

The Prevention of Depressive Symptoms in Children and Adolescents: A Meta-Analytic Review

Jason L. Horowitz and Judy Garber
Vanderbilt University

Research on the prevention of depressive symptoms in children and adolescents was reviewed and synthesized with meta-analysis. When all 30 studies were included, selective prevention programs were found to be more effective than universal programs immediately following intervention. Both selective and indicated prevention programs were more effective than universal programs at follow-up, even when the 2 studies with college students were excluded. Effect sizes for selective and indicated prevention programs tended to be small to moderate, both immediately postintervention and at an average follow-up of 6 months. Most effective interventions are more accurately described as treatment rather than prevention. Suggestions for future research include testing potential moderators (e.g., age, gender, anxiety, parental depression) and mechanisms, designing programs that are developmentally appropriate and gender and culturally sensitive, including longer follow-ups, and using multiple measures and methods to assess both symptoms and diagnoses.

Keywords: depression, prevention, meta-analysis, adolescents, children

Depression during childhood and adolescence is a significant public health concern, affecting about 1% to 2% of prepubertal children and about 3% to 8% of adolescents (Costello et al., 1996; Kovacs, 1996; Lewinsohn, Clarke, Seeley, & Rohde, 1994). Child and adolescent depression has a chronic, episodic course and is associated with many negative outcomes, including substance abuse, academic problems, cigarette smoking, high-risk sexual behavior, physical health problems, impaired social relationships, and a thirty-fold increased risk of completed suicide (Birmaher et al., 1996; Brent et al., 1988; Le, Munoz, Ippen, & Stoddard, 2003; Rohde, Lewinsohn, & Seeley, 1994; Stolberg, Clark, & Bongar, 2002). In addition, early onset depression increases the risk of subsequent depressive episodes later in adolescence and adulthood, with recurrence rates ranging from 45% to 72% over 3 to 7 years (Emslie et al., 1997; Harrington, Fudge, Rutter, Pickles, & Hill, 1990; Lewinsohn, Rohde, Klein, & Seeley, 1999; Rao, Hammen, & Daley, 1999; Weissman et al., 1999).

Because of the high costs associated with pediatric depression, the past 10 years has seen a growing interest in its prevention. This trend has been catalyzed by both a mandate issued by the Institute of Medicine (Mrazek & Haggerty, 1994) and a natural downward extension of treatment research (Gladstone & Beardslee, 2000). The Institute of Medicine report classified prevention programs into three distinct categories on the basis of the populations to whom the interventions are directed. Universal preventive interventions are administered to all members of a target population. Selective prevention programs are given to members of a subgroup of a population whose risk is deemed to be above average. Indicated preventive interventions are provided to individuals who manifest subclinical signs or symptoms of a given disorder.

Universal interventions for preventing depression typically have been conducted in schools and have included as many as 1,500 children (Spence, Sheffield, & Donovan, 2003). The format usually has involved large-group presentations or curricular modifications. General strengths associated with universal interventions include avoiding the stigma of singling out individuals for treatment and relatively low dropout rates (Spence et al., 2003). Universal prevention programs with adolescents (e.g., Clarke, Hawkins, Murphy, & Sheeber, 1993) have focused on cognitive and behavioral skills training, including cognitive restructuring, anxiety management, relaxation, problem-solving skills, emotion-focused coping, anticipating consequences, and assertiveness. Universal interventions with elementary school-age children (e.g., Ialongo et al., 1999; Kellam et al., 1994) have sought to prevent depression by implementing mastery learning and behavioral management programs.

Selective interventions target individuals at elevated risk for depression as a function of family factors such as divorce (Gwynn & Brantley, 1987; Wolchik et al., 1993), parental death (Sandler et al., 1992), parental depression (Beardslee et al., 1997), or parental alcoholism (Roosa et al., 1989), environmental factors such as

Jason L. Horowitz and Judy Garber, Department of Psychology and Human Development, Vanderbilt University.

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Correspondence concerning this article should be addressed to Jason L. Horowitz or Judy Garber, Department of Psychology and Human Development, Peabody 512, Vanderbilt University, 230 Appleton Place, Nashville, TN 37203-5721. E-mail: jason.horowitz@vanderbilt.edu or judy.garber@vanderbilt.edu

poverty (Cardemil, Reivich, & Seligman, 2002), or personal characteristics such as a negative cognitive style (Seligman et al., 1999). Because selective samples tend to be diverse, the interventions have been more varied than universal or indicated prevention programs, and they typically target multiple outcomes in addition to reducing depression. Still, there is some similarity across studies in that most have used some form of cognitive-behavioral techniques. These selective intervention programs also have tended to be implemented with smaller groups than have universal programs, and therefore the studies have involved fewer participants.

Indicated interventions are conducted with individuals who already show subclinical signs and symptoms of depression. This adds a step to the recruitment process, as potential participants must qualify for the program through a depression screening procedure. Although this may be more time- and cost-consuming initially, it ensures that those receiving the intervention are at greatest risk. Implementation of the programs has been similar to that of selective interventions, with the norm being a small-group format. The majority of these interventions (e.g., Clarke et al., 1995, 2001; Jaycox, Reivich, Gillham, & Seligman, 1994) have taught cognitive techniques, such as developing a more flexible thinking style and making more realistic, less pessimistic attributions. Such programs also commonly teach problem-solving skills, goal setting, perspective taking, information gathering, and decision making. One study (Forsyth, 2001) implemented an interpersonally oriented program, which focused on the resolution of role disputes and working through role transitions. Table 1 presents a summary of characteristics of all studies reviewed in the current meta-analysis.

Several qualitative reviews have summarized and synthesized findings across studies of interventions aimed at preventing depression in youth (Garber & McCauley, 2002; Gillham, Shatté, & Freres, 2000; Munoz, Le, Clarke, & Jaycox, 2002). Such reviews, however, are not able to address questions about effect size. Differences in sample sizes can make similar effect sizes significant in some cases but nonsignificant in others. Moreover, effect sizes can vary widely among those studies that find significant results. A meta-analysis can amalgamate effect sizes from different studies with various numbers of participants to allow more precise conclusions.

Meta-analysis has been used to aggregate data on prevention programs for various problems in childhood and adolescence such as substance abuse (Cuijpers, 2002; Gottfredson & Wilson, 2003), behavioral and social problems (Durlak & Wells, 1997), HIV transmission (Albarracin et al., 2003), and suicide (Dew, Bromet, Brent, & Greenhouse, 1987). In particular, meta-analyses have provided information regarding whether programs should target high-risk or universal samples (Gottfredson & Wilson, 2003). One meta-analysis included some depression prevention programs for children and adolescents (Jané-Ilopis, Hosman, Jenkins, & Anderson, 2003), although it also included treatment studies, studies that targeted the improvement of protective factors for depression or mental illness, and studies that aimed to reduce risk factors related to depression. Moreover, Jané-Ilopis et al. used multiple effect sizes from the same study and effect sizes from adult samples but did not review the majority of the studies of children and adolescents included in the current meta-analysis. Thus, in contrast to the earlier meta-analysis by Jané-Ilopis et al., the present article specifically targeted programs aimed at preventing depressive symptoms in children and adolescents, using only one effect size from the same sample.

In addition to comparing effect sizes across studies, meta-analytic reviews allow for the examination of other characteristics that can influence effect sizes. Because there is some evidence that boys and girls respond differently to different types of preventive interventions (e.g., Reivich, 1996), the current meta-analysis examined sex differences. In addition, given the preponderance of cognitive approaches to preventing depression, the effect of age was investigated because children of different ages, with diverse cognitive abilities, may not respond the same way. Following the definition of adolescence as the second decade of life (Steinberg & Lerner, 2004), we included depression prevention studies with participants through age 20. In addition, we examined two other variables that could influence the effects of the interventions: length of the programs and length of the follow-ups. It is possible that some prevention programs would be more effective if they continued for a longer period of time (e.g., Clarke et al., 1993) or that the effects of the program will only become apparent after a sufficiently long follow-up period, during which changes in depressive symptoms would be expected to occur (e.g., Gillham, Reivich, Jaycox, & Seligman, 1995).

Finally, although a meta-analysis can compare effect sizes across studies, any given study can produce a significant effect size in multiple ways. An increase in depressive symptoms in the control group and no change in symptoms in the intervention group could yield an effect size identical to that produced by a decrease in symptoms in the intervention group and no change in the control group. However, these two patterns of results would be interpreted quite differently.

Gillham et al. (2000) suggested that the term *prevention* be reserved for those programs that result in a diminished expected increase in symptoms or disorders relative to controls, whereas studies that result in a decline in the level of depression relative to controls should be referred to as *treatment*. Other researchers (e.g., Cardemil et al., 2002) have referred to effects observed immediately after intervention as treatment and those observed at follow-up as prevention. In general, prevention studies have not addressed this issue, and a meta-analysis alone cannot be used to make such distinctions. Therefore, to differentiate between prevention effects and treatment effects, we examined the trajectories of depressive symptoms for both the intervention and control groups for each of the studies that provided such data.

In summary, the current article assessed the efficacy of 30 studies aimed at preventing depressive symptoms in children and adolescents and used meta-analysis to examine their relative effect sizes. In particular, we compared the differential efficacy among universal, selective, and indicated prevention programs. We also explored potential moderators including age, sex, length of intervention, and length of follow-up. Finally, we examined whether the effects produced by the interventions are better characterized as treatment or prevention, and we recommend several directions for future research.

Method

Search Procedures

Three methods of obtaining relevant studies were used. First, a computer search of PsycINFO for all years in the database was conducted. The
(text continues on page 408)

Table 1
 Summary of Descriptive Characteristics and Results of Studies

Study	Type	Sample	<i>N</i>	Age (years)	% Female	Length of intervention	Post-intervention effect size	Effect size at follow-up closest to 6 months	Effect size at last follow-up	Summary of intervention
Clarke et al. (1993), Study 1	U	9th and 10th graders	662	<i>M</i> = 15.4	42.2%	Three 50-min sessions in consecutive health classes	0.06	-0.06 (3 month)	-0.06 (3 month)	Two educational lectures and one videotape describing symptoms, causes, and treatments of depression
Clarke et al. (1993), Study 2	U	9th and 10th graders	380	<i>M</i> = 15.1	46%	Five 50-min sessions in consecutive health classes	0.09	0.14 (3 month)	0.14 (3 month)	Depression education and behavioral training: Increase pleasant activities; chart relation between mood and activities
Kellam et al. (1994)	U	1st graders	575	4.7-9.4; <i>M</i> = 6.3	49%	Continual implementation of curricular alterations over school year	-0.01	— ^a	— ^a	Mastery learning program to improve reading competence: Group-based approach to mastery and a more flexible corrective process
Hains & Ellmann (1994)	U	High school volunteers	21	NR	76%	4 group and 9 individual 50-min sessions	0.36	-0.04 (2 month)	-0.04 (2 month)	Stress inoculation training using cognitive behavioral strategies: Cognitive restructuring, problem solving, anxiety management
Cecchini (1997); Johnson (2000)	U	5th graders	100	NR	NR	Eight 50-min group sessions two times a week	0.11	-0.15 (12 month)	-0.15 (12 month)	Improve interpersonal relationships, social skills, strategies for erasing negative thoughts; mood monitoring
Petersen et al. (1997)	U	6th-9th graders	335	NR	NR	Sixteen 40-min group sessions	-0.12	NA	NA	Penn State Adolescent Study: Teach adaptive emotional, cognitive, and behavioral stress responses

(table continues)

Table 1 (continued)

Study	Type	Sample	<i>N</i>	Age (years)	% Female	Length of intervention	Post-intervention effect size	Effect size at follow-up closest to 6 months	Effect size at last follow-up	Summary of intervention
Ialongo et al. (1999)	U	1st graders	678	NR	46%	Continual implementation of curricular alterations over school year	NA	NA	NA	Classroom centered program: Curriculum changes, improve behavior management strategies; family-school partnership training for teachers and parents
Pattison & Lynd-Stevenson (2001)	U	5th and 6th graders	66	9–12; <i>M</i> = 10.4	52%	10 weekly 2-hr group sessions	–0.01	0.40 (8 month)	0.40 (8 month)	Penn Prevention Program: One group with cognitive component first, one with social component first
Lowry-Webster et al. (2001)	U	5th–7th grade Australian students	594	10–13	53%	Ten weekly 1-hr group sessions	0.17	— ^a	— ^a	A family-based group cognitive-behavioral program targeting anxiety: Teaches physiological, cognitive, and behavioral coping; teaches parents child management, discipline skills
Shochet et al. (2001)	U	Year 9 ^b Australian students	260	12–15; <i>M</i> = 13.5	53%	RAP-A; Eleven weekly 40–50-min group sessions RAP-F: 3+ parent sessions	0.39	0.25 (10 month)	0.25 (10 month)	Resourceful Adolescent Program: School-based resilience program using both a cognitive-behavioral and an interpersonal approach; family program includes parallel parent education
Spence et al. (2003, 2005)	U	Grade 8 Australian students	1,500	12–14; <i>M</i> = 12.9	52%	Eight weekly 45-min sessions	0.29	0.03 (12 month)	0.03 (48 month)	Problem Solving for life program: School-based program teaching cognitive restructuring and problem-solving skills
Merry et al. (2004)	U	Years 9 and 10 ^b students in New Zealand	364	13–14; <i>M</i> = 14.2	52%	Eleven sessions conducted in school	0.02	–0.13 (6 month)	0.05 (18 month)	Adaptation of Resourceful Adolescent Program for children in New Zealand

Table 1 (continued)

Study	Type	Sample	N	Age (years)	% Female	Length of intervention	Post-intervention effect size	Effect size at follow-up closest to 6 months	Effect size at last follow-up	Summary of intervention
Gwynn & Brantley (1987)	S	Children of divorced parents	60	9–11	50%	Eight weekly group sessions	1.37	— ^a	— ^a	Educational support group: Divorce education, encouragement of emotional expression, and problem-solving skills training
Roosa et al. (1989)	S	Children of alcoholics	81	9–13; M = 10.3	50%	8 weekly group sessions	0.41	— ^a	— ^a	Education about alcoholism, activities to improve self-esteem, and emotion-focused coping strategies
Sandler et al. (1992)	S	Children whose parent died less than 2 years ago	72	7–17; M = 12.4	49%	9 family and 6 parent-only sessions	0.24	— ^a	— ^a	Family bereavement program: Grief workshop, family advisement program targeting parental demoralization, parental warmth, stable positive events, and stress management
Wolchik et al. (1993)	S	Children of divorced parents	94	8–15; M = 10.6	39%	2 individual and 10 weekly group sessions	–0.06	— ^a	— ^a	Parent-only intervention: Improve the mother-child relationship, teach discipline skills, schedule positive activities, improve child's contact with father
Beardslee et al. (1997)	S	Children of parents with an affective disorder	52	8–15; M = 11.5	40%	6–10 meetings with parents, child, or both	0.20	0.42 (18 month)	0.42 (18 month)	Cognitive education program: Increase understanding within family, educate about mood disorders; control condition received two 1-hr lectures
Seligman et al. (1999)	S	College freshmen with low ASQ scores	235	NR	52%	8 weekly 2-hr group sessions and 6 individual sessions over next 2 years	0.32	0.12 (6 month)	0.25 (36 month)	Cognitive-behavioral program: Cognitive restructuring, empirical hypothesis testing, behavioral activation; and interpersonal skills training

(table continues)

Table 1 (continued)

Study	Type	Sample	<i>N</i>	Age (years)	% Female	Length of intervention	Post-intervention effect size	Effect size at follow-up closest to 6 months	Effect size at last follow-up	Summary of intervention
Quayle et al. (2001)	S	7th and 8th grade Australian girls	47	11–12	100%	8 weekly 80-min sessions	–0.62	0.62 (6 month)	0.62 (6 month)	Adaptation of Penn Prevention Program for Australian children
Cardemil et al. (2002), Study 1	S	Low-income Latino children	49	<i>M</i> = 11.3	45%	Twelve weekly 90-min group sessions	0.99	1.24 (6 month)	1.24 (6 month)	Modified Penn Resiliency Program: Changed ethnicity of children in examples, focused on problems specific to low-income families, single-parent homes, and managing interpersonal conflict
Cardemil et al. (2002), Study 2	S	Low-income African American children	106	<i>M</i> = 10.9	55%	Twelve weekly 90-min group sessions	0.16	0.31 (6 month)	0.31 (6 month)	Modified Penn Resiliency Program: Changed ethnicity of children in examples, focused on problems specific to low-income families, single-parent homes, and managing interpersonal conflict
Jaycox et al. (1994); Gillham et al. (1995)	I	Children with depressive symptoms and/or family conflict	143	10–13; <i>M</i> = 11.4	46%	Twelve weekly 90-min group sessions	0.18	0.32 (6 month)	0.20 (36 month)	Penn Prevention Program: Cognitive component teaches link between thoughts and feelings; social problem-solving component teaches goal setting, perspective taking, decision making, generation of action alternatives
Clarke et al. (1995)	I	Children with depressive symptoms	150	<i>M</i> = 15.3	70%	Fifteen 45-min group sessions conducted three times a week	0.31	–0.07 (6 month)	–0.01 (12 month)	Cognitive-behavioral program: Identifying and challenging automatic negative thoughts and development of effective coping strategies

Table 1 (continued)

Study	Type	Sample	<i>N</i>	Age (years)	% Female	Length of intervention	Post-intervention effect size	Effect size at follow-up closest to 6 months	Effect size at last follow-up	Summary of intervention
Reivich (1996); Shatté (1996)	I	Children with depressive symptoms	152	12–14; <i>M</i> = 12.7	47%	Twelve weekly 2-hr group sessions	0.12	0.40 (4, 8 month)	0.22 (12 month)	Penn Optimism Program: Identical to the Penn Prevention Program; Penn Enhancement Program: Affect-focused program with emphasis on emotional expression
Lamb et al. (1998)	I	Rural high school students	41	14–19; <i>M</i> = 15.8	56%	Eight weekly sessions	0.70	— ^a	— ^a	Cognitive skills program: Coping, problem-solving, and communication skills
Forsyth (2001)	I	College students with depressive symptoms	59	18–25; <i>M</i> = 19.4	97%	Four group sessions	1.51	1.95 (12 month)	1.95 (12 month)	Interpersonal program: Role transitions, role disputes, and emotional expression
Clarke et al. (2001)	I	High-risk children with depressive symptoms	94	13–18; <i>M</i> = 14.6	60%	Fifteen 1-hr group sessions	0.41	0.47 (15 month)	0.04 (24 month)	Cognitive-behavioral program: Cognitive restructuring, specifically targeting parent-related beliefs
Yu & Seligman (2002)	I	Chinese youth with depressive symptoms or family conflict	220	8–15; <i>M</i> = 11.8	45%	10 weekly 2-hr group sessions	0.23	0.30 (3 month)	0.30 (3 month)	Modified Penn Optimism Program: Adapted for use with Chinese children
Freres, Gillham, Hamilton, & Patton (2002)	I	Children with depressive symptoms	268	11–12	53%	Twelve 2-hr group sessions	–0.06	0.16 (6 month)	0.03 (24 month)	Penn Resiliency Program: Same as Penn Prevention Program
Freres, Gillham, Reivich, Shatté, & Seligman (2002)	I	6th and 7th graders with depressive symptoms	74	NR	36%	Eight 2-hr group sessions for children; six 90-min sessions for parents	0.07	0.56 (6 month)	0.56 (6 month)	Shortened Penn Resiliency Program with all the same components for children; parents were taught the core skills their children were learning but at an adult level

Note. U = universal; S = selective; I = indicated; NR = not reported; NA = not available; RAP-A = Resourceful Adolescent Program—Adolescents; RAP-F = Resourceful Adolescent Program—Family. ASQ = Attributional Style Questionnaire; PPP = Penn Prevention Program.

^a Follow-up was not conducted. ^b In the Australian and New Zealand educational systems, Year 9 is equivalent to U.S. Grade 8, and Year 10 is equivalent to U.S. Grade 9.

keywords "depression" and "prevention" were entered, and the resulting list was examined manually to identify studies of children and adolescents. To prevent publication bias and to obtain all relevant studies, we included unpublished dissertations. Although this introduces the potential problem of using studies that have not undergone peer review, such studies were deemed important because of the relatively nascent nature of this field. Dissertations were obtained through interlibrary loan or by contacting the author directly. All relevant dissertations were obtained, and analyses showed no significant difference in effect size between unpublished dissertations and published studies. Second, references from all located depression prevention studies and reviews were examined. Finally, a manual search was conducted of any journal in which another study used had been published, dating back to 1971. This included the *Journal of the American Academy of Child and Adolescent Psychiatry*, *Prevention and Treatment*, the *Journal of Adolescent Research*, *Archives of General Psychiatry*, *Psychological Science*, *Psychology in the Schools*, the *Journal of Cognitive Psychotherapy*, the *American Journal of Community Psychology*, *Behavior Research and Therapy*, *Development and Psychopathology*, *Behavior Change*, *Family Relations*, the *Journal of Clinical Child Psychology*, and the *Journal of Consulting and Clinical Psychology*.

Criteria for inclusion of a study in the meta-analysis were the following: (a) one of the stated goals had to involve preventing depressive symptoms and/or disorders in children or adolescents; (b) the study had to include a comparison of an active intervention with a control condition; (c) participants had to be randomly assigned to the intervention or control group; (d) studies had to measure depressive symptoms with a generally accepted measure; and (e) the study had to include participants under age 21.

Coding of Studies

All studies were coded for type of intervention, total number of participants, mean age, percent female, length of intervention, and length of follow-up. Independent coding was done by the first author, Jason L. Horowitz, and a postdoctoral researcher. Overall agreement for the two coders was .96. For all categorical variables, kappas were greater than .80. All disparities were resolved by consensus.

Computation of Effect Sizes

Effect sizes were computed by dividing the difference between the posttreatment depression scores of the control group and the intervention group by the standard deviation of the control group. Although some researchers favor using a pooled standard deviation, Weisz and colleagues (Weiss & Weisz, 1990; Weisz, Weiss, Han, Granger, & Morton, 1995) found that one effect of treatment may be to make variability greater in the treatment group than in the control group. They suggested using the standard deviation of the control group when such heterogeneity is observed. Therefore, in this meta-analysis, the standard deviation of the control group was preferred to the pooled standard deviation. The statistic created by this procedure is Cohen's *d* (Cohen, 1977), by which an effect size of .2 is considered small, .5 is considered moderate, and .8 is considered large.

When the necessary data were not included in the printed articles, data were requested from the authors. If authors were unable to provide the data, we used the procedures offered by Smith, Glass, and Miller (1980) for computing an effect size on the basis of other statistical data. If an article reported no significant results or offered no explanatory statistics, and if the authors could not provide the data, we used a conservative estimate of 0 for the effect size. This occurred for one study (Ialongo et al., 1999) and for the follow-up effect size but not for the posttreatment effect size of another study (Petersen et al., 1997).

Following the example of other meta-analyses (Weiss & Weisz, 1990; Wilson, Lipsey, & Derzon, 2003), we maintained independence of effect sizes by using only one effect size from each participant sample in the

analysis. Two studies (Kellam et al., 1994; Pattison & Lynd-Stevenson, 2001) included two control groups in their comparisons. In these cases, the means and standard deviations of the two control groups were pooled before being compared with the intervention group. Two studies (Reivich, 1996; Shochet et al., 2001) used two variations of an intervention and a control group. Because the variations used did not differ on any of the characteristics measured in the current meta-analysis, the intervention effects were pooled in reference to the one control group.

A few studies broke down their results into subgroups, such as showing differential effects for high-anxious versus low-anxious children. Because this was rarely done, effect sizes for the meta-analysis were computed by studies using all participants. The possibility of differential effectiveness by level of anxiety, however, is an important consideration that we discuss later.

All included studies used some kind of self-report measure of depressive symptoms. Few studies used other measures of depression such as diagnostic interviews. Because the only method of assessment used consistently across all studies was self-report, only effect sizes for self-report measures were included in the meta-analysis.

Studies varied in the length of time that passed before follow-up measures were taken. Some studies measured outcome variables only immediately postintervention. These studies were included in the analyses of immediate effects but not in the analyses of long-term effects. For those studies that conducted follow-up analyses, the most common length of time was 6 months. Follow-ups ranged from as short as 2 months to as long as 3 years. The present meta-analysis involved two approaches: (a) effect sizes were computed for each study at the follow-up that was closest to 6 months (range = 3 to 8 months). This was done to compare different intervention effects without biasing the results by the length of the follow-up. (b) We computed an effect size for each study at the last conducted follow-up and used the length of follow-up as a separate variable. This was done to incorporate as much longitudinal information as possible and to assess the effects of prevention programs over time.

In all cases, a correction for small sample bias and weighting procedures was used on the basis of Hedges and Olkin (1985). The procedures these authors recommend give greater weight to effect sizes from larger samples and those with less variance. To accomplish this, we weighted effect sizes by the inverse of the variance of the effect size. All techniques used for data analysis followed the recommendations of Hedges (1994).

Results

Distribution of Effect Sizes

A summary of all effect sizes is presented in Table 1. Positive effect sizes represent lower levels of depressive symptoms for participants in the intervention group as compared with controls. Effect sizes at postintervention ranged from -0.62 to 1.51. The weighted overall mean effect size was 0.16, which is considered small (Cohen, 1977). Only six studies reported negative effect sizes. The distribution was significantly heterogeneous ($Q = 92.65, p < .01$), indicating a need to subdivide studies. At follow-up, effect sizes ranged from -0.15 to 1.95. The weighted overall mean effect size was 0.11. Only four studies reported negative effect sizes at follow-up. The distribution again was significantly heterogeneous ($Q = 84.12, p < .01$).

Type of Intervention

There was a significant main effect for type of intervention at postintervention, $\chi^2(2, 27) = 7.11, p = .03$, such that the weighted mean effect size for selective prevention programs (mean effect size = .30) was greater than the weighted mean effect size of

universal prevention programs (mean effect size = .12). There also was a nonsignificant trend for indicated prevention programs (mean effect size = .23) to produce greater effect sizes than universal programs, $\chi^2(1, 19) = 2.82, p = .09$. The indicated and selective programs were not significantly different, $\chi^2(1, 16) = 0.54, p = .46$.

There also was a significant main effect for type of intervention at follow-up, $\chi^2(2, 20) = 25.82, p < .001$; the weighted mean effect sizes for both selective prevention programs (mean effect size = .34) and indicated prevention programs (mean effect size = .31) were greater than the weighted mean effect size of universal prevention programs (mean effect size = .02). The difference between selective and indicated programs again was not significant, $\chi^2(1, 11) = 0.08, p = .78$.

When the two samples with college students (Forsyth, 2001; Seligman et al., 1999) were removed from the analyses, the difference at posttreatment between selective prevention programs (mean effect size = .29) and universal prevention programs (mean effect size = .12) was marginally significant, $\chi^2(1, 18) = 3.43, p = .06$. Indicated (mean effect size = .18) and universal programs were not significantly different at posttreatment, $\chi^2(1, 19) = 1.14, p = .29$. At follow-up, however, the main effect for type of intervention was significant even without the two studies with college students, $\chi^2(2, 17) = 25.06, p < .001$. Weighted mean effect sizes for both selective prevention programs (mean effect size = .56) and indicated prevention programs (mean effect size = .25) were still greater than the weighted mean effect size of universal prevention programs (mean effect size = .02). In addition, selective programs were more effective than indicated programs, $\chi^2(1, 9) = 4.68, p = .03$.

Sex of Participants

Following the suggestion of Hedges (1994), weighted regression analysis was used to examine all continuous variables. Sex was operationalized as the percentage of participants in each study who were female. At postintervention, there was a significant effect for sex, $F(1, 26) = 5.39, p = .03, \Delta R^2 = .17$, indicating that studies with a greater percentage of female participants had greater effect sizes. This effect was not significant when the two studies with college students were removed from the analyses. At follow-up, there was no effect for sex, $F(1, 19) = 1.28, p = .27, \Delta R^2 = .06$.

Age of Participants

At postintervention, there was a significant effect for age of participants, $F(1, 28) = 4.78, p = .04, \Delta R^2 = .15$; greater effect sizes were found for programs implemented with older participants. This effect was not significant when the two studies with college students were removed from the analyses. At follow-up, there was no effect for age, $F(1, 21) = 0.05, p = .83, \Delta R^2 = .002$.

Length of Follow-Up and Length of Intervention

There was no effect for number of months of the follow-up on the effect size at the last follow-up, $F(1, 20) = 1.01, p = .33, \Delta R^2 = .05$, and no effect for the number of sessions included in the intervention on the effect size either at postintervention, $F(1,$

$26) = 0.02, p = .90, \Delta R^2 < .001$, or at 6-month follow-up, $F(1, 20) = 2.50, p = .13, \Delta R^2 = .11$.

Prevention Versus Treatment

To be judged a prevention effect required the following: (a) an increase in depressive symptoms among members of the control group and (b) no increase or a diminished increase of symptoms in the intervention group. None of the studies of universal interventions met the first criterion; rather, depression scores for the control groups as well as the intervention groups in these studies were very static over time, which is consistent with the finding that the weighted mean effect size for universal studies was only .12 at postintervention and .02 at 6-month follow-up. One universal prevention study (Pattison & Lynd-Stevenson, 2001) found a moderate effect size of .40 at the 8-month follow-up, but this was the result of a decrease in symptoms in the intervention group and therefore would be classified as a treatment effect.

For selective studies, most showed a treatment effect; that is, a decrease in depression scores for those in the intervention group. Even the selective studies with large effect sizes (e.g., the 1.24 effect size of Cardemil et al., 2002) would be classified as treatment. Only one selective study (Quayle, Dzuirawiec, Roberts, Kane, & Ebsworthy, 2001) showed a prevention effect such that the control group showed an increase in depression scores. Regarding studies of indicated programs, the two with the largest effect sizes (0.47 from Clarke et al., 2001; 1.95 from Forsyth, 2001) were clear examples of treatment effects. In contrast, three studies (Freres, Gillham, Hamilton, & Patton, 2002; Jaycox et al., 1994; Reivich, 1996) showed prevention effects such that there was an increase in depressive symptoms for the control group and no increase or a decrease in depressive symptoms for the intervention group.

Discussion

The current meta-analysis showed a wide range in the degree of success of programs aiming to prevent depressive symptoms in children and adolescents. Although there were some extreme scores, the majority of effect sizes at both postintervention and 6-month follow-up represent small to moderate effects. At postintervention, selective prevention programs were more effective than universal programs, and there was a nonsignificant trend for indicated prevention programs to be more effective than universal programs as well. Both selective and indicated prevention programs were significantly more effective than universal programs at follow-up.

This latter finding can be partly explained by differences in the level of symptoms found in the control groups. In universal samples, control participants often do not show a high enough level of depressive symptoms at follow-up to demonstrate a preventive effect for the intervention. In contrast, in selective and indicated studies, the sample is chosen on the basis of risk status or sub-clinical symptoms and therefore is likely to have a higher level of depressive symptoms at baseline as well as to show an increase in level of depressive symptoms over time. An example can be seen by comparing the results of the Pattison and Lynd-Stevenson (2001) evaluation of the Penn Prevention Program with a universal sample to the original study (Jaycox et al., 1994), conducted with

an indicated sample. Mean scores on the Children's Depression Inventory (CDI; Kovacs, 1985) for the Penn Prevention Program groups following intervention were comparable (7.6 for control vs. 8.4 for PPP), but the mean CDI scores of the control group in the original Penn Prevention Program study continued to rise over time and were significantly higher at the last follow-up (13.3 vs. 8.1) than that of the universal replication.

Although universal programs avoid the initial step of screening for risk, they involve delivering services to large numbers of individuals with relatively small need. Moreover, the number of participants required to show a significant statistical effect of a universal intervention typically is huge and hardly feasible (Cuijpers, 2003). The current meta-analysis showed that depression prevention programs that target selective or indicated child and adolescent samples may be more practicable and beneficial in the long run than those that target universal samples. It is possible, however, that although universal programs yield low effect sizes, they still could be cost-effective if they are able to prevent even a small number of cases of depression at comparatively low cost. Appropriate cost-effectiveness analyses contrasting the relative costs and benefits of the different types of prevention programs need to be conducted.

Even within selective and indicated studies, however, there was variability in effect sizes. Thus, other factors such as age and gender of participants can affect the success of these programs. The current study found greater effect sizes at postintervention for studies with older participants and a higher percentage of female participants, although these results were no longer significant when the two studies of college students were excluded. Nevertheless, these age and sex trends in response to depression prevention programs should be studied further.

The current meta-analysis found no effect for length of intervention or length of follow-up. The lack of variability in length of the interventions (range = 3 to 16 sessions; $M = 10.5$; median = 11) may account for this null finding. With regard to length of follow-up, there was great disparity across studies. Whereas some studies conducted follow-ups at only 2 months, others continued as long as 36 months. Even if a prevention program is effective, this might not be evident after only 2 months, in part because it may take time for the control group to show increases in symptoms. Furthermore, an intervention that is effective at a short-term follow-up but rapidly loses its effect will appear more successful than it is without a long-term follow-up. Future prevention research should follow the example of Gillham and Reivich (1999) and Seligman et al. (1999), who conducted follow-up assessments every 6 months for 36 months or Spence, Sheffield, and Donovan (2005) who collected data every 12 months for 4 years. Moreover, epidemiological studies showing the rise in depression around age 13 to 15 years (e.g., Hankin et al., 1998) indicate that studies of prevention programs targeting younger children may need to follow them longer until they are through this age period of increasing rates of depression in order to show a prevention effect.

Prevention or Treatment?

The present meta-analysis compared effect sizes across studies, but cannot, by itself, be used to determine whether the significant effect sizes were the result of an increase in depressive symptoms in the control group and no increase or a diminished increase in

symptoms in the intervention group (i.e., prevention) or the result of a decrease in symptoms in the intervention group and no change in the control group (i.e., treatment). None of the studies of universal interventions met the criteria to be considered prevention. Depression scores for both the control and intervention groups tended to be quite stable over time. Universal (e.g., Pattison & Lynd-Stevenson, 2001) and selective studies (e.g., Cardemil et al., 2002) that did show moderate effect sizes were best classified as treatment as a result of significant decreases in depression scores for the intervention group. One selective study showed a prevention effect (Quayle et al., 2001), that is, the control group increased in depression scores and the intervention group did not. The best evidence of true prevention of depression came from studies with indicated samples. Three indicated studies (Freres, Gillham, Hamilton, & Patton, 2002; Jaycox et al., 1994; Reivich, 1996) demonstrated true prevention effects. Their control groups showed an increase in depressive symptoms, whereas their intervention groups showed no increase or a decrease in depressive symptoms. Thus, of all 30 studies whose explicitly stated aim was the prevention of depression in children and adolescents, only 4 showed evidence of an actual prevention effect.

Many studies made it difficult to find evidence of prevention because they failed to conduct long enough follow-ups. Only 12 of the 30 studies reviewed here conducted a follow-up past 6 months. A prevention effect might have been found in some of the other studies had more time passed. For example, the preventive effect in the Jaycox et al. (1994) study was not evident until the 18-month assessment. It also is possible for a study to show both a short-term treatment effect and a longer term prevention effect over time.

One important question is, Are these programs effective? That is, do they show a significant difference between the intervention and no-intervention groups? The distinction between treatment and prevention does not change the conclusions drawn from this meta-analysis that many of these programs have been successful and that, in general, selective and indicated programs have larger effect sizes than universal programs. It is important to note, however, that thus far such success may best be thought of as the reduction, and thus treatment, of depressive symptoms rather than the prevention of increases in depressive symptoms in vulnerable individuals.

A second important question is, Do these programs prevent depression? The current analysis indicates that there is yet very little evidence to support the idea that they do. Only 4 of 30 studies met the criteria to show evidence of prevention. There may be both methodological and substantive reasons for this. Most prevention protocols mirror established treatment protocols. Researchers should consider whether the mechanisms targeted to treat depression are the same as those that should be targeted to prevent it. Additionally, in order to maximize their ability to find evidence of prevention, future studies should consider focusing on indicated populations in particular and should conduct more and longer follow-up evaluations to allow time for the possible preventive effects to occur. Furthermore, future prevention studies themselves should report whether the effects they produce are treatment or prevention effects.

Priorities for Prevention of Depression Research

The present meta-analysis indicates that a growing number of empirically tested programs aimed at preventing depression have

shown low to moderate effects, with most reducing rather than preventing increases in levels of depressive symptoms. Several important questions remain that can guide future research on the prevention of depression in youth.

Who should be the target of depression prevention programs?

This meta-analysis showed that selective and indicated programs had greater effects than universal programs. Although we argue that it is premature to abandon universal programs for preventing depression, focusing particularly on high-risk populations makes sense at this time. On the basis of findings from epidemiological, developmental, and clinical studies, particularly important risk factors for depression include being a female adolescent (Hankin et al., 1998), being the offspring of depressed parents (Goodman & Gotlib, 1999), having elevated levels of depressive and/or anxious symptoms (e.g., Pine, Cohen, Gurley, Brook, & Ma, 1998), and being exposed to certain stressors such as parental divorce or loss (e.g., Sandler et al., 1992). Thus far, however, none of these risk factors has been found to moderate the relation between intervention and outcome. Although some programs were more effective for female participants and older adolescents, these findings were mainly due to the inclusion of one or two studies with college student samples. Thus, despite the fact that adolescent girls are at increasing risk for depression and certainly should be the target of prevention efforts, it remains possible that prevention programs also could be effective with boys and preadolescents, although larger control groups may be needed to show such effects.

With regard to anxiety, two studies (Hains & Ellmann, 1994; Lowry-Webster et al., 2001) found that children with higher levels of anxiety or arousal experienced a greater reduction in depressive symptoms. Although such results might support the idea that children with anxiety constitute a good target for depression prevention programs, these findings also might have been attributable to higher levels of depressive symptoms occurring in the context of anxiety, rather than to the effect of anxiety per se. Future prevention studies should explore whether reducing anxiety in children with different baseline levels of depressive symptoms actually decreases the risk of subsequent depression.

Recommendation 1: Studies testing the efficacy of programs for preventing depression should examine whether certain risk factors (e.g., parental depression, subsyndromal depressive symptoms, gender, age, anxiety) moderate the relation between the intervention and depression. Selective and indicated studies that target samples on the basis of some risk factors then should examine the role of other, nonselective risk factors as possible moderators. Analysis of moderators can begin to identify for whom interventions are most effective.

How do depression prevention programs need to be modified to accommodate individual differences? If certain individual characteristics (e.g., age, gender, ethnicity, cognitive ability) moderate the effects of preventive interventions on depression, how, then, should programs be modified to increase their efficacy for more individuals? To date, only the Penn Prevention Program (Jaycox et al., 1994) has been investigated with different ethnic groups and has been found to be successful with Latino (Cardemil et al., 2002) and Chinese (Yu & Seligman, 2002) children but not with African American children (Cardemil et al., 2002). Whether and how depression prevention programs should be modified to be more culturally sensitive is an important issue for future study. In addition, more descriptive research is needed to identify risk

factors and processes that predict depression in different cultural groups.

Recommendation 2: Findings from basic research on the epidemiology, phenomenology, course, and etiology of mood disorders that highlight differences associated with developmental level, gender, and ethnicity should guide modifications in programs aimed at preventing depression. That is, prevention programs need to be adapted to make them developmentally appropriate, gender and culturally sensitive, and amenable to being delivered at a level commensurate with the cognitive abilities of the participants.

By what processes is depression prevented in children and adolescents? Thus far, most depression prevention studies have compared an active intervention to a no-contact or wait-list control group, so it is not possible to determine what aspect of the intervention accounted for positive findings. Studies that compare two or more active interventions or an intervention that controls for nonspecific factors can begin to address this issue. For example, Merry et al. (2004) included an active attention-placebo control that was similar in structure to their primary intervention but focused on participants having fun and did not include elements thought to actively prevent depression. In addition, dismantling studies that contrast different components of an intervention can help identify active ingredients underlying change.

Depression prevention studies also have varied in the extent to which they have included measures of potential mediators of the relation between the intervention and the outcome. For example, several successful prevention programs have taught cognitive restructuring techniques (e.g., Clarke et al., 2001; Jaycox et al., 1994). However, without measuring change in cognitions, one cannot conclude that this was the mechanism that accounted for the effect. Other processes, such as the social support afforded by a group intervention, could be the active ingredient(s).

Better measurement of processes gives a more complete picture of the effects of a prevention program even if it does not successfully prevent depression. Some studies that included multiple outcome variables found that their programs did affect risk factors associated with depression, even if they showed little or no effect on depression per se (e.g., Ialongo et al., 1999; Sandler et al., 1992; Wolchik et al., 1993). These programs appeared to at least affect the hypothesized mediators, such as achievement, coping skills, or improved interpersonal relationships. It may take more time to see the effect of these mediators on depressive symptoms, and thus a longer follow-up might be necessary. Conversely, it also is possible that although the intervention may affect the hypothesized mediator(s), these variables may not then influence the outcome.

Recommendation 3: Studies of depression prevention programs should examine mechanisms by (a) contrasting alternative interventions that experimentally manipulate hypothesized mediators and (b) testing whether the hypothesized mediators are affected by the intervention and, if so, whether they indeed mediate the relation between the intervention and outcome. Identifying the mechanisms through which interventions work will facilitate the development of more effective and efficient prevention programs (Kraemer, Wilson, Fairburn, & Agras, 2002).

How can depression prevention programs be enhanced to produce stronger and more enduring effects? Evidence from this meta-analysis indicates that current depression programs have low to moderate effects at best, and they are generally short-lived. The

longest lasting effects were found for the Penn Prevention Programs (Gillham et al., 1995) for up to 2 years. More often it has been the case that postintervention effects diminish after 6 to 12 months. Depression prevention programs can be strengthened in several ways.

First, given that the causes of depression likely are multifaceted, prevention programs need to target multiple components, particularly negative cognitions, interpersonal relationships, and responses to stress (Garber, in press). Cognitive interpersonal models (e.g., Gotlib & Hammen, 1992), suggest that depressed individuals have negative cognitions especially within the social domain, which then serve to exacerbate and perpetuate interpersonal difficulties and depression. Therefore, prevention programs that teach and integrate cognitive, coping, and social skills (e.g., Jaycox et al., 1994) may be more effective than those that focus on only one domain, although this remains to be explicitly tested.

Second, given that families with a depressed member tend to have dysfunctional interaction patterns (Garber, 2005; Goodman & Gotlib, 1999; Kaslow, Deering, & Racusin, 1994), interventions for preventing depression in youth should attempt to enhance the family environment. Although some programs have included parents (Beardslee et al., 1997; Freres, Gillhman, Reivich, Shatté, & Seligman, 2002; Lowry-Webster et al., 2001; Wolchik et al., 1993), only one study (Shochet et al., 2001) systematically investigated the addition of a parent component to the prevention program evaluated. Shochet et al. found that participants in both the Resourceful Adolescent Program—Adolescent and the Resourceful Adolescent Program—Family had fewer depressive symptoms than controls and that there was no significant difference between the two intervention groups. The family component included stress management training, information on normal adolescent development, and strategies to promote family harmony and manage conflict. The family program, however, was hampered by very low attendance by parents; only 10% attended all three sessions, and 64% did not attend any. In the treatment literature, Clarke and colleagues (Clarke, Rohde, Lewinsohn, Hops, & Seeley, 1999; Lewinsohn, Clarke, Hops, & Andrews, 1990) similarly found that adding a parent group to a cognitive-behavioral therapy program for currently depressed adolescents was no more effective than a cognitive-behavioral therapy group alone, but here too parent attendance rates were very low. Clarke and colleagues, however, have not yet tested the incremental contribution of a parent component to their prevention program.

Thus, existing evidence is inconclusive about the benefits of including parents in depression prevention programs, and what particular parenting components have the greatest preventive effect. An important next step in the development of depression prevention programs would be to explicitly target parenting behaviors that are most likely to contribute to depression in children (e.g., criticism, rejection, withdrawal, intrusiveness). This then could supplement the child-focused components of the interventions that more directly address children's cognitions, communication, and coping strategies.

Recommendation 4: The development of prevention programs should be guided by theory, particularly those theories that recognize the role of multiple interacting intrapersonal, interpersonal, and contextual factors. Depression prevention programs should

systematically investigate various combinations of interventions that aim to alter these different risk factors and processes.

What methodological questions still need to be addressed in future depression prevention studies? One important process-related issue is who leads the interventions (Weisz et al., 1995). Most of the programs reviewed here used mental health professionals or graduate students, and therefore there was not enough variability to examine the effect of type of group leader on outcome. Because many of the successful programs are highly manualized and conducted in schools, it is possible that others, particularly teachers or school counselors, can provide the interventions with the same level of competence. Indeed, Spence et al. (2003, 2005) found that teachers competently implemented the Problem-Solving for Life program in the schools.

Second, what is the optimal timing and duration of follow-up for detection of a preventive effect? Part of the answer to this will depend on the age at which the intervention begins. Ideally, preventive interventions should occur prior to the documented increase in depressive symptoms (about age 13–14 years) and continue through the period during which the rates of symptoms and disorders would be expected to rise (e.g., ages 15–18) among individuals in the control condition. How much to intervene before age 13 will depend on the developmental demands of the program and how enduring the effects of the intervention are likely to be.

Another important methodological issue concerns the measurement of depression. The present meta-analysis examined the effect of prevention programs on depressive symptoms rather than diagnoses because the majority of studies measured only symptoms (see Clarke et al., 2001; Spence et al., 2003, for exceptions). The Institute of Medicine (Mrazek & Haggerty, 1994) defines prevention as an intervention that prevents a clinically diagnosable disorder. Considering that disorders are usually the standard for treatment and prevention research, it is unfortunate that so few studies of depression prevention to date have obtained diagnoses. This is partially due to the relative ease with which symptoms can be assessed and the comparative cost of doing clinical interviews at multiple points with large samples. In addition, because of the low base rate of depressive disorders in children, statistical power for detecting prevention effects would be even lower for analyses of diagnoses than for changes in symptoms unless huge numbers of participants were included (Cuijpers, 2003).

The absence of information about diagnoses, however, does not diminish the importance of the findings based on symptom measures. Depressive symptoms alone comprise a meaningful outcome in children and adolescents. Indeed, taxometric analyses (Hankin, Fraley, Lahey, & Waldman, 2005) suggest that depression may be more accurately represented as a dimensional, rather than a categorical, construct. Subclinical depressive symptoms in youth constitute a risk for subsequent depressive disorders (Clarke et al., 1995; Pine, Cohen, Cohen, & Brook, 1999) and predict an increased risk of substance use, academic failure, dropout, and teen pregnancy (Gillham et al., 2000). Moreover, moderate levels of depression have been found to persist for years in some children (Twenge & Nolen-Hoeksema, 2002). Thus, prevention of depressive symptoms, regardless of whether or not a clinical diagnosis is warranted, is a goal worthy of study.

The primary measure used to assess depressive symptoms in prevention studies has been the Child Depression Inventory (CDI; Kovacs, 1985). One limitation of the CDI, however, is that three

items measure externalizing symptoms. Several of the programs reviewed here had elements related to the prevention of behavior problems (e.g., problem solving, decision making). Thus, some of the effects found using the CDI as the outcome measure might have been partially due to changes in externalizing symptoms. In addition, given the high rate of comorbidity with depression (Angold, Costello, & Erkanli, 1999) and the fact that the skills taught in several of the depression prevention programs reviewed here also may help prevent other problems, measures of these other conditions should be included as well.

Recommendation 5: Prevention studies should use basic findings about depression to inform important methodological decisions such as the selection of when to intervene, when and for how long to conduct follow-up assessments, and the choice of outcome measures. Multiple measures of both depressive symptoms and disorders as well as other problems (e.g., externalizing) should be included in prevention trials whenever possible.

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