Factors that influence Psychosocial Functioning in Adolescents with Sickle Cell Disease.

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Abstract:

Objective: To examine whether psychosocial factors play a more important role than biomedical risk factors in predicting adolescent adaptation to sickle cell disease (SCD) ; to determine whether psychosocial factors moderate the relationship between biomedical risk factors and adaptation.

Methods: Ninety African American adolescents from the multisite Cooperative Study of Sickle Cell Disease were recruited to complete a battery that included measures of psychosocial status and psychological adaptation. Data regarding their health status were collected from medical records.

Results: The findings revealed that intrapersonal (self-esteem, social assertiveness), stress-processing (use of social support), and social ecological factors (family relations) were significant predictors of adaptation; however, biomedical factors did not predict adaptation. There was no evidence that psychosocial factors moderated the relationship between biomedical risk factors and adaptation.

Conclusions: Psychosocial factors proved to be better predictors of adaptation than biomedical risk factors. Additional research is needed to better understand the nature of the interrelationships among biomedical risk factors, psychosocial factors, and adaptation.

Key words: sickle cell; adjustment to chronic illness.

Article:

Sickle cell disease (SCD), a chronic hereditary condition, refers to a group of disorders in which an abnormal hemoglobin is present (Charache, Lubin, & Reid, 1989). The symptoms may include pain episodes that can require hospitalization. Repeated episodes may lead to life-threatening complications (i.e., major organ damage). Sickle cell disease is mainly found among individuals whose heritage can be traced to Africa, India, the Middle East, or the Mediterranean. It has been estimated that 1 in 650 African American newborns (Thompson, 1995) or 65,000 African Americans (Charache, Lubin, & Reid) has SCD.

Increased life expectancy due to recent medical advances has heightened the need to understand more fully the psychosocial aspects of living with sickle cell (Fischoff & Jenkins, 1987) and the threat to adaptation during adolescence (Armstrong, Lemanek, Pegelow, Gonzalez, & Martinez, 1993; LePontois, 1986). Adaptation is used here in a broad sense to refer to both psychological functioning (e.g., anxiety, depression) and personal adjustment (e.g., behavior problems, other externalizing disorders) (Brown, Doepke, & Kaslow, 1993).

Previous researchers have demonstrated that chronic illness in general (Lavigne & Faier-Routman, 1992) and sickle cell disease in particular (Bennett, 1994; Brown, Doepke, et al., 1993; Brown, Kaslow, et al., 1993; Gil, Wilson, & Edens, 1997; Kliewer & Lewis, 1995; Thompson, 1995; Thompson et al., 1999; Thompson, Gil, Burbach, Keith, & Kinney, 1993) are risk factors for adolescent adaptation. Yet previous findings have been inconsistent regarding whether SCD adolescents differ from person depression or other internalizing disorders (Brown, Kaslow, et al., 1993; Iloeje, 1991; Morgan & Jackson, 1986; Siegel, Golden, Gough, Lashley, &

Secker, 1990), anxiety (Schoenherr, Brown, Baldwin, & Kaslow, 1992; Treiber, Mabe, & Wilson, 1987), externalizing disorders (Brown, Kaslow, et al., 1993), and social relations (Morgan & Jackson, 1986; Noll, Bukowski, Davies, Koontz, & Ris, 1992).

One explanation for the inconsistent findings is that adolescents with SCD or other chronic illnesses may indeed vary in their psychosocial adaptation to the unpredictability and seriousness of the complications (e.g., intermittent pain crises) and the various treatment issues associated with the condition. In fact, in recent studies using within-group designs, researchers have documented this variability in adaptation to sickle cell (Thompson et al., 1993; Thompson, Gil, Keith et al., 1994).

Two related explanations for these inconsistent findings appear possible. First, it is conceivable that variability in the biomedical status (i.e., medical severity) of the participants across studies of sickle cell disease may account for the discrepant findings on adaptation. However, studies that include both biomedical and psychosocial factors in the same regression models provide convincing evidence that biomedical factors account for very little of the variability while psychosocial factors account for considerably more of the variability in adaptation to sickle cell (Thompson et al., 1993; Thompson et al., 1999) and other chronic illnesses (Bennett, 1994; Lavigne & Faier-Routman, 1993). One objective of this study is to examine in a national sample the hypothesis that psychosocial factors explain more of the variability in adaptation than biomedical factors (i.e., medical severity).

A second possible explanation for this inconsistency is that previous research may not have considered the possibility that psychosocial factors contribute to the variability in adaptation in two markedly different ways. First, psychosocial factors may be directly associated with adaptation. However, in addition, psychosocial factors could conceivably moderate or buffer the effects of medical factors on adaptation. If this buffering effect occurs, then the lack of psychosocial resources may increase the likelihood that significant medical problems are associated with less favorable adaptation. Nevertheless, it may also be important to demonstrate that psychosocial factors do not play a moderator role. In fact, such findings may suggest that psychosocial factors are even more important than moderator models suggest. A moderator model would suggest that psychosocial factors are primarily important when severe medical conditions are threatening adaptation. However, the absence of a moderator role would suggest that the importance of psychosocial factors for predicting adaptation is fairly independent of medical severity.

Two theoretical models from the stress and coping literature provide a useful framework for conceptualizing the interrelationships of biomedical factors, psychosocial factors, and adaptation. The transactional stress and coping model by Thompson is grounded in ecological-systems theory. Thompson and colleagues conceptualize a relationship between chronic illness (a potential stressor) and adaptation that varies as a "function of biomedical, developmental and psychosocial processes" (Thompson & Gustafson, 1996, p. 143). Variables consistent with this model have been demonstrated to account for 30% to 68% of the variance in psychosocial adaptation among children with sickle cell disease (Thompson et al., 1993) as well as cystic fibrosis (Thompson, Gustafson, George, and Spock, 1994; Thompson, Gustafson, Hamlett, & Spock, 1992).

Another risk-resistance model, the Disability-Stress-Coping Model, also includes biomedical factors along with psychosocial resistance factors (Wallander & Varni, 1992; Wallander, Varni, Babani, Banis, & Wilcox, 1989). Three types of psychosocial resistance factors—intrapersonal, stress-processing, and social-ecological—are proposed in the Wallander model. A major strength of this model is the inclusion of additional social-ecological factors as Thompson and Gustafson (1996) proposed in their own critique of the transactional stress and coping model. Prior literature has applied this model to adaptation among children with sickle cell disease (Brown, Doepke, et al. 1993) and other chronic illnesses such as diabetes, JRA, and spina bifida (Wallander & Varni, 1992; Wallander et al., 1989).

Both theoretical models imply that psychosocial factors might serve as potential protective mechanisms by buffering the impact of the stressor (e.g., medical severity) on adaptation. The existing body of research

associated with these models has made a substantial contribution by identifying psychosocial variables associated with variability in adaptation. However, the issue of whether psychosocial resistance factors buffer the relationship between biomedical risk factors and adaptation is largely unexplored among adolescents with sickle cell (Thompson, 1995). A new wave of studies is now needed to examine this issue. Therefore, a second objective of this research is to examine whether psychosocial factors moderate the relationship between biomedical risk factors and adaptation (psychological functioning).

Self-esteem (Gray, Genial, & Tambolane, 1980; Kumar, Powers, Allen, & Haywood, 1976; Thompson et al., 1993; Thompson, Gustafson, George, et al., 1994; Thompson, Gustafson, Hamlett, et al., 1992), assertive communication style (Belgrave & Washington, 1986), and positive outlook (LePontois, 1986; Lewis & Kliewer, 1996; McArnarney, 1985) are three interpersonal factors demonstrated to be associated with adaptation to sickle cell in previous research. The most promising stress-processing factors from previous research include coping strategies (Gil et al., 1991; Gil, Abrams, Phillips, & Keefe, 1989; Nash & Telfair, 1994; Thompson et al., 1993; Thompson et al., 1994) and knowledge about sickle cell (Nash & Telfair, 1994). In addition, family factors are the most frequently studied social-ecological variables (Thompson et al., 1999). Findings from previous research suggest that family factors such as cohesion (Kliewer & Lewis, 1995), organization and control (Burlew, Evans, & Oler, 1989) family support (Wallander & Varni, 1989), and parent-child relationships (Hurtig, Koepke, & Park, 1989) may be important social-ecological factors for understanding adaptation. All these factors may be potential moderators.

Psychological functioning is the measure of adaptation in this study for several reasons. First, previous research has demonstrated that anxiety (Molock & Belgrave, 1994; Treiber et al., 1987), depression (Bennett, 1994; Brown, Kaslow, et al., 1993; Lavigne-Faier & Routman, 1992), and other internalizing disorders (Thompson et al., 1992; Thompson et al., 1993; Thompson et al., 1994; Thompson et al., 1999) are more prevalent among adolescents with SCD than externalizing disorders. Therefore, anxiety and depression may be the most appropriate outcome variables for examining the moderator role of psychosocial factors. Second, depressive symptoms may be particularly important to include in samples of adolescents with chronic illness because of evidence that depressive symptoms may become evident during adolescence (Bennett, 1994; Brown, Kaslow, et al., 1993).

To summarize, this study has the potential to make two contributions to existing literature. First, it examines the hypothesis that psychosocial factors account for more of the variability in adjustment than medical severity in a national sample. Second, this study can potentially extend our understanding of the nature of the interrelationships among biomedical factors, psychosocial factors, and adaptation. Specifically, it examines whether psychosocial factors moderate the relationship between biomedical factors and adaptation.

Method

Sample

Ninety African American adolescents, 46 male and 44 females, between the ages of 14 and 19 and their parents were randomly selected from 12 centers participating in the multisite Cooperative Study of Sickle Cell Disease (CSSCD). All 90 had sickle cell anemia (HbSS). The mean age of the adolescents was 16.8 years. The median education was tenth grade although the education ranged from seventh grade to two years of college. Additional information on the sample is presented in Table I.

Instruments

Outcome Variables. The outcome variables included two measures of psychological adaptation. These variables were anxiety (state and trait) and depression.

The State-Trait Anxiety Inventory (STAI) (Speilberger, Gorsuch, & Lushene, 1970), a 4-point, 40-item scale, was used to measure trait and state anxiety. The STAI rather than the corresponding version for children (STAIC) was administered because the STAI is described as appropriate for adults and adolescents (Speilberger et al., 1970). Moreover, the STAIC was normed on younger children (grades four to six) than those in our

sample (Speilberger, 1973). Although the authors of STAI report adequate psychometric properties, the authors do not report efforts to evaluate these properties in an African American sample. However, data reported by Brown and Duran (1988) provided evidence that the state and trait subscales were distinct in an African American sample. In this study, the Cronbach alphas were .80 and .83 for the state and trait scales, respectively. Depressive symptoms were assessed using the Beck Depression Inventory (BDI), a 21-item forced choice scale. The Beck has been used successfully with adolescents as young as 13 (Albert & Beck, 1975; Teri, 1982). An overall score on the BDI was obtained as well as separate scores on the cognitive and somatic subscales. Beck (1967) demonstrated an acceptable split-half reliability, test-retest reliability, and construct validity. In two outpatient standardization samples in which African Americans were well represented, Beck (1967) reported Cronbach alpha coefficients of .90 (alcoholics) and .88 (heroin addicts).

Table I. Demographic Characteristics

	Participants		Nonparticipants	
Variable	***n***	**(%)**	***n***	**(%)**
Age[a] (years)				
14	9	(10.0)	35	(12.1)
15	25	(27.8)	30	(10.4)
16	22	(24.4)	49	(17.0)
17	13	(14.4)	46	(15.9)
18	10	(11.1)	66	(22.8)
19	11	(12.2)	63	(21.8)
Male gender	46	(51.1)	140	(48.4)
Head of household				
Male gender	29	(32.2)	113	(39.4)
Married	31	(34.4)	113	(39.5)
High school graduate	54	(60.7)	158	(55.8)
Own home	29	(33.3)	110	(38.7)
Household income <$10,000	46	(55.4)	147	(55.9)

Participants ($N = 90$) were selected from adolescent participants in the Cooperative Study of Sickle Cell Disease (CSSCD). Nonparticipants ($N = 289$) were adolescent participants in the CSSCD who were not selected for this study.
[a] $p < .01$.

Predictor Variables. The predictor variables were grouped into three categories based upon the Wallander and Varni (1989) model. The first category, *intrapersonal factors*, included social assertion, self-esteem, and positive outlook.

The Rathus Assertiveness Schedule (Rathus, 1973), a 30-item self-report measure of the extent to which individuals engage in socially assertive behavior, was used to measure social assertiveness. The Rosenberg Self-Esteem Scale, a 10-item unidimensional scale, was used to measure feelings of self-worth/self-concept and personal acceptance (Rosenberg, 1965). Positive outlook was measured using a composite score based on four items developed specifically for this study. These items asked adolescents to predict the effect of SCD on their life goals (e.g., likelihood of marriage, parenthood, employment). The Cronbach alphas for the Rathus Assertiveness Schedule, the Rosenberg Self-Esteem Scale, and on the positive outlook index were .74, .82, and .55, respectively.

The two *stress-processing variables* were coping style and the general knowledge about SCD. The Coping Health Inventory for Parents (CHIP) (McCubbin, McCubbin, Patterson, Cauble, Wilson, & Warwick, 1983) was adapted for this study. The CHIP was designed for parents; however, the items were modified so that the adolescents could self-report about the coping behaviors they used to adjust to their condition. The acceptable Cronbach alphas for the three factors—(1) maintaining family integration (.86); (2) maintaining social support (.86); and (3) communication to understand the medical situation (.79)—provide some support for the adapted version of this scale.

An eight-item (six true/false/don't know and two multiple choice) measure was developed to assess the adolescent's general knowledge about SCD. Scores, on a ratio scale, ranged from a high score of 8 correct to a

low score of 0 correct. Experts in the field were used to support the face validity of the questions for tapping knowledge about sickle cell disease.

The primary *social-ecological variable* was the family environment. The Family Environment Scale (FES; Moos & Moos, 1986) was used to assess how parents and adolescents perceived their family environments. This true-false, 90-item scale asks respondents to indicate whether each statement describes their family.

Table II. Descriptive Data for Study Constructs

Variable	*n*	*M*	*SD*	Ranges	α
Psychological					
State anxiety	76	34.14	8.89	20, 58	.80
Trait anxiety	75	39.19	8.55	23, 62	.83
Beck Depression, overall	87	7.21	7.47	0, 36	.85
Beck Depression, cognitive	88	6.65	5.99	0, 30	.82
Beck Depression, somatic	87	1.67	2.19	0, 10	.63
Intrapersonal					
Rathus, social assertion	90	12.23	21.56	−46, 59	.74
Rosenberg, self-esteem	87	31.21	5.45	14, 40	.82
Positive outlook	90	1.30	1.49	0, 7	.55
Stress processing					
Modified CHIP, family	88	38.33	10.26	11, 57	.86
Modified CHIP, social support	88	33.07	10.39	11, 54	.86
Modified CHIP, medical knowledge	88	13.08	5.50	0, 24	.79
Sickle cell disease knowledge	90	5.00	1.90	0, 8	.60
Social-ecological					
FES, family relations	80	3.40	2.04	−2, 7	.48
Medical severity index	84	15.71	8.87	0, 34	.59

$N = 90$.

This study utilized an index modeled after the Family Relations Index (FRI) proposed by Holahan and Moos (1981) to measure family relationships and supportiveness. The score is derived by subtracting the sum on the conflict scale of the FES from the combined sum of scores on the cohesion and expressiveness subscales. Due to the length of the entire protocol, the shortened versions of all subscales proposed by Moos and Moos (1986) were used to generate the version of the FRI used in this project. The utility of the abbreviated version was supported in a separate sample in which the full FRI was administered to 116 parents of children ages 2 to 5. The abbreviated version of the FRI used in this study was created within that data set. The correlation between the full and abbreviated version of the FRI was .86 ($p < .001$) (additional information available from Kathleen Burlew).

To assess medical severity, a medical index was created that was consistent with the SCD parameters reviewed by Brown et al. (1993). To create the index, the following information was gathered from medical records: fetal hemoglobin level, average acute chest syndrome rate (per year), pain crisis rate (per year), and number of days hospitalized per year for selected SCD-related events (all calculated for the 5 years prior to study entry). A medical severity score was created by dividing each of the four individual scores into deciles and adding the decile scores. The mean, standard deviation, and other information on the index are presented in Table II. Information regarding the presence of a pain crisis in the preceding 2 weeks prior to the administration of the psychosocial battery was also gathered from medical records.

The test-retest and internal consistency reliabilities reported by the authors for the standardized scales above are generally adequate, although the applicability of the scales to a predominantly African American population is unclear. Therefore, Cronbach alphas were computed on all study variables (see Table II). Generally, the Cronbach alphas were at least marginally acceptable (see Table II).

Procedures

A list of all participants in the CSSCD study between the ages of 14 and 19 was generated at each participating

center. To ensure a representative sample, a random sample of 25% of the CSSCD adolescent participants at each site were approached to participate. Of the 379 adolescents who were enrolled in 12 CSSCD study sites, stratified sampling (with replacement for refusals) based on the number of eligible adolescents at each site was used to select a sample among only those adolescents with homozygous sickle cell disease (HbSS).[1] Written consent for participation was obtained from all parents and adolescents.

The research was introduced to potential participants and their parents as a study of SCD and quality of life. In order for an adolescent to participate, a parent also had to agree to complete the other measures for an adjunct study of parents of children and adolescents with sickle cell disease. The instruments were administered to individuals by a social worker or another person specifically trained for the research (i.e., a nurse).

Data Analysis

Data were analyzed using the SAS statistical package (SAS Institute, 1989). T tests were performed to determine whether the outcome variables differed by age or gender. Since no substantial differences were apparent between younger (14-16 years old) and older (17-19 years old) adolescents or between gender groups, these groups were merged for the remaining analyses. A square-root transformation of the Beck outcome variables (total, cognitive, and somatic) was used after residual analysis of the untransformed Beck variables indicated poor fit and violation of model assumptions.

Some cases were eliminated from all or part of the analyses because of missing data. The medical information was unavailable for six of the cases because these patients received their medical care in a separate facility. Eighteen patients did not complete some part of the anxiety (16) or depression (3) measurements. Twelve did not complete one or more of the predictor variables. The 27 patients who had missing data for one or more variables were not significantly different from the 63 with complete data for any of the other demographic or predictor variables included in the study.

Separate multiple regression models were fit for state anxiety, trait anxiety, and depression (overall score and cognitive and somatic subscales). Preliminary analyses were performed to determine which potential predictor variables had a significant bivariate relationship to each of the outcome variables. Backward selection procedures were then used to select the subset of variables that were statistically significant predictors (SAS Proc Reg). Medical severity was retained in all models. Once the variables had been selected, the analyses were repeated using only those variables in order to increase sample size. Finally, to test for moderator effects, the interaction of each of the significant predictors and the medical severity index was added to each regression model. The results are presented in the tables as forward regression so that the contribution of each variable can be shown. A logistic regression was fit to predict the categorical variable of depression (yes/no) using Proc Logistic in SAS.

Results

Descriptive Statistics

The means and standard deviations for the demographic variables appear in Table I; this same information is provided for the study variables in Table II. We did not have access to information on the number or characteristics of those families who refused to participate. However, we compared the demographic data of the 90 participants with that of 289 adolescent CSSCD participants at the same centers who did not participate in this study. The two groups were similar on gender, percentage with male heads of household, and on two socioeconomic indicators: percentage of families that own homes and percentage with household incomes under $10,000. The only significant difference was that the participants were slightly younger. This suggests the sample may be representative of this population.

Approximately 26% of the sample reported scores on the Beck Depression Inventory indicating mild to severe levels of depressive symptoms. The mean for state anxiety in our sample was somewhat lower than the corresponding means in the normative sample of high school students for male (39.45) and female (40.54) high

school students. Similarly, the mean for trait anxiety was also lower than the means reported among high school students for males (40.17) and females (40.97) in the normative sample.

The medical records indicated an average of 6.54 days hospitalized per year and an average of 1.25 pain episodes per year during the 5 years prior to study entry. Bivariate correlations between the outcome variables and the predictor variables are presented in Table III.

Table III. Pearson Product Moment Correlation Between Outcome Variables and Continuous Predictors

	State-trait anxiety		Beck Depression Inventory[a]		
Predictor variables	**State**	**Trait**	**Overall**	**Cognitive**	**Somatic**
Rathus, social assertion	−.43***	−.53***	−.30**	−.33**	−.19
	(*n* = 76)	(*n* = 75)	(*n* = 87)	(*n* = 88)	(*n* = 87)
Rosenberg, self-esteem	−.34**	−.54***	−.60***	−.58***	−.50***
	(*n* = 74)	(*n* = 73)	(*n* = 85)	(*n* = 85)	(*n* = 85)
Positive outlook	.15	.22	.27*	.21*	.35***
	(*n* = 76)	(*n* = 75)	(*n* = 87)	(*n* = 88)	(*n* = 87)
Modified CHIP, family	−.36**	−.36**	−.14	−.10	−.12
	(*n* = 76)	(*n* = 75)	(*n* = 85)	(*n* = 86)	(*n* = 85)
Modified CHIP, social support	−.39***	−.37***	−.22*	−.19	−.11
	(*n* = 76)	(*n* = 75)	(*n* = 85)	(*n* = 86)	(*n* = 85)
Modified CHIP, medical situation	−.27*	−.20	−.05	−.01	−.01
	(*n* = 76)	(*n* = 75)	(*n* = 85)	(*n* = 86)	(*n* = 85)
SCD knowledge	−.38***	−.32**	−.28**	−.26*	−.27*
	(*n* = 76)	(*n* = 75)	(*n* = 87)	(*n* = 88)	(*n* = 87)
Family Relations Index	−.34**	−.49***	−.41***	−.43***	−.25*
	(*n* = 71)	(*n* = 70)	(*n* = 77)	(*n* = 78)	(*n* = 77)
Medical severity	.08	.06	−.00	−.04	−.03
	(*n* = 72)	(*n* = 70)	(*n* = 81)	(*n* = 82)	(*n* = 81)

[a]Square root transformations used.
*$p < .05$.
**$p < .01$.
***$p < .001$.

Regression Models

Anxiety. Separate multiple regression models were fit using a backward elimination procedure to predict state and trait subscales of the STAI. The unstandardized regression coefficients (B), the standardized regression coefficients (ß)), the squared partial correlations (pr-square), R^2, and adjusted R^2 are displayed in Table IV.

The results revealed that the R^2 for the state regression model was significantly different from zero, $F(3, 68) = 8.47$, $p = .0001$. One intrapersonal (social assertiveness) and one stress-processing (social support) factor contributed significantly to the prediction of state anxiety. The more socially assertive the adolescent ($t = -3.2$, $p = .001$) and the more use of social support as a coping strategy ($t = -2.54$, $p = .01$), the less state anxiety. The medical severity index was held in the model but did not make a significant contribution ($p < .23$). Altogether, the model accounted for 27% (24% adjusted) of the variability in state anxiety. To assess whether either the use of social assertion or social support moderated the relationship between biomedical factors and outcomes, the interaction of each with the severity index was then added as two separate terms to the regression model. The resulting R^2 (.27) appears in parentheses in Table IV. The addition of the interactions did not improve the R^2 significantly ($p = .97$).

Table IV. Predictors of Anxiety

Variable	*B*	*SE B*	β	Partial R^2	Model R^2	*p*
State anxiety[a]						
Medical severity index	0.12	0.10	0.12	.01	.01	
Rathus, social assertion	−0.14	0.04	−0.35	.20	.20	.01
CHIP, social support	−0.25	0.10	−0.28	.07	.27	.05
Trait anxiety[b]						
Medical severity index	0.06	0.09	0.06	.00	.00	
Rathus, social assertion	−0.11	0.04	−0.30	.34	.34	.01
Rosenberg, self-esteem	−0.46	0.17	−0.29	.12	.46	.01
Family Relations Index	−1.09	0.40	−0.27	.05	.51	.01
CHIP, social support	−0.17	0.08	−0.20	.03	.54	.05

[a] $n = 72$. Adjusted $R^2 = .24$, $R^2 = .27$ when the social assertion × medical severity and social support × medical severity interaction terms were added to the model.
[b] $n = 64$. Adjusted $R^2 = .50$, $R^2 = .56$ when the social assertion × medical severity, self-esteem × medical severity, family relations × medical severity and social support × medical severity interaction terms were added to the model.

The results revealed that the R^2 for the trait regression model was significantly different from zero, $F(5, 58) = 13.84$, $p = .0001$. Intrapersonal (social assertiveness and self-esteem), stress-processing (social support), and social-ecological (family relations) factors all contributed to the model. Lower trait anxiety scores were associated with greater social assertiveness skills ($t = -2.77$, $p = .007$), higher self-esteem ($t = -2.78$, $p = .007$)), the use of social support as a coping strategy (t = -2.09, p = .04) and the perception of a more supportive family environment ($t = -2.74$, $p = .008$). These variables, along with the nonsignificant contribution of the medical severity index ($p < .53$), explained 56% (50% adjusted) of the variability in trait anxiety. The addition of the products of each of the four psychosocial factors with the medical severity index altogether did not make a significant contribution to the R^2 ($p = .70$).

Depression. A second set of multiple regression models was fit to predict the overall Beck and the cognitive and somatic subscales of the Beck. These scores are all continuous. A square root transformation was applied to these three continuous Beck scores. The unstandardized regression coefficients (B), the standardized regression coefficients (ß), the squared partial correlations (pr-square), R^2, and adjusted R^2 are provided in Table V. The results revealed that the R^2 for the regression model was significantly different from zero for the overall Beck, $F(3, 68) = 23.65$, $p = .0001$, the cognitive subscale, $F(3, 68) = 22.97$, $p = .0001$, and the somatic subscale, $F(2, 69) = 14.74$, $p = .0001$.

Table V. Predictors of Depression

Variable	*B*	*SE B*	β	Partial R^2	Model R^2	*p*
Beck Depression[a]						
Medical severity index	0.01	0.01	0.06	.00	.00	
Rosenberg, self-esteem	−0.16	0.02	−0.61	.45	.45	.001
Family Relations Index	−0.18	0.06	−0.25	.06	.51	.01
Beck subscale of cognitive depression[b]						
Medical severity index	0.01	0.01	0.05	.00	.00	
Rosenberg, self-esteem	−0.14	0.02	−0.59	.43	.43	.001
Family Relations Index	−0.18	0.06	−0.28	.07	.50	.01
Beck subscale of somatic depression[c]						
Medical severity index	0.01	0.01	0.06	.00	.00	
Rosenberg, self-esteem	−0.09	0.02	−0.55	.30	.30	.001

A square-root transformation has been used for the dependent variables.
[a] $n = 72$. Adjusted $R^2 = .49$. $R^2 = .53$ when the self-esteem × medical severity and family relations × medical severity interaction terms were added to the model.
[b] $n = 72$. Adjusted $R^2 = .48$. $R^2 = .53$ when the self-esteem × medical severity and family relations × medical severity interaction terms were added to the model.
[c] $n = 76$. Adjusted $R^2 = .28$. $R^2 = .30$ when the self-esteem × medical severity interaction term was added to the model.

Intrapersonal (self-esteem) and social-ecological (family relations) factors contributed significantly to prediction of overall Beck scores and scores on the Beck cognitive subscale. The more favorable the self-esteem, the fewer depressive symptoms endorsed on the overall Beck ($t = -6.89$, $p = .0001$) and on the cognitive subscale ($t = -6.56$, $p = .0001$). Similarly, better family relations were associated with fewer depressive

symptoms on the overall Beck (t = -2.78, p = .006) and on the Beck cognitive subscale (t = -3.11, p = .002). These two variables, along with the medical severity index, explained 53% (49% adjusted) of the variability on the overall Beck, and 53% (48% adjusted) of the variability on the cognitive subscale. The medical severity index was held in each model but did not make a significant contribution to predicting scores on either the overall Beck (p = .51) or the cognitive subscale (p = .54). The interaction terms (the medical severity index X family relations; the medical severity index X self-esteem) did not significantly increase the R^2 in either the overall Beck (p = .33) or the cognitive Beck (p = .17) models.

Only an intrapersonal factor, self-esteem, contributed significantly to the prediction of scores of somatic depression (t = -5.42, p = .0001). A more favorable self-esteem was associated with fewer somatic symptoms of depression. Altogether, 30% (28% adjusted) of the variability in symptoms of somatic depression was explained by scores on the self-esteem scale and the medical severity index. The medical severity index was retained but did not contribute significantly to the model (p = .79). The product of medical severity and self-esteem did not significantly improve the R^2 (p = .89).

The findings in the above analyses that treated depressive symptoms as a continuous variable were consistent with findings from a separate logistic regression using categorical scores on the overall Beck as an outcome variable. When the Beck is used as a categorical variable, the following cut-off scores are recommended to assign participants to categories on the Beck Depression Inventory: normal (0-9), mild-moderate depression (10-18), moderate-severe depression (19-29), and extremely severe depression (30+) (Beck & Steer, 1987). However, in this study, there were few with sufficient symptoms to be assigned to either the mild, moderate, or severe category. Therefore, these three categories were collapsed. This created a dichotomous (yes/no) measure of depressive symptoms in which participants were categorized as either normal (0-9) or elevated (10+) on depressive symptoms. The same intrapersonal (self-esteem) and social-ecological (family relations) factors that were significant in the model that used the Beck as a continuous score contributed significantly to the prediction of whether participants were in the depressed (mild/moderate/severe) category. Controlling for medical severity, the odds of being depressed increased as both self-esteem (Wald chi-square = 11.34, p = .0008) and the quality of family relations decreased (Wald chi-square = 6.58, p = .01). The interaction terms (self-esteem X medical severity; family relations X medical severity) did not add to the model (p = .87).

Discussion

Previous studies of the psychological functioning of adolescents with SCD demonstrated considerable variability in adaptation to sickle cell disease during adolescence. Some of that research has documented that psychosocial factors explain substantially more of the variability in adaptability than biomedical factors (Bennett, 1994; Lavigne & Faier-Routman, 1993; Thompson et al., 1993). One objective of this research was to examine whether this pattern was evident in a national sample of adolescents with sickle cell disease.

Two theoretical models, the transactional stress and coping model and the disability-stress-coping model, suggest that psychosocial factors can moderate or mediate the relationship between biomedical risk factors and adaptation. Yet little empirical work is available on the nature of the interrelationships among these three variables (Thompson, 1995). Hence, a second objective of this research was to investigate whether psychosocial factors moderate the relationship between biomedical risk factors and adaptation (psychological functioning).

The findings related to the first objective were consistent with previous findings. Consistent with previous findings (Hurtig et al., 1989; Thompson et al., 1993), the contribution of the biomedical index to the model was minimal in every model. Psychosocial factors accounted for more of the variability than biomedical factors in both depressive symptoms and anxiety in this national sample. Moreover, the findings were consistent with the two theoretical models in that several intrapersonal (social assertion, self-esteem), stress-processing (use of social support), and social-ecological (family relations) factors accounted for a significant amount of the variability in adaptation.

Several patterns in the multiple regression also have implications for the theoretical models. In this particular sample, intrapersonal factors accounted for more of the variability, in general, than either social-ecological or stress-processing factors. Nevertheless, the evidence that one intrapersonal factor (self-esteem) and one stress-processing factor (family relations) were each associated with healthier functioning was particularly strong. These two variables were negatively associated with most indicators of depressive symptoms and trait anxiety. The use of social support as a coping strategy and a socially assertive style were associated with less state and trait anxiety.

We had expected SCD knowledge to be a significant psychosocial predictor. In our sample of adult patients with SCD (Burlew et al., submitted), fewer symptoms of depression were evident as knowledge about sickle cell increased. However, no such relationship was present in this adolescent sample. Perhaps the adolescent's own knowledge about the illness is not as important as a supportive and dependable family environment for adolescents. However, the individual's own knowledge about SCD may become more important as one enters adulthood and assumes more personal responsibility for one's own health care.

The second objective addressed the nature of the relationships among psychosocial factors, biomedical factors, and adaptation. Here, the findings were less definitive. The findings do not suggest that the major importance of psychosocial factors is due to any moderator role that psychosocial factors might serve between biomedical factors and adaptation. This is important information for understanding adaptation to sickle cell. It suggests that, even in milder cases, when the medical condition does not pose a threat to adaptation, lack of psychosocial resources could still place individuals at increased risk for poor adaptation to sickle cell disease.

Despite these findings, however, it would be premature to abandon completely the theory that psychosocial factors moderate the relationship between stress and adaptation. Rather, future research is needed to address some specific issues related to this theory. First, our measure of risk was limited to biomedical factors. However, risk factors in the disability stress-coping model include biomedical factors along with functional independence and other stressors (e.g., major life events, daily hassles). Therefore, additional research is needed to examine the nature of the interrelationships among those alternative risk factors, psychosocial factors, and adaptation. Second, additional psychosocial factors are included in both theoretical models (e.g., temperament, cognitive appraisal, health locus of control) that were beyond the scope of this study. Perhaps other psychosocial variables do indeed play a moderator role. Third, psychological adaptation was conceptualized in this study as the absence of psychopathology. We felt it important to use internalizing factors such as anxiety and depression because much of the previous research on psychosocial predictors has focused on internalizing disorders (Thompson et al., 1999). However, other indicators of personal adjustment (e.g.,. social functioning and externalizing behaviors) might be considered in future research. Finally, both theoretical models also indicate that psychosocial factors could conceivably mediate as well as moderate the relation between risk factors and adaptation. This particular study considered only the moderator role.

The predictors of cognitive (self-esteem and family relations) and somatic (self-esteem) depression were similar but not identical. The same intrapersonal variable (self-esteem) predicted both cognitive and somatic symptoms. However, in addition, a social-ecological variable (family relations) predicted cognitive symptoms. Similarly, the same intrapersonal (social assertion) and stress-processing (use of social support) variables predicted state and trait anxiety. However, an additional intrapersonal variable (self-esteem) and a social-ecological variable (family relations) also predicted trait anxiety. Despite the similarities, the differences suggest that state versus trait anxiety along with cognitive versus somatic depression may be tapping different areas of functioning. Hence, it may be useful to include both types of anxiety and both types of depression in future studies.

The findings have several additional implications for our understanding of adaptation to SCD. First, the findings provide further evidence of the importance of psychosocial factors in predicting adaptation. These findings suggest that a focus on the medical aspects of SCD may be insufficient for promoting adaptation for adolescents and their families. Rather, it may be useful to develop interventions that enhance self-esteem, social assertion skills, and promote the use of social support to cope with the challenges presented by sickle cell disease. In

addition, the findings further support the importance of including activities to strengthen family relationships by increasing family cohesion and expressiveness while decreasing conflict.

The findings also reinforce the need to conduct more studies with experimental designs to validate further the link between psychosocial factors such as family relations or the use of social support and adaptation. For example, such research might utilize experimental designs to examine whether interventions aimed at countering the potential impact of the challenges of sickle cell on the family or support groups that increase the availability and use of social support would enhance adaptation among adolescents with sickle cell.

Several limitations in this study ought to be addressed in future research. First, the findings suggest that biomedical factors play only a very small role in predicting adaptation. We caution against accepting this conclusion unconditionally due to the reality that more work is needed on assessing medical severity among sickle cell patients. Psychological researchers may want to collaborate with medical researchers for much of that work. Second, it also may be important to examine the relationship between the perception of medical severity and adaptation. Third, it was necessary for us to use an abbreviated version of the Family Relations Index in this particular study. Even though evidence was provided to support the use of this abbreviated version, future studies that use full scales of the FES such as the conflict or cohesion scales may address slightly different issues regarding the relationship of family factors to adaptation.

A fourth limitation was the fact that psychological adaptation was conceptualized in this study as the absence of psychopathology. Even though it was important to test for moderator effects using the outcome variables that have been demonstrated to be most important in previous research, future research should expand the focus to include other indicators of adjustment such as externalizing behaviors or school achievement.

The fact that, for practical reasons, all instruments in this study were self-report measures may be an additional limitation. However, the use of self-report measures is common in previous studies of adaptation to chronic illness. Nevertheless, as research progresses in this area, it will be important to include other types of measures (e.g., observational and behavioral measures).

Sample size is a common concern in studies of chronic illness. The sample size of this study may also be a limitation. However, based upon power calculations (Cohen, 1988), our study had 80% power to detect an effect size of 0.16.

In conclusion, the findings from this national sample support previous conclusions that a biopsychosocial approach is more conducive to understanding adaptation to sickle cell disease. In fact, the findings demonstrate that psychosocial factors explain more of the variability in adaptation than medical factors in this national sample. However, the findings did not support the theory that psychosocial factors moderate the relationship between medical severity and adaptation. Rather, the relationship between psychosocial factors and adaptation appears to be unrelated to the relationship between medical factors and adjustment.

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Notes:

[1] The number of adolescents from each Comprehensive Sickle Cell Center appears in parentheses : New York, NY (20) ; Boston, MA (1) ; New Haven, CT (2) ; Washington, DC (11) ; Durham, NC (8) ; Miami, FL (8) ; Chicago, IL (18) ; St Louis, MO (3) ; Memphis, TN (7) ; Augusta, GA (6) ; Oakland, CA (4) ; San Francisco, CA (2) ; total = 90.

References:

Albert, N., & Beck, A. (1975). Incidence of depression in early adolescence: A preliminary study. *Journal of Youth and Adolescence*, *4*, 301-307.

Armstrong, F. D., Lemanek, K. L., Pegelow, C. H., Gonzalez, J. C., & Martinez, A. (1993). Impact of lifestyle disruption on parent and child coping, knowledge, and parental discipline in children with sickle cell anemia. *Children's Health Care*, *22*, 189-203.

Beck, A. T. (1967). *Depression: Causes and treatment*. Philadelphia: University of Pennsylvania Press.

Beck, A. T., & Steer, R. (1987). *Beck Depression Inventory manual*. San Antonio: Psychological Corporation.

Belgrave, F., & Washington, A. (1986). The relationship between locus of control and select personality traits in two chronically ill adolescent groups. *Journal of Rehabilitation*, *Fall*, 37-60.

Bennett, D. S. (1994). Depression among children with chronic medical problems: A meta-analysis. *Journal of Pediatric Psychology*, *19*, 149-169.

Brown, M., & Duran, P. (1988). Construct validity for blacks of the STAI. *Measure and Evaluation in Counseling*, *21*, 25-37.

Brown, R., Kaslow, N., Doepke, K., Buchanan, I., Eckman, J., Baldwin, K., & Goonan, B. (1993). Psychological and family function in children with sickle cell and their mothers. *Journal of American Academy of Child and Adolescent Psychiatry*, *32*, 545-552.

Brown, R. T., Doepke, K. J., & Kaslow, N. J. (1993). Risk-resistance-adaptation model for pediatric chronic illness: Sickle cell syndrome as an example. *Clinical Psychology Review*, *13*, 119-132.

Burlew, A. K., Evans, R., & Oler, C. (1989). The impact of a child with sickle cell disease on family dynamics. *Annals of the New York Academy of Sciences*, *565*, 161-171.

Burlew, A. K., Telfair, J., Wright, L., Barbarin, O., Belgrave, F., & Kaluenja, J. (submitted) *Social and personal factors associated with psychological adjustment among adults with sickle cell disease*.

Charache, S., Lubin, B., & Reid, C. D. (1989). *Management and therapy of sickle cell disease* (NIH Publication No.89-217). Washington, DC: National Institutes of Health.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed., pp. 407-444). Hillsdale, NJ: Lawrence Erlbaum.

Fischoff, J., & Jenkins, D. S. (1987). Sickle cell anemia. In J. Noshpitz (Ed.), *Basic handbook of child psychiatry* (pp. 97-102). New York: Basic Books.

Gil, K., Abrams, M., Phillips, G., & Keefe, F. (1989). Sickle cell disease pain: Relation of coping strategies to adjustment. *Journal of Consulting and Clinical Psychology*, *57*, 359-370.

Gil, K. M., Williams, D. A., Thompson, R. J., & Kinney, T. R. (1991). Sickle cell disease in children and adolescents : The relation of child and parent pain coping strategies to adjustment. *Journal of Pediatric Psychology, 16*, 643-663.

Gil, K. M., Wilson, J. J., & Edens, J. L. (1997). The stability of pain coping strategies in young children, adolescents, and adults with sickle cell disease over an 18-month period. *Clinical Journal of Pain*, *13*, 110-115.

Gray, M. J., Genial, M., & Tambolane, W. V. (1980). Psychosocial adjustment of latency-aged diabetics : Determinants and relationship to control. *Pediatrics*, *65*, 60-73.

Holahan, C. J., & Moos, R. J. (1981). Social support and psychological distress: A longitudinal analysis. *Journal of Abnormal Psychology*, *90*, 365-371.

Hurtig, A. L., Koepke, D., & Park, K. B. (1989). Relation between severity of chronic illness and adjustment in children and adolescents with sickle cell disease. *Journal of Pediatric Psychology*, *14*, 117-132.

Iloeje, S. O. (1991). Psychiatric morbidity among children with sickle cell disease. *Developmental Medicine and Child Neurology*, *11*, 1087-1094.

Kliewer, W., & Lewis, H. (1995). Family influences on coping processes in children and adolescents with sickle cell disease. *Journal of Pediatric Psychology*, *20*, 511-525.

Kumar, S., Powars, D., Allen, J., & Haywood, L. J. (1976). Anxiety, self-concept and person and social adjustments in children with sickle cell anemia. *Journal of Pediatrics*, *88*, 859-863.

Lavigne, J. V., & Faier-Routman, J. (1992). Psychological adjustment to pediatric physical disorders: A meta-analytic review. *Journal of Pediatric Psychology*, *17*, 133-157.

Lavigne, J. V., & Faier-Routman, J. (1993). Correlates of psychological adjustment to pediatric physical disorders: A meta-analytic review and comparison with existing models. *Developmental and Behavioral Pediatrics*, *14*, 117-123.

LePontois, J. (1986). Adolescents with sickle cell anemia: Developmental issues. In A. L. Hurtig & C. T. Viera (Eds.), *Sickle cell disease: Psychological and psychosocial issues* (pp. 75-83). Urbana: University of Illinois Press.

Lewis, A. L., & Kliewer, W. (1996). Hope, coping, and adjustment among children with sickle cell disease: Tests of mediator and moderator models. *Journal of Pediatric Psychology*, *21*, 25-41.

McArnarney, E. (1985). Social maturation: A challenge for handicapped and chronically ill adolescents. *Adolescent Health Care*, *6*, 90-101.

McCubbin, H., McCubbin, M., Patterson, J., Cauble, A. E., Wilson, L., & Warwick, W. (1983). CHIP—Coping Health Inventory for Parents: An assessment of parental coping patterns in the care of the chronically ill child. *Journal of Marriage and the Family*, *4*, 359-370.

Molock, S., & Belgrave, F. (1994). Depression and anxiety in patients with sickle cell disease. *Journal of Health and Social Policy*, *5*, 39-53.

Moos, R. H., & Moos, B. S. (1986). *Family Environment Scale manual*. Palo Alto, CA: Consulting Psychologist Press.

Morgan, S. A., & Jackson, J. (1986). Psychological and social concomitants of sickle cell anemia in adolescents. *Journal of Pediatric Psychology*, *11*, 429-440.

Nash, K. B., & Telfair, J. (1994). Sickle cell disease: A biopsychosocial model. In I. L. Livingston (Ed.), *Handbook of black American health: The mosaic of conditions, issues, policies, and prospects* (pp. 123-139). Westport, CT: Greenwood Publishing.

Noll, R., Bukowski, W., Davies, W. H., Koontz, K., & Ris, D. (1992). Social interactions between children with cancer or sickle cell disease and their peers: *Teacher Rating Developmental and Behavioral Pediatrics*, *13*, 187-193.

Rathus, S. A. (1973). A 30 item schedule for assessing assertive behavior. *Behavior Therapy*, *4*, 398-406.

Rosenberg, M. (1965). *Society and the adolescent self image*. Princeton, NJ: Princeton University Press.

SAS Institute, Inc. (1988). *SAS/STAT user's guide*, Release 6.03 edition. Cary, NC: SAS Institute, Inc.

Schoenherr, S., Brown, R., Baldwin, K., & Kaslow, N. (1992). Attributional styles and psychopathology in pediatric chronic illness groups. *Journal of Clinical Child Psychology*, *21*, 380-387.

Siegel, W., Golden, N., Gough, J., Lashley, M., & Secker, I. (1990). Depression, self esteem, and life events in adolescents with chronic diseases. *Journal of Adolescent Health Care*, *11*, 501-504.

Speilberger, C. (1973). *STAIC preliminary manual*. Palo Alto, CA: Consulting Psychologist Press.

Speilberger, C. D., Gorsuch, R. L., & Lushene, R. G. (1970). *The State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.

Teri, L. (1982). The use of the Beck Depression Inventory with adolescents. *Journal of Abnormal Child Psychology*, *10*, 277-284.

Thompson, R. (1995). Commentary: Sickle cell disease. *Journal of Pediatric Psychology, 20*, 403-406.

Thompson, R., Armstrong, F. D., Kronenerger, W., Scott, D., McCabe, M., Smith, B., Radcliffe, J., Colangelo, L., Gallagher, D., & Wright, E. (1999). Family functioning, neurocognitive functioning, and behavior problems in children with sickle cell disease. *Journal of Pediatric Psychology, 24*, 491-498.

Thompson, R. J., Jr., Gil, K. M., Burbach, D. J., Keith, B. R., & Kinney, T. R. (1993). Role of child and maternal processes in the psychological adjustment of children with sickle cell disease. *Journal of Consulting and Clinical Psychology*, *61*, 468-474.

Thompson, R. J., Jr., Gil, K. M., Keith, B. R., Gustafson, K. E., George, L. K., & Kinney, T. R. (1994). Psychological adjustment of children with sickle cell disease: Stability and change over a 10-month period. *Journal of Consulting and Clinical Psychology*, *62*, 856-860.

Thompson, R. J., Jr., & Gustafson, K. E. (1996). *Adaptation to chronic childhood illness*. Washington, DC: American Psychological Association.

Thompson, R. J., Jr., Gustafson, K. E., George, L. K., & Spock, A. (1994). Change over a 12-month period in the psychological adjustment of children and adolescents with cystic fibrosis. *Journal of Pediatric Psychology, 19*, 189-203.

Thompson, R. J., Jr., Gustafson, K. E., Hamlett, K. W., & Spock, A. (1992). Psychological adjustment of children with cystic fibrosis: The role of child cognitive processes and maternal adjustment. *Journal of Pediatric Psychology*, *17*, 741-755.

Treiber, F., Mabe, A., & Wilson, G. (1987). Psychological adjustment of sickle cell children and their siblings. *Children's Health Care, 16*, 82-88.

Wallander, J. L., & Varni, J. W. (1989). Social support and adjustment in chronically ill and handicapped children. *American Journal of Community Psychology, 17*, 185-201.

Wallander, J. L., & Varni, J. W. (1992). Adjustment in children with chronic physical disorders. Programmatic Research on a disability-stress-coping model. In A. M. LaGreca, L. Siegel, J. Wallander, & C. Walker (Eds.), *Stress and coping with pediatric conditions* (pp. 279-298). New York: Guilford Press.

Wallander, J. L., Varni, J., Babani, L. Banis, H. T., Varni, J. W., & Wilcox, K. T. (1989). Family resources as resistance factors for psychological maladjustment in chronically ill and handicapped children. *Journal of Pediatric Psychology*, *14*, 157-173.