

**LONG-TERM EFFECTS**  
**FROM A RANDOMIZED TRIAL OF TWO PUBLIC HEALTH**  
**PREVENTIVE INTERVENTIONS FOR PARENTAL DEPRESSION**

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## Abstract

This paper presents an analysis of the long-term effects of a randomized trial conducted to evaluate the effectiveness of two standardized manual-based prevention strategies for families with parental mood disorder: two informational lectures, and a brief clinician-based approach (M = 7 sessions) including child assessment and a family meeting. One hundred five families in which at least one parent suffered from a mood disorder and in which at least one non-depressed child was within the 8-15 year age range were recruited. Parents and children were assessed separately at baseline and every nine to twelve months thereafter on behavioral functioning, psychopathology, and response to intervention. Both interventions produced sustained effects through the sixth assessment point, approximately 4-1/2 years after enrollment, with relatively small sample loss of families (<14%). Clinician-based intervention families had significantly more gains in parental child-related behaviors and attitudes, and in child-reported understanding of parental disorder. Child and parent family functioning, as measured by the Family Relations Inventory, increased for both groups, and internalizing symptoms as measured on the Youth Self Report decreased for both groups, with no significant group differences. These findings demonstrate that brief, family-centered preventive interventions for parental depression may contribute to long-term, sustained improvements in family functioning.

Keywords: parental depression, prevention, adolescents

Adolescent depression is a public health problem of major proportions with point prevalence rates ranging from 0.4% to 8.4% (Fleming & Offord, 1990). The lifetime prevalence of Major Depressive Disorder in adolescents aged 15-18 is 14%. One of the most potent risk factors for adolescent depression is being the child of a depressed parent (Institute of Medicine, 1994); these children are about four times more likely to develop a mood disorder than those of nondepressed parents (Beardslee, Versage & Gladstone, 1998). In fact, by the age of 20, a child with an affectively ill parent has a 40% chance of having an episode of depression, and by age 25, this rate increases to 60% (Beardslee et al., 1993).

Family factors may maintain depression in youth (Brent et al., 1997; Hammen et al., 1999) and are more salient than peer relationships in predicting adolescent depressive symptoms (Sheeber, Hops & Davis, 2001). Moreover, adverse family environments are among the most consistent risk factors for adolescent depression (Evans & Seligman, 2005). Yet, to date, intervention and prevention approaches to adolescent depression have used individual or group approaches focusing on enhancing adolescent's coping strategies (e.g., Clarke et al., 2001; Jaycox et al., 1994) rather than family-based approaches. Although several studies incorporate traditional family therapy approaches to the *treatment* of child/adolescent depression with some success (e.g., G.S. Diamond, Reis, G.M. Diamond, Siqueland, & Isaacs, 2002; Nelson, Barnard, & Cain, 2003), a recent meta-analysis (Horowitz & Garber, 2006) indicated only three other family-based interventions targeting children/adolescents who are not yet clinically ill (Sandler et al., 1992; Shochet et al., 2001; Gillham & Reivich, 1999). None of these programs focused on children of depressed parents. Given the strong effects of parental depression on family functioning, evidence suggesting family participation may enhance the benefits of treatment for depressed adolescents (Diamond et al., 2002), and evidence of the benefits of family change in

promoting resilience in teens (Asarnow et al., 2002, Diamond et al., 2002; Shochet et al., 2001), consideration of a family-based approach is warranted.

Over the past fifteen years, our group developed and tested two family-based preventive interventions for families with parental depression: informational lectures, and a brief clinician-based approach. We chose to focus on *selective* prevention (Institute of Medicine, 1994) (i.e., targeting youth at elevated risk as a function of parental mood disorder) (Beardslee, 1998). Our approaches targeted decreasing some risk factors inherent in the presence of parental mood disorder (e.g., family conflict and lack of parental focus; Beardslee, 2003) and increasing factors demonstrated to be protective when a parent suffers from mood disorders (e.g., increased involvement in outside activities, supportive adult/family relations, and increased family understanding; Beardslee & Podorefsky, 1988).

Given the high rates of parental depression and the need for large-scale programs that could be widely disseminated, our approaches were designed to be used by a range of practitioners from different disciplines working from different theoretical schools. Because parents with depression are likely to present in settings other than mental health clinics while seeking services for their children (US Department of Health and Human Services, 2000), the interventions were designed to be used by those who treat adults, children and families in a wide variety of settings (e.g., pediatric clinics, primary care clinics, community settings). We targeted improved recognition and treatment of depression in youth as an intervention outcome because most youth with depression are neither identified nor treated (US Dept of Health and Human Services, 2000), and we included education for parents about treatment for depression in adulthood. Effective treatment of parents with depression reduces rates of child difficulty when compared to parents not receiving adequate treatment (Weissman et al., 2006).

After standardizing the approaches, we conducted a pilot study in which the sample ( $n=36$  families) was randomized on a 1/3:2/3 basis. The findings from this sample demonstrated the safety (Beardslee et al., 1997) and feasibility of the approaches (Beardslee et al., 1993). Given that greater effects were observed in the clinician group, and that both interventions were safe and useful, we undertook a randomized trial with a larger sample, with equal numbers of families assigned to each intervention condition. Again, relative to lecture group families, the clinician-facilitated group displayed greater parental change in variables believed to mediate resilience in the offspring (e.g., improved child-focus and communication) and greater understanding of parental mood disorder by the children at 18 month (Beardslee et al., 1997) and 30-month follow-up assessments (Beardslee, Gladstone, Wright, & Cooper, 2003), both groups showing sustained gains. A modest reduction in child internalizing symptoms also was found for both groups (Beardslee et al., 2003) at the 30-month follow up interval.

Effective preventive interventions must show long-term effects (Institute of Medicine, 1994; National Advisory Mental Health Council Workgroup on Child and Adolescent Mental Health Intervention Development and Deployment, 2001). In this report, we address whether changes demonstrated at previous intervals (Beardslee et al., 1997, 2003) resulted in longer-term effects for families by presenting analyses from the sixth assessment point, approximately 4-1/2 years after enrollment and randomization.

We hypothesized that: (1) Parent-reported changes in family communication and understanding would be sustained over the longer follow-up interval and greater in the clinician group; (2) improvements in children's understanding of parents' mood disorders would be sustained over the longer follow-up interval and greater in the clinician group; and (3) parental change in communication, functioning, and understanding would predict better understanding of

parental mood disorder on the part of the offspring. Based on our previous analyses, we explored the following hypotheses: (1) Over the follow-up period, adolescents would show decreases in internalizing symptoms despite expected increases in this age group (e.g., Rao et al., 1995); (2) families who participated would be more likely than families in community samples to seek treatment for depressed children; and (3) overall, family functioning would improve from baseline and be sustained over time.

## Methods

Detailed descriptions of the sample, measures, and the two interventions have been presented previously (Beardslee, Versage, Salt, & Wright, 1999; Beardslee et al., 2003).

### *Respondents and Procedures*

Dual- and single-parent families were eligible to participate if they had (1) at least one child 8-15 years of age and (2) at least one parent who had experienced an episode of mood disorder in the 18 months before contact. Parental exclusion criteria were serious current parental substance abuse or dependence, schizophrenia, current severe marital crisis, or other life crises (e.g., hospitalization) that would prevent the family's participation. Families also were excluded if parents were currently in marital or family therapy more than twice monthly. Parents in psychotherapy for depression were **not** excluded. Youngsters were excluded if they had a current or past history of major depression by parental report but *not* excluded if they experienced or were treated for other diagnoses (e.g., ADHD). We enrolled *all* eligible children in each family. All participants provided written informed consent, and the children provided written assent at enrollment in accordance with human subjects' standards. Any individual or family was free to discontinue participation at any point.

The sample consisted of 105 families, including 21 families from the pilot sample and 84 families enrolled after the pilot period.<sup>1</sup> We recruited 53.6% of the sample from a large prepaid northeastern health maintenance organization; mental health practitioners referred an additional 13.05%, 5.22% were recruited from support groups, 9.57% responded to newspaper advertisements, and 18.26% from a variety of other sources. Pilot families were randomized to treatment groups on a 2/3:1/3 basis (clinician-facilitated: lecture). For the other 84 families, a balanced block randomization procedure was used, with blocks of four stratified by family type (single- or dual-parent) (Beardslee et al., 1999). Pilot measures, both pre- and post-intervention, were identical to those used after the pilot period, and there were no significant differences on study measures between the pilot and non-pilot families.

Assessments were conducted initially (T1), immediately after intervention (T2), and regularly 9-12 months thereafter (T3-T6) for all subjects by raters who were blind to assessment of other family members. Retention strategies included flexibility in location and timing of the assessment (within predefined limits) and small payments for each subject. It was impossible for individual assessors to be blind to group status, although child and parent assessors were kept blind to assessment information from each other.

*Intervention design.* Both intervention strategies addressed poor communication, misunderstanding, feelings of guilt and blame, and lack of recognition of depression. To address putative protective factors, both interventions sought to build children's support networks and their outside activities, and to increase shared family understanding (Beardslee, et al., 1999).

Depression was presented as a family illness, and a cognitive framework was used (Beardslee et

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<sup>1</sup> The pilot sample differed from the main sample in one respect. Originally, children were not interviewed directly at baseline in the pilot sample, although responses to the same self-reports were provided by the children. After the pilot study, 21 of the 36 pilot families consented to have their children interviewed. Thus, one year after enrollment, diagnostic data retrospective to time of enrollment were gathered for the children in these 21 pilot families. There were no significant differences on baseline measures for those who remained in the study and those who did not.

al., 1999). Parents were encouraged to get appropriate treatment for depression and, if in treatment, contact was made with their clinicians before starting the preventive intervention in order to ensure coordination of their care. Both intervention groups were offered further sessions or telephone consultation at any point on request; the groups did not differ in their use of these extra sessions/referrals. Manuals specified the delivery of both interventions.

*Clinician-facilitated intervention.* The clinician-facilitated intervention consisted of an average of seven sessions ( $M=6.7$ ,  $SD=1.3$ , range 2-9), including separate meetings with parents and children, and family meetings (Beardslee et al., 1999). Intervenors were licensed social workers or clinical psychologists, trained rigorously in the intervention strategies. Training included case review, discussion of the manual, and, for many of the clinicians, role-playing with actors who played the parts of family members. Throughout the project, ongoing supervision occurred to ensure fidelity. Clinicians presented information about family experiences of mood disorder, obtained a history of parental mood disorder, assessed the functioning and concerns of the children, linked general information to each family's experience of depression, and helped parents prepare for and conduct a family meeting to discuss how the family would deal with depression. Telephone contacts or refresher meetings occurred at 6-9 month intervals reviewing parental illness and addressing concerns that may have emerged during the interval. Using the manual and scripts, we developed a set of scales assessing presence or absence of key intervention action during key sessions (i.e., sessions with the child, planning the family meeting, and the actual family meeting). Ten families' clinical interventions were rated (37 sessions), and an overall score was obtained by summing the three ratings. Raters for fidelity were not project clinicians and had no knowledge of the families' treatment. Overall, interrater reliability using the scales was excellent (intraclass correlations ranged from 0.89 to 0.99), and adherence to the

protocol was similarly stellar (86.4% for the family meeting, 91.7% for the child session) (Beardslee et al., 2003). Difference in adherence among the four clinicians was nonsignificant.

*Lecture intervention.* The lecture intervention consisted of two meetings delivered in a group format without children present and consultation as requested by families. The lecture was based on the same constructs as the clinician-facilitated intervention, but there was no active attempt to link the psychoeducational material to each individual's life experiences. Lecture groups were formed as lecture families enrolled and contained an average of three families per lecture group. The first author delivered each lecture. A standardized scale based on content information from the lecture script was used to measure script adherence. A rater evaluated adherence to the lecture script regarding the presence or absence of 10 items from the first lecture and 27 from the second in the videotapes. Ten videotapes (five of the first lectures and five of the second lectures, randomly selected) were rated, and adherence to the protocol was greater than 95% mean percent agreement (Beardslee et al., 2003).

### *Measures*

*Demographic information.* Family composition, SES, and other demographic data from heads of household at initial assessment were assessed using a measure developed by Larkin and Hirschfeld (Larkin & Hirschfeld, 1997).

*Parental psychopathology.* At T1, the Schedule for Affective Disorders and Schizophrenia - Lifetime Version (SADS-L) (Endicott & Spitzer, 1978), scored according to Research Diagnostic Criteria (RDC) (Spitzer, Endicott, & Robins, 1978), was administered to the parent/s consistent with our previous risk sample (Beardslee et al., 1996). The SADS-L assesses affective illness (i.e., Major Depressive Disorder, Minor Depression, Intermittent Depression, Mania, Hypomania, Schizoaffective Disorders), anxiety disorders, and substance

abuse/dependence. Assessment of potential episodes in intervals after enrollment was done using the Streamlined Longitudinal Interval Continuation Evaluation (SLICE). Psychometric properties for both versions have been well studied and are excellent (Keller et al., 1987).

*Child psychopathology.* Each parent and child/ren were interviewed using the Schedule for Affective Disorders and Schizophrenia for School-Age Children, Epidemiologic Version-Revised (Kiddie-SADS-E-R), Life Time Version (Puig-Antich et al., 1980), and scored using the Diagnostic and Statistical Manual of Mental Disorders, 3<sup>rd</sup> Ed., Revised (DSM-III-R)(American Psychiatric Association, 1987).<sup>2</sup> Information from both interviews was combined using standard procedures described by Orvaschel et al.(1982) to obtain a best estimate diagnosis. A shortened interval version (KSLICE) was used to cover the intervals (Keller, Shapiro, & Lavori, 1984). Reliability and validity properties have been well established (Ambrosini, 2000).

*Parental global functioning.* For each parent, the Global Assessment Scale (GAS) (Endicott et al., 1976) was used to obtain the individual's worst and current levels of functioning during each assessment interval and for the 18 months prior to enrollment. Extensive training was done using vignettes developed by the NIMH-CRB Psychobiology of Depression study.

*Child internalizing symptomatology.* To assess degree of internalizing symptoms we had children rate problem and adaptive behaviors on the Youth Self-Report (YSR) and the Young Adult Self-Report (YASR, used from age 18 forward), (Achenbach & Edelbrock, 1987). The broadband internalizing *t*-scores were used from both YSR and Young Adult Self-Report (YASR, used after age 18) for analyses. Internal consistency estimates were good ( $\alpha$ 's = 0.84, 0.80 for YSR internalizing and YASR internalizing, respectively).

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<sup>2</sup> DSM-III-R diagnoses were used for the offspring to assess more typical childhood and adolescent disorders not covered by RDC criteria (e.g., Attention Deficit Hyperactivity Disorder, Separation Anxiety Disorder) in addition to the affective disorders covered by the SADS.

*Impact of the intervention - parents.* The Semistructured Interview about the Intervention (SII) was administered to each parent at baseline and at each follow-up assessment to assess parent reported changes in eight behaviors (e.g., increased talking with children about depression) and nine attitudes (e.g., increased information about risk and resiliency in children) regarding parenting, family communication, and understanding (Beardslee et al., 1999). We developed the SII to measure the 1) impact of parental affective disorders; 2) impact of the interventions on family functioning; and 3) degree to which the interventions addressed parents' greatest concerns. Raters used information obtained from the SII to identify specific changes in illness-related behaviors and attitudes attributed to project participation. To score positively, a parent must both report change *and* attribute it to the intervention. An interrater-reliability study, with raters blind to group membership, showed excellent intra-class correlations for total behaviors ( $r = 0.94$ ), total attitudes ( $r = 0.92$ ) and total changes ( $r = 0.93$ ) (Beardslee et al., 1997). Internal consistency estimates were good ( $\alpha = 0.78$ ).

*Impact of the intervention - children.* The Semistructured Child Interview (SCI) (Beardslee et al., 1999) provided information about children's perception of changes in the family especially to describe increased understanding. Independent raters, rating 20 transcribed and blinded interviews, generated excellent intra-class correlations ( $r = 0.92$ ; Beardslee et al., 1997). Change in understanding was generated combining the rater's score of improved understanding with the child's Likert rating of change (Beardslee et al., 2003). Internal consistency estimates were good ( $\alpha = 0.84$ ).

*Family functioning - the Family Relationship Inventory (FRI).* To assess overall change in family functioning, the FRI was administered at baseline and at each follow-up assessment to both parents and children. It is a 27-item measure consisting of the three subscales (Cohesion,

Expressiveness, and Conflict) of the relationship dimension of the Family Environment Scale (FES), a widely used, empirically derived measure of family functioning (Holahan & Moos, 1981). Estimates of internal consistency for this study were good ( $\alpha = 0.83$ ).

*Health care contacts.* Treatment involvement at baseline and at follow-up intervals was elicited using a semistructured interview form to explore the impact of outside treatment on outcome variables. Information covered included whether participants had sought a variety of treatments for either medical or psychiatric difficulties (e.g., medication, mental health services, or lay services such as talking to a religious leader) from baseline to the third assessment point and at each interval following. Interviewers elicited the information directly from individual parents, and child assessors interviewed the mothers about their children's participation until the adolescent became a young adult. Young adults provided these data directly.

#### *Data Analytic Strategies*

In families in which there was only one parent with a mood disorder in the 18 months prior to enrollment, that parent was designated the identified patient (IP), and the non-ill parent was designated as a non-identified patient (NIP). When both parents had experienced a mood disorder in the 18 months prior to baseline, the parent with the most severe/chronic mood disorder was designated as IP. Finally, at each assessment, a couple's GAS score, used as a covariate, was created that reflected the lower of the two parents' functioning scores.

Because this study was concerned with developmental outcomes, we created a dichotomized developmental stage variable (younger than 14 vs. 14 and older) calculated at each assessment to examine changes associated with early vs. late adolescence.

Not all outcome measures were obtained from all subjects at each assessment due to time constraints and to assessment mode. In addition, a relatively small amount of non-response was

due to dropout. In order to examine whether the dropouts and missing data affected the outcomes, we created a dichotomous, ‘completion status’ variable for each outcome for each subject. We considered all subjects who had data from all or all-but-one assessment (and who had completed the intervention) as completers, and those with at least one point, but with two or more missing data points, as partial-completers. This variable is included in all regressions to estimate differences in outcomes between subjects with and without complete data and to adjust estimates of treatment group effects for nonresponse due to dropouts and missing data.

*Statistical methods.* Regression analyses were performed with SAS version 9.0 (PROC GENMOD) using generalized estimating equations (GEE). GEE provide unbiased estimates of treatment effects and tests of hypotheses when data are missing at random. GEE test statistics are chi-squared, distributed under the null hypothesis. For parent outcomes, regressions adjusted for parent gender, IP status, couple’s worst GAS score, and completion status. For child outcomes, regressions adjusted for gender, developmental stage, and completion status.

For outcomes measured at baseline and later assessments (parent FRI, child FRI, and child internalizing), we used two linear regression models. First, to test for post-intervention differences in outcomes between treatment groups, we modeled the post-intervention scores, adjusting for baseline score and covariates. Second, we tested for a sustained change from baseline by comparing the average baseline level with the average post-intervention level. We also compared mean outcome levels within the post-intervention assessments to examine the effect of time. Intervention group by assessment interaction effects also were tested for each outcome and were excluded when not significant. For outcomes without a baseline measure (parent behavior and attitude changes, and child understanding), we tested for a treatment group difference by comparing the average outcome between groups across all assessments combined.

We tested for a sustained change by comparing average post-intervention levels for the groups combined. Effect sizes are included to clarify the magnitude of differences in outcomes between treatment groups and to clarify the change in scores between the baseline and follow-up assessments. The effect size is the observed difference expressed in standard deviation units and is calculated by taking the difference in the mean outcome divided by the pooled standard deviation. P-values are two sided and are considered significant at the .05 level.

## Results

### *Sample Composition*

At baseline, the sample consisted of 105 families. Of these, 97 completed the intervention with gradual sample loss over the follow-up assessments (Figure 1). Sample loss did not vary by intervention group. Because all children within the age range in each family were recruited, the number of children is larger than the number of families. Twelve families (20%) in the clinician group and eight families (17%) in the lecture group were single parents. Depending on assessment, parent behavior and attitudes scores were obtained for between 95% and 98% of subjects, FRIs for between 87% and 97% of parents and between 81% and 89% of children, child understanding for between 86% and 89% of the children, and internalizing outcomes for between 82% and 87% of the children. Ninety-one families (87%) completed the T6 assessment point.

The time from baseline to sixth assessment averaged of 53 months for both parents and children. It was longer in the clinician intervention for both parents and children (parents:  $M=50.0$  ( $SD=7.2$ ) vs.  $M=56.1$  ( $SD=10.4$ ) months,  $t(157)=4.11$ ,  $p<.001$ ; children:  $M=49.8$  ( $SD=7.4$ ) vs.  $M=54.8$  ( $SD=9.9$ ) months,  $t(112)=3.21$ ,  $p<.01$ ).

At baseline, 151 (79%) (males 62%, females 93%) parents had ever suffered from a mood disorder, with the most frequently diagnosed condition being Major Depression (males

39%, females 86%). In addition, 102 (54%) parents experienced nonaffective disorders, including anxiety disorders and substance abuse. One hundred ten parents had comorbid disorders (males 52%, females 76%) (Table 1).

In the 18 months prior to baseline, both parents in 30 families (35% of two-parent families) had experienced a mood disorder. The average couple's worst GAS score was 50.4 (SD=11.8; range 25-70) in the same interval, indicating significant family impairment. Similarly, in approximately 44% of couples, at least one parent met criteria for a nonaffective disorder in the past 18 months. The number of active illness episodes in the intervals slowly declined but remained high: 90% of families had at least one mood disorder from T1-T3, 81% from T3-T4, 72% from T4-T5, and 66% from T5-T6. Rates of non-affective disorders showed corresponding decline from T1-T3, 43%, from T3-T4, 37%, from T4-T5 36%, and 31% from T5-T6.

There were no significant differences in baseline demographic or psychopathology between the intervention groups on parent or child variables. The mean age of the parents was 43, of the children 12. The mean worst GAS in the parents was 60. Sixty-four percent of parents had major depression in the clinician group and 65% in the lecture group. The eight families who dropped out between assessment and intervention were not different from the 97 families who completed the intervention on measures of global functioning or SES. There were no significant differences between groups for completion status on any variable.

*Family responses to intervention.* Parents in the clinician intervention group averaged 2.7 more child-related behavior and attitude changes than lecture parents did. Clinician and Lecture means: 7.04 (SD=3.0) vs. 4.39 (SD=2.7), effect size 0.84;  $\chi^2(1) = 52.8, p < .0001$ ). The number of reported changes increased across the four follow-up assessments ( $\chi^2(3) = 88.6, p < .0001$ ). The IP reported 0.95 more changes than did the NIP ( $\chi^2(1) = 4.53, p < .05$ ). Neither

gender nor the couple's worst functioning predicted the parents' response to intervention in this domain. The average number of changes made did not vary by completion status.

*Children's change in understanding of parental illness.* Children in the clinician group scored, on average, 0.7 points higher (Clinician and Lecture means: 3.0 (SD=2.1) vs. 2.3 (SD=2.1), effect size=0.33) on improved understanding of parental mood disorder ( $\chi^2 (1) = 5.0$ ,  $p < .05$ ) than those in the lecture group across the four follow-up assessments. Scores increased significantly over time ( $\chi^2 (3) = 9.0$ ,  $p < .05$ ) but, with developmental stage in the model, this increase was not significant. On average, older children scored 0.6 points higher than younger children did, but the difference did not reach statistical significance. Completion status was a significant covariate, with completers scoring, on average, 0.95 points higher than partial completers ( $\chi^2 (1) = 5.3$ ,  $p < .05$ ).

*Connection between parent change and child change in understanding.* At each assessment, child understanding scores correlated significantly with concurrent couple-level parent changes in behavior and attitudes (Spearman  $r = .26$ ,  $p < .01$ ), and with parent changes at the previous assessment point (Spearman  $r = .28$ ,  $p < .01$ ). Regression analysis indicated concurrent parent changes significantly predicted child understanding ( $\chi^2 (1) = 14.1$ ,  $p < .001$ ), as did previous parent changes ( $\chi^2 (1) = 18.3$ ,  $p < .0001$ ). When both previous and concurrent parent changes were used to predict child understanding, parent changes from the previous assessment significantly predicted child understanding ( $\chi^2 (1) = 13.6$ ,  $p < .001$ ), while concurrent changes were no longer significant. With previous parent changes in the model, neither completion status, nor treatment group were significant predictors of child understanding changes.

*Internalizing symptomatology.* Overall, internalizing  $t$ -scores decreased 3.2 points from baseline levels ( $\chi^2 (1) = 9.0$ ,  $p < .001$ , effect size = 0.32). Baseline levels significantly predicted

later internalizing scores ( $\chi^2(1) = 28.2, p < .0001$ ). With baseline scores in the model, there was no main effect for intervention group, but there was for gender, with girls scoring, on average, 3.7 points higher than boys ( $\chi^2(1) = 8.3, p < .01$ , effect size = 0.37). In addition, completers scored, on average, 4.0 points lower than partial completers ( $\chi^2(1) = 4.1, p < .05$ ).

*Family functioning.* Parent FRI scores increased, on average, 1.0 point from baseline ( $\chi^2(1) = 11.2, p < .001$ , effect size = 0.20). Controlling for baseline FRI, the intervention groups did not differ in post-intervention FRI scores ( $\chi^2(1) = 0.3, p > .1$ ). Baseline FRI values significantly predicted later FRI scores ( $\chi^2(1) = 167.6, p < .0001$ ). Controlling for baseline FRI, there was a main effect for IP status, with non-ill parents scoring 1.4 points higher on average ( $\chi^2(1) = 7.99, p < .01$ ). Couple's worst GAS scores predicted FRI scores ( $\chi^2(1) = 21.9, p < .0001$ ), with higher adaptive functioning levels predicting higher FRI scores. FRI scores averaged 2.2 points higher for subjects with more complete data ( $\chi^2(1) = 9.7, p < .005$ ).

Child FRI scores increased, on average, 2.5 points from baseline ( $\chi^2(1) = 19.3, p < .0001$ , effect size = 0.50). Each post-baseline mean FRI level was significantly higher than baseline in pair-wise post-hoc comparisons, and means at the post-baseline assessments did not differ significantly. Adjusting for baseline FRI, the groups did not differ in post-intervention FRI scores ( $\chi^2(1) = 1.8, p > .1$ ), nor did FRI scores vary by completeness status ( $\chi^2(1) = 0.74, p > .1$ ). Neither gender nor developmental stage predicted levels of family functioning ( $p$ 's  $> .1$ ). A significant effect for baseline FRI was obtained ( $\chi^2(1) = 5.40, p < .05$ ).

*Recognition and treatment of major depression.* Children were excluded from the study when parents reported a history of major depression in the children by brief screening. However, data from the KSADS revealed that 16 children *had* experienced an episode of major depression

prior to baseline. Only two (13%) were treated. Nine experienced an additional episode of major depression after baseline. Six of these nine (67%) received treatment.

Of the 122 children with no baseline major depression, 17 (eight in the clinician-facilitated group and nine in the lecture group) developed at least one episode of major depression after intervention. By T6, 12 of the 17 (71%) children received treatment for at least one episode (6/8 in the clinician group and 6/9 in the lecture group). This suggests recognition was much higher at post-intervention assessments than at baseline (12 of 17 (71%) vs. 2 of 16 (13%), respectively ( $\chi^2(1) = 11.4, p = .001$ )).

### Discussion

This paper presents one of the first reports of long-term, sustained, positive family change in response to family based preventive intervention for parental mood disorders. As hypothesized, parents in the clinician group reported changes in illness-related behaviors and attitudes that were sustained over time and were greater than the lecture group. IP parents reported more changes than NIP parents did, and group differences observed initially at T3 continued through T6. As hypothesized, children's understanding of parental disorder was sustained over time and greater in the clinician group. Parental behavior and attitude changes and degree of child understanding of parental mood disorder was positively associated, suggesting the interventions increased positive family interactions. Given the range of response within intervention groups, parent changes were more effective predictors of child change than group membership. These findings are significant considering children and parents were exposed to high levels of ongoing parental mood disorder, which undermines interpersonal relationships.

Adolescent internalizing symptoms diminished over time in both groups, although they were going through a period of increased risk (Rao et al., 1995) and would be expected to have

increased depressive symptomatology (Lewinsohn, Rohde, & Seeley, 1998). This is an important prevention effect, as internalizing symptoms are predictors of and precursors to major depression. That girls fared worse than boys is consistent with literature indicating that, at puberty, girls begin to display higher levels of depressive symptomatology (Angold et al., 1998).

Unlike the low rates of recognition and treatment for youngsters with major depression prior to baseline, rates of recognition and treatment of subsequent depression after intervention were much higher (including rates for those not previously depressed). Approximately two-thirds of these youngsters were recognized and treated when depressed. We recognize this offers only a rough comparison since before intervention, the children were younger and at an earlier developmental stage. As another comparison, with our own risk study of children drawn from the same HMO over a 4-year period, the majority of whose parents were depressed, only 30% of those with depression were recognized and treated (Beardslee et al., 1996). Future studies with larger samples should examine what strategies are most effective in increasing recognition and treatment for depression in families at high risk for depression.

As hypothesized, positive changes in family functioning also were sustained over time. Increases in parent-reported changes in behaviors and attitudes, and reports by parents and children of improved family functioning on the FRI, provide multi-modal support for this hypothesis. Group differences in family functioning were found on some, but not all, measures. Future analyses will explore within and between group differences to determine which intervention strategy works best for which families.

Several characteristics of the intervention approaches may have contributed to their success. First, our approaches were strength-based (Maton, Schellenbach, Leadbeater & Solarz, 2004). Second, the clinician-facilitated program was flexible and tailored to each family's needs.

Third, these approaches focused on helping the family develop effective problem-solving strategies and coherent narratives about their family's experience with depression (Beardslee, 2003, Focht & Beardslee, 1996, Podorefsky McDonald-Dowdell, & Beardslee, 2001). The value of family narratives in dealing with both physical and mental illness has come to be recognized widely (Pennebaker, 1997). Fourth, parents were assisted in obtaining treatment, and care was coordinated between delivery of preventive intervention services and the clinician providing treatment for depression or other illnesses. Finally, we believe the continued support throughout the study for families in both groups influenced the sustained effects. These included regularly scheduled assessments, consultation as requested, and, in the clinician group, regularly scheduled telephone meetings. The groups did not differ in terms of the use of consultation as requested. We believe regularly scheduled assessments for both groups provide a reminder to subjects of the goals presented in the intervention (Finn & Tonsager, 1997). We recommend regularly scheduled follow-ups and availability of consultation as requested from families as part of any long-term prevention strategy.

Although findings from this study are promising, there are both strengths and limitations in our design that have important implications for future work in this area. First, we chose two forms of intervention compatible with public health approaches and did not elect to have a control group without intervention. We believed not offering families some form of intervention would be unethical. Future studies should explore the effects of less intensive models of intervention than these two to separate the effects of time and treatment in a clearer fashion. Given that these youngsters are passing through a crucial developmental epoch in adolescence (Conger & Ge, 1999; Ge & Conger, 1999), such designs would also allow exploration of normal

developmental changes these youngsters are undergoing versus changes from receiving preventive intervention.

Second, the observed effects, while sustained, were small on some measures, as might be expected from a relatively brief intervention. For any individual measure, there was some sample loss. We have addressed the sample loss in two ways: (1) the general estimating equations take into account all available data; and (2) completion status did not affect any group comparisons. The possibility of response bias on the respondents' part should be considered, although this would operate equally for both interventions. The clinician intervention group was followed slightly longer than the lecture group because it took longer to deliver and to schedule follow-up assessments, which could be done only after the clinician follow-up; however, given the lengthy follow-up period, this is unlikely to have affected the results. We also recognize that the assessors could not be blind to the intervention that the subjects received although we have demonstrated independent reliability for the ratings. Future studies using less intensive questionnaires can assess the outcomes blind to assessment conditions.

Third, in addition to participating in these interventions, a large number of families received other treatment (e.g., medications for depression) over the follow-up period, and positive changes from this treatment may have contributed to the sustained effects we report. In examining the effects of outside treatment, we found that participation in outside therapies did not affect the group differences in measures designed to detect changes in mediating variables (i.e., changes in parental behaviors and attitudes and childrens' improved understanding of parental mood disorder). However, families who sought and received outside therapy reported *poorer* functioning on the FRI and on the YSR/YASR. It is likely that the association between treatment seeking and either family functioning or youth internalizing symptomatology simply

indicates that those who were more ill sought and received treatment. Our measure of outside treatment was a broad nonspecific measure. Future studies should explore in greater detail the kind of treatment parents receive and its impact on preventive interventions.

Finally, the sample was largely white and middle class, and all had health insurance. Similarly, rates of alcohol and substance abuse were low, a reflection of the inclusion/exclusion criteria. Replication of these findings in other samples representing different cultural and economic backgrounds and different levels of access to care is needed. Our initial, randomized replication with a sample of single-parent, urban women of color showed similar effects to our initial comparison trial (Podorefsky et al., 2001). The intervention has also been used in the Netherlands, has been adapted for widespread use in Finland (Beardslee et al., in press), and has been successfully incorporated into a manual-based, cognitive behavioral, family-centered treatment for youngsters with depression and inflammatory bowel disease (Szigethy et al., 2005).

In developing our preventive interventions, planning for dissemination was essential; thus, we chose strategies that could be used by a wide range of clinicians. In Finland, dissemination has occurred, as over 40 master trainers have been trained in the use of these and related methods and they, in turn, have trained over 500 clinicians (Solantaus, 2006). Detailed manuals and training tapes are available from the first author.

Conducting this project made us aware of three larger health care systems problems. First, in contrast to countries with national health insurance (Beardslee et al., in press), this country focuses far less on prevention than on treatment, despite evidence for the effectiveness for a wide array of prevention strategies (Institute of Medicine, 1994). Secondly, while completely compatible, prevention and clinical treatment require different orientations and approaches. Our prevention approach was timed to be delivered when the family was not in

crisis and to focus on acquiring new skills. It was based on the principle that parents could be effective parents despite depression. It also addressed concerns that might arise several years from the point of intervention, as opposed to treatment designed to have maximal effect during acute illness (Beardslee, 1998). Third, it remains difficult to combine child and parent care clinical approaches and/or reimbursement strategies in either prevention or treatment studies (Beardslee & Knitzer, 2004) because health care systems are not integrated to encourage this, and clinicians often are trained to focus on either children or parents, but not on both.

Although the findings of this current project are promising, there is still more to be learned about the effectiveness of these interventions and more methodologically rigorous work needs to be done in part before full-fledged dissemination is appropriate. Future research should also examine relative cost-effectiveness of various prevention approaches.

Given the chronic nature of depression and evidence of sustained long-term effects from these relatively brief standardized public health interventions, incorporation of family psychoeducational preventive approaches should be considered as part of clinical care for parents with depression. Future clinical and research programs should consider involving the entire family; teaching focused problem-solving strategies; helping parents be effective in enhancing strengths in their youngsters; including parental treatment referral as part of intervention and monitoring the effectiveness of parental treatment for depression; providing support for staff to learn and employ new methods; developing shared understanding of the illness by families; and providing long-term resources to families.

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Table 1. Individual Parental Psychopathology.

	Lifetime Diagnosis	18 Mos. – Baseline	T1 – T3	T3 – T4	T4 – T5	T5 – T6
Any Affective Disorder						
Males /	53 (62%)/	43 (51%)/	33 (42%)/	30 (39%)/	29 (39%)/	24 (34%)/
Females	98 (93%)	92 (88%)	79 (81%)	62 (66%)	52 (58%)	47 (54%)
Major Depression						
Males /	33 (39%)/	25 (29%)/	16 (21%)/	13 (17%)/	15 (20%)/	15 (21%)/
Females	90 (86%)	72 (69%)	61 (63%)	46 (49%)	41 (46%)	33 (38%)
Minor Depression						
Males / Females	33 (39%)/ 33 (31%)	15 (18%)/ 19 (18%)	17 (22%)/ 21 (22%)	17 (22%)/ 14 (15%)	16 (22%)/ 14 (16%)	13 (19%)/ 15 (17%)
Intermittent Depression						
Males / Females	5 (6%)/ 9 (9%)	5 (6%)/ 9 (9%)	5 (6%)/ 11 (11%)	4 (5%)/ 7 (7%)	4 (5%)/ 5 (6%)	3 (4%)/ 5 (6%)
Mania						
Males / Females	5 (6%)/ 12 (11%)	2 (2%)/ 5 (5%)	2 (3%)/ 4 (4%)	1 (1%)/ 5 (5%)	1 (1%)/ 1 (1%)	0 / 2 (2%)
Hypomania						
Males / Females	4 (5%)/ 13 (12%)	3 (4%)/ 11 (10%)	2 (3%)/ 6 (6%)	3 (4%)/ 3 (3%)	0 / 4 (4%)	1 (1%)/ 3 (3%)
Schizoaffective Disorder	3 (4%)/	2 (2%)/	0 /	1 (1%)/	1 (1%)/	0 /
Males / Females	1 (1%)	1 (1%)	1 (1%)	0	0	0
Generalized Anxiety Disorder	17 (20%)/	5 (6%)/	4 (5%)/	6 (8%)/	4 (5%)/	4 (6%)/
Males / Females	24 (23%)	7 (7%)	9 (9%)	5 (5%)	6 (7%)	5 (6%)
Phobic Disorder						
Males / Females	11 (13%)/ 24 (23%)	8 (9%)/ 15 (14%)	8 (10%)/ 15 (15%)	7 (9%)/ 12 (13%)	7 (9%)/ 10 (11%)	3 (4%)/ 9 (10%)
Panic Disorder						
Males / Females	7 (8%)/ 16 (15%)	1 (1%)/ 7 (7%)	1 (1%)/ 6 (6%)	1 (1%)/ 7 (7%)	2 (3%)/ 3 (3%)	0 / 4 (5%)
Obsessive Compulsive Disorder	2 (2%)/	0 /	1 (1%)/	0 /	1 (1%)/	3 (4%)/
Males / Females	3 (3%)	0	0	0	0	0
Alcohol Abuse						0 /
Males / Females	12 (14%)/ 6 (6%)	1 (1%)/ 2 (2%)	1 (1%)/ 1 (1%)	1 (1%)/ 1 (1%)	1 (1%)/ 1 (1%)	0
Drug Abuse						
Males / Females	15 (18%)/ 9 (9%)	0 / 0	0 / 0	0 / 0	0 / 0	0 / 0
% Sample with Comorbid Disorders	44 (52%)/ 76 (72%)	14 (16%)/ 38 (36%)	16 (21%)/ 33 (34%)	16 (21%)/ 25 (27%)	13 (18%)/ 18 (20%)	12 (17%)/ 17 (20%)

**Figure 1. Study Flow – Intent to Treat Design**

