluating the prevention program in this paper.

Measures

All children completed a battery of questionnaires 3 times (in addition to the screening questionnaires) during this phase of the study. The 3 administrations took place at pre-test (approximately 3 months after the screening), at post-test (3 months after pre-test), and at 6-month follow-up evaluation (6 months after post-test).

At each interval questionnaires were sent to parents, asking them to complete the packet and return it by mail. Behavioral reports on school report cards covering the treatment period for all children in the treatment and wait-list groups were also examined as an independent outcome measure.

Depressive symptoms. Children completed two self-report questionnaires assessing current depressive symptoms: the Children's Depression Inventory (CDI; Kovacs, 1985), as described above, and the Reynolds Children's Depression Scale (RCDS; Reynolds, 1989). Unlike the CDI, the RCDS assesses frequency rather than severity of depressive symptoms. Reynolds reports that the RCDS is both reliable and valid as indicated by its high correlations with other depression measures (Reynolds, 1992). In the present study there was a high correlation between the CDI and RCDS at each assessment point (Pre-test: $r = 0.77$, post-test: $r = 0.62$, follow-up: $r = 0.75$; all P's $\leqslant 0.001$). In order to reduce the number of analyses, these two scales were combined by converting the raw scores to z-scores (based on the pre-test mean and standard deviation) and then summing them. The summed z-scores were used as an overall *depressive symptom score* in all of the main analyses. However, for the sake of clarity, the raw score means and standard deviations of the CDI and RCDS will be presented in Tables and Figures. In addition, since both of these scales assess depressive symptoms only for the past 2 weeks, a *retrospective report of depressive symptoms* was created from the CDI and given to children at the 6-month follow-up. This scale first asks children to identify the period in the past 6 months when they were feeling the worst ("feeling a little more down or sad than usual"), and then asks them to base their answers on that time period. The questions do not differ from the CDI, except that they are in the past tense. This scale enabled us to measure depressive symptoms during the 6-month interval between post-test and follow-up. It should be noted that the retrospective report of depression may have questionable validity, since it is unclear whether children can accurately recall and evaluate depressive symptoms in the past.

Behavioral conduct at home. In order to assess children's behavior at home, parents completed the Child Behavior Checklist (CBCL; Achenbach, 1991), a standard 119-item checklist. On this scale, parents are instructed to indicate if each behavior is 'not true', 'somewhat or sometimes true', or 'very or often true' of their child. The child's T-score is calculated based on sex and age, and contains two components: *internalizing behavior*, including social withdrawal, somatic complaints and depressive symptoms; and *externalizing behavior*, including aggressive, hyperactive and delinquent behavior.

Teacher reports of classroom behavior. Teachers' behavioral reports on the children's report cards were collected for all children who participated in the treatment and wait-list groups. On this measure of *classroom behavior*, teachers indicated 'commendable effort', 'satisfactory', or 'needs improvement' for 6 items, which we grouped into three 2-item subscales: self-discipline (accepts responsibility for self and practices self-discipline); peer relations (shows consideration for others and works well with others); and conduct (follows class and school rules and shows respect for property). Each item was rated on a 3-point scale, creating a possible range of 0–6 for each 2-item scale. Thus, a high score on this measure means better classroom behavior. Scores were calculated for the academic period ending just after the pre-test evaluations, and for the academic period covering the treatment period and ending just after the post-test evaluations. Since the 3 subscales were highly correlated with each other (r's ranging from 0.29 to 0.72; all P's $\leqslant 0.01$), we averaged the 3 subscale scores to create an *overall classroom behavior* score. Teacher reports could not be

used at the 6-month follow-up, because half of the children had moved into the seventh grade where these ratings are no longer used.

Explanatory style. Children also completed the Children's Attributional Style Questionnaire (CASQ; Kaslow, Tannenbaum & Seligman, 1978), a 48-item forced-choice questionnaire which assesses explanatory style for both positive and negative hypothetical events. The questionnaire measures whether attributions made about positive and negative events are stable or unstable, global or specific, and internal or external. Three summary scores were calculated: a composite score for positive events (CP), a composite score for negative events (CN), and an overall score based on the difference between these two (CP–CN). On both CP and CN scales, a higher score means more internal, stable and global explanations. Thus, high scores on CP and on CP–CN indicate an optimistic explanatory style, while high scores on CN indicate a pessimistic explanatory style.

Statistical procedures

This study employed a mixed-method nested design, with unbalanced groups. That is, children were nested within school groups, which were nested within treatment conditions. We followed Hopkins (1982) recommendation that both group and child effects be examined in analyses of covariance (ANCOVA's) or multivariate analyses of covariance (MANCOVA's). This approach results in slightly reduced degrees of freedom but takes into account the role of group effects in treatment outcome. In each analysis, the initial level of depressive symptoms or behavior problems was statistically controlled, and change in the individual child was evaluated as a function of whether the children participated in a treatment or control condition. In addition, school group effects nested within the treatment conditions were controlled.

In the subsequent analyses, one-tailed P-values are reported for between-group analyses when there was a clear, unidirectional prediction that the treatment group would do better than the control group (two-tailed P-values are noted when used).

RESULTS

Attrition

There was some attrition of subjects at post-test and follow-up from both the treatment and control groups. Analyses of the data revealed that there were no differences between treated and control children who dropped out of the project before the post-test evaluation in terms of the pre-test dependent measures. Similarly, the treated and control children who dropped out of the project before the follow-up evaluation did not differ from each other in terms of the dependent measures at pre-test or post-test.

Age and sex effects

There were no main effects of sex or age of the child on the treatment effects discussed below, and no interactions of age or sex with treatment participation were found.

Primary analyses

Depressive symptoms. The results showed significant relief from and prevention of depressive symptoms for children who participated in the treatment groups as compared to controls. In addition to statistically significant changes, qualitative analyses revealed meaningful clinical changes in depressive symptoms.

Children in the treatment group reported significantly fewer depressive symptoms immediately following the program than children in the control group (Relief of Depressive Symptoms—Hypothesis 1). In addition, at the 6-month follow-up, children in the treatment group reported fewer depressive symptoms than did controls. The treatment group also reported fewer depressive symptoms in their retrospective reports of the worst period of depressive symptoms than the control group (Prevention of Depressive Symptoms—Hypothesis 2). Scores on each measure at post-test and follow-up are shown in Table 2, and changes over time are indicated in Table 4.

Specifically, an ANCOVA showed that treatment resulted in fewer depressive symptoms at post-test [$F(1,106) = 3.70$; $P \leq 0.05$]. Similarly, a MANCOVA of follow-up depressive symptom scores and retrospective reports of depressive symptoms showed a significant treatment effect

Table 4. Changes over time in symptoms within treatment and within control groups

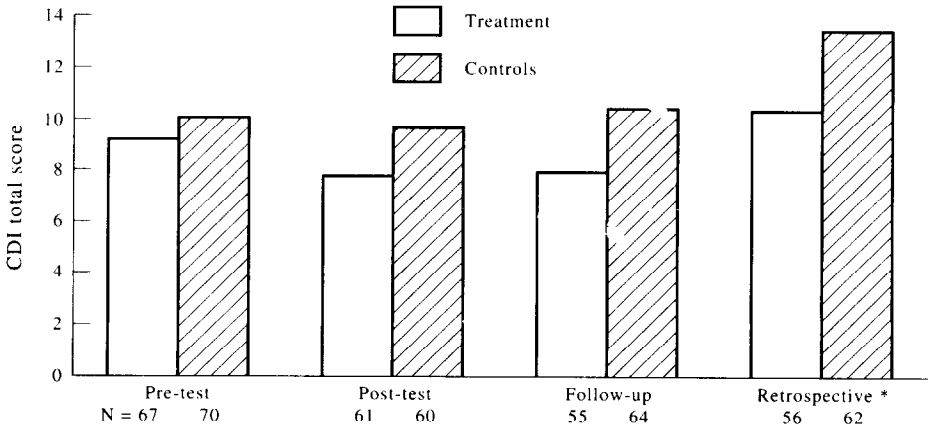
Measure / Group	Pre-test to post-test *df*	Pre-test to post-test *t*-value	Pre-test to follow-up *df*	Pre-test to follow-up *t*-value
Children's Depression Inventory Score				
Treatment	59	2.09*	53	1.19
Controls	58	0.33	59	−0.79
Reynolds Children's Depression Scale Score				
Treatment	58	3.18**	52	1.27
Controls	60	1.60	60	0.18
Explanatory Style (CP−CN)				
Treatment	58	−1.17	54	−1.01
Controls	62	−1.10	61	−0.75
Internalizing Behavior Problems at Home				
Treatment	30	3.29**	30	3.52***
Controls	37	2.10*	37	2.85**
Externalizing Behavior Problems at Home				
Treatment	30	2.08*	28	3.18**
Controls	37	2.37*	37	0.90
Classroom Behavior				
Treatment	65	−2.78**		N/A
Controls	23	0.56		N/A

*$P \leqslant 0.05$; **$P \leqslant 0.01$; ***$P \leqslant 0.001$.

[Wilks' $\lambda = 0.955$, $F(2,100) = 2.34$; $P \leqslant 0.05$]. In all of these analyses depressive symptoms at pre-test were covaried to control for the initial level of symptoms and school group effects were statistically controlled. The composite depressive symptom score was used to reduce the number of analyses. Figure 1 displays the changes in depressive symptoms by group in terms of raw scores on the CDI.

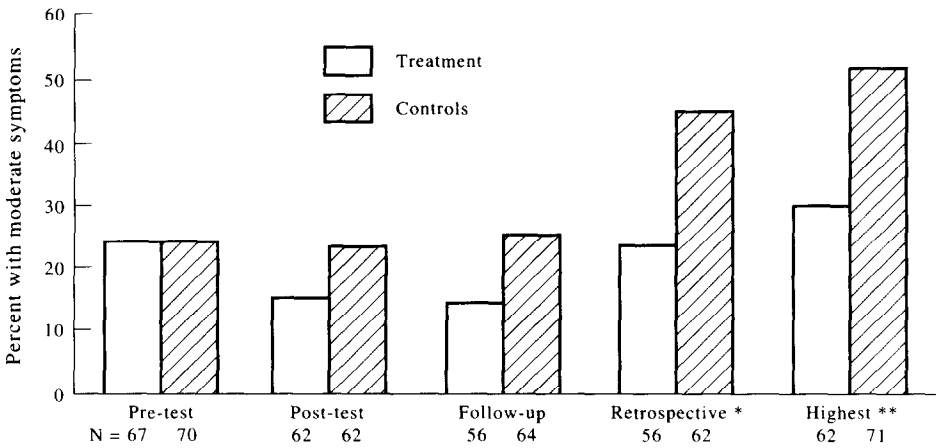
Qualitative changes on the CDI are discussed to better understand the clinically meaningful changes in depressive symptoms. Cut-off scores of 13 or 15 are generally used with the CDI to indicate clinically-relevant levels of symptoms (Reynolds, 1992). A cut-off of 15 indicating 'moderate depressive symptoms' is used in the present study since it is closer to one standard deviation above the pre-test mean in our data (mean = 9.5, SD = 6.6). At pretest, 24% of both treatment and controls were at or above this cutoff (see Fig. 2). At post-test, the percentage was reduced to 15% of treated children vs 23% of controls (Fisher one-tailed exact probability = 0.36). At the follow-up, only 14% of treated children had moderate symptoms, as compared to 25% of controls (Fisher one-tailed exact probability = 0.17). Thus, treatment reduced the number of children in the 'moderate' range of depressive symptoms from 24 to 15%, and this reduction was maintained at follow-up.

In addition, only 23% of treated children but 44% of control children reported on the retrospective report of depressive symptoms a period when they felt at least moderate levels of depressive symptoms during the 6 months after post-test (Fisher one-tailed exact probability



* Retrospective = report of worst 2 weeks between post-test and follow-up

Fig. 1. Depressive symptoms by group.



* Retrospective = report of worst 2 weeks between post-test and follow-up;
** Highest = highest reported score after pre-test

Fig. 2. Percent of children with moderate depressive symptoms by group.

$\leqslant 0.05$). Finally, since depressive symptoms tend to be episodic, the highest level of depressive symptoms reported by each child at post-test, follow-up, or in the retrospective reports was also examined. This measure captures the worst report of depressive symptoms, regardless of how long after the treatment period they occurred. Of children in the treatment group, 29% reported at least moderate depressive symptoms at some point after treatment, as compared to 51% of controls (Fisher one-tailed exact probability $\leqslant 0.05$). In the treatment group, the average highest report of depressive symptoms was 11.9 (SD = 8.0), while the control group reported a significantly higher level of depressive symptoms with a mean of 15.0 (SD = 7.4) ($t = -2.36$; $P \leqslant 0.01$).

Conduct problems. Overall, results did not support the immediate relief of conduct problems at home among children in the treatment group. At post-test, a MANCOVA of internalizing and externalizing behavioral problems revealed no treatment effect when the initial level of problems and school group effects were controlled [Wilks' $\lambda = 0.982$; $F(2,59) = 0.55$, NS]. These results are displayed graphically in Fig. 3 and the means T-scores are reported in Tables 2 and 4.

At follow-up, a MANCOVA of externalizing and internalizing behavioral problems also failed to show an overall treatment effect in reducing behavioral problems in the treatment group [Wilks' $\lambda = 0.893$; $F(2,59) = 1.71$, NS, see Fig. 3], controlling for pre-test levels of problems and school-group effects. Whereas there is no definitive support for the overall hypothesis that behavior problems would be prevented, follow-up ANCOVA's showed that parents reported fewer externalizing conduct problems in the treated children than controls [$F(1,60) = 3.02$, $P \leqslant 0.05$), but that

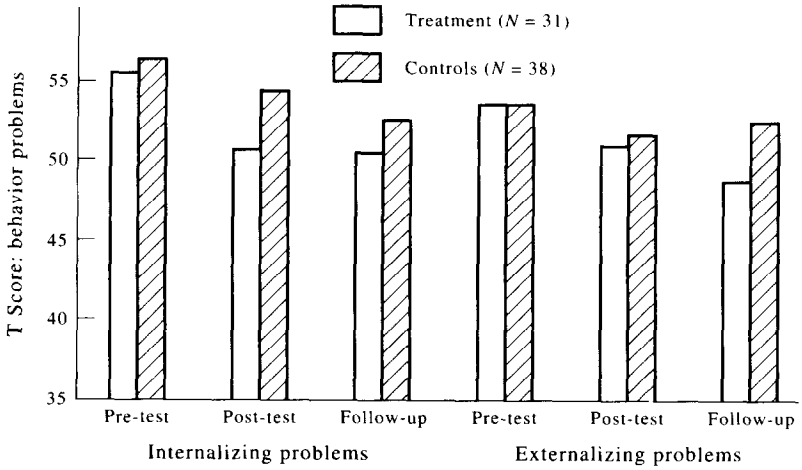


Fig. 3. Parent reports of behavior problems by group.

there was no significant effect for internalizing behavior problems at follow-up [$F(1,60) = 0.41$, NS].

Hypothesis 5, predicting improved classroom behavior, was supported. Teachers reported significantly better classroom behavior in participants than in controls at post-test [$F(1,81) = 3.41$, $P \leqslant 0.05$]. Since no teacher reports were available at follow-up, prevention of classroom behavioral problems could not be tested.

There are two caveats offered regarding the conduct data. First, compliance was low. Only 42% of parents of treated children and 52% of parents of controls returned the questionnaires at all three intervals. The preceding ANCOVA's, therefore, were based on the reduced sample of the children whose parents completed the CBCL at all three assessment points in order to make the results more clear. It is important to note, however, that there was no significant difference at pre-test on depressive symptoms or teacher ratings of conduct between the group of children whose parents completed the questionnaires and those whose parents did not. Second, the teacher reports were unavailable in the no-participation control group reducing the control group size from 74 to 24.

Secondary analyses

Explanatory style. Contrary to our expectations, children in the treatment groups did not show any changes in the composite explanatory style scores as a result of treatment. There were no group differences at any of the three assessments, and no significant changes in either group over time (see Table 5). Because the negative-stable dimension of explanatory style is both theoretically and empirically the most important dimension of the reformulated learned-helplessness theory of depression, we looked at changes in this dimension specifically (Alloy, Abramson, Metalsky & Hartlage, 1988; Seligman, 1991). Children who participated in the treatment program were less likely to attribute negative events to stable, enduring causes at the end of treatment [$F(1,94) = 5.48$; $P \leqslant 0.05$] and at follow-up [$F(1,99) = 6.49$, $P \leqslant 0.05$, see Table 6]. These analyses controlled for both pre-test scores in explanatory style and school-group effects.

The change in the tendency to attribute a stable cause to negative events was investigated as a possible mediator of the treatment effect on depression. Children's ability to explain negative events with less stable explanations at the end of treatment was significantly associated with the decrease in

Table 5. Means and group differences in explanatory style: composite scores

Measure / Group	Pre-test Mean (SD)	Post-test Mean (SD)	Follow-up Mean (SD)
Composite Score (CP–CN)			
Treatment	5.55 (3.9)	6.02 (5.3)	6.14 (4.0)
	($N = 67$)	($N = 61$)	($N = 57$)
Controls	4.93 (3.7)	5.66 (4.1)	5.46 (4.8)
	($N = 71$)	($N = 65$)	($N = 65$)
Composite of Negative Events (CN)			
Treatment	7.49 (2.6)	7.34 (3.3)	6.69 (2.8)
	($N = 67$)	($N = 61$)	($N = 57$)
Controls	7.80 (2.5)	7.83 (2.5)	7.75 (3.0)
	($N = 71$)	($N = 65$)	($N = 65$)
Composite of Positive Events (CP)			
Treatment	13.04 (2.7)	13.36 (3.6)	12.99 (3.5)
	($N = 67$)	($N = 61$)	($N = 57$)
Controls	12.80 (2.6)	13.49 (3.0)	13.20 (3.3)
	($N = 71$)	($N = 65$)	($N = 65$)

Table 6. Means and group differences in explanatory style: individual dimensions

Measure / Group	Pre-test Mean (SD)	Post-test Mean (SD)	Follow-up Mean (SD)
Negative events			
Stable			
Treatment	2.64 (1.5)	2.22 (1.5)	1.80 (1.4)
	($N = 67$)	($N = 61$)	($N = 57$)
Controls	2.39 (1.2)	2.51 (1.5)	2.43 (1.4)
	($N = 71$)	($N = 65$)	($N = 65$)
Internal			
Treatment	2.74 (1.5)	2.76 (1.7)	2.71 (1.6)
	($N = 67$)	($N = 61$)	($N = 57$)
Controls	2.90 (1.4)	3.08 (1.7)	3.09 (1.8)
	($N = 71$)	($N = 65$)	($N = 65$)
Global			
Treatment	2.11 (1.3)	2.30 (1.4)	2.18 (1.1)
	($N = 67$)	($N = 61$)	($N = 57$)
Controls	2.48 (1.5)	2.30 (1.3)	2.23 (1.5)
	($N = 71$)	($N = 65$)	($N = 65$)
Positive events			
Stable			
Treatment	3.88 (1.4)	4.15 (1.7)	3.97 (1.8)
	($N = 67$)	($N = 61$)	($N = 57$)
Controls	3.94 (1.6)	3.96 (1.7)	3.79 (1.6)
	($N = 71$)	($N = 65$)	($N = 65$)
Internal			
Treatment	4.81 (1.5)	4.74 (1.4)	4.92 (1.5)
	($N = 67$)	($N = 61$)	($N = 57$)
Controls	4.73 (1.3)	4.94 (1.3)	5.07 (1.3)
	($N = 71$)	($N = 65$)	($N = 65$)
Global			
Treatment	4.31 (1.3)	4.48 (1.6)	4.09 (1.6)
	($N = 67$)	($N = 61$)	($N = 57$)
Controls	4.06 (1.3)	4.52 (1.5)	4.33 (1.6)
	($N = 71$)	($N = 65$)	($N = 65$)

depressive symptoms at both post-test and follow-up. In both cases ANCOVA's controlling for pre-test levels of depressive symptoms and school-group effects and examining a single independent variable (either treatment participation or the change in explanatory style) showed that both changes in explanatory style and treatment were significantly associated with a reduction in depressive symptoms at post-test and follow-up. However, when both independent variables were entered into the same ANCOVA, the change in explanatory style was a significant predictor of decreases in depression, whereas the treatment variable became non-significant. Examination of the changes in R^2 showed that the unique variance associated with the change in the negative-stable dimension accounted for a significant proportion of the variance in change in depressive symptoms, even when treatment participation was controlled [$F(1,98) = 4.99$, $P \leqslant 0.05$ at post-test; $F(1,103) = 5.29$, $P \leqslant 0.05$ at follow-up]. On the other hand, when the explanatory style change was controlled, the unique variance associated with treatment participation failed to account for a significant proportion of the variance in depression at either post-test or follow-up [$F(1,98) = 1.61$ and $F(1,103) = 2.11$, respectively]. Thus, it appears that children's ability to attribute negative events to more temporary causes mediated the impact of treatment in decreasing depressive symptoms.

As expected, the negative-stable dimension of explanatory style was unrelated to changes in classroom behavior at post-test, or the effect on externalizing behavior at follow-up. In all cases, the treatment variable remained a significant predictor of changes in behavior when the change in explanatory style was controlled.

High- and low-parental conflict groups. There were better treatment effects for those children who were experiencing more parental conflict at home, as measured by their reports at the screening phase of the study. The sample was divided into two groups at the median for parental conflict. Whereas children in the high-conflict group showed reduced depressive symptoms as a function of treatment immediately after the program [$F(1,55) = 5.04$, $P \leqslant 0.05$] and on the two follow-up measures [Wilks' $\lambda = 0.869$; $F(2,51) = 3.83$, $P \leqslant 0.05$], there was no treatment effect in the low-conflict group [$F(1,55) = 0.36$ and $F(2,52) = 0.50$, respectively]. There was a similar pattern of results for internalizing symptoms, where parents of treated children reporting high parental conflict reported fewer internalizing symptoms as compared to controls at the end of treatment [$F(1,38) = 2.99$, $P \leqslant 0.05$], but this did not remain significant at follow-up [$F(1,36) = 1.66$, NS]. There was no treatment effect on internalizing symptoms among children reporting low parental conflict [$F(1,37) = 1.01$ and $F(1,36) = 0.00$, respectively]. Interestingly, a different pattern was found for externalizing symptoms. There was no treatment effect for children experiencing a high degree of parental conflict at home at post-test or follow-up [$F(1,38) = 0.04$ and $F(1,36) = 1.68$, both NS], and no immediate treatment effect for children reporting low levels of parental conflict [$F(1,37) = 0.10$, NS]. However, there was a significant treatment effect at follow-up for children from homes with low parental conflict, where parents reported reduced externalizing behavioral problems in treated children as compared to controls [$F(1,36) = 3.12$, $P \leqslant 0.05$]. In the analyses reported in this section, pre-test levels of symptoms were controlled, but the nested school-group effects were not controlled because of the substantial reduction in degrees of freedom in relation to the split-group number of children.

Although there appears to be a somewhat stronger pattern of results among those children who were experiencing family conflict at home, there were no significant interactions between level of parental fighting and treatment for any of the measures discussed above.

High- and low-symptomatic groups. Since children entered the program with different levels of symptoms, the sample was divided into high- and low-symptomatic groups, in order to see which children benefitted most from the prevention program.

First, the sample was split at the median depressive symptom score at pre-test. In the high-symptom group, the treated group showed a trend towards fewer symptoms at post-test, and significantly reduced depressive symptoms at follow-up. Specifically, an ANCOVA failed to show any significant treatment effect in the high symptom group between pre-test and post-test, since depressive symptoms decreased significantly in both groups [$F(1,55) = 1.62$, NS]. This change in both groups may partly reflect a drift towards the mean or spontaneous remission of symptoms. A MANCOVA showed that treatment significantly prevented symptoms at follow-up and in retrospective reports [Wilks' $\lambda = 0.866$; $F(2,50) = 3.88$, $P \leqslant 0.05$].

In the low symptom group, there was a trend towards prevention of depressive symptoms, but this trend was not significant. An ANCOVA of depressive symptoms at post-test, controlling for pre-test levels, showed a non-significant treatment effect [$F(1,55) = 2.02$, NS], and a MANCOVA of the two depressive symptom measures at follow-up also failed to show a treatment effect [Wilks' $\lambda = 0.977$; $F(2,53) = 0.62$, NS]. This group, not surprisingly, had a much lower rate of depressive symptoms at each interval. We will have to wait for longer term follow-up data in order to evaluate prevention among children with few depressive symptoms. Again, these analyses controlled for pre-test scores but not for the school-group effects.

As with the parental conflict split, there appeared to be a stronger pattern of results in the group experiencing more symptoms at pre-test. However, tests of the interaction between symptom level and treatment participation failed to show any significant interactions. Thus, while it does not appear that the treatment is differentially more effective in the group experiencing more symptoms.

DISCUSSION

The Penn Prevention Program relieved depressive symptoms immediately after treatment. More importantly, the treated children had fewer depressive symptoms than controls at the 6-month follow-up. While behavioral problems were not reduced overall, externalizing behavior problems reported by parents were reduced in the treatment group 6 months after the program ended. We interpret the difference in depressive symptoms between treated and untreated groups at post-test to mean that treatment relieved ongoing symptoms, and interpret the differences at follow-up to mean that the treatment prevented depressive symptoms and externalizing behavior problems from recurring.

We hypothesize that children in the treatment group used the skills they had learned in the Penn Prevention Program after the program was over to fend off depressive symptoms in the wake of bad events. For instance, we found evidence that treated children's increased ability to attribute temporary (unstable) causes to negative events mediated the treatment effect in relief and prevention of depressive symptoms. While the causal direction of these relationships remains unclear, it does appear that explanatory style change, at least for the negative-stable dimension, may be as important an ingredient in therapy for children as has been previously found for adults. This study lends support to the view that the negative-stable dimension of explanatory style may have an important etiologic or maintaining role in the expression of depressive symptoms, and is not merely another symptom. Other researchers have also found a mediational role for explanatory style in the development of depressive symptoms (Cole & Turner, 1993).

Since explanatory style was unrelated to changes in behavioral problems, what caused the relief and prevention of behavior problems? Children who participated in the prevention groups appeared to gain a much better sense of how to handle problems, especially interpersonal problems, by the end of the program (Jaycox, 1994). They also appeared to be more confident in their ability to deal with conflict. Parents' and children's responses on evaluations of the Penn Prevention Program support these ideas, with both parents and children noting improvements in the child's ability to handle problems at the end of the program.

There have been several other early intervention projects that reported decreases in depressive symptoms in children or adolescents and maintenance of those gains following the intervention. These intervention studies targeted children who were moderately depressed (Butler *et al.*, 1980; Kahn *et al.*, 1990; Reynolds & Coats, 1986) or in the moderately to severely depressed range (Lewinsohn *et al.*, 1990; Stark *et al.*, 1987; Stark *et al.*, 1991). In contrast to these studies, children in the present study were identified as at-risk for depression by measuring current depressive symptoms as one risk factor and parental conflict as the other. Thus, we attempted to identify children at risk for depression proactively, although about a quarter of the children in the program did have symptoms which would put them in the range of 'moderate' depressive symptoms.

In addition, these intervention studies ranged from having no follow-up (Butler *et al.*, 1980), to a follow-up of 4–8 weeks (Kahn *et al.*, 1990; Reynolds & Coats, 1986; Stark *et al.*, 1987), with two exceptions. First, a study of treatment for adolescent depression showed a trend towards continued improvement up to 2 years after treatment (Lewinsohn *et al.*, 1990). In addition, Stark and colleagues conducted a 7 month follow-up of cognitive-behavioral treatment for depressed children

as compared to traditional counseling, but the results indicated the cognitive-behavioral group no longer differed from the traditional counseling group at 7 months follow-up (Stark *et al.*, 1991). Our results show successful prevention a full 6 months after the end of the program. Given the results of this study together with evidence of prevention of depression in adolescents (Clark *et al.*, 1993), a cognitive-behavioral approach to prevention appears quite promising.

Recognizing the overlap between depressive symptoms and conduct problems, we included techniques geared towards both. The results of this study support this broad approach: both depressive symptoms and behavior problems were impacted through treatment.

The treatment effects were somewhat stronger, among the children who entered the program with relatively more depressive symptoms, and among those who reported a higher level of parental conflict at home. The effects in high-depressive symptom group are most comparable to effects in studies of moderately depressed children or clinically referred children cited above. However, it is important to note that no attempt was made to diagnose depression in the identified children, and that the present study speaks only to alleviation and prevention of depressive symptoms as opposed to a depressive disorder. There are at least three plausible explanations for why symptomatic children would benefit most from the program. First, there may be a floor effect. That is, there is simply more room for change in children who have more problems to begin with. Second, those children who had the most problems may have been more motivated to learn the skills. Third, those children currently experiencing problems may have had more opportunities to practice and use the skills.

Prevention effects in the low symptom group remain unclear as yet. Children who entered the project with few depressive symptoms remained relatively symptom-free, regardless of whether they were in the treated or control group. Low symptom children show fewer symptoms across time, so the longer-term follow-up data, which tracks them as they enter adolescence, will be necessary to evaluate the prevention effects for this group of children. The reader is referred to Gillham, Reivich, Jaycox and Seligman (1994) for the results of the long-term follow-up data.

Since there was no placebo control-group in this study, it is not possible to rule out non-specific effects, such as expectation of gains, group cohesion, or adult attention, to explain the observed changes in symptomatology, as found in other studies (Fine, Forth, Gilbert & Haley, 1991; Reynolds & Coats, 1986). A more thorough investigation of this prevention program, using random assignment and a placebo control group, is clearly warranted. Another limitation of our treatment is that it was conducted after school and by members of our research team. Thus, the skills taught were not modeled for the child by a significant figure in his or her life, such as a parent or teacher. In future versions of this program, we hope to involve parents and teachers in order to provide the child with a role-model who can demonstrate the skills on a daily basis. This type of ongoing modeling and feedback may boost and strengthen the long term effects.

In conclusion, by targeting the presumed cognitive underpinnings of depression and associated behavioral problems, we were able to relieve and prevent depressive symptoms and behavior problems in school children. We believe that the skills taught in our program can be delivered by parents and teachers. It is our hope that the prevention of depressive symptoms and conduct problems for school children will now be done on a broad scale, using our techniques, to help alleviate a major public health problem.

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