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Childhood Depression Subscales Using Repeated Sessions on Children's Depression Rating Scale – Revised (CDRS-R) Scores

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Abstract

Background: Although acute treatments have been shown to be effective in treating early-onset depression, only one-third or thereabouts reach a remission within 3 months. Unfortunately, delayed time to remission in early-onset depression leads to poorer therapeutic outcomes. Clearly, there is a need to identify, diagnose, and provide effective treatment of a depressed patient quickly. A sophisticated understanding of depression subscales and their change over time with treatment could enhance pathways to individualized treatment approaches for childhood depression.

Objective: Previous studies have found that the clinician-measured instrument, Children's Depression Rating Scale-Revised (CDRS-R) measures multiple subscales (or components) of depression. The aim of this study was to see how these subscales may change over the course of a 12-week study. This knowledge will help determine if dimensions/subscales of childhood depression (paralleling the adult literature) using the subscales derived from factor analysis procedure is useful.

Methods: We examined two clinical trials in which youth ($n = 234$) with major depressive disorder (MDD) were treated openly with fluoxetine for eight sessions spread over 12 weeks. The CDRS-R was completed based on clinician interviews with parent and child at each session. Classical test theory and component analysis with associated parallel analysis (oblique rotation) were conducted on each week's scores.

Results: Although more factors were needed for the baseline and first two therapy sessions, a two-factor solution sufficed thereafter. Depressed facial affect, listless speech, and hypoactivity best defined Factor I, whereas sleep problems, appetite disturbance, physical symptoms, irritability, guilt, and weeping best defined Factor II. All other symptoms cross-loaded almost equally on the two factors. The scale's reliability (internal consistency) improved from baseline to exit sessions ($\alpha = 0.65$–0.91). As a result, the clinicians' assessments of the various symptoms became more highly related to one another. This caused the first eigenvalue to increase from 3.24 to 7.38 and the variance explained to increase (%) from 19% to 43% over sessions. These two factors may reflect 1) clinician-observed signs and 2) reported symptoms of depression.

Conclusions: Factor analysis of CDRS-R data in a single session consistently generates a complex and difficult to interpret structure of at least three factors. This makes it very difficult to understand what these factors measure. However, when gathered over additional sessions, the CDRS-R structure tends to simplify to two factors. The reasons for this simplification are as yet unclear and in need of further study.

Introduction

Childhood depression causes significant psychological distress to patients and their families. The effects of depression include problems in the areas of daily functioning, family life, learning, and relationships, suicidal ideation, suicide attempts and nonsuicidal self-injurious behaviors (Birmaher et al. 2007; Emslie et al. 2010; Vitello et al. 2011). In a 2011 nationwide self-report survey, 28.5% of youth between the ages of 14 and 18 reported having felt sad and hopeless almost daily for ≥ 2 weeks. Furthermore, adolescents reported lifetime and past year major depressive episodes of 12.8% and 8.1% respectively (CDC Youth Risk Behavior Surveillance System 2011). Childhood depression often runs a chronic, recurrent course and has been associated with adult depression. Depression is currently characterized as the second leading global cause of lifetime disability in adults. Its impact on

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years lived with disability and economic burden exceeds those associated with major categories of noncommunicable diseases, diabetes, cardiovascular disease, chronic respiratory disease, and cancer (Becker & Klein 2013).

Unfortunately, even with efficacious and intensive treatment (i.e., combination of medication and therapy), only about one third of depressed patients achieve remission within 3 months. Even then, the presence of residual symptoms leaves them vulnerable to relapse (Curry et al. 2010; Kennard et al. 2006). Multiple factors, including biological heterogeneity, age differences, comorbid illnesses, environmental factors, and variable symptom presentation, influence the selection of effective treatments. Currently, treatment is essentially given on a trial and error basis. In the adult depression literature, homogenous subtypes (melancholic, atypical) developed for the purpose of diagnosis and treatment of depression, using pattern recognition of symptoms (*Diagnostic and Statistical Manual of Mental Disorders,* 4th ed., Text Revision [DSM-IV-TR]) have had limitations in their ability to predict response to current treatments, thus far (American Psychiatric Association 2000). To date, studies have found melancholic depression to be associated with hypothalamic–pituitary–adrenal (HPA) axis dysregulation, whereas atypical depression is associated with lower cortisol levels and metabolic dysregulation (Wong et al. 2000; Gold and Chrousos 2002; Stetler and Miller 2011; Lamers et al. 2012).

The National Institute of Mental health launched the Research Domain Criteria project (RDoC), to address this problem. The project conceptualizes mental illness as a disorder of brain circuits, and seeks novel approaches to evaluating mental illnesses with the use of integrated techniques in genomics and neuroscience to compliment current clinical observations in patient populations (Insel et al. 2010). To this end, the identification of consistent dimensions of childhood depression using data-driven clinical observations may enhance our understanding of its psychopathology when integrated with neuroscience and genomic techniques.

An approach being used in depression studies is that of data-driven methods employed to evaluate dimensions within depressive illness. One such technique is the latent factor analysis (LFA), a type of latent analysis. LFA is a complex array of structure- analyzing procedures used to identify interrelationships among a large set of observed variables that are then grouped into smaller sets of variables, through data reduction. The variables groups have common characteristics, and are known as factors, subscales, or dimensions (Nunnally and Bernstein 1994; Pett et al. 2003).

These methods may provide additional information by deciphering reliable dimensions that exist within reported depressive symptoms, with potential utility in evaluating how symptom dimensions may correlate with brain circuits and biomarkers, providing clinicians and researchers with the following: A deeper appreciation of the underlying pathophysiologic mechanisms of depressive illness, identification of new treatment targets, and subsequent translation of integrated results from research to clinical decision making. The exploratory factor and principal components analysis models (which are types of LFA) are particularly advantageous in examining the heterogeneity of disease syndromes, as they are designed to discover structure in the absence of pre-existing hypothesis about subtypes (Lubke and Muthen 2005).

Adult depression studies evaluating subtypes using LFA methods have yielded multiple results. The LFA models revealed a range of factors (two to seven factors), with three factor solutions frequently occurring: Cognitive, affective (depressed mood, anhedonia, feelings of worthlessness, poor concentration, suicidality), and somatic (fatigue, appetite, sleep, psychomotor disturbance) dimensions were apparent (de Jonge et al. 2007; Roest et al. 2011; de Jonge et al. 2012; van Loo et al. 2012; Manian et al. 2013). In a systematic review of adult depression studies, van Loo and colleagues report that evidence is lacking for consistent, depressive symptom dimensions/subtypes, as different symptoms grouped together across multiple studies analyzed. Probable explanations may include: Differences in populations evaluated (e.g., inpatient vs. outpatient, postpartum depression), questionnaires used (self- vs. clinician- rated), study design and methodology (cross-sectional study designs, analysis using one session), and theoretical modeling choices preceding analyses (exploratory, cluster, principal components analyses and combined).

Despite these issues, the use of LFA method still holds promise in enabling understanding of psychopathology and developing new treatment targets for treatment utility, using a modified approach by studying dimensions longitudinally for change with treatment in clinical trials. In a combined sample of bipolar and unipolar depressed subjects (Mitchell et al. 2013), lamotrigine was helpful in alleviating the dimensions of depressive cognitions and psychomotor retardation in subjects with bipolar depression. Results from adult studies have informed and guided similar studies of dimensions in childhood depression (Guo et al. 2006; Jain et al. 2007; Bernstein et al. 2010; Tao et al. 2010).

One of the measures frequently used in clinical trials for childhood depression assessment disease severity and changes in depression symptomatology is the clinician- rated Childhood Depression Rating Scale-Revised (CDRS-R) (Poznanski et al. 1984; Poznanski and Mokros 1996). Of its 17 items, 14 are rated based on the child, parent, and clinician's assessment of the child's mood during the days or weeks prior to the interview, whereas the last 3 items are the clinician's assessment of the child's nonverbal behavior during the same interview. Because of the unique features of observed and reported symptoms in this questionnaire, the total score offers a robust assessment of depression severity and improvements with intervention. Using exploratory factor analysis, including a Promax rotation, Guo et al. (2006) described five factors using data from a baseline CDRS assessment score in depressed children. These factors were: Observed depression (tempo of speech, hypoactivity, depressed affect), reported depression (weeping, depressed feeling, low self-esteem), anhedonia (social withdrawal, inability to have fun), morbid thoughts (morbid ideation, suicidal ideation), and somatic symptoms (excessive fatigue, sleep, physical complaints, schoolwork problems). Jain et al. (2007) noted that the internal consistency of individual items when the baseline and exit CDRS scores were examined, improved remarkably at the end of the study. However, they found a three-factor solution to be more suitable. Using a cross-sectional study approach of a convenience sample ($n = 140$) of depressed and nondepressed children from an outpatient psychiatric clinic, Bernstein et al. (2010) also observed that the CDRS-R exhibited at least two factors in their sample using principal component analysis, where items 1–14 loaded on factor one whereas items 15–17 loaded on factor two. The three-factor solution was considered to be statistically difficult to interpret because of the sparseness of salient variables (variables having large loadings) on the second and third factors. This study was unable to make conclusions about the two factor structure derived, because of similar complications observed in adult studies as outlined (Bernstein et al. 2010).

A critical theme seen to evolve in the abovementioned studies is that items measuring ''observed'' symptoms of depression on the CDRS-R (factor 2, items 15–17) are consistently delineated as a separate factor. These tentative findings raise important questions

about depressive illness and patients' perceptions of their illness, which may vary from person to person. We are yet to understand the impact of these dimensions on depression treatment outcomes. Consequently, examining longitudinal changes of depressed childrens' perception of their symptoms, a frequently neglected facet in depression research, may contribute to our understanding of childhood depression and further the development of effective, efficient treatments for depression.

This study examines a homogenous population of depressed children using the repeated testing of factor analysis of the CDRS-R, of eight consecutive sessions during a clinical intervention. We intend to evaluate the derivation factor structure over time, as well as the impact of the factors on baseline demographic and clinical characteristics.

Methods

Evaluable sample

We examined two relapse prevention clinical trials in which youth with major depressive disorder (MDD) were treated openly with fluoxetine for the first 12 weeks (Emslie et al. 2008; Kennard et al. 2008). In one trial, 168 youth (ages 8–17 years) were treated with fluoxetine 10–40 mg for 12 weeks, and responders were eligible to enter into a double-blind discontinuation phase (Emslie et al. 2008). In the other trial, 66 youth (ages 11–17 years) were treated with fluoxetine 10–40 mg for 12 weeks, and responders were randomized to either begin relapse prevention cognitive behavioral therapy (CBT) in addition to medication treatment, or to continue on fluoxetine alone (Kennard et al. 2008). Patients in both studies were treated identically for the visits utilized in these analyses (first 12 weeks), and the data are reported by visit. Primary outcomes for both studies have been reported previously (Emslie et al. 2008; Kennard et al. 2008).

Measures

Across both trials, 234 youth, ages 7–17 years, entered a 12 week open label trial of fluoxetine before randomization. Because not all patients came for all visits, sample sizes varied from 187 to 234. Patients presented to see a clinician every week for the first 4 weeks, and then biweekly through week 12 (weeks 1, 2, 3, 4, 6, 8, 10, and 12). Severity of depression symptomatology (based on the CDRS-R) and global improvement (based on the Clinical Global Impressions – Improvement [CGI-I]) were assessed at each visit by the treating clinician.

CDRS-R and CGI

The CDRS-R is a 17 item semistructured clinician-rated scale that incorporates depressive symptoms from the patient, caregiver, and clinician for the first 14 items. The last three items are inferred by the clinician alone. The items for sleep, appetite, and tempo of speech are rated 1–5 but all others are rated from 1 to 7 with higher scores indicating increased pathology (Poznanski et al. 1996).

Individual items on the CDRS-R are summed to create a total score (range 17–113). A score ≥ 40 corresponds to moderate to severe depression, and defined eligibility for entry into the studies.

The CGI (Guy 1976) assesses clinical severity and improvement, each with a seven point scale (lower values being more favorable). At intake, only severity is rated. In subsequent assessments, both severity and improvement are rated. This is a standard scale for psychopharmacologic research, and a CGI improvement score of 1 (very much) or 2 (much) improved is considered an acceptable response to acute treatment, as is a clinical severity rating of ≤ 3. The intraclass correlation for CGI-I as a continuous variable is 0.93, and if used as a categorical variable, it is $\kappa = 0.95$ (Guy 1976). For the present analyses, remission was defined as a CDRS-R ≤ 28 and a CGI score of 1 or 2.

Statistical methods

The data were analyzed as follows.

First, demographics were analyzed using 1) SAS Proc Means, 2) Proc Freq, and 3) Proc TTest (version 9.3). Part 1 generated means and standard deviations of continuous variables; part 2 generated distributions and tests of group difference for categoric variables, and part 3 generated tests of group difference for continuous variables.

Next, classical test theory (CTT) analysis generated a mean and item/total correlation for each item or domain, a coefficient α internal consistency reliability, a scale mean, and the standard deviations for the scale as a whole within each session. SAS Proc Reliability (version 9.3) was used.

Finally, component analysis, with an associated parallel analysis to define dimensionality (Horn 1965; Humphreys and Ilgen 1969; Humphreys and Montanelli 1975; Montanelli and Humphreys 1976), was used to define the scale's dimensionality in each session. In parallel analysis, random data matrices consisting of uncorrelated normal deviates having the same number of ''variables'' (17) and ''subjects'' (from 187 to 234) as the real data were generated. There are several variants in the technique. In the present case, 50 such randomizations were generated per sample and the successive eigenvalues (scree) averaged. The simulated scree is then compared with the scree obtained from the real data. Specifically, a one dimensional solution is one in which the eigenvalue of the first real principal component exceeds the eigenvalue of the (averaged) first randomly generated principal component, and the reverse is true of the subsequent eigenvalues. More than one principal component eigenvalue exceeding the eigenvalues of subsequent randomly generated eigenvalues means that the questionnaire is multidimensional. Factors extracted were subjected to an oblique (Promax) rotation to allow for easier interpretation of the results. SAS Proc Factor (version 9.3) was used.

Results

Of the 234 youth included in the analysis, 55.6% were male, most were Caucasian (73.9%), and the mean age was 12.6 ± 2.9 years. The average baseline CDRS-R score reflected moderate to severe depression (57.7 ± 8.0). All 234 subjects had a baseline CDRS-R score, and 215 had a CDRS-R exit score, which occurred after 12 weeks of treatment on fluoxetine or at the time of exiting the study. Table 1 describes demographic and clinical variables observed at baseline.

Table 2 indicates that the mean scores decreased over sessions by a factor of $\sim$50%. However, the standard deviations remained relatively constant. Coefficient α was a moderately low 0.65 at baseline. It improved nearly monotonically at each subsequent session, so that the exit α was 0.91. Likewise, the first eigenvalue (λ_1) progressively increased from 3.24 to 7.38, implying that the variance it explained increased from 19% to 43%. This means that the test measured overall depression as opposed to individual unrelated items more strongly over time (the question of whether the patients became more or less depressed is a totally separate one, discussed subsequently). The baseline session (session 0) generated

Table 1. Baseline Demographic and Clinical Characteristics

Variable	*Total (n = 234)*
Age (years)	12.6 ± 2.9
Children	86 (36.8%)
Adolescents	148 (63.2%)
Gender	
Male	130 (55.6%)
Female	104 (44.4%)
Ethnicity/Race	
Caucasian	173 (73.9%)
African American	21 (9.0%)
Hispanic	32 (13.7%)
Other	8 (3.4%)
Number of depressive episodes	
1	160 (68.4%)
2	59 (25.2%)
3	12 (5.1%)
4	3 (1.3%)
Duration of current depressive episode (wks)	25.5 ± 21.5
Number of comorbid diagnoses	1.1 ± 1.0
0	66 (28.2%)
1	100 (42.7%)
2	50 (22.6%)
3+	18 (7.7%)
Suicidal behavior during episode	
None	49 (21.0%)
Wishes	81 (34.8%)
Thoughts	77 (33.0%)
Plans	19 (8.2%)
Attempts	7 (3.0%)
Baseline CDRS-R Total Score	57.7 ± 8.0
Baseline CGI Severity	4.9 ± 0.7

CDRS-R, Children's Depression Rating Scale-Revised; CGI, Clinical Global Impressions.

five factors according to parallel analysis, but they decreased to three factors at sessions 2 and 3, and stabilized at two factors thereafter. Also, the scale mean decreased from 57.71 to 28.14. The scale standard deviation increased slightly, but most of this change occurred between sessions 0 and 1; changes past this point were erratic in direction.

Table 2. Sample Sizes

Session	n	*Mean*	*SD*	α	$_1$	%	*Number of factors (NF)*
0	234	57.71	8.08	0.65	3.24	0.19	5
1	224	49.95	9.98	0.81	4.60	0.27	3
2	213	42.51	10.54	0.86	5.38	0.32	3
3	212	37.73	10.84	0.88	6.04	0.36	2
4	220	34.29	10.55	0.89	6.34	0.37	2
6	212	31.73	10.4	0.90	6.75	0.40	2
8	201	29.33	9.14	0.88	6.38	0.38	2
10	187	27.43	9.31	0.90	6.86	0.40	2
12	215	28.14	10.22	0.91	7.38	0.43	2

Children's Depression Rating Scale (CDRS) Mean (mean), CDRS standard deviation (SD), coefficient α, first eigenvalue ($_1$), percent'age of variance explained by the first factor (%), and number of factors (NF) inferred from parallel analysis.

Table 3. CDRS Factor Pattern and Structure from Exit Session (Session 12)

	Pattern		*Structure*		
Item	*I*	*II*	*I*	*II*	h^2
1	0.31	0.45	0.55	0.62	0.45
2	0.58	0.38	0.78	0.69	0.72
3	0.55	0.34	0.73	0.63	0.62
4	0.16	0.51	0.42	0.60	0.37
5	-0.05	0.68	0.30	0.65	0.43
6	0.44	0.37	0.63	0.60	0.50
7	-0.08	0.50	0.18	0.46	0.22
8	-0.02	0.76	0.37	0.74	0.55
9	-0.17	0.55	0.11	0.46	0.23
10	0.34	0.59	0.64	0.76	0.66
11	0.47	0.49	0.72	0.74	0.70
12	0.28	0.47	0.53	0.62	0.44
13	0.47	0.17	0.56	0.41	0.33
14	0.01	0.68	0.37	0.68	0.47
15	0.88	-0.06	0.85	0.40	0.73
16	0.98	-0.22	0.86	0.29	0.78
17	0.99	-0.25	0.86	0.26	0.78
Factor variance			0.36	0.34	0.53
Factor correlation			0.52		

CDRS-R, Children's Depression Rating Scale.

Table 3 contains the Promax factor pattern and structure obtained from the exit session (session 12). At this point, depressed facial affect, listless speech, and hypoactivity best define Factor I, whereas sleep problems, appetite disturbance, physical symptoms, irritability, guilt, and weeping best defined Factor II. All other symptoms cross-loaded almost equally on the two factors. The distribution of items into each factor seems to reflect the type of information, with factor I items stressing signs of depression, and factor II items stressing symptoms of depression.

A Varimax rotation is produced by default when a Promax rotation is generated in SAS. Had the factor correlation been relatively low, we would have accepted the Varimax rotation for simplicity. However, as Table 3 notes, the Promax factor correlation was 0.52, hence we felt that a Promax rotation provided a better description of the factor structures.

We then examined the relation between scores on the two components as derived from week 12 and demographic variables presented in Table 1. None were significant.

Discussion

To our knowledge in both childhood and adolescent depression studies, this is the first longitudinal study of evaluation of the changes in the factor structure of the clinician rated CDRS-R following a clinical trial intervention in a homogenous sample using repeated sessions. All subjects were formally diagnosed with depression using a structured interview. One (of several) interpretations of the factor structure is that Factor I measures *signs* of depression (depressed facial affect, listless speech, and hypoactivity) and Factor II measures *symptoms* of depression (sleep problems, appetite disturbance, physical symptoms, irritability, guilt, and weeping) as previously reported (Bernstein et al. 2010). Once again, items 15–17 separated from the other items as one factor, although in this study it was on factor I instead of factor 2, underscoring the consistency of this finding across several studies (Bernstein et al. 2010). Using these labels for convenience, it

implies that changes in *observed signs* vary somewhat independently from changes in *reported symptoms*. However, the fact that the two factors were highly correlated ($r = 0.52$) means that these groups overlap considerably, possibly as a result of the shared effect of other items that did not load onto the factor structure. The items measuring reported depression, anhedonia, excessive fatigue, impaired schoolwork, low self-esteem, and morbid and suicidal ideation loaded almost evenly on both factors (cross-loaded) implying that they reflected both components (see Table 3). Therefore, the two factors are distinguished by the items using only the clinician's observation *in vivo* and the clinician's assessment of reported symptoms from the parent and child.

This result may allow for the discussion and exploration of questions about dimensions in childhood depression, as any of the abovementioned or other findings may have significant clinical implications as to the mechanism involved in the development of childhood depression. If the dimensions are found to be consistent in other studies, they can be integrated with biomarkers, leading to an approach that may contribute to the fund of knowledge about the pathophysiology of childhood and adult depression.

Previous factor analyses of the CDRS-R have described it as multidimensional (Guo et al. 2006; Jain et al. 2007; Bernstein et al. 2010). However, these studies had either gathered data from a single session or a pair of sessions (baseline and exit), subjects had been randomized to different interventions, studies had lacked structured diagnostic interviewing, or the sample had been obtained from a heterogeneous population of depressed and nondepressed children. As noted in Table 2, the baseline session yielded a five factor solution, consistent with previous reports (Guo et al. 2006). The structure then leveled out at two factors by session 3. It is difficult to construct three or more reliable subscales from a set of 17 items, because there will be at most an average of five items/subscale. However, 17 items can be enough to form two reliable subscales if they divide nearly equally on the subscales and relate well to their respective subscale scores.

It is not yet clear why this simplification occurred during our analyses of this sample of patients. We postulate the following: First, it may have been a representation of increasing signal and decreasing noise, hence providing sharper precision of what these two factors do measure. Second, patients may take a ''path of least resistance'' in reporting each individual symptom and simply report each item in terms of whether they feel good or bad (''halo''). A halo effect is defined as the tendency to respond to the items globally rather than specifically, and is a cognitive bias in which one's perception of something or someone is influenced by their overall impression of the person/thing (Wells, 1907; Thorndike 1920; Bird et al. 2000; Myford and Wolf 2004; Hatala et al. 2011; Marais and Andrich 2011). For example, depressed subjects who have anhedonia and social withdrawal, and are socially averse and eager for the interview to conclude, may hastily and consistently rate all items based on their mood at that time. In essence they may ''feel bad;'' hence all other symptoms are rated in terms of how bad they feel, ignoring whether or not they feel motivated.

Third, their added insight may lead them to see their depression as a unified whole rather than a set of minimally related symptoms. For example, depressed subjects prior to the initial evaluation are aware of their sad mood, but unaware that the symptoms of depression also include fatigue, physical symptoms, sleep problems, and appetite and weight changes. After evaluation and with subsequent visits, as they are asked repeatedly about all depression symptoms on the CDRS-R, they begin to understand their individual symptoms as part of the depression syndrome instead of as separate entities.

A fourth alternative leading to a similar interpretation of the results is that the depression treatment may decrease some specific symptoms more than other specific symptoms, if different symptoms have different likelihoods of responding to the treatment. These alternatives all lead to a simplification of the factor structure, but by very different mechanisms. Because the section of data analyzed does not include cognitive therapy or psychoeducation to teach patients the inherent relation among the symptoms, it is difficult to conclude that one or more of these alternatives can be attributed to the subscales derived from the factor analysis. Nonetheless, this issue is one for future research to decide. We lack data at this point to decide among the four alternative interpretations we have listed.

Limitations

Study limitations include the limited sample size. Results from procedures such as factor analysis, which involve estimating a large number of parameters, should be verified in larger samples. Unfortunately, we were not able to ascertain the number of raters who collected, clinical data from patients during the study, nor verify their training and consistency, nor were we able to test for inter-rater reliability in both studies. All pharmacotherapy visits were conducted by child psychiatrists. If the raters were different, we are assuming that the variance was the same, although there is no way of testing this.

Conclusions

The complex structure we found at baseline is similar to that previously reported (Pozanski et al. 1984; Guo et al. 2006), both of which were conducted after a single baseline session. Guo et al.'s method (maximum likelihood) was somewhat different from the present (principal components), but we suggest that this is not what is responsible for the difference noted at later sessions, as both investigators concluded that the structure was complex at the end of a single test administration. Bernstein et al. (2010) reported a two factor structure in the exit session of a study in which the scale was also administered in a baseline session. This is precisely the number of factors we observed, starting with the third therapy session. There was a difference in the items that loaded on the two factors in either study, which may be accounted for by the heterogeneity of depressive symptoms and severity of the sample in the study by Bernstein et al.

Clinical Significance

We are examining the two factors in a different sample of depressed children, The Treatment of Adolescents with Depression Study (March et al. 2004) to examine how stable the factors are and whether they might differentially relate to clinical outcomes. The ultimate goal is to match treatments to subtypes of childhood depression (should they exist). This particular clinical trial examined four intervention groups treated with antidepressants, CBT, combined treatment, or placebo in a factorial design over 12 weeks. In addition, it seems important to consider what produces the change in factor structure with experience. We hope our future research will further understanding about depression in children, so as to improve its treatment.

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