

Multidisciplinary Approach to Child and Adolescent Depression

GUEST EDITORS: BETTINA F. PIKO, ROBERT MILIN, RORY O'CONNOR, AND MICHAEL SAWYER





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Depression Research and Treatment

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Guest Editors: Bettina F. Piko, Robert Milin, Rory O'Connor, and Michael Sawyer



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Editorial

Multidisciplinary Approach to Child and Adolescent Depression

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In collaboration with a qualified international scientific team, we are pleased to launch this special issue which contains papers from epidemiological aspects to clinical implications with special emphasis put on psychological models and theories.

The special issue on child and adolescent depression is also an arena for multidisciplinary and covers a wide range of viewpoints and contexts. Hopefully, with this volume, we provide a platform for a need of subsequent communication between basic sciences and clinical experiences as the most significant challenge of the 21st medical research. This is particularly true in case of depression research where integration of basic biological sciences with possible application areas is fundamental.

Depression contributes to morbidity and mortality across the life course; therefore it is particularly important to detect influences of depressive symptoms in childhood since these may have longer-term adverse effects on the psychosocial adjustment of adults [1]. The frequency of depressive symptoms increases markedly in adolescence [2]. Both biological-hormonal changes and environmental factors are likely to contribute to this increase [3]. As research results suggest, early adolescence is a time when the prevalence of depressive symptoms increases markedly, particularly among females [4, 5]. This is due to intensified gender socialization in this age period [6].

Not surprisingly, adolescent depression has become a focus in current scientific research. However, there is

evidence that depressive symptoms tend to begin before puberty. As a result, more attention should be paid to depression in children and the preadolescent period as well. Unfortunately, we know much less about the contextual factors of child and adolescent depression. We need more research into different aspects of this phenomenon including a multidisciplinary/biopsychosocial approach. Critical evaluations and review articles are particularly useful for integrating empirical research results. Early detections and interventions of mood disorders are perhaps a greatest challenge for preventive psychiatry and mental health promotion. As a consequence, we have lots of undiscovered fields in child and adolescent psychiatry and psychology related to child and adolescent depression. Apart from this editorial, a total of 11 articles are offered to the readers in this issue; most of them are theoretically based research papers with two reviews and studies on psychological models, comorbid states, and prevention issues.

Longitudinal studies are particularly relevant for investigating the role of psychological models which may help us understand underlying mechanisms of how adolescent depression may develop. In a longitudinal study, Z. Gutkovich and his colleagues investigated the role of anhedonia and pessimistic attributional style in a sample of hospitalized depressed adolescents. Anhedonia (the diminished capacity to experience pleasure) and pessimistic attributional style (the manner in which a person explains the cause of an event in a negative way) are of special interest in relation

to major depression. As the authors found, anhedonia is a critical characteristic of adolescent depression, one that could be intimately involved in the pathogenic mechanism of the depressive episode through association with pessimistic attributional style.

In the next paper, published by A. H. Mezulis et al., prospective associations between negative emotionality, rumination, and depressive symptoms was examined in a community sample of youth followed longitudinally from birth to adolescence. The authors applied a cognitive model of depression where ruminative response style represented a cognitive vulnerability. Results found that greater negative emotionality in infancy was associated with more depressive symptoms at age 15, and rumination significantly mediated the association between them for girls but not for boys.

The third longitudinal study, carried out by Y. M. Sanchez and coworkers, aimed to investigate association between adverse life events and depressive symptoms in African American youth. In addition, control-related beliefs were added as a mechanism linking life event stress and depression which had received little attention before. Findings suggest that control-related beliefs mediated the association between adverse life events and depressive symptoms, and a significant adverse life events → control-related beliefs → depressive symptoms indirect effect. In addition, violent life events, rather than nonviolent events, also may have predicted depressive symptoms.

Next, in this special issue, an exciting review focuses on magnetic resonance spectroscopy. G. Kondo and his team provided a thorough and critical overview of studies in this field. They concluded that, because it offers a noninvasive and repeatable measurement of relevant *in vivo* brain chemistry, MRS has the potential to provide insights into the pathophysiology of adolescent major depression MDD, as well as the mediators and moderators of treatment response.

As it has already been noted here, preadolescent child depression is getting more and more attention. In an exciting paper of M McCabe et al., the focus has been laid to this age group and the psychosocial functioning in relation to depression. The authors concluded that it was harder for the social network of the children to detect the problems of children with elevated depressive symptoms, who did not meet the diagnostic criteria. Laypeople usually differentiated between the “clinical” and “normal” groups. The authors recommended that it might be important to implement intervention programs including “at-risk” children as well.

The next papers deal with comorbidity. L.-G. Lundh and colleagues provided longitudinal research data on depressive symptoms and deliberate self-harm in a community sample of adolescents. There was support for a bidirectional relationship between depressive symptoms and self-harm in the girls; whereas in boys, there was only support for a unidirectional relationship, depressive symptoms being a predictor of increased self-harm one year later.

In terms of comorbidity, substance use disorders occupy a special place. The next three papers, not surprisingly, deal with this type of comorbidity. H. O. Anderson and A. M. Libby reported on a study on depression with and without

comorbid substance dependence among young adults. As it was concluded, frequent use of substances significantly increased the likelihood of subsequent depression with comorbid substance dependence compared to depression alone.

In the next study carried out by T. Pirkola and coworkers, the authors aimed at examining the differences between depressed psychiatric adolescent outpatients with and without cooccurring alcohol misuse in psychosocial background, clinical characteristics, and treatment received during one-year followup. Alcohol misuse well indicated family problems and had a deleterious effect on treatment attendance.

Alcohol misuse is a serious problem amongst adolescents in many countries. It is a particularly serious problem amongst indigenous communities. The study of S. H. Stewart and colleagues examined the relationship between hopelessness, depression and excessive drinking in a Canadian population of adolescents, the majority of whom were aboriginal youth. The results from the study showed a strong relationship between hopelessness and depressive symptoms. They also showed a strong relationship between depressive symptoms and “drinking to cope”. In this context, it appeared that excessive alcohol intake was being used to both reduce unpleasant feelings of depression and block pessimistic thoughts commonly associated with depression. The results from the study suggest that focusing on hopelessness, depressive symptoms and drinking to cope may be important for programs trying to reduce alcohol misuse among aboriginal youth.

Finally, the last two papers deal with prevention issues. There is limited evidence that prevention programs for depression are effective among adolescents [7]. However, given current knowledge about adolescent help seeking, it is important that prevention and early intervention programs include elements that aim at improving the quality of the social support offered by peers and family members to adolescents experiencing depressive symptoms. Teachers, family members, and peers should also be encouraged to actively engage with young adolescents experiencing depressive symptoms rather than waiting for them to initiate help seeking. This is particularly important for adolescents experiencing higher levels of depressive symptoms who may not initiate help seeking themselves. A promising approach in this area is the use of mental health first-aid programs being developed in Australia [8]. Mental health first-aid programs focus on key steps which can be taken by teachers, family members, and peers to support adolescents with depression while guiding them to professional sources of help. The first paper that is a review carried out by F. Rice and A. Rawal focused on basic research evidence from community, clinical, and high-risk populations that identified cognitive mechanisms and emotional regulation as key processes involved in the onset and maintenance of depression. Finally, C. A. McCarty et al. reported on a novel intervention design. Despite the preliminary nature of the intervention, applying positive thoughts in school-based prevention programs seemed promising.

We hope that our readers will enjoy this special issue.

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Research Article

Anhedonia and Pessimism in Hospitalized Depressed Adolescents

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This longitudinal study investigates whether anhedonia and pessimistic attributional style represent a clinical state or a trait in hospitalized depressed adolescents. 81 consecutive adolescent inpatients were screened with the Beck Depression Inventory (BDI) and the clinician-rated Major Depressive Disorder (MDD) criteria sheet. 51 patients with BDI score ≥ 10 and/or ≥ 4 symptoms on MDD criteria sheet were assessed at Time 1 upon admission, with 39 patients (78%) assessed at discharge (Time 2) with the Pleasure Scale for Children and Children's Attributional Style Questionnaire—Revised. Anhedonia and pessimism at admission were associated with BDI scores at admission and discharge as well as number of depressive symptoms and depression severity. MDD diagnosis was associated with anhedonia, but not with pessimism. Pessimism—but not anhedonia—improved significantly by discharge. Results suggest that while some adolescents exhibit enduring anhedonia, pessimistic attributional style appears to be a concomitant feature of an acute depressive state.

1. Introduction

Anhedonia, the diminished capacity to experience pleasure, is of special interest in relation to major depression because diminished pleasure is a core manifestation of the disorder [1] and is associated with higher suicidal risk [2]. Anhedonia in adults is correlated with self-reported depressive measures and is associated with the diagnosis of depression but not other diagnoses [3]. Research with adults [3] has been consistent with Klein's hypothesis [4] about the existence of a qualitatively distinct subtype of major depression that has been variously defined as "truly anhedonic", or "endogenomorphic." It has been shown that adult patients with this subtype of depression are more severely depressed than depressed patients with a more normal pleasure capacity [5]. Recovered adult anhedonic depressed patients still had a lower pleasure score upon recovery than normally hedonic patients [5]. It is possible that some patients have a characterological anhedonic trait that predisposes them to endogenomorphic depressive states [5, 6]. In contrast to the

patients with "endogenomorphic" depression, patients with situational depression have preserved hedonic capacity [7]. Yet recent studies showed that acute stress reduces reward responsiveness and had been linked to anhedonic behaviors thus raising the possibility of "situational" anhedonia [8].

Available research with children has demonstrated that anhedonia is consistently associated with the diagnosis of Major Depressive Disorder [9]. However, in contrast to adult studies, research with children has failed to find a correlation between self-report depression measures and anhedonia [9] or self-reported enjoyment and interest, possibly related to anhedonia [10]. Interpretation of these data remains unclear and can be related to the characteristics of the samples or developmental differences [9]. In particular, prepubertal children have lower capacity than adolescents or adults to report on their depressive symptoms [11].

Very little is known about anhedonia in depressed adolescents. In a study of nonreferred adolescents Carey et al. [12] did not find an association between self-reported depression and frequency of engaging in pleasant activities. On the

other hand, it has been demonstrated that clinical picture of adolescent depression is more similar to adult depression, in particular, adolescents experience greater anhedonia compared to children [11, 13]. The concept concerning the existence of two qualitatively distinct subtypes of depression among adults (i.e. “exogenomorphic” or “situational” versus “endogenomorphic” or “truly anhedonic” depression corresponds to the notion of Garland and Weiss [14] who proposed that there may be two subgroups of adolescent depression: mild, situational, placebo-responsive depressions; and severe, genetically loaded, treatment-resistant depressions.

Dysfunctional or pessimistic attributional style is closely related to such concepts as learned helplessness and external locus of control and, along with the anhedonia, has been accorded a pivotal role in conceptualizations of Depressive Disorder [15]. Attributional style is the manner in which a person explains the cause of negative and positive events. This concept has its roots in the learned helplessness model of depression and Beck’s cognitive theory of depression [16]. Attributing the cause of uncontrollable bad events to internal, stable and global factors and to a lesser degree, the opposite style for attributing good events to external, unstable and specific factors, is a maladaptive or pessimistic attributional style.

Pessimistic attributional style has been shown to be associated with depression in all age groups, and particularly in adolescents, for example, in a cross-sectional study by Gotlib et al. [17]. In our previous work [18] we have discussed the association between dysfunctional attributional style, sense of subjective incompetence and demoralization [19]. Our present study attempts to add to the literature by examining stability of pessimistic attributional style over the course of intensive inpatient treatment.

Recently published, by the American Academy of Child and Adolescent Psychiatry, Practice Parameter for the assessment and treatment of children and adolescents with depressive disorders [20] presented the prevailing view on the role of attributional style in childhood depression: “negative attributional style (is)... associated with poor outcome [21, 22].” “The onset and recurrences of Major Depression may be moderated by the presence of stressors. However, the effects of these stressors also depend on the child attributional styles.” The very term “attributional style” implies that it is a permanent characteristic.

However, the relationship between dysfunctional attributional style and Depressive Disorder appears to be complex and there is controversy among researchers. Abramson et al. [15] postulated that maladaptive attributional style is “depressogenic.” Lau and Eley [23] consider attributional style as a risk marker of genetic effects for adolescent depressive symptoms. Lewinsohn and colleagues [24] postulated that a bout of depression will lead to the development of a stable pessimistic explanatory style, a possibility that they have labeled the “scar hypothesis.” Nolen-Hoeksema [25] demonstrated in a longitudinal study with children in a nonclinical sample that after the first depressive episode attributional style became more dysfunctional than prior to the onset of depression, providing support for a scar hypothesis. There are several ways in which pessimistic style

could develop during a depressive episode. The deficits in school performance and peer interactions that children often show when depressed could convince a child that he or she has low abilities, is unlikable, and generally is not able to control important outcomes in life [22].

Other research contradicts the scar hypothesis. In particular Asarnow and Bates [26] found in the sample of child psychiatric inpatients in the cross-sectional study that children with remitted depression have an explanatory style that was as optimistic as normal controls. In summary, while there is no doubt that pessimistic explanatory style is associated with depression, there remains a controversy in studies concerning the nature of the relationship between pessimistic style and depression, and whether pessimistic attributional style is a state marker of depression or a trait-like characteristic. We did not find studies addressing the “scar hypothesis” in adolescents with the treated depressive episode, that is, whether a depressive episode in treated adolescents leaves a “scar” such as a stable pessimistic explanatory style.

To date, there have been no longitudinal studies on adolescent anhedonia. The longitudinal studies on adolescent attributional style are very scarce [27, 28] and concerned with nonreferred, nontreated adolescents. These studies provided controversial results. In the study of Stevens and Prinstein [27], attributional style measured at Time 2 in 11 months since the outset of the study appeared to be a stable characteristic. Schwartz et al. [28] who measured attributional style in one year since the outset of the study found that “youth experienced significant changes in their attributional styles over time (from adaptive to maladaptive and vice versa).”

To our knowledge there have been no studies examining attributional style and anhedonia pre- and post-treatment in depressed adolescents. Further investigation of anhedonia and attributional style in adolescent depression can be of theoretical and practical importance. It is important to understand whether anhedonia relates to a subtype of depression among adolescents, and whether anhedonia and attributional style represent a clinical state or a trait. This understanding can be important for the prognosis at an acute phase, and for prognosis and treatment planning after the resolution of acute depressive symptoms.

The objective of the current study was to examine the relationship between self-reported depressive scores, clinical diagnosis of major depression, subsyndromal depression, anhedonia and attributional style in a sample of adolescent inpatients with depressive symptomatology at admission and at discharge.

Based on clinical impressions we hypothesized that many cases of depression in hospitalized adolescents are situational depressions and are phenomenologically similar to demoralization. We expected that pessimistic attributional style as well as anhedonia would be associated with the severity of self-reported depression and with the diagnosis of depression upon admission. The latter was based on a premise that demoralized patients may have an acute anhedonic state in the midst of a depressive episode. We hypothesized that both

anhedonia and pessimistic attributional style would improve upon discharge.

2. Methods

2.1. Sample and Procedure. Eighty-one consecutive adolescent inpatients were screened for depressive symptomatology upon their hospital admission to the inpatient acute care adolescent psychiatric ward, during a five month period. Patients were administered the Beck Depression Inventory (BDI) [29] within three days of admission and their treating clinicians completed a Major Depressive Disorder (MDD) criteria sheet. The MDD criteria sheet is a checklist of the symptoms according to DSM-IV criteria for major depression, which includes a depression severity rating and duration criteria. MDD criteria sheet allows to document depressive symptoms elicited by clinician according to DSM-IV criteria for a Major Depressive Disorder. It had been used previously for the clinical and research purposes in the institution where study had been carried out (unpublished data). Patients who completed questionnaires and did not have mental retardation or neurological impairment were included. Four patients (5%) refused to participate, 4 mentally retarded patients were excluded and one patient was unable to participate because of an acute catatonic state at time of admission. None of the four patients who refused to participate was judged by their clinician as being depressed. The remaining 71 patients completed the BDI and their treating clinicians completed the MDD criteria sheet. Fifty-one of these 71 patients met the inclusion criteria of having a BDI score of 10 or above and/or having at least four symptoms on the MDD criteria sheet (three symptoms in addition to essential symptoms of depression or anhedonia) and were enrolled into the study. The decision to include patients with near threshold MDD was made based on the fact that subsyndromal depression causes significant functional impairment in adults [30] and adolescents [31]. It also provided an opportunity to compare patients who met full DSM-IV criteria for MDD with the patients who had only subsyndromal depression. Those patients who were enrolled were administered the Pleasure Scale for Children (PLS) [9] and the Children's Attributional Style Questionnaire—Revised (CASQ-R) [32] at admission (Time 1) and at the discharge (Time 2), and repeated BDI at the time of the discharge. The questionnaires were completed in one session in the presence of one of the investigators (Z.G. or R.E.). Some patients had very long hospital stay due to disposition problems. To increase comparability of the data patients whose discharge was delayed after remission of their symptoms due to placement problems were tested a second time one month after their admission. Clinicians were blind to the scores on the self-rated scales at the time of the completion of the MDD sheet. Final diagnosis was established based on DSM-IV criteria by the treatment team led by one of the investigators (R.D.), who were blind to the self-rated scales. Data were collected via chart review on subject's final psychiatric diagnosis, age, gender, ethnicity, and family composition. Major stressors were assessed based on parent, patient and clinician report. Thirty-nine patients

TABLE 1: Description of the sample ($N = 51$).

Mean age, y (SD)	14.9 (1.59)
Male, n (%)	14 (27.5)
Ethnicity, n (%)	
Caucasian	35 (69)
African-American	11 (22)
Hispanic	3 (6)
Asian	1 (2)
Indian	1 (2)
Family status, n (%)	
Living with both biological parents	16 (31)
Living with a single biological parent	20 (39)
Living in stepfamilies	8 (16)
Living in a foster family	6 (12)
Living in a group home	1 (2)
Mean length of stay, days (SD)	18.22 (25.45)
Primary Diagnosis, n (comorbid with depressive Disorder)	
MDD	27
Other depressive disorder: depressive disorder NOS or adjustment disorder with depressed mood	9
Bipolar disorder	11 (0)
ADHD	6 (2)
ODD	10 (8)
Anxiety disorder	10 (7)
Eating disorder	3 (2)
Dysthymia	4 (0)
Substance abuse	7 (4)
Psychosis	11 (6)

(78%) had testing with the BDI, CASQ-R and PLS at the time of their discharge (Time 2). Patients who were tested at time 1 only ($N = 12$) had very short hospital stays and most of them were discharged precipitously against medical advice. Patients who have been tested at Time 1 and Time 2 had at least a one week interval between testing. Patients who had testing at Time 1 and at Time 2 did not differ on any sociodemographic variables from patients who had testing only at Time 1. Patients who were tested only at Time 1 tended to have less depressive symptoms and lower depression severity. 11 consecutive inpatients who did not have depressive symptomatology were tested upon admission only, with the BDI, CASQ-R and PLS and their clinicians completed MDD criteria sheet. These subjects constituted a comparison group and were included into some analyses.

The study sample characteristics are described in the Table 1. 29 out of 36 patients (81%) with any acute Depressive Disorder (MDD being subsumed under Depressive Disorder group, Dysthymia being excluded) had comorbid diagnoses. Out of 27 patients with MDD 13 patients had comorbid diagnoses (48%). All fifteen patients who had concerning depressive symptomatology ($BDI \geq 10$) but less

TABLE 2: Correlation coefficient between measures.

Scores on independent measures, Time 1	BDI, Time 1	BDI, Time 2	Number of depressive symptoms	Depression severity
Pleasure Scale	-.40**	-.33*	-.38**	-.32*
CASQ-R, Total	-.59***	-.48***	-.32*	-.27*
CASQ-R	.55**	.45**	.22	.21
CO-NEG				
CASQ-R				
CO-POS	-.52**	-.42**	-.35**	-.27*
Number of stressors in last 6 months	-.08	.02	.02	.20
Number of remote stressors	-.03	.16	.20	.39**

* $P < .05$; ** $P < .01$; *** $P < .001$.

than four symptoms on MDD criteria sheet had primary diagnoses other than Depressive Disorder. The study was approved by Long Island Jewish Medical Center Institutional Review Board. All subjects provided assent and parents or legal guardians provided consent.

2.2. Instruments. We used Beck Depression Inventory to assess self-reported depressive symptomatology. BDI has been shown to be a reliable measure of self-reported depression in adolescents [33]. We chose the conventional cut-off score of 10 [33] to identify the presence of clinically concerning depressive symptoms. The primary measure for anhedonia was the Pleasure Scale for Children (PLS), the 39-item measure which was developed by Kazdin [9]. This questionnaire is a list of events and activities that children would normally find rewarding. The items reflect three categories similar to anhedonia scales for adults [3, 34]: physical anhedonia or activities involving physical pleasures (e.g. eating a favorite meal, lying in bed etc.); social anhedonia or activities involving other persons (e.g. talking to friend, being told how great one looks) and other activities that do not fall within the above (e.g. related to interest and achievement). The child or adolescent is asked to decide whether the activity would make him or her feel “very happy”, “happy” or “would not matter.” We made slight modifications of the scale to make language more appropriate to use with adolescents, for example, “special present” instead of “new toy” or “someone asks you to hang out with them” instead of “play”. The primary measure for attributional style was Children’s Attributional Style Questionnaire—Revised which is a shortened 24-item version [32] of the original 48-item questionnaire [35]. The CASQ, also referred as referred to as the KASTAN-CASQ [35], is the primary measure of attributional style for youth ages 8 to 18. Psychometric properties of the CASQ-R have been investigated by Thompson et al. [36] who concluded that it is a psychometrically adequate measure. The scale uses a forced choice format to assess causal attributions.

Each item presents a hypothetical situation followed by two statements regarding why the event happened. Adolescents choose the response that best explains why they believe that event might have occurred. Three dimensions of attributions (i.e. internal-external, global-specific, stable-unstable) are assessed. An equal number of items assessed each dimension for both good and bad outcomes. Composite attributional style can be calculated based on all of the items for negative events (“composite negative”) and all of the items for positive events (“composite positive”). A sample item that measures internality while holding stability and globality would be, for example, “A good friend tells you that he hates you.” Choices are (a) my friend was in a bad mood that day (external) (b) I was not nice to my friend that day (internal). Attributing negative events to internal, stable, and global factors and the opposite style for attributing positive events is associated with lower CASQ scores, reflective of less adaptive or more depressogenic attributions.

2.3. Data Analysis. Pearson correlations with one-tailed tests were computed for continuous variables. Independent samples T -tests were performed to assess group differences. Paired Sample T -tests were used to assess change over time on dependent variables. Partial correlation between Pleasure Capacity scores and CASQ scores while controlling for BDI was conducted. A separate analysis was performed for the patients with the most significant clinical improvement defined as a 50% decrease from the initial BDI score.

3. Results

Pleasure scale scores and CASQ-R scores showed significant inverse correlation with the BDI scores at Time 1 and Time 2, number of depressive symptoms and depression severity as rated by clinician (see Table 2). Independent sample T -tests demonstrated a significant difference in Pleasure scale scores between patients with the diagnosis of the Major Depressive Disorder ($N = 27$) ($\bar{x} = 70.63 \pm 16.02$) and

TABLE 3: Correlation between attributional style and pleasure capacity.

	Patients with depressive disorder (<i>N</i> = 36 for Time 1; <i>N</i> = 28 for Time 2)		Patients without depressive disorder (<i>N</i> = 15 for Time 1; <i>N</i> = 11 for Time 2)	
	PLS, Time 1	PLS, Time 2	PLS, Time 1	PLS, Time 2
CASQ, Time 1	.69***	.51**	.28	-.11
CASQ, Time 2	.50**	.47**	.47	.00

P* < .01; *P* < .001.

TABLE 4: Difference over time.

	Normative data (scale range)	\bar{x} (SD) Time 1	\bar{x} (SD) Time 2	<i>t</i>	<i>P</i>
BDI	< 10 (0–63)	20.92 ± 13.16	11.15 ± 9.56	5.56	< .001
PLS	No normative data (39–117)	75.37 ± 5.30	78.16 ± 14.29	–1.76	< .05
CASQ-R	4.87 ± 3.39 (–12 to +12)	1.69 ± 5.29	3.36 ± 5.01	–2.83	< .01
CASQ-R CO-NEG	≈2.7 ± 1.9 (0 to –12)	4.55±2.78	3.79 ± 2.76	2.04	< .05
CASQ-R CO-POS	≈7.6 ± 2.2 (0 to + 12)	6.24±3.11	7.15 ± 3.06	–2.52	< .01

those without such diagnosis (*N* = 24) (\bar{x} = 80.71 ± 12.75) (*t* = 2.46, *df* = 49, *P* < .01). Pessimistic attributional style was not associated with the diagnosis of Major Depressive Disorder.

CASQ-R scores were strongly associated with Pleasure scale scores (*r* = .69, *P* < .001) in the adolescents with a diagnosis of Depressive Disorder even when controlling for the BDI score; there was no association between those variables for patients without the diagnosis of Depressive Disorder (see Table 3).

Differences in Pleasure Scale scores and CASQ-R scores at Time 1 and Time 2 were tested via Paired Sample *T*-tests, which indicated significant changes in CASQ-R scores and Pleasure Scale scores over time (see Table 4). Table 4 also summarizes attributions about bad events and good events at Time 1 and Time 2.

A separate analysis performed for the patients with the most significant clinical improvement defined as a 50% decrease from the initial BDI score (*N* = 19) indicated even more significant increases in CASQ-R scores at the time of discharge approaching levels for normal epidemiological controls [35, Kaslaw, personal communication] (\bar{x} = 1.05 ± 5.65 at Time 1 versus \bar{x} = 4.26 ± 4.66 at time 2; \bar{x} = 4.87 ± 3.39 for normal epidemiological control. There was no difference in the change of the Pleasure Capacity over time among significantly improved patients and the rest of the study sample.

Independent samples *T*-tests demonstrated significant differences on Pleasure scale scores and CASQ-R scores between the patients with depressive symptomatology (study sample, *N* = 51) and patients without depressive symptomatology (comparison group, *N* = 11) (see Table 5). Adolescents in the comparison group had higher mean

scores on a Pleasure Scale and differed significantly from the depressed adolescents. Patients in the comparison group had significantly more optimistic attributional style than patients in the study sample. The difference in attributional style was across both dimensions, attributions for positive and for negative events.

Scores on self-reported and clinician-rated measures did not correlate with sociodemographic variables except for a modest correlation between remote stressors and depression severity (*r* = .39, *P* < .01).

There was no significant difference on Pleasure Scale scores and CASQ-R scores between adolescents with “pure” MDD (*N* = 14) and those with MDD and comorbid disorders (*N* = 13).

4. Discussion

At admission, anhedonia and pessimistic attributional style were associated with self-reported depression, a number of depressive symptoms and depression severity. Anhedonia, but not pessimistic style, was associated with the diagnosis of Major Depressive Disorder. One possible explanation might be that while pessimistic style is associated with an overall level of psychological distress, it is not a core feature of MDD in adolescents. The finding that anhedonia is associated with the diagnosis of depressive disorder is consistent with previous research with adults [1] and children [9]. Our results contradict findings that anhedonia was not associated with self-reported depression in children [9] and with involvement in pleasurable activities by adolescents [12]. However, this finding is consistent with studies with adults that reported association between anhedonia and self-reported depression and depression severity [3]. Our

TABLE 5: Group comparison.

	Non-depressed controls (scale range)	\bar{x} (SD) Study sample ($N = 51$)	\bar{X} (SD) Non-depressed inpatients ($N = 11$)	t	P
BDI	≤ 10 (0–63)	20.92 ± 13.16	3.27 ± 3.23	-8.47	$< .001$
PLS	No normative data (39–117)	75.37 ± 15.30	84.82 ± 15.85	1.85	$< .05$
CASQ-R	4.87 ± 3.39 (–12 to +12)	1.69 ± 5.29	6.36 ± 2.69	-2.83	$< .001$
CASQ-R CO-NEG	$\approx 2.7 \pm 1.9$ (0–12)	4.55 ± 2.78	2.6 ± 1.8	-2.18	$< .05$
CASQ-R CO-POS	$\approx 7.6 \pm 2.2$ (0–12)	6.24 ± 3.11	9.0 ± 1.7	2.84	$< .01$

results are intuitive and consistent with the notion that compared to depression in children, the clinical picture of adolescent depression is more similar to adult depression and is consistent with previous reports that adolescents experience greater anhedonia compared to children [11, 13]. We also believe that these results may have to do with the characteristics of our sample, that is, sample of seriously depressed hospitalized adolescents compared to non-hospitalized patients in the study of Carey et al. [12], and with the better ability of adolescents to report depressive symptoms.

These findings have important clinical ramifications. Several studies for example, Fawcett et al. [2] have shown that severe anhedonia is associated with suicidal risk and may respond to pharmacotherapy and therapeutic interventions such as cognitive therapy. Klein [4] suggested that “truly anhedonic” depressions show better response to treatment with antidepressants than do other types of depressions. The results of our study suggest potential usefulness of assessing anhedonia as related to hospitalization and discharge criteria.

There was a significant difference in anhedonia and attributional style between inpatients with and without depressive symptomatology. For attributional style, this difference was found across both dimensions, that is, attributions for positive and for negative events. Adolescents in the comparison group were much less anhedonic and more optimistic than patients in the study sample. These findings support further the association between both anhedonia and pessimistic style with depressive symptomatology. We do not have ready explanation for the fact that non-depressed inpatients were more optimistic than remitted depressed inpatients and had higher CASQ-R scores than normal epidemiological controls for children. It could be due to our small sample size or that remitted depressed adolescents may still have residual pessimistic traits. Also, one may speculate that non-depressed adolescent inpatients might have an abnormally optimistic style. More extensive research of non-depressed adolescent inpatients is needed and would clarify this issue.

It is intriguing that anhedonia and pessimism were strongly associated in adolescents with a diagnosis of any Depressive Disorder, even when controlling for BDI score.

There was no association between those variables for patients without the diagnosis of Depressive Disorder. Nurcombe et al. [37] question if Depressive Disorder in adolescence is a distinct categorical entity—making an analogy to patients in a general hospital that have fever, but who no physician identifies as having “Major Fever Disorder.” Our results are consistent with the findings of Kazdin [9] who demonstrated in a cross-sectional study that children high in anhedonia showed a dysfunctional attributional style. Those children showed less active involvement in seeking rewarding events, lower expectations for positive outcomes and more attributions for these outcomes. A strong association between anhedonia and helplessness in adolescents with the diagnosis of Depressive Disorder might suggest the possibility of a causal and bidirectional relationship between them and their involvement into pathogenic mechanism of depression. There is a striking similarity between the learned helplessness animal model and the more recently developed anhedonia model. In both models, animals are exposed to stressful stimuli, which are noxious stimuli in the learned helplessness model and chronic unpredictable stress in the anhedonia model [38]. This similarity raises questions that await future research, in particular if there may be a common pathway linking stressful stimuli to helplessness and anhedonia.

Even though the study was not designed to formally validate the Pleasure Scale for use in adolescents, it provides some evidence of its validity. We have compared our data with the results of Kazdin [9]. The means are quite similar for non-depressed inpatient adolescents in our study and non-depressed inpatient children in the Kazdin sample ($\bar{x} = 84.8$ versus 84.6 , resp.) and anhedonia is slightly higher in depressed adolescents compared to depressed children ($\bar{x} = 75.4$ versus 78.8 , resp.), which is consistent with reports that depressed adolescents have greater anhedonia compared to children [11, 13].

Anhedonia improved over the course of patients’ hospital stay but, contrary to our hypothesis, while statistically significant this change does not appear to be clinically meaningful (about a 3 point increase for the scale with the score range 39 to 117). There was no difference in change in anhedonia between significantly improved patients and the rest of the study sample. Our pre-study hypothesis was based on our

expectation that most cases of adolescent depression are phenomenologically similar to demoralization; consistent with the notion by Garland et al. [14] that majority of the cases of adolescent depression are situational depressions. Patients with situational depression have preserved hedonic capacity, even though during acute state their demoralization can be superficially mistaken for an anhedonia [7]. The notion by Garland and Weiss [14] was based on the data on a nonreferred sample. It appears to be a feasible explanation that there is a much higher proportion of patients with non-situational depression in the sample of in-patient depressed adolescents than in nonreferred adolescents. Another possible explanation is that normally hedonic depressed patients actually do not show acute anhedonia in the midst of their acute depressive episode as measured by the pleasure scale. This would be consistent with Clark et al. [5] who found that normally hedonic adult depressed inpatients assessed on admission had Pleasure scores only slightly below those of normal subjects and below their own scores upon recovery.

Pessimistic style improved significantly (with about 1.7 point increase) and this improvement appears to be more clinically meaningful on a scale ranging from -12 to $+12$. This change affected both attributions for bad events and good events. Interestingly, improvement for attributions for positive events was more robust and approached the normal levels. One can speculate that while recovering from depression adolescents would still attribute bad outcomes to themselves but would be able to take credit for good outcomes. Patients who had a significant improvement in depression defined as a decrease in BDI of more than 50% from the initial score had even more impressive improvement in pessimism: with approximately a 2.7 increase in the score, approaching epidemiological normal controls. It is important to note that adolescents who showed significant improvement were as pessimistic upon admission as the rest of the sample. Scores upon discharge were approaching levels for the epidemiological controls, especially so for the significantly improved group. This is a finding that contradicts the scar hypothesis, which holds that depression leaves scars in explanatory style, and even recovered children never regain their optimism. Our findings are consistent with the study of Asarnow and Bates [26] who showed that remitted children inpatients were as optimistic as a never depressed group. Importantly, Asarnow and Bates [26] noted that the remitting depressive group was composed primarily of children whose depressions rapidly remitted following hospitalization and separation from their family and school environments. Limitation of that study was that data on initial (pre-treatment) scores were not available. Nolen-Hoeksema [25] argued that subjects in that study could have never had a pessimistic style. Our subjects had a demonstrated pessimistic style upon admission so this change did take place over the brief period of hospitalization. This is a fascinating finding which shows that explanatory style in adolescents is a flexible and dynamic feature. One might speculate that developing cognitions of adolescents with newly formed capacity for abstraction are very dynamic. This would make them more vulnerable to pessimism but also more capable of recovery. Depressed adolescents in

our sample showed high capacity for remoralization in a protective therapeutic setting. The fact that pessimistic style did not discriminate between patients with the diagnosis of Major Depressive Disorder while anhedonia did probably means that anhedonia is a more primary, core characteristic of depression in adolescents and attributional style is less specific to the diagnosis of depression. It is conceivable that there is an overlap between constitutional factors reflected by anhedonia and demoralization reflected by pessimism.

Lewinsohn et al. [39] in their prospective study of adolescent Major Depressive Disorder among nonreferred adolescents have shown that depressotypic attributional style predicted a future psychopathology in formerly depressed young men. Due to the nature of their sample, depressotypic cognitions have been measured in nontreated adolescents. Lewinsohn et al. [39] commented that important issues that need to be addressed in future research are whether depressotypic cognitions are measured at intake or at the end of treatment. Our findings further underscore the importance of this distinction. We suggest that future research should assess prognostic value of both pre-and post-treatment attributional style and perhaps the prognostic value of its change over the course of treatment. Further research is needed to assess change in pessimistic attributional style separately in hedonic and anhedonic depressed adolescents.

Scores on self-reported and clinician-rated measures did not correlate with sociodemographic variables. This was contrary to our expectations, because one would think that depression might be associated with a number of major stressors or with an unfavorable family situation. That no relationship was found for family situation might be because the entire sample was disadvantaged in this regard, with only 30% of adolescents living in an intact biological family; there was not enough variability within the sample. It is also possible that the environmental component is not as decisive as factor in the development of adolescent depression. Similarly there was no association between recent stressors and depressive variables. However, there was a modest correlation between remote stressors and depressive severity ($r = .39$, $P < .01$). This is consistent with the findings of Nolen-Hoeksema [25] that negative life events play a larger role in the development of depression in earlier life, and later it is personal characteristics that play an important role, with life events becoming less important.

Our data should be interpreted in the context of several limitations of our present study. The first limitation is the lack of normative data for adolescents on anhedonia. Second limitation is the small size of the study sample and a very small size of comparison group and heterogeneity of the study sample with the high number of comorbid disorders. Further research is needed to determine if these data can be replicated or generalized to other clinical populations for example, less severely disturbed adolescents. However comorbidity did not appear to affect Pleasure Scale scores and CASQ scores. Third limitation is that we did not use more stringent instruments such as Research Diagnostic Criteria to establish the diagnosis of Major Depressive Disorder. Another limitation is that we did not have further follow-up data on our subjects and did not investigate if

improvement in attributional style was lasting or related to a protective milieu and was thus short-lived.

5. Conclusions

We suggest that anhedonia is a critical characteristic of adolescent depression, one that could be intimately involved in the pathogenic mechanism of the depressive episode through association with pessimistic attributional style; also there may be a subgroup of depressed adolescents who exhibit enduring anhedonia, which might create vulnerability to future bouts of depression.

Our results suggest that contrary to the "scar hypothesis," pessimistic attributional style is not an enduring trait in depressed adolescents but rather a concomitant feature of an acute depressive state. Our findings suggest that hospitalized depressed adolescents may have superimposed demoralization that is highly responsive to intensive inpatient psychiatric treatment with its protective supportive therapeutic milieu.

Our findings underscore the importance of maintaining treatment gains such as improvement in optimism. Further research should have longitudinal designs, in particular including post-episode data.

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Research Article

Rumination Mediates the Relationship between Infant Temperament and Adolescent Depressive Symptoms

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This study examined prospective associations between negative emotionality, rumination, and depressive symptoms in a community sample of 301 youths (158 females) followed longitudinally from birth to adolescence. Mothers reported on youths' negative emotionality (NE) at age 1, and youths self-reported rumination at age 13 and depressive symptoms at ages 13 and 15. Linear regression analyses indicated that greater NE in infancy was associated with more depressive symptoms at age 15, even after controlling for child gender and depressive symptoms at age 13. Moreover, analyses indicated that rumination significantly mediated the association between infancy NE and age 15 depressive symptoms in the full sample. When analyzed separately by gender, however, rumination mediated the relationship between NE and depressive symptoms for girls but not for boys. The results confirm and extend previous findings on the association between affective and cognitive vulnerability factors in predicting depressive symptoms and the gender difference in depression in adolescence, and suggest that clinical interventions designed to reduce negative emotionality may be useful supplements to traditional cognitive interventions for reducing cognitive vulnerability to depression.

1. Introduction

Adolescent depression is a major mental health problem. Depression increases in the transition to adolescence, such that while fewer than 6% of youth under age 11 will experience a depressive episode, nearly 20% of youth will experience a depressive episode by age 18 [1, 2]. In addition, up to 65% of adolescents report subclinical depressive symptoms at any given time, and extensive research has demonstrated that both mild-to-moderate depressive symptoms and diagnosable depressive episodes predict greater academic and interpersonal problems, substance use, and depressive episodes in adulthood [3, 4]. Adolescent depression also confers risk for future depression, with nearly 70% of adolescents experiencing another episode within five years [5]. Within adolescence, the early to middle adolescent period from ages 11 to 15 is of particular salience to depression researchers. During this time, depression rates surge for all youth and a marked gender difference emerges

such that by age 15 girls are twice as likely as boys to become depressed [2].

Extensive research on the etiology of adolescent depression has demonstrated multiple vulnerability factors contributing to the rise in depressive symptoms as well as the emergence of the gender difference in depression during this developmental period. Both affective (e.g., temperament) and cognitive (e.g., rumination, cognitive style) factors have been found to confer vulnerability to adolescent depression. In their ABC Model of depression in adolescence, Hyde et al. [6] integrated affective and cognitive models of adolescent depression by hypothesizing that the specific temperamental trait of negative emotionality contributes to the development of maladaptive cognitive responses, such as rumination, that become habitual across the adolescent transition and subsequently confer vulnerability to depression. The current study examines this hypothesis longitudinally in a sample of community youth followed from infancy into adolescence.

Temperament is conceptualized as biologically-based, relatively stable individual differences in emotional, behavioral, and attentional reactivity and regulation [7]. These individual differences are hypothesized to be present early in infancy and childhood, and relatively stable across the lifespan. Negative emotionality (NE) is defined as a constellation of temperamental characteristics including high frequency and intensity of negative affective states such as fear, frustration, and distress. In infancy, children high in NE tend to display strong startle responses to new or aversive stimuli as well as high distress to novel or frustrating situations. Later in childhood, youth high in NE tend to dislike or avoid novel situations, become highly distressed in novel or frustrating situations, and display negative emotions such as fear, distress, and frustration more frequently and/or intensely than other children [8, 9]. Numerous studies have demonstrated an association between NE and depression among adults, adolescents, and children [10–13]. Specifically, NE has been demonstrated to be both concurrently associated with and prospectively predictive of depression in adolescence [10, 14].

The cognitive model of depression suggests that individual differences in cognitive responses to negative events may predispose individuals to becoming depressed when faced with such events. One such cognitive vulnerability is a ruminative response style [15, 16]. Rumination may be broadly defined as a passive and perseverative attentional focus on negative stimuli, including sad, depressed, or negative emotions, stressful events, and self-critical or otherwise negative thoughts. Nolen-Hoeksema originally defined rumination broadly as “repetitively focusing on the fact that one is depressed; on one’s symptoms of depression; and on the causes, meanings, and consequences of depressive symptoms” [15, page 569]. In recent years, researchers have identified many subtypes of rumination differentiated primarily by the content upon which an individual is ruminating. One such subtype of rumination has been termed *depressive rumination*. Depressive rumination is defined as a passive and perseverative focus on negative emotions such as sadness and depressed mood [17–20]. Depressive rumination reflects an involuntary coping response in which one’s attention following a stressor is “directed passively and perseveratively toward the negative emotions elicited by the stressor” [21, page 977]. As such, depressive rumination is essentially emotion-focused and can be differentiated from other rumination subtypes, including brooding, which is ruminative focus on negative or self-critical thoughts [22] and from rumination about other negative emotions such as anger [18, 23].

Rumination on sadness and depressed feelings is believed to prolong and exacerbate depressed mood by increasing the salience of the negative emotions being attended to. Not surprisingly, then, depressive rumination has been demonstrated to prospectively predict both the onset and duration of depression among adolescents [24, 25]. However, we understand less about the factors contributing to individual differences in depressive rumination. Given the emotion-focus of depressive rumination, individual differences in negative emotionality may be associated with individual

differences in the tendency to ruminate on those negative emotions.

In their ABC Model of depression in adolescence, Hyde et al. [6] integrated affective and cognitive models of depression by suggesting that youth high in negative emotionality would, over time, develop more negative cognitive responses to stressful events. This hypothesis integrates basic research linking affective reactions to stressful events to the cognitive processing of those events, and further frames the integration within the developmental trajectory of adolescent depression. Weiner [26] noted that affective processing of negative events precedes higher-order cognitive processing of the event, such as making attributions about causality. Several studies have demonstrated that affective responses to stressful events subsequently affect cognitive processing of those events. For example, high negative affect is associated with greater subjective appraisal of ambiguous or novel events as stressful [27]. High negative affect is also associated with greater interpretation of events as catastrophic, greater attention to the negative event, greater self-focus, and more negative expectancies [28–30]. Over time, this pattern of more negative and depressogenic cognitive responding may become habitual and consolidate into a trait of cognitive vulnerability to depression such as rumination. Several researchers have suggested that cognitive vulnerabilities to depression emerge and consolidate in early to middle adolescence, a timing that is consistent with the increase in depressive symptoms among youth [31, 32].

This hypothesized developmental link between negative emotionality and rumination suggests that the predictive relationship between negative emotionality and depression may be mediated in part by rumination. In recent years, a handful of studies have examined this hypothesis empirically. Feldner et al. reported that, among adults, negative emotionality was significantly correlated with rumination [33]. A similar correlation has been observed among adolescents as well [34]. Verstraeten et al. tested the full mediation model among adolescents and found that rumination did significantly mediate the relationship between negative emotionality and depression, but only when constructs were analyzed concurrently rather than prospectively [35]. To the best of our knowledge, only one study has examined the relationship between negative emotionality, rumination, and depression prospectively among adolescents. Mezulis et al. recently found that the prospective relationship between negative emotionality in early adolescence (age 12) and later depressive symptoms (age 15) was mediated by rumination at age 14, even after controlling for depressive symptoms at ages 12 and 14 [36]. A limitation of this study, though, is that all measures were self-reported by the youth in adolescence, leaving unanswered the question of whether indices of temperament early in life are associated with the development of rumination and subsequent depression in adolescence.

Finally, it is as yet unknown whether prospective relationships among negative emotionality, rumination, and depression may contribute to our understanding of the emergent gender difference in depression in adolescence. Although many studies identify a gender difference in rumination that

has been found to partially mediate the gender difference in depression in adolescence (see [6] for a review), most studies fail to demonstrate a significant gender difference in negative emotionality in infancy or childhood [37]. It is possible that negative emotionality may contribute to the development of rumination amongst girls but not boys, perhaps because of how coping responses to negative mood are socialized differently among boys and girls [38]. Thus, we hypothesized that the prospective relationship between negative emotionality and rumination may be stronger for girls than boys.

The current study extends the extant literature by examining rumination as a mediator of the relationship between negative emotionality in infancy and depressive symptoms in adolescence in a prospective study of community youth followed longitudinally from infancy to age 15. The conceptual model is presented in Figure 1. Specifically, we hypothesized that:

- (1) negative emotionality in infancy would predict depressive symptoms in mid-adolescence (age 15);
- (2) the relationship between infant negative emotionality and adolescent depressive symptoms at age 15 would be mediated by rumination at age 13, even after controlling for depressive symptoms at age 13;
- (3) the relationship between infant negative emotionality and rumination at age 13 would be stronger for girls than boys.

2. Method

2.1. Participants. Participants were 301 adolescents (158 female) in the United States who have been part of the longitudinal Wisconsin Study of Families and Work since birth (formerly named the Wisconsin Maternity Leave and Health Project; [39]). Participants were originally recruited from the Madison and Milwaukee, Wisconsin areas and currently reside in a range of communities, including a large Midwestern city, a small Midwestern city, several small towns, and rural areas. Of participants in the present study, 90.0% were White, 4.0% American Indian/Alaskan, 3.0% African American, 1.7% Asian/Pacific Islander, 1.0% Hispanic, and 0.3% were members of another group. Participants are ethnically representative of the communities from which they were recruited, and their families are socioeconomically similar to families in the United States.

Data were collected at age one and during the summer following grades seven (mean age = 13.52, SD = 0.33; summers of 2004 and 2005) and nine (M = 15.50, SD = 0.33; summers of 2006 and 2007). The present study includes participants who completed all measures used in the study. Participants who remained in the study at adolescence did not differ from those who discontinued participation prior to adolescence in terms of race/ethnicity, family income, or parents' depressive symptoms.

2.2. Procedure. When the participating children were 12 months of age, their mothers completed a written questionnaire to assess the children's temperament. At ages 13 and 15,

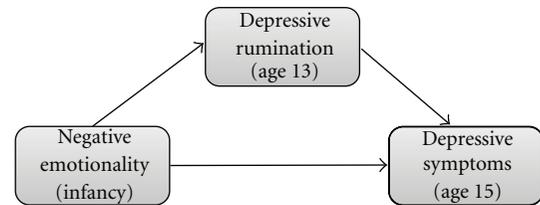


FIGURE 1: Conceptual model.

participants completed a number of questionnaires administered on a laptop computer during in-home visits. These questionnaires included measures of depressive symptoms in the past two weeks and rumination tendencies.

2.3. Measures

2.3.1. Negative Emotionality. Negative emotionality was measured using the distress to limitations (anger), distress to novelty (fear), and startle subscales of the Infant Behavior Questionnaire (IBQ; [40]). The 39 items of these subscales asked mothers to rate their children's responses to specific behaviors (e.g., child became upset when having face washed) in the past two weeks on a seven-point scale ranging from 1 (*never*) to 7 (*always*). Negative emotionality was calculated by averaging the mean scores on each of the three subscales. Rothbart reported internal consistencies for children 12 months of age as .78 for distress to limitations and .81 for distress to novelty. In the present study, internal consistency was .84 for distress to limitations, .74 for both distress to novelty and startle, and .84 for the combined set of items.

2.3.2. Rumination. Depressive rumination was assessed at age 13 using a short form of the Ruminative Response Scale (RRS) of the Response Style Questionnaire (RSQ; [41]). The original RRS includes 22 items in which respondents are asked how often they engage in ruminative responses when they feel sad or down, with responses rated on a 4-point Likert scale from 1 (*almost never*) to 4 (*almost always*). In the current study, we used five items specifically assessing rumination about negative affect. Items include "When I feel sad or down, I think about how alone I feel" and "When I feel sad or down, I think about how hard it is to concentrate". The five items utilized were selected based upon consultation with Nolen-Hoeksema at the time of study design (personal communication, 2001) as representing a selection of rumination items that excluded automatic negative thoughts and emphasized instead rumination about sad, depressed, or down affect. The exclusion of items that include negative automatic thoughts is preferable because it creates a purer measure of depressive rumination that is focused on the affective component of depressive symptoms, rather than the cognitive component. The full RRS has been used with adolescents in several prior studies [42, 43]; in the present study, internal consistency was .73 for the depressive rumination subscale.

2.3.3. Depressive Symptoms. Depressive symptoms were assessed at ages 13 and 15 with the Children’s Depression Inventory (CDI; [43]). The CDI includes 27 items assessing common affective, behavioral, and cognitive symptoms of depression. For each of the 27 items, adolescents were asked to pick which of three statements best described them in the past two weeks. The three statements represent differing levels of symptom severity; for example, youth select one of the following three statements: “I was sad once in a while,” “I was sad many times,” or “I was sad all the time.” Each item is scored 0, 1, or 2, and answers to individual items were summed, such that a CDI score could range from 0 to 54. The CDI has demonstrated good internal consistency (typically .71 to .89) and adequate test-retest reliability (typically .72 to .87) [44–46]. In the present study, internal consistency was .83 at age 13 and .86 at age 15.

2.4. Data-Analytic Technique. Analyses were conducted in SPSS as a series of regression models (and in the case of the indirect effect, confidence intervals) using the macro command set developed by Preacher and Hayes [47] to test mediation models that include covariates. A mediation model suggests that the relationship between the predictor variable (here, negative emotionality) and the outcome variable (here, depressive symptoms at age 15) is partially or completely accounted for by some mediating variable (here, rumination at age 13). With the mediator in the model, the predictor and outcome variables are not expected to be directly related, but rather *indirectly* related through the effect of the predictor variable on the mediator, and then the mediator variable on the outcome variable.

Common tests of indirect effects in mediation models, such as the Sobel test, assume that the sampling distribution of an indirect effect is normally distributed; however, this assumption typically holds only for quite large sample sizes. To avoid violating this assumption, the procedure developed by Preacher and Hayes uses a nonparametric approach that does not require multivariate normality to explicitly test the indirect effect. Specifically, this procedure employs a bootstrap method, in which the original data are sampled (with replacement) 5000 times. The indirect effect coefficients generated from these 5000 samples are then ordered numerically. The low and high values that cap the middle 95% of the results represent the bounds of a 95% confidence interval. If zero is not within this 95% confidence interval, then there is evidence of an indirect effect between the predictor variable (e.g., negative emotionality) and outcome variable (e.g., depressive symptoms) at the standard Type I error rate of $\alpha = .05$.

We examined the hypothesized mediator model three times: once for the entire sample, and then separately for boys and girls.

3. Results

3.1. Descriptive Statistics. Table 1 displays descriptive statistics for overall negative emotionality, rumination, and depressive symptoms, separately by gender. Table 2 displays

TABLE 1: Descriptive statistics for overall negative emotionality, rumination, and depressive symptoms, by gender.

Variable	Girls		Boys		Comparison	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>t</i>	<i>P</i>
NE (infancy)	2.94	.58	2.84	.56	1.46	.146
Rumination (13)	2.02	.55	1.77	.53	3.91	.000
Depression (13)	4.89	5.21	3.79	4.18	2.01	.046
Depression (15)	5.74	5.82	3.84	4.95	3.03	.003

TABLE 2: Correlation matrix for overall negative emotionality, rumination, depressive symptoms, and gender.

	1	2	3	4
(1) NE (infancy)		.09	.08	.14
(2) Rumination (13)	.17*		.23**	.15
(3) Depression (13)	.10	.52**		.45**
(4) Depression (15)	.07	.45**	.62**	

Note. Correlations significant at $P < .05$ denoted by *. Correlations significant at $P < .01$ denoted by **. Correlations are above the diagonal for boys and below the diagonal for girls.

correlations between negative emotionality, rumination, and depressive symptoms, again separately by gender. As seen in Table 2, the patterns of correlations varied markedly for girls and boys. Therefore, analyses were computed first for the entire sample and then separately for girls and boys.

3.2. Temperament, Depressive Rumination, and Depressive Symptoms. The present study examined a mediation model in which depressive rumination at age 13 was hypothesized to mediate the relationship between negative emotionality in infancy and depressive symptoms at age 15. Regression equations tested each pathway depicted in Figure 1. In addition, each equation controlled for prior depressive symptoms at age 13 and gender. Depressive symptom scores were log transformed prior to analysis to account for their skewed distribution.

Results supported our hypothesized mediation model. The effect of negative emotionality on depressive symptoms at age 15 was significant, $b = .09$, $t(300) = 2.23$, $P = .03$. Additionally, as expected, the path from negative emotionality to rumination was significant; participants who were higher in negative emotionality during infancy reported greater tendencies to ruminate as adolescents, $b = .14$, $t(300) = 2.57$, $P = .01$. Similarly, the path from rumination to depressive symptoms was significant; adolescents who reported more rumination at age 13 had more depressive symptoms at age 15, even after controlling for earlier symptoms, $b = .12$, $t(300) = 2.99$, $P = .003$. With rumination in the model, the direct effect of negative emotionality on depressive symptoms at age 15 was nonsignificant, $b = .02$, $t(300) = .61$, $P = .54$. Additionally, the confidence interval for the effect of the indirect pathway via rumination did not include “0” (.004 to .037), indicating a significant mediated pathway. The overall mediation model was significant, $F(4, 296) = 34.14$, $P < .001$, and accounted for approximately 32% of the

variance in depressive symptoms ($R^2 = .32$, adjusted $R^2 = .31$). Thus, our model of the relationship between negative emotionality, rumination, and depressive symptoms was supported by the data.

3.2.1. Temperament, Rumination, and Depressive Symptoms among Girls. Given the marked differences in the correlations among variables for boys and girls in our sample, we also examined the hypothesized mediation model separately by child gender to determine if the relationships among variables were comparable across gender. The overall model for girls was significant, $F(3,154) = 24.61$, $P < .001$, and explained 32% of the variance in depressive symptoms ($R^2 = .32$, adjusted $R^2 = .31$). The effect of infant negative emotionality on depressive symptoms at age 15 failed to reach statistical significance, likely as a result of the reduced sample size ($b = .08$, $t(157) = 1.48$, $P = .14$). However, the path from negative emotionality to rumination was significant; as expected, girls who were higher in negative emotionality during infancy reported greater tendencies to ruminate as adolescents, $b = .16$, $t(157) = 2.09$, $P = .04$. Similarly, the path from rumination to depressive symptoms was significant; girls who reported more rumination at age 13 had more depressive symptoms at age 15, even after controlling for symptoms at age 13, $b = .16$, $t(157) = 2.86$, $P = .005$. Finally, the confidence interval for the indirect pathway via rumination did not include “0” (.004 to .061), indicating that rumination significantly mediated the relationship between negative emotionality and depressive symptoms among girls.

3.2.2. Temperament, Rumination, and Depressive Symptoms among Boys. The overall model for boys was significant, $F(3,139) = 16.04$, $P < .001$, and explained 26% of the variance in depressive symptoms ($R^2 = .26$, adjusted $R^2 = .24$). The effect of infant negative emotionality on depressive symptoms at age 15 was comparable to that observed among girls and also failed to reach statistical significance, also likely as a result of the reduced sample size ($b = .08$, $t(142) = 1.32$, $P = .19$). However, in contrast to girls, the effect of negative emotionality on rumination was not significant; boys who were higher in negative emotionality during infancy did not report greater tendencies to ruminate as adolescents, $b = .09$, $t(142) = 1.11$, $P = .27$. Furthermore, the effect of rumination on depressive symptoms was also not significant; rumination at age 13 did not predict depressive symptoms at age 15 among boys, $b = .08$, $t(142) = 1.36$, $P = .18$. Finally, the confidence interval for the indirect pathway via rumination did include “0” (−.005 to .030), indicating that rumination did not significantly mediate the relationship between negative emotionality and depressive symptoms among boys.

4. Discussion

This study examined the relationship between temperament, rumination, and depressive symptoms prospectively in a community sample of youth followed from infancy to age

15. The primary purpose of the study was to empirically test one of the integrative hypotheses of the ABC Model of adolescent depression which asserts that affective vulnerability to depression, that is, temperament, contributes to the development of cognitive vulnerability to depression, that is, rumination [6]. This hypothesis suggests that individuals who are temperamentally predisposed to respond to stressful events with intense and prolonged negative affect will subsequently allocate more attentional resources to those events, and that this pattern of affective-cognitive processing of stressful events will, over time, consolidate into the stable cognitive vulnerability of rumination. The present study extends prior examinations of rumination as a mediator of the relationship between temperament and depression by examining infant temperament as it predicts later rumination and depressive symptoms.

4.1. Infant Negative Emotionality and Adolescent Depression. Numerous studies have examined the temperamental construct of negative emotionality as it predicts depressive symptoms and disorders [7, 13]. However, only a handful of studies have examined the predictive relationship between infant or early childhood temperament on depression in adolescence. Moffitt et al. [48] reported that behavioral inhibition, one component of negative emotionality, at age 3 prospectively predicted depression diagnoses by age 21. Similarly, Lonigan et al. found that childhood negative emotionality at age 9 predicted depressive symptoms at age 16 [49]. Consistent with these prior studies, we similarly found that greater negative emotionality in infancy was significantly associated with greater depressive symptoms in adolescence.

4.2. Linking Affective and Cognitive Processes to Adolescent Depression. The primary purpose of this study, however, was to examine the mechanism by which early individual differences in negative emotionality develop into later depressive symptoms. In adolescence, we know that individual differences in affective and cognitive responses to stressful events may differentiate individuals for whom mood disturbances are transient from individuals for whom that mood disturbance persists and develops into a depressive response. Teasdale’s differential activation hypothesis [30] suggests that some individuals are more likely to respond to negative affect with the activation of negative thoughts and rumination. Teasdale labeled this individual difference in the extent to which negative cognitive processing is elicited by negative affect as *cognitive reactivity*, and suggested that for these cognitively reactive individuals, a vicious cycle between negative affect and negative thinking will ensue that eventually leads to depression. The ABC Model presents a theoretically consistent hypothesis that emphasizes the *developmental* relationship between negative affect and cognitive processing, suggesting that it is early individual differences in affective responding to stress that set the developmental stage for this vicious cycle of affective and cognitive processing to ensue. We hypothesized that children who are temperamentally high in negative emotionality will

become adolescents who are high in cognitive vulnerability, including rumination.

Only a handful of studies have examined whether individual differences in negative emotionality predict individual differences in cognitive vulnerability to depression. Several studies have examined the relationship between neuroticism, rumination, and depression among adults [50–55]. Neuroticism is a personality trait which is similar to, but more broad than, the temperamental construct of negative emotionality; neuroticism is associated with high negative emotionality as well as high stress sensitivity and worry [56].

Few studies have examined the relationship between the specific construct of negative emotionality and rumination. Our findings contribute to a small but growing body of literature suggesting that negative emotionality is an important contributor to rumination among adolescents. Our findings are consistent with those of Chang [34] and Verstraeten et al. [35] demonstrating a significant association between these constructs, and providing further evidence that the relationship between temperament and depression may be mediated by cognitive processes.

Interestingly, although the mediation model was supported for the entire sample, follow-up analyses by child gender suggested that the prospective association between infant negative emotionality and adolescent depressive symptoms may be mediated by rumination among girls but not among boys. This finding suggests, consistent with the ABC Model of the gender difference in adolescence depression, that there may be multiple processes contributing to the gender difference in depression. In our sample, girls and boys did not differ on mother-reported infant negative emotionality, a finding consistent with a recent meta-analysis examining gender differences in temperament [37]. However, boys and girls did differ on rumination in early adolescence, with girls reporting greater rumination than boys. Our findings suggest that early negative emotionality may contribute to later ruminative tendencies among girls but not boys. It is interesting to speculate on this gender divergence; one possible explanation may be how parents respond to displays of negative emotionality differently for sons and daughters. Recent research has suggested that mothers may be more likely to direct attention to and encourage discussion of negative emotions, particularly fear, distress, and sadness, when interacting with their daughters than with their sons, and that this gender difference in parenting style may contribute to the gender difference in rumination [38].

It is also important to note that negative emotionality is only one of multiple constructs of temperament associated with vulnerability to depression. Prior research has also implicated low positive emotionality in the etiology of depression [12], although that relationship is not hypothesized to be mediated by cognitive vulnerability [6]. Recent research has also suggested that the relationship between negative emotionality and cognitive vulnerability may itself be moderated by other regulatory components of temperament. For example, effortful control is another feature of temperament conceptualized as “the ability to inhibit a dominant response to perform a subdominant response” [7, page 137], and thus is a self-regulatory process that

requires effortful or voluntary control of both attention and behavior to modulate emotional experience and expression [57]. Some have found that effortful control may moderate the relationship between the affective reactivity of negative emotionality and rumination [35]. Although the present study did not include a measure of effortful control with which to examine this hypothesis, it would be an interesting elaboration for future prospective studies.

4.3. Clinical Implications. Adolescent-onset depression is associated with both concurrent deficits in adaptive functioning and prospective risk for future depressive episodes. The vast majority of youth experiencing depression in adolescence will have another episode within five years [5]. Unfortunately, treatments for adolescent depression lag behind those for other disorders and more than half of youth fail to respond to currently available interventions [58]. The majority of current depression interventions emphasize techniques designed to reduce or eliminate depressogenic cognitive processes such as rumination. Continued evidence suggesting that individual differences in negative emotionality significantly contribute to individual differences in rumination suggest that interventions designed to reduce individuals’ negative affect may be helpful in treating or preventing depression as well. Relaxation training may be effective in attenuating youths’ automatic and intense negative affective responses, and a growing body of research suggests that mindfulness-based interventions may improve emotion regulation through improving the individual’s ability to respond to stressful situations reflectively rather than automatically. Mindfulness has been shown to be effective in treating depression, possibly through its effects in decreasing rumination [59, 60]. Kabat-Zinn [61] also suggested that the slow and deep breathing taught in mindfulness training may reduce physiological reactivity and the subjective emotional and physiological feelings of distress. Although few studies have examined the effects of mindfulness on affective responding, Goldin and Gross [62] recently reported that individuals with social anxiety reported less negative affect during a breath-focused mindfulness task compared to a distraction-focused task. Similarly, Charbonneau and Mezulis [63] recently reported that among college females high in negative emotionality, a brief intervention teaching emotion regulation strategies such as deep breathing and progressive muscle relaxation strategy was effective in reducing rumination. In summary, a greater understanding of the relationship between affective and cognitive vulnerability to depression may suggest that clinical interventions targeting negative emotionality or emotional reactivity may be effective in reducing cognitive vulnerability to depression as well.

4.4. Limitations and Future Research. While our study demonstrates links between temperament, rumination, and depressive symptoms, several limitations should be noted. First, the study examined rumination narrowly defined as perseverative attention on negative emotions. Conceptually, the depressive rumination subtype of rumination is most logically linked with negative emotionality, as the strong

negative emotions experienced by individuals high in this temperamental feature may particularly elicit the attentional focus on negative emotions that comprises depressive rumination. However, future research may want to consider the relationship between negative emotionality and other forms of rumination. Of particular interest may be brooding, which is defined as rumination on negative or self-critical thoughts. Recent evidence specifically links the rumination dimension of brooding with depression among adolescents and may be one specific facet of the cognitive reactions to stress elicited by negative emotionality [64, 65]. Second, our sample was a community sample of predominantly Caucasian youth. The relationships among our constructs may be different among high-risk or clinical samples. Finally, we note that the pathway from negative emotionality to depression, mediated by rumination, demonstrated in the current sample is but one of multiple developmental trajectories implicated in the etiology of adolescent depression. As extensively reviewed in other studies, there are multiple temperamental and cognitive factors that contribute to adolescent depression both within and across individuals. Low positive emotionality is another temperamental factor implicated in the etiology of depression, as well as other cognitive vulnerability factors such as negative cognitive style (as reviewed in [6]). Thus, the negative emotionality-rumination-depression link examined here is undoubtedly but one pathway to adolescent depression.

Despite these limitations, our findings continue to implicate temperament in the development of cognitive vulnerability to depression. These findings contribute to our ability to identify at-risk individuals as well as design interventions targeting both affective and cognitive processes in adolescent depression.

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Review Article

Review: Magnetic Resonance Spectroscopy Studies of Pediatric Major Depressive Disorder

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Introduction. This paper focuses on the application of Magnetic Resonance Spectroscopy (MRS) to the study of Major Depressive Disorder (MDD) in children and adolescents. *Method.* A literature search using the National Institutes of Health's PubMed database was conducted to identify indexed peer-reviewed MRS studies in pediatric patients with MDD. *Results.* The literature search yielded 18 articles reporting original MRS data in pediatric MDD. Neurochemical alterations in Choline, Glutamate, and N-Acetyl Aspartate are associated with pediatric MDD, suggesting pathophysiologic continuity with adult MDD. *Conclusions.* The MRS literature in pediatric MDD is modest but growing. In studies that are methodologically comparable, the results have been consistent. Because it offers a noninvasive and repeatable measurement of relevant *in vivo* brain chemistry, MRS has the potential to provide insights into the pathophysiology of MDD as well as the mediators and moderators of treatment response.

1. Introduction

Led by a major research initiative from the U.S. National Institute of Mental Health (NIMH), the hunt is on for biomarkers in psychiatry [1]. A biomarker is a characteristic that is objectively measured and evaluated as an indicator of normal biological processes, pathogenic processes, or pharmacological responses to a therapeutic intervention [2]. The readiness of biological markers to serve as features, risk factors, or diagnostic criteria is of significant concern in the development of the DSM-V [3], and the research agenda for DSM-V emphasizes the need to translate research findings into a new classification for psychiatric disorders [4, 5]. In psychiatry, biomarkers could be used to detect and assess or to predict the development of psychiatric disorders—and more importantly, biomarkers could be used to inform treatment decisions [6]. It has been argued that the DSM-V should be structured to permit incorporation of well-replicated findings from neuroscience, by creating

mechanisms to flexibly evaluate genetic markers or neuroimaging results rather than waiting for publication of the DSM-VI [7]. A consensus has emerged that advances in the assessment, treatment, and prevention of brain disorders are likely to originate from studies based on clinical and translational neuroscience [8].

In the search for these translational tools, neuroimaging is a strong candidate to aid psychiatry in its quest to join the other specialties in medicine in utilizing tests anchored in biology for delivering care and developing new interventions. Advances in child psychiatry neuroimaging have begun to provide a scientific infrastructure for understanding numerous psychiatric disorders [9, 10]. Because it can define the neural structures and pathways that mediate illness and its progression, imaging has the potential for use in clinical decision making and disease monitoring [11]. Owing to the fact that depression is not associated with gross tissue pathology or with unambiguous animal models for spontaneous and recurrent episodes, the availability of research tools to

noninvasively assess the brain is critical to elucidating the neurobiology of mood disorders [12]. In addition to the scientific insight they provide, the neuroimaging methods employed in child and adolescent psychiatry are noninvasive and have proven to be safe [13].

Child and adolescent psychiatry investigators have adopted a number of neuroimaging approaches. The purpose of this paper is to review the findings reported using Magnetic Resonance Spectroscopy (MRS) in the study of pediatric Major Depressive Disorder (MDD). As the name suggests, MRS is a procedure that allows measurement of relevant neurochemistry. Its application to the study of MDD is therefore of particular interest, because mood disorders are illnesses of “state” [14, 15]. If MRS is able to define and validate neurochemical biomarkers, then results of MRS studies may one day be utilized to diagnose mood disorders and to monitor treatment response.

MDD is a significant global health problem, ranking 3rd on the World Health Organization’s list of the most common causes of disability worldwide [16]. In the U.S., the National Comorbidity Survey Replication study found that the peak age of onset for mental disorders is 14 years, with MDD most commonly emerging during adolescence [17, 18]. With an annual incidence of 2% in children and 4% to 8% in adolescents [19], and a cumulative lifetime prevalence of up to 20% [20], MDD in children is associated with academic failure, social impairment, substance abuse, and suicide attempts [19]. Compared with healthy controls, depressed adults are more likely to have had a depressive episode in adolescence [21]. Adding to the morbidity and mortality experienced by patients and their families, pediatric MDD imposes a substantial economic burden on society [22]. The personal and financial toll is amplified by the fact that only 50% of patients with MDD are diagnosed before reaching adulthood [23]. Therefore, novel diagnostic and treatment tools for patients in the critical adolescent stage of development are urgently needed [24–27].

The paper begins with a brief description of how MRS data is acquired, the information that is obtained, and how that information differs from the results of other neuroimaging methods. Following a review of MRS studies in pediatric MDD, the paper concludes with suggested directions for further study.

2. Magnetic Resonance Spectroscopy: A Primer

In 1946, Felix Bloch at Stanford University and Purcell at the Massachusetts Institute of Technology independently demonstrated the phenomenon of Nuclear Magnetic Resonance (NMR) [28, 29]. It was known that the nuclei of certain elements, for example, hydrogen (^1H) and phosphorus (^{31}P), have magnetic properties and spin. Bloch and Purcell showed that nuclei containing an odd number of nucleons (i.e., protons + neutrons) could absorb energy at a specific resonance when placed in a strong magnetic field. Based upon the principles of NMR, magnetic resonance imaging (MRI) was introduced to clinical medicine in the 1970s [30]. The principles of NMR were used to develop magnetic resonance spectroscopy (MRS), which enabled characterization

of living tissues based upon their chemical constituents [31]. Whereas MRI depicts the spatial distribution of protons of water, which are transformed into a visual representation of anatomy for interpretation, MRS detects various molecules present at concentrations on the order of mM (millimolar) [32]. For a review of the scientific principles underlying MRS, the interested reader is referred to the referenced publications [33–36].

The key distinction between MRI and MRS is the type of information the magnetic resonance signal is used to encode. MRI studies create anatomical images whereas MRS provides *quantitative* biochemical information about the tissue under study. Rather than high-resolution images, MRS data are presented as graphical spectra, with the area under each peak representing the relative concentration of nuclei detected for a given atomic species, for example, hydrogen or phosphorus. The x -axis of graphed MRS data denotes the frequency shift localizing the metabolite in parts per million. MRS spectra peaks correspond with specific chemical compounds of interest. Thus, MRS non-invasively provides a repeatable measure of chemical concentration data in living tissues, including the human brain. For the patient, the procedure is identical to a clinical study except for the amount of time spent in the scanner; the duration ranges from less than 10 minutes for acquisition of proton spectra to approximately 30 minutes for a phosphorus scan. MRS scans use no radiation, which allows for repeated measurement at different times in the course of a patient’s illness, for example, prior to the start of treatment and at the point that remission is achieved. Figure 1 shows an example of ^1H -MRS spectrum obtained from the anterior cingulate cortex of a patient with MDD.

The chemicals quantifiable with ^1H -MRS include the following: N-Acetyl-Aspartate (NAA), creatine (Cr), choline (Cho), myoinositol (mI), and lactate (Lac). The term “GLX” (Glx) is used to designate the single peak containing the amino acid neurotransmitters Glutamate (Glu), Gamma-Aminobutyric Acid (GABA), and Glutamine (Gln) [37], because ^1H -MRS signals from Glu and Gln are complicated by the interaction of neighboring protons and the pH dependence of chemical shift [38]. Glu is the major excitatory neurotransmitter in the human brain and was first measured in 1992 [39]. Brain *in vivo* concentrations of Glu are approximately 8–13 times that of GABA, and the ratio of Glu/Gln ranges from 2.4–3.8; therefore, alterations in Glx are typically attributed to altered Glu concentrations [40]. GABA, the major inhibitory neurotransmitter in the brain, has an ^1H -MRS peak that can be separated from Glx at magnetic field strengths ≥ 2 Tesla using spectral editing technique or 2-D J-resolved spectra [41].

NAA is the most prominent ^1H -MRS peak and is found only in the nervous system [42]. It is a marker of neuronal density or function, osmoregulation, and energy homeostasis; there is a direct relationship between NAA synthesis, oxygen consumption, and ATP production in the central nervous system [43]. NAA may also play a critical role in myelin production within oligodendrocytes [44]. Reduction in NAA levels measured by ^1H -MRS is a recognized marker of neuronal loss or dysfunction in several psychiatric and

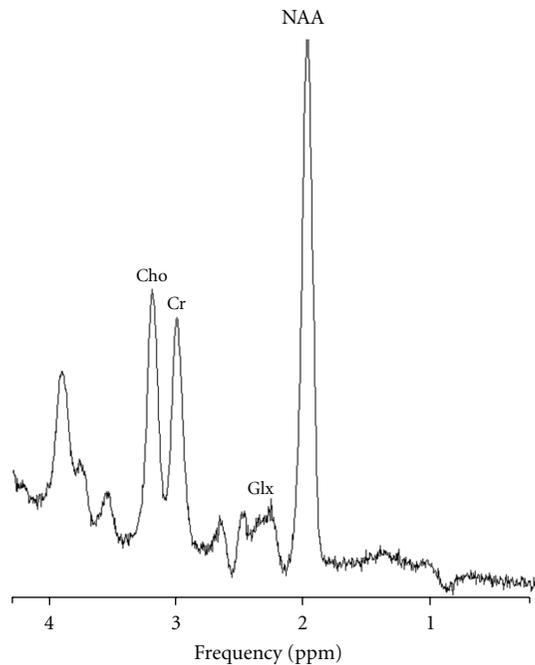


FIGURE 1: Representative proton (^1H) magnetic resonance spectrum of the anterior cingulate cortex at 3 Tesla (TR = 2000 ms, TE = 135). Cho = Choline; Cr = Creatine; Glx = Glutamine + Glutamate; NAA = N-Acetyl Aspartate; ppm = parts per million.

neurological disorders including drug abuse, schizophrenia, traumatic brain injury, stroke, epilepsy, multiple sclerosis, neoplasm, HIV encephalopathy, and Alzheimer's disease [43].

The Cr peak reflects the sum of the Cr and Phosphocreatine (PCr) peaks, an important limitation that will be discussed below. The equilibrium maintained between Cr and PCr is determined by the cellular demand for the high-energy phosphate stored as creatine phosphate [45]. As its level is considered to be constant, Cr is often used as an internal standard for comparison [46]. The Cho peak contains four membrane- and myelin-related chemicals [36]: phosphorylethanolamine (PE), phosphorylcholine (PC), glycerophosphorylethanolamine (GPE), and glycerophosphorylcholine (GPC). Cho is a metabolic marker of membrane density and integrity, that is, phospholipid synthesis and degradation [30]. Neuropathology characterized by cell membrane breakdown liberates Cho and increases the free Cho pool, contributing to an increased resonance in neurodegenerative disorders [45]. In traumatic brain injury, Cho levels increase in relation to the severity of neuronal injury resulting from the breakdown of membranes and myelin [47]. Finally, an elevation in the Cho resonance within brain lesions has been accepted as a sign of malignancy [48, 49]. Myo-inositol (mI) is a sugar involved in the regulation of neuronal osmolarity, the metabolism of membrane bound phospholipids, and in the phosphoinositide (PI) secondary messenger pathway [46]. Myo-inositol is considered a marker of glial proliferation, and an increase in mI resonance may be a proxy for increased inflammation in the brain [50].

Under normal circumstances, lactate (Lac) is present in the brain at concentrations too small to be detected using ^1H -MRS. However, if the aerobic oxidation mechanism fails and anaerobic glycolysis is triggered—such as brain ischemia, hypoxia, seizure activity, and metabolic disorders—Lac levels rise significantly [30]. Typically, the Lac peak can be observed as an inverted doublet at an echo time of 135 ms at 1.3 ppm. It has been shown that lactate becomes elevated if large numbers of inflammatory cells are activated [51]. In the past two decades, ^1H -MRS has progressed from the laboratory into routine use in the treatment of cancers of the brain and prostate [33]. Mood disorders may join the list of disease states in which clinicians can make use of ^1H -MRS, if the current pace of neuroimaging research in psychiatry is maintained.

^{31}P -MRS is a related neuroimaging method that acquires the resonance spectra of phosphorus rather than hydrogen. Although MRS can be performed on a variety of nuclei such as carbon, nitrogen, fluorine, and sodium, only the nuclei of phosphorus (^{31}P) and hydrogen (^1H) exist *in vivo* in sufficient concentrations for routine clinical evaluation [52]. Studies employing ^{31}P -MRS have indicated possible abnormalities in membrane high-energy phosphate metabolism, phospholipid metabolism, and intracellular pH in mood disorders [32].

In ^{31}P -MRS spectra of the brain, seven chemical peaks are resolved; these are phosphomonoester (PME), inorganic phosphate (Pi), phosphodiester (PDE), phosphocreatine (PCr), and alpha-(α), beta-(β), and gamma-(γ) nucleoside triphosphate (NTP) [32, 53, 54]. Figure 2 displays an example of the spectra that is acquired.

The β -NTP peak is measured as a proxy for ATP, the principal energy source in brain. The phosphomonoester (PME) peak contains the signals from numerous metabolites, including those related to membrane phospholipid synthesis such as phosphocholine (PC) or phosphoethanolamine (PE) [55] and sugar phosphates such as glycerophosphate or inositol phosphates [56]. In the PME region, PE is the most abundant and PC is the second most abundant metabolite [57]. The membrane breakdown products glycerophosphocholine and glycerophosphoethanolamine contribute to the PDE peak [58], but most of the signal in the *in vivo* PDE peak arises from membrane phospholipid itself [59, 60], marking PDE as a marker of neuronal integrity. The Pi peak appears between the PME and PDE peaks. Pi appears in many metabolic pathways. Although the Pi peak contains both PO_4^{-1} and PO_4^{-2} , they register as one peak due to the rapid exchange between these two molecules. The position of this peak reflects the equilibrium between PO_4^{-1} and PO_4^{-2} , a fact that allows investigators to calculate brain pH from the chemical shift of the Pi peak [61]. Because the phosphate ions exist in the intracellular space, this calculated pH reflects intracellular pH (pHi). The PCr peak is the most prominent peak in the ^{31}P -MRS spectra of the brain [32]. PCr conveys high-energy phosphates from the mitochondria to the cytosol. When an ATP molecule is consumed, PCr transfers its high-energy phosphate group to ADP (adenosine diphosphate), thus replenishing ATP via the creatine kinase reaction. In this regard, PCr behaves

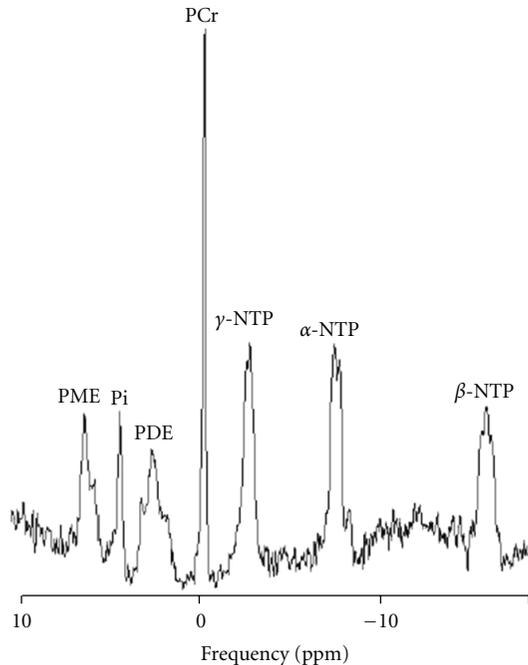


FIGURE 2: Phosphorus (^{31}P) magnetic resonance spectrum of the whole brain at 3 Tesla (TR = 3000 ms, TE = 2.3 ms). PME = phosphomonoester; Pi = inorganic phosphate; PDE = phosphodiester; PCr = phosphocreatine; NTP = Nucleoside Triphosphate; ppm = parts per million.

as a buffer of ATP [62]. PCr is abundant in tissues with rapidly variable energy demands, that is, brain and muscle tissue. This ATP buffer is absent in tissues where energy demands are constant, such as liver tissue. NTP forms three distinct peaks—alpha- (α) , beta- (β) , and gamma- (γ) nucleoside triphosphate—of which the doublet of the γ -ATP peak is resolved in ^{31}P -MR spectra [32]. ATP is the bioenergetic substrate for many biochemical processes in the brain and is present at a much higher concentration—on the order of 1.8 mM—than any other NTP [63].

For a number of practical reasons, proton spectroscopy is the most widely employed MRS method in psychiatric research. Protons are abundant in organic structures, and their nuclei have high magnetic sensitivity. In 1995, ^1H -MRS became widely available when the U.S. Food and Drug Administration (FDA) approved the software for an automated and inexpensive MRS sequence protocol, the PROton Brain Examination (PROBE) [31, 64], which can be run without dedicated research personnel on a standard MRI scanner [31]. The majority of ^1H -MRS studies have been conducted using MRI scanners operating at magnetic field strengths of 1.5 Tesla or less, which is less than optimal for ^{31}P -MRS. However, 3 Tesla MRI was approved by the FDA in 2000 [65] and is becoming widely accessible to major medical centers. The increased availability of scanners with 3 or 4 Tesla magnetic fields will improve the sensitivity of MRS studies [66]. At present, to conduct MRS brain scans that target other nuclei of interest to psychiatrists—such as phosphorus or lithium—requires specialized equipment (though not a separate MRI machine!) and research

expertise. These obstacles will not be insurmountable, if ^{31}P -MRS, in particular, proves to be a valid and reliable measure of one or more translational biomarkers in the affective disorders. To date, unlike physicians in other fields of medicine, psychiatrists do not benefit from working with objective measures of illness and recovery, such as blood pressure or hemoglobin A1c. In the competition to define the first validated biomarker for use in clinical psychiatry, MRS investigators have joined the pursuit along with their colleagues in genetics, neuroscience, and other branches of neuroimaging.

In recent years, multiple studies have reported regional and global hypometabolism in subjects experiencing a major depressive episode, which could be related to the pathophysiology of mood disorders [67]. The literature describes several abnormalities of bioenergetic metabolism in adults, primarily decreased baseline levels of β -nucleoside triphosphate and total NTP, in the basal ganglia and the frontal lobes of MDD subjects compared with healthy control subjects [63, 68, 69]. Our group recently reported baseline PCr levels could be a predictor of depression treatment outcomes [67]. Thus, ^{31}P -MRS provides investigators with a robust methodology [70], with which it is now possible to test specific hypotheses regarding the neurobiology of mood disorders in adults. MDD is a common and disabling illness that often begins in adolescence [23, 71–73]. Given the well-established safety of MRI, further study of pediatric mood disorders utilizing ^{31}P -MRS represents a rational endeavor in an attempt to expand the evidence base in medicine.

3. Materials and Methods

3.1. Search Strategy. A literature search using the U.S. National Library of Medicine's PubMed database was conducted to identify peer-reviewed neuroimaging MRS research studies of children and adolescents with MDD that were published between January 1966 to March 2010. The following terms were included in the search: “magnetic resonance spectroscopy,” “depressive disorder,” “mood disorder or affective disorder,” and “child or adolescent or pediatric or early-onset.” We performed a backward search of bibliographic references from the identified articles to ensure the inclusion of relevant articles. A forward citation search for identified studies was also performed. Studies that recruited a mixture of adults and children were not included. All relevant articles published in English were included, and due to the small number of studies no methodological exclusion criteria were applied.

4. Results and Discussion

The literature search yielded 18 articles reporting original MRS data in pediatric MDD: 13 studies in MDD and 5 in children and adolescents with mood disorders not meeting full diagnostic criteria for MDD. Table 1 presents the published MRS studies in pediatric MDD, all of which employed ^1H -MRS.

TABLE 1: Magnetic resonance spectroscopy studies of pediatric major depressive disorder.

Study	Sample	Field strength	Voxel size & location	Findings
Caetano, et al. 2005 [83]	-14 MDD (mean age 13.6)-22 Healthy Controls (mean age 13.3)	1.5 Tesla	8cc in the Left Dorsolateral Prefrontal Cortex	↓ glycerophosphocholine + phosphocholine (GPC + PC; or choline-containing compounds) in MDD patients. ↑ Inositol and myo-Inositol levels in MDD patients. In MDD patients Glutamate level is inversely correlated with Duration of Illness as well as Number of Episodes
Farchione et al. 2002 [76]	-11 treatment-naive MDD patients (mean age 14.3)-11 healthy controls (mean age 14.3)	1.5 Tesla	0.8 cc in the Left and Right Dorsolateral Prefrontal Cortex	↑ Cho was observed in left (but not right) DLPFC in MDD patients versus healthy controls (32.5% higher). No difference in NAA or Cr were observed between case-control pairs
Gabbay et al. 2009 [89]	-7 MDD patients (mean age 16.2) with Melancholic Features-7 patients (mean age 16.9) with non-melancholic, reactive MDD-6 healthy comparison patients (mean age 16.2)	3 Tesla	10 cm anterior-posterior (AP) × 7 cm left-right (LR) × 6 cm inferior-superior (IS) = 420 cm ³ in the Left Putamen and Right Caudate	No significant correlations were found in the healthy control group or in the MDD group as a whole. In Melancholic patients, plasma Kynurenine concentrations were positively correlated with Right Caudate total Choline. In Melancholic patients, plasma 3-hydroxyanthranilic acid (3-HAA), a neurotoxic intermediate of the Kynurenine Pathway) was positively correlated with Left Putamen total Choline
Gabbay et al. 2007 [86]	-14 adolescents (mean age 16.2 years) who had symptoms of MDD for 8 weeks and a score ≥40 (mean=63.6) on the Children's Depression Rating Scale—Revised-10 healthy comparison patients	3 Tesla	16 (anterior-posterior) × 16 (left-right) voxels, each anominal 0.75 cm ³ in the left and right caudate, putamen and thalamus	Adolescents with MDD had significantly ↑ concentrations of choline (2.11 mM versus 1.56 mM) and creatine (6.65 mM versus 5.26 mM) in the left caudate. No other neurochemical differences were observed between groups
Kusumakar, et al. 2001 [75]	-11 MDD (mean age 16.7)-11 Healthy Controls (mean age 16.6)	1.5 Tesla	Multivoxel: 6x6 placed in anterior medial temporal region (amygdala)	↓ Left amygdala Cho-Cr ratios in MDD patients compared with controls. Left amygdala NAA-Cr and right amygdala Cho-Cr and NAA-Cr did not differ significantly between patients with depression and control patients
MacMaster et al. 2008 [87]	11 MDD, 11 Healthy Controls	1.5 Tesla	0.8 mL voxel in the left and right Medial Temporal Cortex	↓ N-acetyl-aspartate in the left medial temporal cortex (27%) in MDD patients versus healthy controls
MacMaster and Kusumakar, 2006 [84]	-12 MDD-11 Healthy Controls (10-18 y/o; 7 females and 5 males per group)	1.5 Tesla	4cc in the right prefrontal cortex	↑ Right prefrontal cortex Choline/Creatine ratio in MDD compared with healthy controls ($p = .007$)
Mirza et al. 2004 [80]	-13 psychotropic-naïve with MDD (mean age 15.5)-13 healthy controls (mean age 15.4)	1.5 Tesla	2 × 1.5 × 1 cm = 3 cc volume centered on the anterior cingulate cortex	Anterior cingulate glutamatergic (Glx) concentrations were significantly ↓ (19% decrease) in MDD patients versus controls (9.27 +/- 0.43 versus 11.47 +/- 0.26, respectively, $p = .000$)

TABLE 1: Continued.

Study	Sample	Field strength	Voxel size & location	Findings
Mirza et al. 2006 [85]	18 pediatric patients with major depressive disorder 9 to 17 years of age, 18 case-matched healthy controls, and 27 patients with obsessive-compulsive disorder 7 to 16 years old	1.5 Tesla	0.8 mL voxel in the medial thalamus	↑ left and right medial thalamic creatine-phosphocreatine concentrations in patients with OCD compared with both healthy controls and patients with MDD. Creatine-phosphocreatine concentrations did not differ significantly between patients with MDD and healthy controls
Rosenberg et al. 2005 [82]	14 MDD patients (mean age 15.6) 14 healthy controls (mean age 15.5)	1.5 Tesla	2 × 1.5 × 1 cm = 3cc centered on the Anterior Cingulate Cortex	↓ anterior cingulate glutamate in MDD patients compared with controls ($p = .0002$; 23% decrease) No difference in anterior cingulate glutamine
Rosenberg, et al. 2004 [81]	-14 psychotropic-naïve patients with MDD without OCD (mean age 15.6)-20 non-depressed, patients with OCD (mean age 11.4)-14 healthy controls (mean age 15.5)	1.5 Tesla	2 × 1.5 × 1 cm = 3mL in the Anterior Cingulate Cortex	↓ Anterior cingulate glutamatergic (Glx) concentrations were significantly in OCD patients (15.1% decrease) and MDD patients (18.7% decrease) compared with controls ($p = .002$)
Smith et al. 2003 [77]	-18 drug naïve outpatients with MDD without OCD (mean age 14.4)-27 drug-naïve patients with OCD (mean age 10.3)-18 healthy controls (mean age 14.4)	1.5 Tesla	0.8 mL voxel in the left and right medial and lateral thalamus	↑ left and right medial thalamic Choline concentrations in OCD patients compared with both healthy controls and patients with MDD
Steingard, et al. 2000 [74]	-17 MDD patients (mean age 15.8)-28 healthy controls (mean age 14.5)	1.5 Tesla	15 mm x 15 mm x 15 mm (3.38cm ³) voxel in the Left Orbitofrontal Cortex	↑ Choline/Creatine ratio in MDD compared with controls ↑ Choline/NAA ratio in MDD compared with controls

Following up on their report of structural abnormalities in the brains of children hospitalized for depression, Steingard and colleagues reported in 2000 finding increased choline/creatine ratios and increased choline/NAA ratios in the left orbitofrontal cortex of adolescents with MDD compared with controls [74]. This suggested that brain cytosolic choline may be increased in depressed adolescents, independent of corresponding structural changes, results that were consistent with studies in adults with MDD. This report was followed in 2001 by Kusumaker's finding of decreased choline/creatine ratios in the left amygdala [75], which again implicated choline in the pathophysiology of pediatric MDD. In 2002, Farchione et al. studied the left and right dorsolateral prefrontal cortex in medication-naïve adolescents with MDD and healthy controls. A significant increase in choline was observed in left—but not right—dorsolateral prefrontal cortex in MDD patients versus controls (32.5% higher) [76].

In a three-armed ¹H-MRS study, Smith et al. compared medication-naïve MDD patients with Obsessive-Compulsive Disorder (OCD) patients and healthy controls. Following up on a prior finding of choline alterations in OCD, the investigators studied the thalamus and found increased choline concentrations bilaterally in the medial thalamus in pediatric OCD patients compared with both MDD patients and controls; they found no difference in medial thalamic choline between MDD patients and control subjects [77]. The potential of MRS in pediatric mood disorders was confirmed by these initial studies in pediatric MDD, which joined studies in adult MDD and preclinical animal research in implicating choline alterations in the disorder [78, 79].

Two pediatric MDD ¹H-MRS studies were published in 2004. Mirza et al. compared medication-naïve patients with healthy controls and found a 19% decrease in glutamatergic (Glx) concentrations in the anterior cingulate

cortex in the MDD patients [80]. A second study of the anterior cingulate cortex found lower Glx concentrations in both MDD patients (18.7% decrease) and OCD patients (15.1% decrease) compared with healthy controls [81]. In the following year, two additional studies were published. Rosenberg et al. separated the Glutamate and Glutamine peaks and found 23% lower Glutamate concentrations in the anterior cingulate cortex of MDD patients compared with controls ($P = .0002$) [82]. Caetano and colleagues conducted a case-control study of the left dorsolateral prefrontal cortex and reported decreased choline-containing compounds and increased myoinositol concentrations in patients with MDD [83]. MacMaster and Kusumakar reported results of their ^1H -MRS study of the right prefrontal cortex in 2006, finding that choline/creatinine ratio was elevated in MDD patients compared with healthy controls [84]. That same year, Mirza et al. published a study comparing patients with OCD with MDD and control subjects. The OCD patients demonstrated increased choline/Phosphocreatine concentrations in the left and right medial thalamus [85].

In recent years, investigators have expanded the range of structures and metabolites under study. Gabbay et al. reported in 2007 that adolescents with MDD had increased concentrations of both choline and creatine in the left (but not the right) caudate nucleus [86]. In 2008, MacMaster et al. were the first to report alterations in NAA in pediatric MDD, finding a 27% decrease in NAA in the left medial temporal cortex in affected subjects versus healthy controls [87]. Finally, Gabbay and colleagues obtained proton spectra with a 3 Tesla scanner and produced findings that begin to parse the subtypes of pediatric mood disorders on a neurobiological level by focusing on immune system dysregulation in pediatric MDD [88]. The investigators reported that in patients with melancholic features, plasma Kynurenine levels were positively correlated with choline concentrations in the right caudate nucleus and plasma 3-hydroxyanthranilic acid, a neurotoxic intermediate of the Kynurenine Pathway, was positively correlated with left putamen total choline [89].

Taken together, the consistency of findings in pediatric MDD validates the utility of MRS as a translational tool for studying pediatric MDD and adds to the converging lines of evidence suggesting that pediatric MDD is continuous with adult MDD [90].

5. Conclusions

A decade ago, Hendren et al. reviewed the neuroimaging literature in child and adolescent psychiatry and called for researchers to “identify clear structure/function hypotheses when studying childhood mental disorders that use but go beyond DSM diagnoses [91].” Echoing this sentiment, the research agenda for DSM-V emphasizes the need to translate findings from clinical neuroscience research into a new classification system based upon pathophysiology and etiological processes [92, 93]. As it continues to mature as a method, MRS is positioned to make a contribution to this evolution in psychiatry. *In vivo* MRS is the only noninvasive imaging technique capable of directly assessing the living biochemistry in

localized brain regions [94]. Studies of depressed children and adolescents have a number of advantages compared with studies of adults: the effects of statistical covariates such as repeated episodes, duration of illness, multiple medications, and normal aging are avoided. Thus, MRS may be a translational research tool capable of partially obviating the developmental and environmental confounders that have made research in child psychiatry a difficult challenge.

Researchers working with pediatric MDD populations have reported MRS findings that implicate Glx, NAA, and choline (including its correlation with immune system metabolites) in the neurobiology of MDD. Altered levels of choline would be consistent with altered neural plasticity as well as animal models of depression and antidepressant response [95]. No study identified in our literature search used repeated measures MRS scans to ascertain if there are changes in neurometabolite concentrations when a child with MDD responds to treatment, that is, whether the baseline differences in Glx, NAA, and choline are differences of “state” or “trait”. Pavuluri and Sweeney have argued for the importance of obtaining functional neuroimaging measures before and after pharmacologic intervention [9], and the feasibility of this approach is demonstrated in several published studies of depressed adults [67, 96, 97]. Results from such studies will shed light on the mechanisms by which antidepressants work and may provide new treatment targets for drug development [13, 98]. In addition to pointing toward the *mechanisms* of illness recovery (i.e., the “mediators” [99]), Pavuluri also emphasizes the potential for neuroimaging studies to identify *predictors* of treatment outcome [9] (i.e., the “moderators” [99]). Identification of the mediators and moderators of pediatric MDD treatment would serve the NIMH’s stated goal of moving toward personalized care.

Research in mood-disordered adults [67, 68, 100–103], combined with preclinical animal studies of depression [104–107], has given rise to the bioenergetic hypothesis, which proposes that altered energy metabolism is a reversible correlate of pathogenesis of mood disorders [108–110]. Converging lines of evidence from electron microscopy, gene expression, genotyping, and sequencing studies implicate mitochondrial dysfunction in MDD [111]. Neuroimaging studies using MRS have convincingly demonstrated that MDD is associated with mitochondrial dysfunction [73, 77–79, 112]. More specifically, a growing body of research shows that MDD subjects have decreased beta nucleoside triphosphate (b-NTP) and increased levels of phosphocreatine (PCr) compared with healthy controls [73, 78, 79]. Furthermore, successful treatment of MDD with antidepressants is associated with normalization in both NTP and PCr levels [79, 112].

Contemporary understanding of the neurobiology of depression is focused on imbalances in neural circuits [12], cellular plasticity and resilience [113, 114], and impaired neurotrophic signaling cascades [115] (for an excellent review, see Carlson et al. [116]; for the conceptual framework, see and Manji et al. [117] and Duman et al. [118]). As a research tool, MRS is unique in its ability to perform *in vivo* quantification of the neurometabolite indicators of neuronal

integrity, mitochondrial functioning, cellular membrane turnover, and signaling cascades [66]. Finally, MRS is the only known method for *in vivo* measurement of gamma-aminobutyric acid (GABA) concentration [41, 119], which plays an increasingly central role in our conceptualization of mood disorders [120–123]. As the field matures, it has the potential to play a major role in delineating the neurobiology of MDD.

5.1. What Are the Limitations of MRS? While MRS studies have the potential to provide unique insights into the neurobiology of pediatric MDD, technical limitations must be acknowledged. As with all neuroimaging modalities, the number of patients enrolled in a given study tends to be small. Comparison between studies (and therefore replication of key findings) is difficult, due to differences in spectra acquisition protocols and the fact that calculation of the area under a given spectral peak is open to considerable interpretation [36]; the optimal method for quantification of neurochemicals has yet to be determined. In addition, the generalizability of MRS studies is limited by potential confounds such as medication effects, duration of illness, comorbidity, and gender [124]. At present, MRS has less temporal and spatial resolution compared with MRI and functional MRI (fMRI) [125]. The volume of the brain—called the “voxel”—that is sampled in a typical ¹H-MRS study is ≤ 8 mL (larger for ³¹P-MRS scans). This is problematic in the light of current models of depressive disorders, which suggest the presence of altered interactions between hierarchically distributed neural networks which are widely distributed throughout the brain [126]. Another issue is the relatively small number of metabolites that can be assayed using MRS. “Key players” such as dopamine, serotonin, and norepinephrine are not visible to an MRS scanner [38]. Finally, scanning times are relatively long (up to 90 minutes), which increases the burden on research participants and limits patient acceptance.

5.2. What Would a Child Psychiatrist Want to Know about MRS? With the current emphasis on translational research in medicine, the question of science’s relevance to practicing clinicians is ever-present. Busy child and adolescent psychiatrists are faced on a daily basis with questions that further study utilizing MRS has the potential to answer.

- (i) *How can we distinguish bipolar disorder (BD) from MDD?* Chang et al. posed the question of whether neuroimaging will be used to diagnose BD in a 2006 publication, concluding that use of MRS variables such as mI or NAA for diagnosis is problematic at present, due to the variable of mood state and the lack of standardized methods for performing and interpreting the scans [112]. If children with both BD and MDD were followed and scanned longitudinally, MRS would be able to test the hypothesis that the major depressive episodes experienced by both groups of patients have the same neurochemical basis.

- (ii) *What is a problem of state versus a problem of trait?* The ability of MRS to measure neurochemical changes that parallel changes in patients’ clinical presentation is now well established in children and adults. In addition, further study of children and adolescents would help determine whether differences in depressed patients are the result of altered development across the life span, or if they can be documented early in development, serving as a risk factor for which prevention strategies might be employed [74].
- (iii) *Can MRS help clinicians assess the effects of medication?* Our literature search did not find any publications utilizing MRS to measure changes in the brain in pediatric MDD treatment studies.
- (iv) *What is the specificity of MRS in mood disorders?* The results thus far suggest that MRS can find neurochemical differences between mood disorders and OCD [77], Intermittent Explosive Disorder [127], and Attention-deficit Hyperactivity Disorder (ADHD) [128].

An illustrative example of the potential for *specificity* offered by MRS is the case of Glx and ADHD. Investigators have found elevated Glx in brain regions of interest in ADHD patients compared to healthy controls. Duman et al. [118] found increased Glx in the right prefrontal cortex and striatum of ADHD subjects, Courvoisier [119] documented increased Glx in the left and right frontal lobes, and Klempan et al. [120] found elevated Glx concentrations in the striatum treatment-naïve ADHD patients. In addition, Carey et al. have shown that changes in glutaminergic tone occur with ADHD treatment [129, 130]. In contrast to the findings in ADHD, Rosenberg [81] and Mirza [80] have shown that Glx is reduced in MDD, and Moore et al. [128] found that pediatric Bipolar Disorder with and without ADHD is differentiated by Glx concentrations in the anterior cingulate cortex.

5.3. What Should Future MRS Studies in Pediatric Mood Disorders Look Like? Some twenty years ago, Bottomley enumerated the “trouble with spectroscopy papers” [131], lamenting the fact that a research tool with such precision had given rise to a medical literature whose findings are not easily reconciled. Noting the lack of standards for the conduct and reporting of clinical MRS research, Bottomley recommended that authors provide objective, rigorously quantified results and that spectra acquisition protocols be described in sufficient details that experiments could be reproduced [131]. More recently, Kreis [132] and Taylor [133] have argued cogently for the field of clinical MR spectroscopy to establish a set of scientific and quality assurance guidelines. This process is underway in oncological neuroradiology [134–136], but widely accepted standards have yet to be established in psychiatry. As shown in Table 1, the MRS literature in pediatric MDD presents the reader with a diversity of techniques, anatomical reference points, and neurochemical findings. Future studies would benefit

from standardized protocol design and data reporting: as Leibenluft [13] has observed, psychiatric neuroimaging articles can be “confusing, tedious to read and . . . boring”—even for researchers in the field. For MRS studies of mood disorders to become a truly *translational* body of research, investigators will need to communicate their findings to the wider psychiatric community in a clear and intuitive manner; establishment of an expert consensus regarding best practices in MRS research and reporting would be a significant early step toward that goal.

Two important gaps in the medical literature were identified by the PubMed search on which this paper is based. The first is that there were no peer-reviewed publications reporting ^{31}P -MRS data in pediatric MDD. ^{31}P -MRS has the unique ability to measure high-energy phosphorus metabolites *in vivo* [63, 68, 69, 137], which is important because dysfunction in neuronal energy metabolism may be one mechanism of depression [109]. Second, no pediatric MDD clinical trials were identified in which MRS brain scans were used as a repeated measure, that is, performed pre- and posttreatment in order to learn which, if any, MRS metabolites were altered by the study treatment intervention. In contrast to their absence in the pediatric MDD literature, repeated measures MRS scans have often been incorporated into treatment studies of pediatric Bipolar Disorder [138–141] and related conditions [142, 143]. Incorporating ^{31}P -MRS methodology and repeated measures study design into pediatric MDD clinical trials could provide fundamental insights into the neurobiology of MDD.

In conclusion, MRS is an emerging translational research tool for the study of pediatric MDD. The bulk of published MRS depression studies have been done in adult populations, but mood disorders often begin in adolescence and there is much to be learned by studying young people who are early in the course of the disorder. The non-invasive nature and relative safety of MRS make this possible. Well-designed future MRS studies in pediatric MDD will shed light on the neurobiology of depression, help to define the physiologic mechanisms illness and recovery, and identify treatment targets for the development of new interventions.

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Research Article

Adverse Life Events and Depressive Symptoms in African American Youth: The Role of Control-Related Beliefs

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The association between experiences of adverse life events and adolescent depressive symptoms has been well documented. However, this association is not consistently observed in urban and low income African American youth. In addition, mechanisms linking life event stress and African American adolescents' depressive symptoms have received little attention. This study examined past year violent and nonviolent life events assessed in 6th grade as predictors of 7th grade depressive symptoms among a community epidemiologically defined sample of 447 (47% girls) urban African American adolescents. Depressive symptoms were assessed twice, at a 1-year interval, and initial depressive symptoms were controlled in the analyses. Control-related beliefs were examined as mediators of the association between life events and depressive symptoms, and gender was examined as a moderator of the association between control-related beliefs and depressive symptoms. Associations among study variables were examined in a series of models, from general to more specific. A model in which nonviolent and violent life events were examined separately and control and contingency beliefs examined as one latent variable was the most informative about the etiology of depressive symptoms in a sample of urban, African American youth. Implications of the findings for preventive interventions and future research are discussed.

1. Adverse Life Events and Depressive Symptoms in African American Youth: The Role of Control-Related Beliefs

Adolescent depression is a serious mental health issue that has been linked with several serious consequences such as academic difficulties, interpersonal difficulties, substance use problems, and the development of comorbid symptoms and disorders including anxiety and disruptive behavior disorders [1]. Thus, there has been interest in understanding the factors associated with adolescent depression in order to inform prevention and treatment efforts. Etiological research has identified adverse life events as important factors in the development of adolescent depression (e.g., [2–5]). While this link between stress and depression has been well documented, life event stress has not consistently been linked with depressive symptoms for urban and low income youth despite their high rates of exposure to adverse life events [4, 6]. Moreover, it is not clear whether similar

mechanisms influence the development and maintenance of depression in response to experiences of adverse life events for these youth as compared to youth from other backgrounds [4]. This study examines control-related beliefs as a potential mechanism through which experiences of adverse life events may lead to the development of depressive symptoms in urban African American youth. Results from this research will help clarify questions about the etiology and maintenance of depressive symptoms in these youth and inform the design of culturally relevant interventions for African American youth.

1.1. Life Event Stress and Depressive Symptoms in Adolescents. Numerous studies have found that life event stress predicts increases in depressive symptoms, syndromes, and disorders in adolescents (e.g., [2–5, 7]). In fact, life event stress has been shown to be a stronger predictor of depressive syndromes and disorders than depressed mood [7]. Patton et al. [8] found that adolescents who experienced one negative life

event were at a fivefold increased risk of developing stable Major Depressive Disorder compared to controls. Those who experienced multiple negative events had an eightfold increase in risk of developing stable depressive disorder [8]. One process by which negative life events can lead to increases in youth depressive outcomes is that major life events can lead to other, more enduring, daily stressors.

Some of the available research with low-income, minority youth supports the link between adverse life events and depression (e.g., [4, 9]). For example, there is evidence that negative life events across a variety of domains, including economic, family, peer, discrimination, neighborhood/violence, and school domains, are positively associated with depressive symptoms in ethnically diverse urban youth in cross-sectional and prospective studies [4, 10]. On the other hand, other research suggests that urban African American youth residing in dangerous contexts are less likely to display their distress through internalizing behaviors than externalizing behaviors [11]; for example, they may express distress in ways that are less likely to be viewed as a sign of weakness (e.g., crying) [12]. Relatedly, the type of life events youth experience can impact whether or not they display depressive symptoms in response to adverse life event stress [11, 13]. For example, research suggests that violent life events are related to youth externalizing symptoms, while nonviolent life events are related to youth internalizing symptoms [14]. These differences in youth adjustment based on the type of life event experienced suggest that it is important to consider the type of life event experienced when examining the link between life events and depressive symptoms, and the distinction between violent and nonviolent life events appears to be important for understanding specificity in responses to stress.

1.2. Cognitive Theories Explaining Link between Life Event Stress and Depressive Symptoms. Cognitive theories of depression highlight the controllability of stressors to explain the stress-depression link. For example, learned helplessness theory [15] posits that individuals who experience life events perceived to be beyond their control may develop an expectation that future life events also will be uncontrollable, leading to a series of cognitive and motivational deficits resulting in depressed affect [15]. Similarly, the hopelessness theory of depression [16] proposes that some people characteristically infer that negative life events are caused by internal, stable, and global forces. Beck's cognitive theory of depression posits that some individuals have a negative cognitive schema, characterized by a negative view of themselves, the world, and the future, which is characteristic of depression and can be triggered by individuals' experiences of adverse life events [17]. The contingency-competence-control (C-C-C) model integrates these types of cognitions into one model [18, 19]. This model posits that perceived control, the perception that one has the ability to produce desired outcomes, is influenced by both contingency, perceptions about the degree to which an outcome is dependent on the behavior of people in general, and competence, perception of one's ability to produce the desired outcome [18, 20]. While perceived control and perceived competence are perceptions about

the self, perceived contingency involves perceptions about youth in general (e.g., other African American adolescents). According to the C-C-C model, perceived contingency and competence are expected to significantly predict, but not fully account for, perceived control, since other factors also may contribute to perceived control [18].

Studies based on the C-C-C model have produced mixed results about the role that perceived contingency and control play in depression development. In a cross-sectional study of ethnically diverse adolescents ages 8 to 17, Weisz et al. [18] found that perceived contingency and perceived competence were significantly associated with perceived control. Furthermore, perceived contingency and competence were associated with adolescent depression, but perceived control was not. In their prospective study of predominantly Caucasian adolescents ages 10–14, Muris et al. [20] found that perceived contingency and perceived competence predicted perceived control. However, unlike Weisz et al. [18] study, perceived competence and perceived control predicted depression, but perceived contingency did not. These studies show that perceived control is influenced by perceived contingency and perceived competence. However, due to the mixed findings regarding the direct relationship of perceived control and contingency on depression in these studies, it is unclear what common and specific roles control and contingency beliefs may play in adolescent depressive symptoms. For this reason, Han et al. [21] have noted that it is important to examine the specific relations of different types of control-related beliefs (e.g., control and contingency) with internalizing symptoms. Thus, the present study examines general and specific associations between control and contingency beliefs and depressive symptoms to better understand the role of these cognitions in urban, African American youth.

The few studies examining the applicability of cognitive theories of depression to low-SES, ethnically diverse youth suggest that control cognitions do affect depression development in these youth. Reinemann and Teeter Ellison [22] found that when youth experienced low levels of negative life events, those with a more internal locus of control reported lower levels of anhedonia than those with an external locus of control. However, for youth who experienced high levels of negative life events, an internal locus of control did not have these buffering effects; specifically, youth with elevated negative life events reported similar rates of anhedonia whether or not they had an internal locus of control. Thus, under conditions of high stress, an internal locus of control may be less able to buffer against depressive symptoms. In contrast, Cowen et al. [23] found that urban youth who displayed resilience in the face of highly stressful conditions reported significantly more use of internal locus of control than nonresilient youth. These studies suggest that an internal locus of control may be an important resource for youth exposed to adverse life events, but these protective effects may be limited for urban, minority youth living under highly stressful conditions.

In addition to mixed findings regarding the role of cognitions in depression development in minority youth, methodological problems limit our understanding of how control cognitions affect psychological adjustment in these

youth. Because studies have used different terminology for conceptually similar cognitions (e.g., internal and external locus of control; contingency, competence, and control beliefs), it is difficult to determine which types of control cognitions predict the development of youth depressive symptoms. While exceptions exist (e.g., [4, 22]), most studies have examined control-related cognitions with predominately Caucasian and middle class samples; thus, it is difficult to determine whether and how control-related beliefs play a role in the development of depression in low SES, ethnically diverse samples. For example, ethnic minority youth living in disadvantaged neighborhoods may perceive that others similar to themselves have little control over their environment given their knowledge of many uncontrollable events experienced by similar others (e.g., victimization by violence). If so, contingency beliefs may be particularly relevant for these youth. Finally, studies examining the role of control-related beliefs in depression development primarily have used cross-sectional designs, limiting our knowledge about whether these cognitions lead to changes in depressive symptoms.

1.3. Present Study. It is recognized that adverse life events can lead to low perceptions of control and contingency. This may be particularly true for youth living in disadvantaged neighborhoods, who often experience life events beyond their control (e.g., neighborhood violence) and may come to believe that their behaviors do not necessarily yield desired life outcomes; these lowered control beliefs may be linked to depressive symptoms. Thus, the goal of the current study was to examine whether control-related beliefs mediate the relationship between adverse life events and depressive symptoms in a sample of urban, low-SES, African American adolescents. Because prior research has found that nonviolent life events are more strongly associated with youth internalizing symptoms than violent life events [14], nonviolent and violent life events were examined separately. Prior research has shown that gender differences in cognitive styles, such as increased use of rumination in girls, help to explain the gender difference in adolescent depression [24–26]. Similarly, gender differences in control-related beliefs may help explain gender differences in adolescent depressive symptoms if these beliefs are more strongly linked with depressive symptoms for girls than boys. To test this possibility, gender was examined as a moderator of the association between control-related beliefs and depressive symptoms.

Three models were tested, moving from a general model to more specific models, in order to understand whether information about the type of life event experienced (i.e., violent or nonviolent) or the type of cognition (i.e., control or contingency) improved prediction of depressive symptoms. The first model, the *General Model*, examined whether adverse life events predicted depressive symptoms and whether control-related beliefs mediated this relationship. The second model, the *Separate Life Events Model*, examined violent and nonviolent life events separately to determine whether these types of life events predicted depressive symptoms differently; it was expected that nonviolent life

events would be more strongly associated with depressive symptoms than violent life events [14]. The third and most specific model, the *Separate Life Events and Separate Beliefs Model*, examined whether nonviolent and violent life events predicted depressive symptoms and whether control and/or contingency beliefs mediated these associations. Like Model 2, this model examined violent and nonviolent life events separately. In addition, this model examined control and contingency beliefs as separate mechanisms to help clarify the mixed findings for the C-C-C model [18, 20]; specifically, this model tests whether personal control has a stronger association with depressive symptoms than beliefs about group level control (contingency beliefs). By examining specific types of life events (i.e., violent and nonviolent) and mechanisms (i.e., control versus contingency beliefs) that have been deemed important in the development of depressive symptoms, this research informs our understanding of the etiology of depression in African American adolescents.

2. Method

2.1. Participants and Sampling Design. Participants were drawn from a larger study that evaluated two school-based preventive interventions targeting early learning and aggressive, disruptive behavior [27]. Three first grade classrooms in each of nine Baltimore City public elementary schools were randomly assigned to one of the intervention conditions or to a control condition. The interventions were provided over the first grade year. Of the 678 children who participated in the intervention in the Fall of 1993, 585 (86.3%) were African American. Of the 585 African American children who participated in grade 1, 76% ($N = 447$) completed face-to-face interviews in the 6th grade and 7th grade and reported about their experiences with adverse life events, control-related beliefs, and depressive symptoms. These 447 youth comprised the sample of interest and included 52.6% boys ($n = 235$) and 47.4% girls ($n = 212$). The majority of participants were from lower SES backgrounds, with 72% receiving free or reduced lunch ($n = 320$). At the sixth grade assessment, the mean age of participants was 11.77 ($SD = 0.35$) with a range of 10.63 to 13.12 years. There were no differences between the 447 participants included in this study and the 138 children from the original sample who did not provide data in 6th and 7th grade in terms of gender, percentage receiving free or reduced lunches, or intervention condition ($P_s > .05$). t -tests indicated no differences between the two groups' 1st grade depressive symptoms, anxiety symptoms, or aggressive behavior ($P_s > .05$). Nine participants who participated in the 6th grade assessment did not provide data in grade 7; there were no differences between these 9 participants and the 447 participants included in the present study in terms of participants' gender, receipt of free or reduced lunch, intervention condition, or age.

2.2. Procedure in Grades 6 and 7. Permission for participation was obtained through written informed consent by at least one guardian and assent by the participating youth. Each spring, a team of project interviewers conducted

standardized interviews with consented youth who provided assent in a private location within the school. Those youth who had dropped out of school or failed to attend were interviewed at a location of their choice. Face-to-face interviews also were conducted with youth within a 90-mile radius of Baltimore. For youth outside this radius, phone interviews were conducted. Participants reported about their experience of adverse life events, control and contingency beliefs, and depressive symptoms. The timing of measurement of these variables was selected in order to best inform a mediation model. Specifically, in 6th grade, participants reported about life events happening in the past year and their current control and contingency beliefs. Depressive symptoms were examined in 7th grade. The time elapsed between 6th and 7th grade measurement points was one year. The study procedures were approved by the Johns Hopkins University Institutional Review Board.

2.3. Measures

2.3.1. Demographics. Information regarding participant age, gender, and receipt of free or reduced lunch (as an indicator of socioeconomic status) was collected. Intervention status (i.e., participation in intervention or control condition in first grade) also was recorded.

2.3.2. Adverse Life Events. Experiences of adverse life events were assessed in 6th grade using a modified version of the Life Events Questionnaire Adolescent Version (LEQ-A; adapted from [28]), a self-report checklist of the occurrence of stressful life events within the last year. The LEQ-A was modified for this study in order to include a broader range of life events relevant to adolescence and family-related stressors by adding items from the Adolescent Perceived Events Scale (APES) [29] and the Adolescent-Family Inventory of Life Events and Changes (A-FILE) [30]. In addition, the LEQ-A was modified to allow a test of the “cost of caring” hypothesis; according to this hypothesis, women may be more likely than men to be distressed by another’s adversity (e.g., friends, family) in addition to their own, thus leading to higher rates of depression in women [31, 32]. For this reason, the LEQ-A was modified for this study in order to assess three sets of events including: (1) events experienced directly; (2) events experienced by family members; (3) events experienced by friends. Given prior research showing that family and peer life events are salient adolescent stressors that impact youth psychological adjustment, all three types of events (i.e., self, family, and peer events) were included in the study [9, 33]. This life events measure assesses discrete and identifiable life events such as witnessing or being a victim of neighborhood violence, parental job loss, and eviction from the home. The sum of all adverse life events endorsed (“yes” = 1; “no” = 0) was used as an indicator of individual stress levels for Model 1. The sum of nonviolent events and the sum of violent life events were used in the more specific models (i.e., Model 2 and Model 3). A sample item of a nonviolent life event is “Did one of your parents lose his or her job this past year?”; a sample item of a violent life event is “were you or a family member shot or stabbed during this past year?”

2.3.3. Depressive Symptoms. Depressive symptoms were assessed in 6th and 7th grades using the Baltimore How I Feel (BHIF; [27]), a 45-item self-report measure of depressive and anxious symptoms. Adolescents reported the frequency of these symptoms over the last two weeks on a 4-point scale (0 = Never; 3 = Most times). Item content for the depression subscale was designed based on the diagnostic and statistical manual of mental disorders, third edition, revised [34] criteria or drawn from other existing child self-report measures including the hopelessness scale for children [35], the depression self-Rating scale [36], and the children’s depression inventory [37]. The major types of depressive symptoms assessed include symptoms of depressed mood (e.g., “I felt very unhappy”), anhedonia (e.g., “I had a lot of fun”), and depressive cognitions (e.g., “I felt that it was my fault when bad things happened”). The internal consistency alpha for the depression subscale was .82 in 6th grade and .83 in 7th grade, indicating that this is a reliable measure. The BHIF depression subscale was significantly associated with a middle school diagnosis of major depressive disorder on the diagnostic interview schedule for children IV (DISC-IV), suggesting it is a valid measure of depressive symptoms [38].

For this study, a depressive symptom latent variable was created using three item parcels created from 15 items from the BHIF. Item parcels, or the mean of several items assumed to be conceptually similar, are often preferred over observed variables because they are more likely to meet assumptions of maximum likelihood estimation procedures that are used in SEM, provide more precise estimates of parameters because they reduce the complexity of measurement models, and simplify models by reducing the number of parameters [39]. The three depressive symptom item parcels were used as indicators of the depressive symptoms latent variable.

2.3.4. Perceived Control. Perceptions of control were assessed in 6th grade using a slightly modified version of the Multidimensional Measure of Children’s Perceptions of Control [40]. This modified version includes 24 self-report items assessing beliefs about causes of events, including beliefs about whether events are under one’s control or the control of external sources (e.g., “I can get really good grades if I try”; “I cannot stay out of trouble no matter how hard I try”; “If other kids are mean to me, I cannot make them stop.”). Modifications included the addition of a behavioral domain and exclusion of a global domain. Thus, this modified measure assesses beliefs about academic, behavioral, and social domains of perceived control. Youth responded using a 4-point Likert scale (0 = Not at all true; 3 = Very true). Internal consistency alphas for the perceived control measure ranged from .76 to .79 for 6th and 7th grades. For this study, the academic, behavioral, and social subscales were used as indicators of the perceived control latent variable.

2.3.5. Perceived Contingency. Perceived contingency was assessed in 6th grade using the perceived contingency scale for children [41], a 30-item self-report measure that focuses on perceived contingencies for people in general (e.g., “Kids can work hard in school and still get bad grades”; “Kids

get yelled at even if they behave”; “Some kids are well-liked and some aren’t; it doesn’t matter how hard they try.”). Academic, behavioral, and social domains were assessed, and youth responded using a 4-point Likert scale (0 = Not at all true; 3 = Very true). According to Weisz et al. [19] internal consistency alphas for the academic, behavioral, and social contingency subscales were .69, .75, and .74, respectively, and .86 for the full scale [19]. The academic, behavioral, and social contingency subscales were used as indicators of the perceived contingency latent variable.

3. Data Analytic Strategy

3.1. Structural Equation Modeling. Structural equation modeling (SEM) using *Mplus* 5.1 [42] was used to examine the hypothesized associations between study constructs and maximum likelihood estimates were obtained. Overall model fit was evaluated using multiple indicators including: Chi Square, the Comparative Fit Index (CFI), the Tucker Lewis Index (TLI), and the Root Mean Square Error of Approximation (RMSEA). According to [43], goodness of fit Chi-square ratio values (Chi-square to degrees of freedom ratio) less than 3 indicate good model fit. A relatively good fit between the hypothesized model and the observed data was indicated by CFI and TLI above .95 and RMSEA less than .06 [44]. For these analyses, study variables assessed the same year (i.e., adverse life events, perceived control, and perceived contingency) were allowed to correlate in order to account for bias resulting from shared method variance. Intervention status, lunch status, and prior depressive symptoms (6th grade) were controlled in all models by regressing 7th grade depressive symptoms on these variables.

Mediated effects were tested based on guidelines presented by Holmbeck [45], which are based on guidelines by Baron and Kenny [46]. These analyses included examination of the simple association between the predictor (adverse life events) and the outcome (depressive symptoms), controlling for 6th grade depressive symptoms, to test whether there was a significant association in the hypothesized direction. Paths from the predictor (adverse life events) to the mediator (control-related beliefs) and from the mediator (control-related beliefs) to the outcome (depressive symptoms) also were tested. Each path must be significant in the hypothesized direction for mediation to be present. As described in Baron and Kenny’s [46] guidelines, if the previously significant simple association between the predictor and the outcome becomes nonsignificant when the mediator is taken into account, there is support for mediation. To provide an additional test of mediation, the Sobel test [47] was used to test the significance of the indirect effect for each model.

Multiple group analysis was used to test gender differences in the association between control-related beliefs and depressive symptoms. For these analyses, the overall fit of the hypothesized model was tested under two conditions: (a) when the path between the control-related beliefs and depressive symptoms was constrained to be equal for boys and girls (i.e., constrained model) and (b) when there were no constraints on the path between control-related beliefs and depressive symptoms for boys and girls, and the

association could vary as a function of gender (i.e., freely estimated model). A significant improvement in model fit, indicated by a significant difference in Chi-square model fit between the free and constrained model, suggests that gender moderates the association between control-related beliefs and depressive symptoms.

4. Results

4.1. Descriptive Statistics. Means, standard deviations, and ranges for the total sample and separately by gender are presented in Table 1. Boys ($M = 2.52$) reported significantly more total experiences of adverse life events than girls ($M = 2.07$), $t = 2.25$, $P < .05$. Boys ($M = 1.67$) reported more experiences of violent life events than girls ($M = 1.37$); this difference was marginally significant ($t = 1.95$, $P = .052$). Boys ($M = 7.93$) and girls ($M = 7.94$) reported similar levels of experiences of nonviolent life events ($t = -0.025$, $P > .05$). Almost all boys (97.8%) and girls (99.2%) in the sample reported experiencing at least one nonviolent life event. More than half of boys (72.2%) and girls (58.2%) reported experiencing at least one violent life event. Boys ($M = 43.93$) and girls ($M = 44.29$) reported similar levels of control beliefs ($t = -0.56$, $P > .05$). Boys ($M = 37.46$) and girls ($M = 36.92$) also reported similar levels of contingency beliefs ($t = 0.81$, $P > .05$). Reports of 7th grade depressive symptoms were similar for boys ($M = 10.57$) and girls ($M = 11.54$) ($t = -1.39$, $P > .05$). Correlations among all study variables are presented in Table 2. Adverse life events were significantly positively correlated with 7th grade depressive symptoms for boys and girls. While life events were negatively correlated with control and contingency beliefs for girls, they only were significantly related to contingency beliefs for boys. Control and contingency beliefs were positively correlated for boys and girls. Both control and contingency beliefs were negatively correlated with 7th grade depressive symptoms for boys and girls.

4.1.1. Mediation Analyses. Three sets of mediation analyses were conducted: (1) the *General Model*, mediation of the association between adverse life events and 7th grade depressive symptoms by control-related beliefs (control and contingency together); (2) the *Separate Life Events Model*, mediation with violent and nonviolent life events examined separately; (3) the *Separate Life Events and Separate Beliefs Model*, mediation with violent and nonviolent life events examined separately, and control and contingency beliefs examined separately. These analyses were performed using latent variables representing the control-related beliefs and 7th grade depressive symptoms. The *General Model* used a life event latent variable while the *Separate Life Events Model* and *Separate Life Events and Separate Beliefs Model* used two observed variables, nonviolent life events and violent life events. The control-related beliefs latent variable used in the *General Model* and the *Separate Life Events Model* was created from three control beliefs subscales and three contingency beliefs subscales. The perceived control and perceived contingency latent variables used in the *Separate Life Events and Separate Beliefs Model* were created from

TABLE 1: Mean and standard deviations of study variables for total sample and by gender.

Variable	Total		Boys	Girls	<i>t</i> -test
	<i>M</i> (SD)	Range ^a	<i>M</i> (SD)	<i>M</i> (SD)	
Life events (6th)	2.30 (2.14)	0–14	2.52 (2.32)	2.07 (1.90)	2.25*
Control (6th)	44.11 (6.92)	18–54	43.93 (6.95)	44.29 (6.89)	–0.56
Contingency (6th)	37.20 (7.03)	13–54	37.46 (6.84)	36.92 (7.23)	0.81
Depressive symptoms (7th)	11.02 (7.66)	0–51	10.57 (6.82)	11.54 (8.50)	–1.39

Note. *N* = 456 for life events grade 6; *N* = 456 for control grade 6; *N* = 456 for contingency grade 6; *N* = 474 for depressive symptoms grade 7.

^aRefers to observed range.

**P* < .05.

TABLE 2: Correlations of study variables for boys and girls.

Variable	1	2	3	4	5	6	7	8
(1) Life events (6th)	—	–.20**	–.13	.42**	.25**	.01	–.05	.14*
(2) Control (6th)	–.19**	—	.49**	–.51**	–.45**	–.01	.08	.06
(3) Contingency (6th)	.21**	.56**	—	–.27**	–.26**	–.11	.04	.02
(4) Depressive Sx (6th)	.24**	–.44**	–.32**	—	.58**	–.01	–.15*	.03
(5) Depressive Sx (7th)	.17**	–.36**	–.31**	.55**	—	–.00	–.10	.16*
(6) Age (6th)	.01	–.11	–.02	–.03	.05	—	–.07	.10
(7) Intervention status	–.10	–.03	–.05	.03	.04	.01	—	.04
(8) Lunch status	.00	–.01	.02	.04	.10	.04	–.22**	—

Note. Correlations for girls are above the diagonal. Correlations for boys are below the diagonal. Sx refers to symptoms. *N* = 447 for life events grade 6, control grade 6, contingency grade 6, depressive symptoms grade 6, depressive symptoms grade 7, age grade 6, and intervention status; *N* = 444 for lunch status.

P* < .05. *P* < .01 (1-tailed).

their respective subscales. Fit indices for the saturated models (all paths included) are summarized in Table 3. Prior to conducting the mediation analyses, measurement models were examined to determine the adequacy of loadings of the indicators on the life events, control-related beliefs, perceived control, perceived contingency, and 7th grade depressive symptoms latent variables to be used in the SEM. All loadings of the indicators for the latent variables in all models were significant (*P*s < .001).

4.2. General Model. The association between life events and 7th grade depressive symptoms, controlling for 6th grade depressive symptoms, lunch status, and intervention status, was tested first and this model was a good fit to the data: ($\chi^2(13) = 24.43, P < .05; CFI = 0.98; TLI = 0.96; RMSEA = 0.04$). There was a marginally significant association between life events and 7th grade depressive symptoms ($\beta = 0.13, P = .068$). Although the association between life events and 7th grade depressive symptoms was only marginally significant, mediation was still possible; therefore, the full model was examined. The full model (Figure 1), with paths from adverse life events to 7th grade depressive symptoms, from adverse life events to control-related beliefs, from control-related beliefs to 7th grade depressive symptoms, and from prior depressive symptoms (6th grade) to 7th grade depressive symptoms ($\chi^2(68) = 248.77, P < .001; CFI = 0.86; TLI = 0.81; RMSEA = 0.08$) explained 35% of the variance in 7th grade depressive symptoms ($R^2 = 0.353, P < .001$). Intervention status ($\beta = -0.01$) and lunch status ($\beta = 0.09$) were not associated with 7th grade depressive symptoms (*P*s > .05). There was a significant,

positive association between 6th grade depressive symptoms and 7th grade depressive symptoms ($\beta = 0.44, P < .001$). The path from adverse life events to control-related beliefs ($\beta = -0.34, P < .001$) and the path from control-related beliefs to 7th grade depressive symptoms ($\beta = -0.26, P < .001$) were significant in the hypothesized direction. The association between adverse life events and 7th grade depressive symptoms was reduced when including control-related beliefs in the model ($\beta = 0.07, P = .316$), suggesting that control-related beliefs mediated the association between adverse life events and 7th grade depressive symptoms. The Sobel test [47] was used to test the adverse life events → control-related beliefs → 7th grade depressive symptoms indirect path. Results indicated that the adverse life events → control-related beliefs → 7th grade depressive symptoms indirect effect was significant ($z = 3.00, P < .01$).

To test whether gender moderated the association between control-related beliefs and 7th grade depressive symptoms, a model in which this path was freely estimated for boys and girls ($\chi^2 = 343.311$) was compared to a model in which this path was constrained to be equal for boys and girls ($\chi^2 = 344.995$). The freely estimated and constrained models were not significantly different ($\chi^2_{diff}(1) = 1.68, P > .05$), indicating that gender does not moderate the association between control-related beliefs and 7th grade depressive symptoms.

4.3. Separate Life Events Model. The associations between nonviolent and violent life events and 7th grade depressive symptoms, controlling for 6th grade depressive symptoms, lunch status, and intervention status, were examined first.

TABLE 3: Goodness-of-fit indices for mediation—saturated models (all paths included).

Model	χ^2/df	CFI	TLI	RMSEA
Saturated general model	3.66	0.86	0.81	0.077
Saturated separate life events mode	3.98	0.80	0.75	0.082
Saturated separate life events and separate beliefs model	2.40	0.92	0.88	0.056

Note. CFI: Comparative Fit Index; TLI: Tucker-Lewis Index; RMSEA: Root mean square error of approximation.

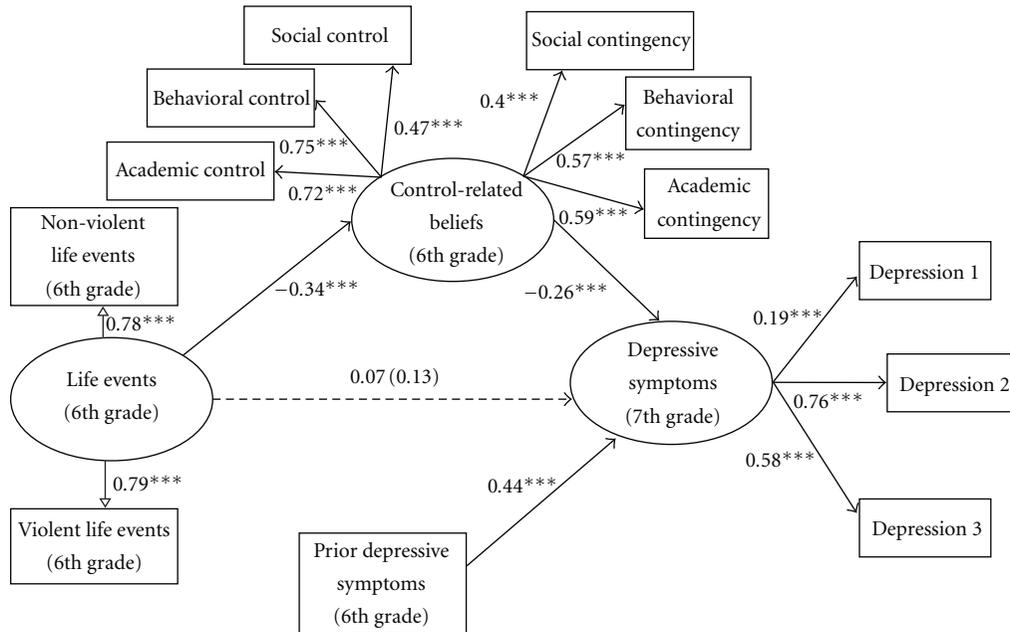


FIGURE 1: Mediation analyses for the *General Model*. The simple association between 6th grade life events and 7th grade depressive symptoms is in parentheses. This model explained 35% of the variance in 7th grade depressive symptoms ($R^2 = 0.353$, $P < .001$). *** $P < .001$.

The model fit indices for these simple associations indicated good model fit: $\chi^2(10) = 21.63$, $P < .05$; CFI = 0.95; TLI = 0.91; RMSEA = 0.05. There was a significant association between violent life events and 7th grade depressive symptoms ($\beta = 0.14$; $P < .05$), but no significant association between nonviolent life events and 7th grade depressive symptoms. The full model (Figure 2), with paths from nonviolent life events and violent life events to 7th grade depressive symptoms, from nonviolent and violent life events to control-related beliefs, from control-related beliefs to 7th grade depressive symptoms, and from 6th grade depressive symptoms to 7th grade depressive symptoms ($\chi^2(64) = 254.80$, $P < .001$; CFI = 0.80; TLI = 0.75; RMSEA = 0.08) explained 35% of the variance in 7th grade depressive symptoms ($R^2 = 0.346$, $P < .001$). Intervention status ($\beta = -0.01$) and lunch status ($\beta = 0.09$) were not significantly associated with 7th grade depressive symptoms ($P_s > .05$). The association between 6th grade depressive symptoms and 7th grade depressive symptoms was significant ($\beta = 0.46$, $P < .001$). The path from violent life events to control-related beliefs was significant in the hypothesized direction ($\beta = -0.17$, $P < .01$), but there was no significant association between nonviolent life events and control-related beliefs ($\beta = -0.11$, $P > .05$). The path from control-related

beliefs to 7th grade depressive symptoms was significant in the hypothesized direction ($\beta = -0.26$, $P < .001$). The association between nonviolent life events and 7th grade depressive symptoms remained nonsignificant. The previously significant simple association between violent life events and 7th grade depressive symptoms became nonsignificant when control-related beliefs were included in the model, suggesting mediation. To provide an additional test for mediation, the indirect path from violent life events \rightarrow control-related beliefs \rightarrow 7th grade depressive symptoms was tested using the Sobel test [47]. The Sobel test [47] indicated that the violent life events \rightarrow control-related beliefs \rightarrow 7th grade depressive symptoms indirect effect was significant ($z = 2.13$, $P < .05$).

To test whether gender moderated the association between control-related beliefs and 7th grade depressive symptoms, a model in which this path was freely estimated for boys and girls ($\chi^2 = 340.942$) was compared to a model in which this path was constrained to be equal for boys and girls ($\chi^2 = 342.227$). The freely estimated and constrained models were not significantly different ($\chi^2_{diff}(1) = 1.29$, $P > .05$), suggesting that gender does not moderate the association between control-related beliefs and 7th grade depressive symptoms.

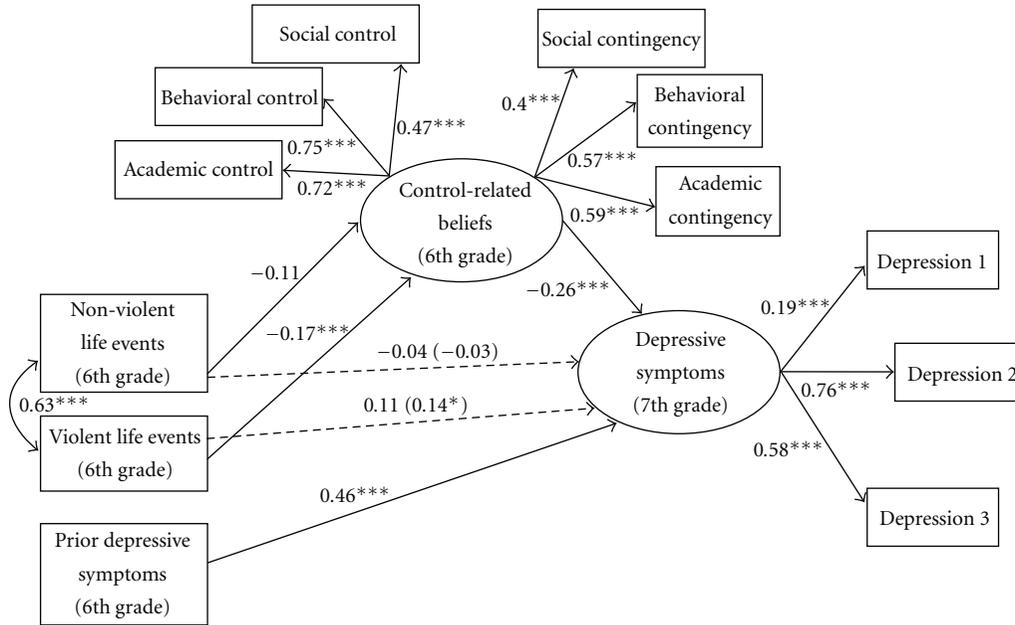


FIGURE 2: Mediation analyses for the *Separate Life Events Model*. The simple associations between 6th grade nonviolent and violent life events and 7th grade depressive symptoms are in parentheses. This model explained 35% of the variance in 7th grade depressive symptoms ($R^2 = 0.346$, $P < .001$). * $P < .05$; ** $P < .01$; *** $P < .001$.

4.4. *Mediation Analyses for the Separate Life Events and Separate Beliefs Model.* The associations between nonviolent and violent life events and 7th grade depressive symptoms, controlling for 6th grade depressive symptoms, lunch status and intervention status, were examined first. The model fit indices for these simple associations indicated good model fit: $\chi^2(10) = 21.63$, $P < .05$; CFI = 0.95; TLI = 0.91; RMSEA = 0.05. There was a significant simple association between violent life events and 7th grade depressive symptoms ($\beta = 0.14$, $P < .05$), but no significant simple association between nonviolent life events and 7th grade depressive symptoms ($\beta = -0.02$, $P > .05$). The full model (Figure 3), with paths from nonviolent life events and violent life events to 7th grade depressive symptoms, from nonviolent and violent life events to perceived control and perceived contingency beliefs, from perceived control and perceived contingency beliefs to 7th grade depressive symptoms, and from 6th grade depressive symptoms to 7th grade depressive symptoms ($\chi^2(58) = 139.21$, $P < .01$; CFI = 0.92; TLI = 0.88; RMSEA = 0.06) explained 40% of the variance in 7th grade depressive symptoms ($R^2 = 0.401$, $P < .001$). Intervention status ($\beta = -0.01$) and lunch status ($\beta = 0.09$) were not associated with 7th grade depressive symptoms (P s $> .05$). There was a significant association between 6th grade depressive symptoms and 7th grade depressive symptoms ($\beta = 0.39$, $P < .001$). The paths from nonviolent life events to perceived control beliefs ($\beta = -0.12$, $P > .05$) and from nonviolent life events to perceived contingency beliefs ($\beta = -0.06$, $P > .05$) were not significant. The association between nonviolent life events and 7th grade depressive symptoms remained nonsignificant ($\beta = -0.04$, $P > .05$). The paths from violent

life events to perceived control beliefs ($\beta = -0.14$, $P < .05$) and from perceived control beliefs to 7th grade depressive symptoms ($\beta = -0.33$, $P < .05$) were significant in the hypothesized directions. The path from violent life events to perceived contingency beliefs ($\beta = -0.20$, $P < .01$) also was significant in the hypothesized direction, but the path from perceived contingency beliefs to 7th grade depressive symptoms was not significant. In this model, the previously significant simple association between violent life events and 7th grade depressive symptoms was not significant. Thus, perceived control beliefs emerged as a possible mediator for the association between violent life events and 7th grade depressive symptoms. To provide an additional test for mediation, the Sobel test [47] was used. The Sobel test [47] indicated that the violent life events \rightarrow perceived control beliefs \rightarrow 7th grade depressive symptoms indirect effect was not significant ($z = 1.54$, $P > .05$).

To test whether gender moderated the associations between perceived control beliefs and 7th grade depressive symptoms, a model in which this path was freely estimated for boys and girls ($\chi^2 = 219.560$) was compared to a model in which this path was constrained to be equal for boys and girls ($\chi^2 = 220.063$). The model with the path from perceived control beliefs to 7th grade depressive symptoms constrained to be equal for boys and girls was not significantly different than the freely estimated model ($\chi^2_{diff}(1) = 0.50$, $P > .05$). To test whether gender moderated the associations between perceived contingency beliefs and 7th grade depressive symptoms, a model in which this path was freely estimated for boys and girls ($\chi^2 = 219.560$) was compared to a model in which this path was constrained to be equal for boys and girls ($\chi^2 = 219.565$). The model

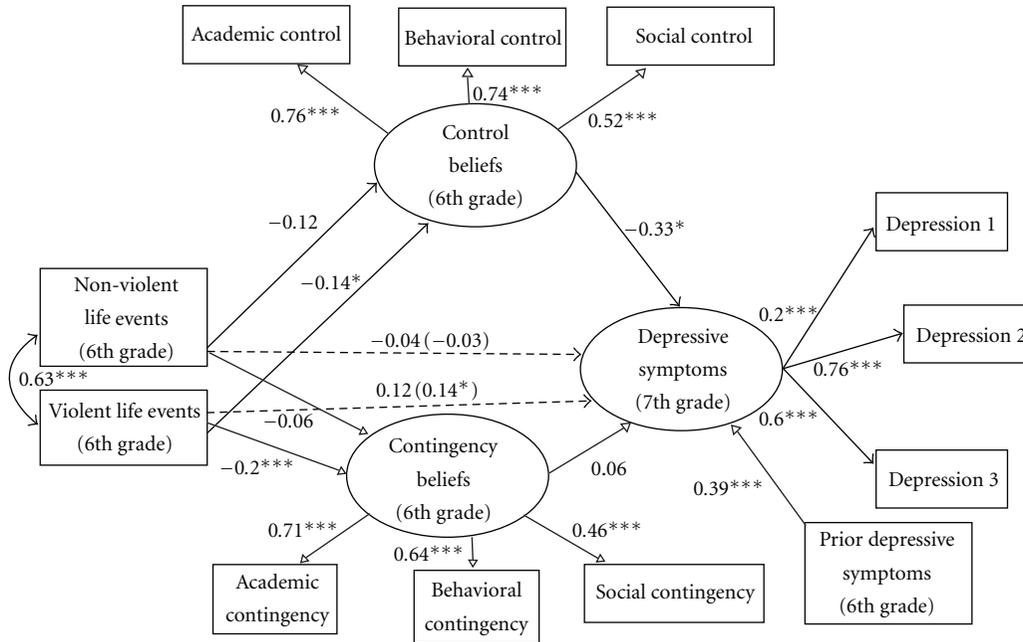


FIGURE 3: Mediation analyses for the *Separate Life Events and Separate Beliefs Model*. The simple associations between 6th grade nonviolent and violent life events and 7th grade depressive symptoms are in parentheses. This model explained 40% of the variance in 7th grade depressive symptoms ($R^2 = 0.401, P < .001$). * $P < .05$; ** $P < .01$; *** $P < .001$.

in which the path from perceived contingency beliefs to 7th grade depressive symptoms was constrained for boys and girls was not significantly different than the freely estimated model ($\chi^2_{diff}(1) = 0.005, P > .05$). These results suggest that gender does not moderate the association between perceived control beliefs and 7th grade depressive symptoms or the association between perceived contingency beliefs and 7th grade depressive symptoms.

5. Discussion

Research regarding the role of life events in depression development for urban African American adolescents has yielded mixed results [2–5], and little is known about the mechanisms that influence the development and maintenance of depression for urban African American youth [4]. The present study examined whether control-related beliefs mediate the association between adverse life events and depressive symptoms in a sample of urban, low-SES, African American adolescents, and whether gender moderated the association between control-related beliefs and depressive symptoms. Both general and specific relationships between adverse life events, control-related beliefs, and depressive symptoms were examined.

5.1. The General Model. Results from the *General Model* revealed that control-related beliefs mediated the association between adverse life events and depressive symptoms, and a significant adverse life events → control-related beliefs → depressive symptoms indirect effect. Although these results should be interpreted with caution because there only was a tendency for significance for the original simple association

between adverse life events and depressive symptoms, these results suggest that examining general control-related beliefs is a useful way to understand the role of control-related cognitions in the development of depressive symptoms in African American youth. Findings suggest that it may be appropriate for treatment interventions addressing youth depressive symptoms to target both types of cognitions together when working with urban, African American youth. However, in order to provide a clearer understanding of the role of life events and cognitions in youth depressive symptom development, more specific models were examined.

5.2. The Separate Life Events Model. Results from the *Separate Life Events Model* indicated that violent life events were significantly associated with depressive symptoms, but nonviolent life events were not. This is counter to some previous research indicating that experiences with violent life events are related to youth externalizing symptoms while nonviolent life events are related to internalizing symptoms in youth (e.g., [14]). These findings indicate that violent life events may be an appropriate screener for urban, African American youth at risk for developing depressive symptoms. Moreover, these findings highlight the importance of examining violent and nonviolent life events separately, in contrast to much prior research that has examined total life events experienced, without examining the differential impact of life events by type.

Violent life events, rather than nonviolent life events, also may have predicted depressive symptoms because it may be difficult for youth to find effective coping strategies to deal with violent life events as compared to nonviolent life events.

For example, for urban youth exposed to violence, strategies such as approach coping may be useful in benign situations but may be less adaptive in violent situations when these coping strategies might lead to negative outcomes, such as future violence exposure and greater distress [48, 49]. In a recent study, Brady et al. [50] found that among African American and Latino boys who had been exposed to community violence, those who had an array of coping strategies, such as modifying behavior without confronting others, had positive long-term adjustment, including less distress, compared to those who had limited coping strategies. For individuals exposed to violent life events then, it may be useful to have an array of coping strategies that they can call upon in order to protect them from negative outcomes. Relatedly, it may be beneficial for depression intervention efforts targeting urban youth to include components that teach youth various coping strategies that they can use in violent as well as nonviolent situations.

Findings showed that control-related beliefs are one mechanism through which violent life events impact youth depressive symptoms. Urban, African American youth living in dangerous settings, may be particularly vulnerable to feelings that they and others like them have little control over their environments because they may often experience stressors that they may in fact have little control over (e.g., community violence exposure), which in turn impacts their psychological adjustment. In fact, researchers have argued that because inner-city youth are often exposed to pervasive and uncontrollable stressors on a daily basis, they may be especially prone to believe that they have little control over events in their lives [4, 23]. Although this perception may in many instances be realistic, it likely has a negative impact on youth's future responses to adverse life events and can lead to increased depressive symptoms [4]. Results from the present study suggest that urban, African American youth may benefit from learning ways to identify which areas of their lives they in fact do have control over, as well as learning various coping strategies to manage situations in which they have little control (e.g., neighborhood violence).

5.3. The Separate Life Events and Separate Beliefs Model. Findings from the *Separate Life Events and Separate Beliefs Model* indicated that examining perceived control and perceived contingency beliefs separately was not a useful way to examine the role of cognitions in youth depression development. Specifically, while there was initial evidence that perceived control beliefs mediated the association between violent life events and depressive symptoms, a test of the indirect effect was not significant, suggesting no mediation. Thus, it seems that examining perceived control and perceived contingency beliefs together is a more useful approach to understanding the development of African American youth's depressive symptoms than examining these beliefs separately.

5.4. Which Model Is Most Informative? In these data, the *Separate Life Events Model* performed better than the *General Model* and the *Separate Life Events and Separate Beliefs Model*. First, by examining life events separately, this model

provided useful information regarding the varied utility of different types of life events (nonviolent versus violent) in predicting youth depressive symptoms (compared to the *General Model*). However, the most specific model, the *Separate Life Events and Separate Beliefs Model*, which separated control and contingency beliefs, did not provide additional information regarding the mechanisms involved in depression development. Therefore, the *Separate Life Events Model* is considered to be the superior model in this study.

5.5. Gender as a Moderator of the Association between Control-Related Beliefs and Depressive Symptoms. It was hypothesized that the association between control-related beliefs and depressive symptoms would be stronger for girls than boys as previous research has shown that gender differences in cognitive styles, such as increased use of rumination in girls, help to explain the gender difference in depression [24–26]. However, gender did not moderate the association between control-related beliefs and depressive symptoms in this sample. One explanation for this finding is that, unlike rumination, boys and girls may have a similar response to feeling that they have little control over desired outcomes. In other words, low-perceived control over one's environment and negative evaluations of one's abilities, capacities, and worth (i.e., control beliefs) may have similar relations to adolescent psychopathology for both boys and girls [21]. Future research should attempt to further our understanding of how control-related beliefs lead to depressive symptoms in boys and girls.

5.6. Implications. Findings suggest that prevention efforts targeting low-income urban youth should assess recent experiences of adverse life events as a means to screen individuals at risk for depression [2]. These interventions should pay special attention to individuals who have experienced violent life events since these types of events seem to play a crucial role in the development of depressive symptoms for urban youth. Findings also suggest that interventions should target control and contingency beliefs. Interventions that focus on individuals' maladaptive cognitions, such as cognitive behavioral therapy (CBT), a well-established treatment for adolescent depression [51], can be used to modify youth's control and contingency cognitions. Depression intervention and prevention efforts also should focus on helping adolescents prepare for and manage experiences of violent life events. Helping youth develop new and effective coping strategies for dealing with experiences with violence may be one way to provide them the tools they need to deal with these frequent stressors. However, more research is needed to understand which coping strategies may be "adaptive" for the contexts in which urban youth live [49]. For example, while studies have generally found that approach coping is linked to better psychological adjustment than avoidant coping, recently there has been increasing recognition that this may not be true for all populations [48, 49]. It may be more adaptive for youth residing in dangerous settings to use avoidant coping than to approach the problem; for example, if a youth approaches a dangerous situation they may be

more likely to encounter other negative outcomes such as exposure to violence (Grant et al. [52]). Therefore, future research should examine the utility of different types of coping strategies for urban, African American youth residing in dangerous neighborhoods.

5.7. Strengths and Limitations. This study adds to the existing body of research on life events and depression symptom etiology in a number of ways. This study used a prospective, longitudinal design with a sample of predominantly low income, African American adolescents. Due to this design, this study was able to address questions of causality and directionality, more so than previous cross-sectional studies with ethnically diverse youth (e.g., [4]). This study also addressed the potential confounding effect of including cognitive appraisals of life events when assessing stress by using a frequency count of life events without inclusion of cognitive appraisal. This methodology allowed the examination of the individual effects of stressors and cognitions on the development of depressive symptoms and testing of mediational hypotheses. This study also provides a better understanding of the etiology of depressive symptoms in an understudied population, urban, African American youth.

The study strengths should be considered in the context of some limitations. The reliance on self-report measures for assessing experiences of adverse life events, control-related beliefs, and depressive symptoms can be problematic because self-report measures may be vulnerable to response bias. For example, youth's depressive symptoms may affect their reports of experiences of adverse life events. While it would have been advantageous to include multiple informants to assess experiences of adverse life events, it should be noted that other informants may be less beneficial when assessing depressive symptoms and control-related beliefs, since these constructs are not readily observable by others, including parents [18].

5.8. Future Directions. These findings suggest several directions for future work. Research has found a positive association between life event stress and youth externalizing behaviors such as aggression, hyperactivity, and conduct problems [53]. Future studies should examine whether control-related beliefs mediate the association between adverse life events and externalizing behaviors in low-income, African American youth. Future research also should examine whether life event stress experienced by youth leads to negative outcomes in general or whether specific types of life event stress lead to specific negative outcomes. There is some evidence that the types of life events experienced by youth are differently associated with internalizing versus externalizing behaviors. For example, in their review of studies examining specificity of stressors on youth psychological outcomes, McMahan et al. [54] found several studies that reported specific relationships between exposure to violence and externalizing outcomes and between divorce or marital conflict and internalizing outcomes. For urban, African American youth, it is important to examine how adverse life events may impact externalizing symptoms in addition to internalizing symptoms. For example, for African American

adolescents who reside in neighborhoods characterized by high levels of community violence, it may be more adaptive to display distress through externalizing behaviors than internalizing behaviors [11]. Thus, future research should examine internalizing and externalizing symptoms, including the possibility of co-occurring symptoms.

Another direction for future research is to examine whether other cognitions influence depressive symptoms. For example, like control-related beliefs [18, 20], cognitions such as self-efficacy beliefs also may play an important role in adolescent depressive symptoms for urban, low-income youth. Researchers should also work to further understand when control-related beliefs begin to play an important role in depression development. Because youth experience cognitive changes such as increased egocentrism and heightened self-consciousness in early adolescence [55], it will be useful to further explore when the effects of control-related beliefs on depressive symptoms begin to emerge, as this information will not only increase our understanding of the etiology of depressive symptoms in youth but also help inform depression prevention and intervention efforts.

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Research Article

Depressive Symptoms and Psychosocial Functioning in Preadolescent Children

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The current study was designed to determine the percentage of children “at-risk” of depression or evidencing clinical levels of depression. In addition, the study examined how the “at-risk” and the clinical groups differed from children who demonstrated no depressive symptoms on positive and negative affect, four aspects of self-concept, and peer ratings of popularity. Respondents were 510 children (270 boys 240 girls) who ranged in age from 7 to 13 years (mean = 9.39). The results demonstrated that 23% of children were either in the “at-risk” or clinical range of depression. Children in both the clinical and the “at-risk” range demonstrated higher negative affect but lower positive affect and lower self-concepts than children in the normal range. However, children’s peers only differentiated between the “clinical” and “normal” groups. It is harder for peers, and other informants such as teachers and parents, to detect the problems of children with elevated depressive symptoms but who do not meet the diagnostic criteria. It is important to implement intervention programs for children who evidence depression symptoms, as well as “at-risk” children. “At-risk” children with elevated levels of depressive symptoms may be more disadvantaged, as their problems are less likely to be detected and treated.

1. Introduction

The clinical presentation of depressive symptoms in children largely parallels that of adults [1, 2]. However, as outlined below, there are some differences in the presentation of these symptoms across the life span [3]. The diagnostic and statistical manual of mental disorders [4] provides a summary of the most widely accepted constellation of depressive symptoms associated with each depressive disorder. The two most prevalent in childhood, and therefore most relevant to the current research, are major depressive disorder (mdd) and dysthymic disorder (DD) [4]. It is important to obtain a better understanding of the prevalence of depressive disorders in childhood, the prevalence of those at-risk of developing depression, and the factors in childhood that are associated with these depressive symptoms.

Epidemiological studies of community samples have reported the prevalence of MDD in children to range from 0.4–2.5%, while the prevalence of DD has been reported to range from 0.6–1.7% (e.g., [5–7]). However, the number of children exceeding cutoff scores for clinically significant

levels of depressive symptoms as assessed by the children’s depression inventory (CDI) has been shown to range from 20 to 24% [8, 9].

Symptoms associated with depression can cause significant impairment across emotional, physical, behavioral, cognitive, and interpersonal functioning [10, 11]. Poor peer relationships, low self-concept, and high negative affect have been strongly associated with depression in preadolescent children [12–14]. Depressed children have been found to demonstrate lower rates of prosocial behavior [15], have poor friendship quality [16], and tend to elicit negative reactions and rejection when interacting with peers [15]. Additionally, depressed children are reported to be sensitive to negative social cues, incorporating this feedback into their social perception [12]. Depression in children has also been associated with poor self-concept, with children tending to evaluate themselves negatively, to have low expectations for performance, more stringent criteria for failure, and a lower perceived self-competence [17]. Poor self-concept has also been correlated with a wide range of negative outcomes, including higher rates of suicide, loneliness, depression,

social anxiety, and alienation in childhood and adolescence [18].

The above research has demonstrated an association between depression and poor interpersonal relationships, poor self-concept, high negative affect, and a lack of positive affect. However, a comparison of the psychosocial functioning of the “at-risk” group of preadolescents with elevated depressive symptoms to that of the normal and a clinical group has yet to be examined. This is important as researchers have argued that treatment may be appropriate for children that evidence functional impairment even though children may not meet diagnostic criteria for depression [11, 19]. These “at-risk” children with elevated levels of depressive symptoms may suffer continuing problems and may be more disadvantaged as their problems are less likely to be detected and treated. Also, given the imperfections of the DSM nosology [19], it is important to also consider those with impairing symptoms. For example, Gotlib et al. [11] has highlighted the importance of being clinically sensitive to adolescents who presented with elevated levels of depressive symptomatology, but who did not meet diagnostic criteria for a depressive disorder, as they reported marked difficulties in psychosocial functioning.

The current study examined the relationship between depressive symptoms and the above variables among preadolescent children (i.e., children aged between 8 and 11 years). Particular emphasis was placed on the examination of the psychosocial functioning of children who reported depressive symptoms in the normal, “at-risk” and clinical range of depressive symptoms. We firstly examined whether the level of depressive symptoms was similar in boys and girls and across year levels, and whether there would be any interaction between these two variables. That is, are there gender differences in levels of depression among children, and is the trajectory of change with increasing age different for boys and girls. These gender differences according to grade have yet to be fully evaluated in previous studies, and they have implications for the clinical management of depression among children.

Further, it was hypothesised that negative affect, poor self-concept and poor peer popularity would become more severe as the level of depressive symptoms increased, with those in the clinical range demonstrating significantly more problems in their interpersonal relationships, self-concept and affect, compared to those “at-risk”, who would evidence more problems than those in the normal range. We also included a peer-report measure of peer acceptance/popularity, as the detection of depressive symptoms by peers is critical. Children with depression see themselves and their environment in a negative light. Peers detect this negativity and then dislike interacting with them [20].

2. Method

2.1. Participants. The 510 participants (270 boy, 240 girls) were enrolled in Grades 3 to 6 at six primary schools in urban regions in Melbourne, Australia. These schools included students from diverse socioeconomic and cultural backgrounds. The only demographic information gathered

was on the child’s sex, age in years, and grade level. There were 106 boys and 102 girls in grade 3 ($M = 8.27$ years, $SD = 0.48$), 67 boys and 48 girls in grade 4 ($M = 9.32$ years, $SD = 0.50$); 60 boys and 47 girls in grade 5 (10.13 years, $SD = .40$), and 60 boys and 43 girls in grade 6 (9.35 years, $SD = 1.18$).

2.2. Materials

2.2.1. The Children’s Depression Inventory (CDI). The CDI is a 27-item self-report measure of severity of depressive symptoms in children as young as seven. The CDI is a childhood extension of the Beck depression inventory [21]. In the present study, one modification was made to the original CDI, which was the removal of the item that assesses suicidal ideation. This item was removed because of ethical considerations (the question was of concern to some of the schools) and this is in line with other previous studies [22–24]. Scores ranged from 0 to 52, with higher scores indicating severe levels of depressive symptoms and scores 12 or below indicating depressive symptoms in the normal range [25]. Since scores of greater than 19 were considered to be in the clinical range, participants who obtained scores of 13–19 were classified as being “at-risk” for depression. The CDI has demonstrated good validity, high internal consistency and test-retest reliability in the measurement of depressive symptoms [26]. The Cronbach’s alpha coefficients in the current study were .89 for the total sample and for both boys and girls.

2.2.2. Positive and Negative Affect Schedule for Children (PANAS-C). The PANAS-C [27] assesses positive and negative affect in children. It is a 20-item self-report measure consisting of two scales: a 10-item positive affect scale and a 10-item negative affect scale. Scores on the PANAS-C range from 10 to 50 on each scale. High scores on the negative affect and positive affect scales indicate elevated level of negative affect and positive affect, respectively. Laurent et al. [27] reported high internal consistency, good construct validity, and convergent and discriminant validity for the PANAS-C with children aged between 8 and 18 years. The Cronbach’s alpha coefficients in the current study were .73 for boys and .74 for girls for the positive affect scale, and .82 for boys and .85 for girls for the negative affect scale.

2.2.3. Perceived Competence Scale for Children (PCSC). The PCSC [28] is a self-report instrument that assesses a child’s self-concept across four domains: academic (academic performance), social (confidence with and acceptance by peers), sporting (sporting and outdoor activities), and global self-worth (being sure of oneself and what one does). The PCSC contains seven items in each subscale, with a total of 28 items. Participants responded using a four-point Likert scale with possible responses of false, mostly false, mostly true and true. Scores are summed and averaged for each subscale, resulting in separate subscale means. High scores on each of these subscales indicated positive levels of self-concept. Harter [28] reported good internal consistency for each of the subscales (alphas ranging from .73 to .86) and satisfactory

TABLE 1: Mean scores and main effects of sex and grade and the sex by grade interaction for depressive symptoms, affect, and self-concept.

Outcome	Depressive symptoms	Positive affect	Negative affect	Academic self-concept	Social self-concept	Sporting self-concept	Global self-worth
Sex							
Boys	8.11	38.79	21.31	3.03	3.09	3.06	3.25
Girls	8.59	38.18	22.39	3.00	2.99	2.88	3.24
Standard error	.69	.50	.57	.05	.06	.06	.05
<i>P</i>	.87	.09	.15	1.00	.19	.00	.86
Grade							
Grades 3 & 4	8.92	38.77	22.61	2.99	2.97	2.94	3.23
Grades 5 & 6	7.79	38.20	21.09	3.04	3.11	2.99	3.26
Standard error	.94	.55	1.05	0.07	.08	.06	.05
<i>P</i>	.22	.25	.08	.43	.05	.33	.48
Sex by grade							
Boys Grades 3 and 4	9.36	39.49	22.27	2.95	2.96	2.97	3.23
Boys Grades 5 and 6	6.87	38.10	20.36	3.11	3.23	3.14	3.27
Girls Grades 3 and 4	8.47	38.06	22.95	3.03	2.97	2.91	3.29
Girls Grades 5 and 6	8.72	38.29	21.83	2.96	3.00	2.84	3.19
Standard error	1.18	.80	1.20	.09	1.00	.09	.07
<i>P</i>	.05	.11	.48	.03	.05	.03	.12
ICC	4.64%	1.06%	11.43%	3.60%	4.18%	2.91%	0.56%

discriminant validity. The Cronbach's alpha coefficients in the current study for the academic (.73 for boys, .75 for girls), social (.79 for boys, .82 for girls), sporting (.73 for boys, .74 for girls) and global self-worth (.74 for boys, .76 for girls) domains indicated good reliability.

2.2.4. Peer-Report Measure of Peer Acceptance/Popularity. The peer-report measure of peer acceptance/popularity [29] is a sociometric scale that requires each participant to nominate three classmates in response to the following two items: "you like to play with a lot" and "you like to play with the least". However, because of the sensitivity of the second item it was altered to ask the participants to name three classmates "you like to play with a little". For each child, the number of nominations he or she received on each of the two items were added and were standardised for differences in classroom size. This was achieved by dividing the number of nominations received by the number of students in the class in which the child was a member. The peer-report measure of peer acceptance/popularity has been found to have moderate to high levels of short-term stability and concurrent validity [30, 31]. Since there was no retest, and no other measure of peer popularity, it was not possible to calculate these measures in the current study.

2.2.5. Procedure. Ethics Approval was obtained from the University Ethics Committee. An information pack was sent to 24 primary schools, which represented diverse sociocultural areas in Melbourne, Australia. The school principals from six schools agreed participate in the study.

All children in the selected classes were invited to participate in the study ($N = 794$), but written parental consent was required for children to take part in the study. If parents wanted any further information they were asked to contact the researchers.

Parental consent was 67% ($N = 532$, class range 48% to 100%), with 96% of these participants completing the questionnaire. The anonymous questionnaire was completed in class groups of about 20 children. The researcher verbally presented the questionnaire to the class group and students were asked to answer items as they were read out. Children were encouraged to ask questions if they needed to clarify the meaning of an item or if they required assistance. In addition, if any children felt discomfort with any question then they were told that they could leave it out. The school counsellor was also advised about the study so that any child who was distressed in any way could speak to her/him and they were also given the contact number for Kids' Helpline in Australia, which provides a telephone service for children who may want to speak to a counsellor for any problems.

3. Results

The mean level of depressive symptoms reported by the overall sample of participants in the current study fell within the normal range ($M = 8.65$, $SD = 7.56$) [21]. These levels are comparable to those found in other studies that used the complete CDI scale (one item was removed in the current study) [25, 32, 33].

Table 1 displays the adjusted mean, standard error, significance level, and intraclass correlation coefficient (ICC)

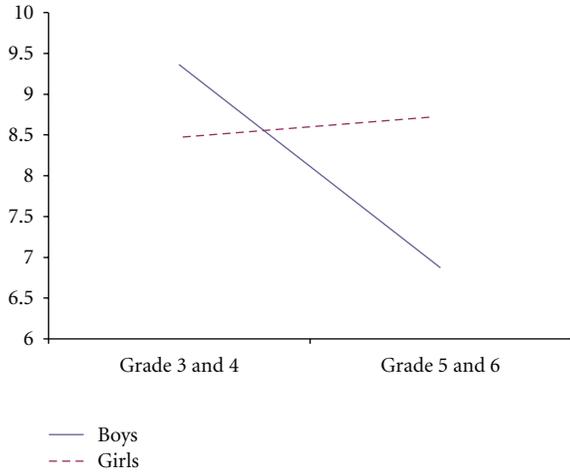


FIGURE 1: Sex by grade interaction for depressive symptoms.

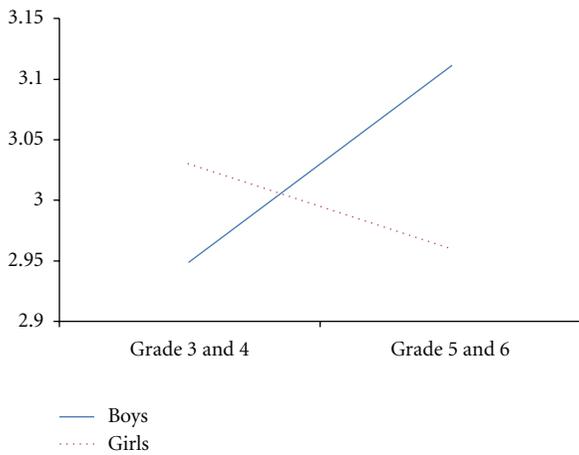


FIGURE 2: Sex by grade interaction for academic self-concept.

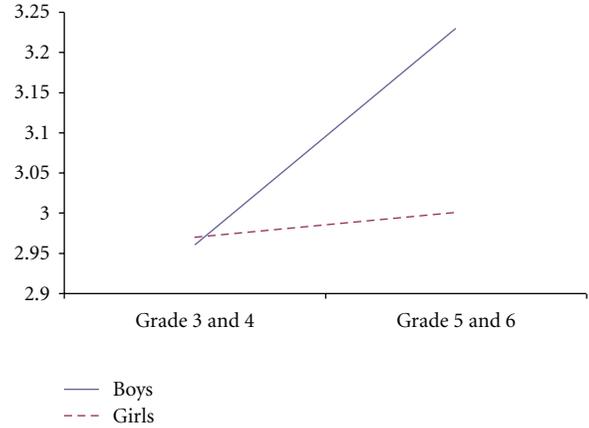


FIGURE 3: Sex by grade interaction for social self-concept.



FIGURE 4: Sex by grade interaction for sporting self-concept.

for the measures of depressive symptoms, affect, self-concept, and peer-rated popularity for the main effects of sex and grade, and the sex by grade interaction. There were no differences between grade 3 and 4, or between grade 5 and 6, thus these respective grades were combined. ICC is a measure of the extent to which observations are not independent of a grouping variable (e.g., schools). It is a ratio of variance between groups in the model to variance within these groups. The presence of a significant intraclass correlation is an indicator of the need to employ multilevel modeling. Higher percentages indicate that the grouping level makes a difference. As the ICC levels were low, a conventional analysis of variance was used.

The findings revealed that there were gender differences in sporting self-concept, with boys scoring higher than girls. Further, Grades 5 and 6 scored higher in sporting self-concept than Grades 3 and 4. These differences are further highlighted in the significant interactions summarized below. There was a significant sex by grade interaction depressive symptoms, and academic, social and sporting self-

concepts (see Figures 1, 2, 3, and 4). Fisher's LSDs ($P < .05$) revealed that boys in Grades 5 and 6 reported significantly lower levels of depressive symptoms than boys in Grades 3 and 4 but higher academic, social and sporting self-concepts. The power for these analyses was low (.10 to .56), but this is not surprising given the small number of children in the "at-risk" and clinical groups.

The number and percentage of participants falling within the normal, "at-risk" and clinical range of depressive symptoms, and their means and standard deviations are presented by sex, grade and for the total sample in Table 2. Just over 77% of students in the sample reported little to no depressive symptoms. Nearly 13% of participants fell within the "at-risk" range for depression, while almost 10% reported depressive symptoms that were in the clinical range.

A second analysis of variance was conducted to determine the relationship between depressive symptoms, positive and negative affect, the four aspects of self-concept and peer-rated popularity. The analyses revealed that as the level of depressive symptoms reported by students increased, levels

TABLE 2: Number of participants falling within the normal, “at-risk”, and clinical range of depressive symptoms and the mean level of depressive symptoms across sex and grade.

		Normal (CDI<13)	“At risk” (CDI 13–19)	Clinical (CDI>19)
Sex				
Boys	M (SD)	5.21 (3.45)	15.52 (1.99)	26.48 (5.54)
	<i>n</i>	205	42	23
	%	75.9	15.6	8.5
Girls	M (SD)	5.49 (3.35)	15.21 (1.82)	26.04 (6.73)
	<i>n</i>	190	24	26
	%	79.2	10.0	10.8
Grade				
Grades 3 and 4	M (SD)	5.51 (3.42)	15.51 (1.84)	26.87 (6.90)
	<i>n</i>	247	45	31
	%	76.5	13.9	9.6
Grades 5 and 6	M (SD)	5.02 (3.37)	15.19 (2.11)	25.17 (4.53)
	<i>n</i>	148	21	18
	%	79.1	11.2	9.6
Total	M (SD)	5.35 (3.40)	15.41 (1.92)	26.24 (6.14)
	<i>n</i>	395	66	49
	%	77.5	12.9	9.6

of positive affect, academic self-concept, social self-concept, sporting self-concept, global self-worth and peer-rated popularity decreased, while levels of negative affect increased. Negative affect (.43), academic self-concept (−.32), social self-concept (−.39) and global self-worth (−.28) demonstrated the largest change per unit increase of depressive symptoms compared to positive affect (−.21) sporting self-concept (−.23) and peer-rated popularity (−.20), which demonstrated more modest changes (see Table 3).

Fisher’s LSD tests indicated that individuals with depressive symptoms in the normal range reported significantly higher levels of positive affect, academic self-concept, social self-concept, sporting self-concept, global self-worth and lower levels of negative affect compared to individuals reporting depressive symptoms in the “at-risk” and clinical range at the $P < .001$ level of significance (see Table 3). Those individuals in the “at-risk” range also reported higher levels compared to those in the clinical range on academic self-concept, social self-concept and global self-worth at the $P < .001$ level, and positive affect and sporting self-concept at the $P < .05$ level, as well as lower levels of negative affect at the $P < .001$ significance level (see Table 3). Individuals falling within the normal range of depressive symptoms were rated by their peers as being more popular than children with clinical levels of depressive symptoms. The popularity of children within the “at-risk” range of depressive symptoms fell between those in the normal and clinical range, and did not differ significantly from either (see Table 3).

4. Discussion

Depressive symptoms reported by the overall sample in the current study fell within the normal range, and were similar

for boys and girls and across year levels. However, there was a sex by grade interaction, with boys in Grades 5 and 6 reporting significantly lower levels of depressive symptoms than boys in Grades 3 and 4. Consistent with this finding, we also showed that boys in Grades 5 and 6 scored higher on academic, social and sporting self-concepts. It is now important to determine what other emotional, social and cognitive changes are occurring among boys in Grades 5 and 6 that may explain these findings.

In total, 23% of children reported depressive symptoms in the “at-risk” and clinical range, and these levels did not differ across sex or grade. The above results are consistent with previous research with children [8, 9, 34], and adolescents [35]. When comparing the current findings to previous epidemiological studies of the prevalence of depressive disorders in community samples of children, it is evident that a much higher proportion of children are presenting with elevated depressive symptomatology than would meet the formal psychiatric diagnostic criteria for a depressive disorder [5–7, 11, 36]. Gotlib et al. [11] highlighted the importance of being clinically sensitive to adolescents who presented with elevated levels of depressive symptomatology. This study highlights the importance of also being clinically sensitive to preadolescents.

Levels of psychosocial functioning were highest for children with depressive symptoms in the normal range, but generally deteriorated as the level of depressive symptoms became more severe. Consistent with the findings of Gotlib et al. [11], the current study found that children with “at-risk” and clinical levels of depression reported psychosocial disturbances, higher negative affect, and reduced positive affect and self-concept, when compared to children presenting with depressive symptoms in the normal range. An important aspect to the findings from the current study is that children who were classified as being “at-risk” of depression demonstrated problems in their psychosocial functioning, not just those students who were classified as being in the clinical range. These findings are consistent with past research [12–14]. If these symptoms remain in place into adolescence, it is likely that the poor self-concept associated with high levels of depressive symptoms may cause problems in other aspects of the children’s lives.

However, only children with clinical levels of depressive symptoms were rated by their peers as less popular than those who scored in the normal range. It may be that children in the “at-risk” group do not stand out sufficiently to be noticed by their peers. Teachers have also been found to be not able to detect less severe cases. Kleftaras and Didaskalou [34] found that although 30 percent of 5th and 6th grade children evidenced high levels of depressive symptoms, their teachers failed to identify them as being depressed, and attributed behavioral problems to other causes. Thus, many children “at-risk” may be unlikely to obtain appropriate intervention for their depression. These issues now need to be explored further in longitudinal research.

Harrington et al. [37] assessed the continuity of childhood depressive symptoms into adulthood. They found that depressed children and adolescents were at greater risk of developing a depressive disorder in adult life as well as

TABLE 3: Main effect of depressive symptom level on affect, self-concept, and peer-rated popularity.

	Normal (CDI < 13)	“At risk” (CDI 13–19)	Clinical (CDI >19)	ICC	Standard error	P	Normal versus “at-risk” t	Normal versus clinical t	“At risk” versus clinical t
Positive affect	39.35	36.88	34.77	1.48%	1.00	<.001	3.48***	5.65***	2.11*
Negative affect	20.83	24.11	29.46	13.3%	1.06	<.001	4.29***	10.03***	5.05***
Academic self-concept	3.14	2.65	2.28	3.00%	.67	<.001	7.29***	11.28***	3.87***
Social self-concept	3.20	2.52	2.10	3.98%	.73	<.001	9.27***	13.03***	3.93***
Sporting self-concept	3.08	2.69	2.42	1.03%	.76	<.001	5.02***	7.49***	2.45*
Global self-concept	3.39	2.96	2.54	0.71%	.56	<.001	7.62***	13.20***	5.30***
Peer-rated popularity	.21	.19	.18	30.75%	.02	.05	1.49	2.14*	0.66

* $P < .05$; *** $P < .001$.

having an increased risk for psychiatric hospitalisation and psychiatric treatment. This indicates that there may be a continuity of affective disturbance between childhood and adult life. Weissman et al. [38] also investigated the continuity of prepubertal major depressive disorder into adulthood and reported similar results to those of Harrington et al. [37]. These results highlight the importance of addressing depressive symptoms in childhood in order to prevent the continuation of these symptoms into adulthood, with the associated risk of the symptoms developing into MDD.

A limitation of the current study was that it was cross-sectional in nature, and so it was not possible to determine the direction of the relationships between the variables. It is important that the participants are followed up over time in order to determine the extent to which the depressive symptoms continue into adolescence, and also to determine the directional relationships between the variables. While schools were selected to represent diverse sociocultural areas in Melbourne, Australia, we did not collect specific data on socioeconomic background and we were also not able to assess how the ones who participated in this study differed from those who did not. Another limitation is that the number of children in the clinical group was small so the findings need to be verified with a larger sample. It is also possible that the level of risk of clinical depression may have been underestimated due to the removal of the item in the CDI that related to suicidal ideation. The study also relied heavily on self-reports of depressive symptoms and psychosocial functioning. Obtaining information on how others, including parents and teachers, provides additional information on children’s functioning and symptoms [39–41]. Future studies also need to examine the role of other factors such as socioeconomic status, ethnicity, family situations, and academic performance.

In summary, the results suggest that it is important to consider interventions for both “at-risk” and clinically depressed children in order to improve their mood, self-concept and social function, and so attempt to prevent the development of clinical depression in adolescence and adulthood. “At-risk” children with elevated levels of depressive symptoms also demonstrated higher negative affect but lower positive affect and lower self-concepts than children with depressive symptoms in the normal range.

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Research Article

Depressive Symptoms and Deliberate Self-Harm in a Community Sample of Adolescents: A Prospective Study

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The associations between depressive symptoms and deliberate self-harm were studied by means of a 2-wave longitudinal design in a community sample of 1052 young adolescents, with longitudinal data for 83.6% of the sample. Evidence was found for a bidirectional relationship in girls, with depressive symptoms being a risk factor for increased self-harm one year later and self-harm a risk factor for increased depressive symptoms. Cluster analysis of profiles of depressive symptoms led to the identification of two clusters with clear depressive profiles (one severe, the other mild/moderate) which were both characterized by an overrepresentation of girls and elevated levels of self-harm. Clusters with more circumscribed problems were also identified; of these, significantly increased levels of self-harm were found in a cluster characterized by negative self-image and in a cluster characterized by dysphoric relations to parents. It is suggested that self-harm serves more to regulate negative self-related feelings than sadness.

1. Introduction

Research shows that depression is relatively rare in children but becomes more prevalent in adolescence. At the same time, the sex ratio also changes considerably; whereas there is little evidence of gender differences in children, depression during adolescence is significantly more common among girls than boys [1, 2]. Another problem behavior that develops approximately at the same time is deliberate self-harm (henceforth referred to as self-harm), here defined as self-inflicted damage to the surface of one's own body. (It should be noted that the definition used in this study does not include behaviors like taking an overdose or self-poisoning. In this way, it is similar to the proposed diagnosis of "nonsuicidal self-injury" in the DSM-V [3], although our definition differs from this notion by not explicitly excluding suicidal intentions.). The mean age of onset of this kind of self-harm is reported to be around 12–15 years [4, 5], and self-harm is quite frequently reported among young adolescents [6–12]. This raises the question how depressive

symptoms and self-harm are associated, both concurrently and prospectively.

It is commonly assumed that self-harm develops as a symptom of high emotional distress (anxiety, depression, self-hate, etc.), as a way of expressing or regulating this distress [13–16]. This would mean that distressful emotions (e.g., depressive feelings) represent a risk factor for the development of self-harm. A risk factor is generally defined [17] as a measurable variable that must precede a negative outcome and be associated with a higher risk for this outcome, which means that risk factors can only be identified by means of prospective studies. So far, there are only a few prospective studies of emotional risk factors for self-harm. In one study [18], self-reported depressive symptoms at the age of 8 were found to predict acts of self-harm 10 years later in a community-based sample of 2,348 boys. In another study [19], predictors at the age of 12 for acts of self-harm at the age of 15 were studied; the results showed that self-reported internalizing problems and somatic complaints, as well as parent-reported externalizing

problems and aggressiveness, independently predicted self-reported acts of self-harm 3 years later.

In a recent study, however, we [20] failed to find support for emotional symptoms among 13–15-year-old adolescents as a risk factor for increased self-harm. In this study, emotional symptoms were measured by Goodman's [21] Strengths and Difficulties Questionnaire (SDQ). One problem with the SDQ, however, is that its 5-item subscale for measuring emotional symptoms contains only one depression-related item (whereas it contains three items related to nervousness, fear and worry, and one somatic item). It is likely that depressive feelings of guilt, shame, worthlessness, self-disgust, self-contempt, or self-hatred are more of a risk factor for the development of self-harm than are feelings of fear, worry, or nervousness. The SDQ emotional symptoms subscale may therefore be less optimal for detecting any existing prospective association between emotional problems and self-harm. It is quite possible that, although emotional symptoms in general do not serve as a risk factor for the development of self-harm, a *subset* of emotional symptoms do so. One purpose of the present study was to test the hypothesis that depressive symptoms represent such a subset of emotional symptoms.

Another purpose was to analyze subcategories of depressive symptoms and investigate if certain subsets of depressive symptoms, or patterns of such symptoms, are especially associated with self-harm. For example, because self-harm implies an attack towards one's own body, it may be assumed to be more associated with feelings of self-hatred, self-contempt, self-disgust, and so forth than with feelings of sadness, lack of energy, or difficulties in concentration.

Finally, we also wanted to test the possibility that self-harm may serve as a risk factor for increased depressive symptoms. In a previous study [20], we found evidence for a bidirectional relationship between self-harm and psychological difficulties in general; that is, overall psychological difficulties predicted an increase in self-harm one year later and self-harm predicted increased psychological difficulties one year later. Our hypothesis was that such a bidirectional relationship would apply also to the association over time between depressive symptoms and self-harm. There are several reasons to expect that self-harm may be a risk factor for increased depression. First, although self-harm may have an emotion-regulating function and therefore lead to decreased emotional distress as an immediate consequence [14], it may have the opposite effect in a longer time perspective by setting the stage for depression-related processes like rumination, shame, guilt, and regret. Second, when it comes to the attention of others (parents, friends, etc.) that an adolescent deliberately harms herself, these others may respond strongly negatively, thereby causing a deterioration and even disruption of interpersonal relationships in a way that may lead to depressive symptoms.

If there is a bidirectional prospective relationship between depression and self-harm, this would mean that depressive symptoms and self-harm may enter into a self-generating "vicious cycle" where increases in the one variable lead to increases in the other and vice versa. This can be described in terms of a dynamic system, where internal

feedback processes lead to the emergence and stabilization of pathological patterns that include both depression and self-harm. Because evidence suggests that there may be gender differences in both depression and self-harm, we studied this question separately in girls and boys.

To summarize, the present study was carried out (1) to study if there is a bidirectional relationship between depressive symptoms and self-harm in young adolescents, in the sense that depressive symptoms serve as a risk factor for self-harm and vice versa; (2) to investigate different patterns of depressive symptoms, how frequent they are among young adolescents, and how they are related to self-harm. Methodologically, we combined two different approaches. First, we used a variable-oriented approach to study both concurrent and prospective associations between symptoms of depression and self-harm, with the hypothesis that we would find evidence of a bidirectional relation between depression and self-harm. Second, we used a person-oriented approach [22] in accordance with an advanced analytical procedure developed by Bergman [23] to identify different subgroups of adolescents with different patterns of depressive symptoms, and then compared these subgroups to see if they differed on self-harm. We hypothesized that the analysis would identify at least one clear depression-related cluster and that depression-related clusters would contain an overrepresentation of girls and be associated with significantly more self-harm than the other clusters. We also expected that adolescents characterized by some categories of depressive symptoms (e.g., negative self-related feelings) would show more self-harm than others (e.g., those characterized by sadness and lack of energy).

2. Materials and Methods

2.1. Participants. The participants were a community sample from a municipality in the south of Sweden which is fairly representative for the rest of Sweden in terms of the proportions of children living with both of their parents, and their ethnic backgrounds, but slightly more rural than Sweden as a whole, and with a slightly lower income level and educational level [24]. At Time 1, there were 532 students in Grade 7 (mean age 13.7 years) and 520 students in Grade 8 (mean age 14.7 years) in the schools of this municipality (excluding three special schools with place for around 25 individuals with severe school difficulties); 992 of these 1052 students (94%) participated. One year later, at Time 2, 984 students in Grade 8 and 9 participated. Ten individuals were excluded as multivariate outliers with stereotypic response patterns. In total, there were available longitudinal data for 879 participants (450 girls and 429 boys), who represented 83.6% of all students that were available for inclusion at Time 1.

2.2. Instruments. The participants filled out an 11-page questionnaire, which was tailor made for young adolescents and tested in a pilot study with around 200 participants [6, 24]. The questionnaire included a number of self-assessment instruments. The present study used data from four of these

instruments, plus some separate questions. As the measure of deliberate self-harm, we used a short version of Gratz' [25] Deliberate Self-Harm Inventory. To measure depressive symptoms, a Depression Index was constructed on the basis of depression-related items from three separate instruments: the Strengths and Difficulties Questionnaire (SDQ [21]), a modified version of the Emotional Tone Index (ETI [26, 27]), and the Appearance subscale of the Body Esteem Scale for Adolescents and Adults, (BEAA [28]), with the addition of separate questionnaire items concerning sleep, alertness, self-rated health, and views on the personal future.

Deliberate Self-Harm Inventory: 9-Item Version Revised (DSHI-9r). This is a shortened and modified version of the Deliberate Self-Harm Inventory which was originally constructed and validated by Gratz [25] and then translated into Swedish and adapted to adolescents [6, 8]. In this 9-item version of the DSHI, respondents are asked if they have deliberately engaged in any of nine different kinds of direct physical self-harm (cutting wrists, arms, or body areas; minor cutting causing bleeding; carving words, pictures, and so forth into the skin; burning oneself with cigarette, lighter, or match; severe scratching, causing bleeding; sticking sharp objects into the skin; biting oneself so that the skin is broken; punching oneself or banging one's head, thereby causing a bruise; preventing wounds from healing) during the past 6 months. Respondents are instructed to rate the number of times they have conducted these behaviours on a scale from 0 to 6, where 0 is "never" and 6 is defined as "more than five times". A total score (from 0 to 54) on the DSHI-9r can thus be calculated by summarizing the number of times a person reports having engaged in these self-harming behaviours. The internal consistency of the DSHI-9r in the present study was $\alpha = .90$. All nine items of the DSHI-9r correlated with mental health problems as measured by SDQ Total Difficulties (r s ranging from .23 to .33).

Depression Index. Depression-relevant items were selected from the 11-page questionnaire, according to their correspondence with items from standard measures of depression and the DSM-IV criteria for major depression [29]. Because the items came from different instruments with different response formats, the scores on each item were transformed to z -scores. Items referring to positive feelings were reverse scored. The items were then subjected to a principal components analysis with varimax rotation, which identified eight components with eigenvalues >1 ; on the basis of converging results from both a scree plot and parallel analysis, however, the number of components was reduced to 6. On the basis of these, six subscales were constructed (see the appendix): Dysphoric relations to parents (10 items of which 4 referred to positive feelings, $\alpha = .85$), Negative self-image (6 items of which 3 involved positive statements about the self, $\alpha = .85$), Dysphoric relations to friends (6 items which all referred to positive feelings, $\alpha = .73$), Fatigue/somatic complaints (5 items, $\alpha = .70$), Sadness/loneliness (4 items, $\alpha = .67$), and Difficulties in concentration (4 items, $\alpha = .65$). Test-retest correlations

between Time 1 and Time 2 were $r = .71$ for the entire Depression index, $r = .60$ for Dysphoric relations to parents, $r = .68$ for Negative self-image, $r = .51$ for Dysphoric relations to friends, $r = .61$ for Fatigue/somatic complaints, $r = .48$ for Sadness/loneliness, and $r = .61$ for Difficulties in concentration.

2.3. Procedure. This research was conducted after approval by the Regional Ethics Committee at Lund University. Contact was established with school managements via headmasters who gave consent to their schools' participation in the study. Information about the form and purpose of the study was sent by mail to the parents, who were asked to contact the school teachers or the researchers if they did not want their child to participate. Parents as well as children were informed that this was a research project on the situation of young people today, in terms of how they feel, and how they perceive themselves, their feelings, relations, and life situation. The participants were also informed that their participation was voluntary, that they were free to withdraw at any time and without having to give a reason, that their answers were treated confidentially, and that no school personnel would have access to their answers. Contacts were established with representatives from school healthcare in the municipality to facilitate procedures if serious psychological problems or other circumstances related to participants would warrant an intervention. The procedure was considered ethically appropriate on the basis of previous research [30, 31].

The 11-page questionnaire was filled out in school, as part of a separate lecture hour, and was administered by research assistants from Lund University. A teacher was present, but did not participate in the data collection. In order to guarantee the students privacy, their school desks were separated as much as possible. The students were instructed to answer all questions as best they could, but not to think too much about any answer. They were instructed not to write their names anywhere on the questionnaire. After the completion of the questionnaire, it was sealed in an envelope by the student.

2.4. Statistical Analysis. The distribution of total DSHI-9r scores and the scores on two of the depression subscales (Dysphoric relations to parents and Sadness/loneliness) were highly positively skewed and leptokurtic at both Time 1 and Time 2; logarithmic transformations were therefore conducted on these three indexes, resulting in acceptable normal distributions.

To test the hypothesis that depressive symptoms would be a risk factor for self-harm, we used both logistic regression (with incidence of new cases of repeated self-harm, at Time 2 as the dependent variable) and hierarchical linear regression (with DSHI-9r scores at Time 2 as the dependent variable). To test the hypothesis that self-harm would be a risk factor for depressive symptoms at Time 2, we only used hierarchical linear regression.

Cluster analysis was used to group all participants on the basis of their different profiles of scores on the six

depression scales, according to the LICUR procedure [23]. This was done in four steps. First, multivariate outliers were identified by means of the residue procedure in the statistical package for pattern-oriented analyses SLEIPNER 2.1 [32] and removed from further analysis. Second, Ward's hierarchical clustering method was applied. Four criteria presented by Bergman [23] were used to decide on the optimal cluster solution: (a) theoretical meaningfulness of the cluster solution; (b) if there is a distinct drop in the explained error sum of squares (EESS) when a cluster solution is extracted, this suggests that two insufficiently similar clusters were merged to a nonoptimal cluster solution; (c) the number of clusters should not be more than 15 and should not be expected to be less than five; (d) the size of the EESS for the chosen cluster solution should preferably not be less than 67% and at the very least exceed 50%. In addition, the homogeneity coefficient of each cluster should preferably be <1 . Third, a data simulation was undertaken to verify that the explained ESS was higher than what could be expected on a random data set with the same general properties as the data set used in the real analysis. Fourth, a nonhierarchical relocation procedure was carried out in order to improve the homogeneity of the clusters and to increase the variance explained by the cluster solution.

3. Results

As expected, girls showed more evidence of depression than boys; this did not, however, apply to all depression subscales. Gender comparisons by independent samples *t*-test showed that girls scored higher than boys on the total Depression index and on the subscales Negative self-image, Sadness/loneliness, and Fatigue/somatic complaints (all $P < .001$). On the other hand, the boys scored higher on Dysphoric relations to friends ($P < .001$), and there were no significant differences ($P > .05$) on Dysphoric relations to parents or on Difficulties in concentration.

More girls than boys reported self-harm. At Time 1, 45.1% of the girls and 37.9% of the boys ($\chi^2 = 5.1$, $P < .01$) reported that they had harmed themselves deliberately at least once during the past 6 months. *Repeated* self-harm (defined as at least 5 instances of self-reported self-harm) was reported by 20.7% of the girls and by 15.9% of the boys. The stability of DSHI-9r scores from Time 1 to Time 2 was higher for girls ($r = .57$) than for boys ($r = .35$).

Correlational analyses showed that total depression scores and self-harm were moderately to highly associated both in girls ($r = .58$ at Time 1 and $r = .55$ at Time 2) and boys ($r = .39$ at Time 1 and $r = .46$ at Time 2).

3.1. Prediction of New Cases of Repeated Self-Harm. Depressive symptoms at Time 1 predicted the incidence of new cases of repeated self-harm at Time 2 in both girls and boys. Incidence here refers to participants who reported repeated self-harm (defined as at least five instances of self-harm) at Time 2 but had reported *no instance of self-harm* at Time 1. The incidence rate of repeated self-harm was 10.4% (26 of 251) among the girls and 8% (21 of 254) among the

boys. To test the hypothesis that depressive symptoms would predict the incidence of new cases of repeated self-harm, logistic regressions were carried out separately for girls and boys. As seen in Table 1, the model was significant in both genders ($\chi^2(1) = 9.13$, $P = .003$ in girls, and $\chi^2(1) = 5.56$, $P = .018$ in boys), explaining 7.3% of the variance in girls (*Nagelkerke* $R^2 = 0.073$) and 4.8% in boys (*Nagelkerke* $R^2 = 0.048$).

3.2. Bidirectional Associations between Self-Harm and Depression. The hypothesis of a bidirectional relationship over time between self-harm and depressive symptoms was confirmed in girls but not in boys. As seen in Table 2, hierarchical regression analyses among girls showed that, when controlling for self-harm at Time 1, depressive symptoms at Time 1 predicted self-harm at Time 2, and, conversely, when controlling for depressive symptoms at Time 1, self-harm at Time 1 predicted depressive symptoms at Time 2. In boys, however, there was only evidence for a unidirectional relationship; although depressive symptoms predicted self-harm, the reverse was not the case.

3.3. Cluster Analysis. In total, 977 participants had full data on the depression index at Time 1 and were entered into the cluster analysis. Of these, 24 adolescents were identified as multivariate outliers and excluded by the residue procedure. Second, the application of Ward's hierarchical clustering method, together with Bergman's [23] criteria, resulted in the choice of a ten-cluster solution, explaining 58.1% of the total error sum of squares (ESS). Third, a data simulation showed that the explained ESS of the cluster solution was significantly higher than expected by chance ($P < .0001$). Fourth, a non-hierarchical relocation procedure to improve the homogeneity of the clusters resulted in a ten-cluster solution that was found to explain 62.5% of the variance. The homogeneity coefficients of the clusters also were quite good, with most clusters having a homogeneity coefficient of $<.40$ and no cluster having a higher homogeneity coefficient than .82 (low coefficients mean high homogeneity).

Figures 1–4 show the profiles of *z*-scores for each cluster. As seen in Figure 1, the analysis identified two depression-related clusters: one small cluster (called the Depression cluster, $n = 27$), which was characterized by high scores on all depression indexes ($z > 1$ on all scales except on Difficulties in concentration), and one larger cluster (referred to as Mild/moderate depression, $n = 81$), which showed high scores ($z > 1$) on Sadness/loneliness and moderately high scores on all other subscales. As seen in Table 3, the Depression cluster showed a highly elevated depression score ($z = 1.37$), whereas the Mild/moderate depression cluster showed a moderately elevated score ($z = 0.68$).

In addition, there were a number of clusters that showed elevated scores on some depression indexes, without scoring high on total depression. As seen in Figure 2, there was a three-problem cluster that combined high scores on Fatigue/somatic problems, Dysphoric relations to parents, and Difficulties in concentration (called the "Fatigue and problems with parents" cluster, $n = 53$) and a two-problem

TABLE 1: Logistic regressions, predicting incidence of repeated self-harm at Time 2 from depressive symptoms at Time 1, among participants with no self-harm at Time 1.

Variables	B	SE	Wald(1)	OR	95% Confidence intervals	
					Lower	Upper
<i>Girls</i>						
Depressive symptoms	.23	.08	9.20**	1.26	1.08	1.45
Constant	-2.09	.21	96.48***	.12		
<i>Boys</i>						
Depressive symptoms	.23	.09	5.85*	1.25	1.04	1.50
Constant	-2.30	.23	98.05***	.10		

*P <.05, **P <.01, ***P <.001.

TABLE 2: Prospective hierarchical regressions, predicting T2 self-harm from T1 depressive symptoms and T2 depressive symptoms from T1 self-harm.

	Variables	R ² Δ	B	SE B	β	F step
<i>Predicting self-harm at T2 from depressive symptoms at T1 among the girls</i>						
Step 1	T1 self-harm	.35	.61	.04	.60	247.1***
Step 2	Depressive symptoms at T1	.01				6.4*
	T1 self-harm		.55	.05	.53***	
	T1 depressive symptoms		.11	.04	.11*	
<i>Predicting depressive symptoms at T2 from self-harm at T1 among the girls</i>						
Step 1	T1 depressive symptoms	.54	.83	.04	.73	571.4***
Step 2	Self-harm at T1	.01				6.5*
	T1 depressive symptom		.77	.04	.68***	
	T1 self-harm		.12	.05	.10*	
<i>Predicting self-harm at T2 from depressive symptoms at T1 among the boys</i>						
Step 1	T1 self-harm	.17	.42	.04	.41	88.2***
Step 2	Depressive symptoms at T1	.03				52.8***
	T1 self-harm		.35	.05	.35***	
	T1 depressive symptoms		.17	.05	.18***	
<i>Predicting depressive symptoms at T2 from self-harm at T1 among the boys</i>						
Step 1	T1 depressive symptoms	.43	.79	.04	.65***	356.3***
Step 2	Self-harm at T1	.00				2.8
	T1 depressive symptoms		.76	.05	.63***	
	T1 self-harm		.08	.05	.06	

*P <.05, **P <.01, ***P <.001.

TABLE 3: Comparison between the clusters on gender, total depression score, and self-harm.

Cluster	n	Proportion girls/boys	Total depr. z-score	Self-harm (DSHI-9r scores)	
				T1	T2 (n)
Time	T1	T1	T1	T1	
Depression cluster	27	20/7	1.37	21.8	16.2 (21)
Mild/moderate depression	81	53/28	0.68	5.4	5.9 (73)
Fatigue and problems with parents	53	32/21	0.42	7.9	7.9 (43)
Fatigue and problems with friends	65	27/38	0.16	1.5	2.7 (56)
Negative self-cluster	71	45/26	0.20	4.8	5.5 (63)
Sadness/loneliness cluster	90	56/34	0.04	2.0	3.2 (84)
Concentration difficulties cluster	97	37/60	-0.08	2.3	2.5 (87)
Average problems cluster	126	37/89	0.01	2.6	2.2 (113)
No problems cluster	154	87/66	-0.32	0.8	1.9 (150)
Happy and healthy cluster	189	82/107	-0.58	0.8	1.0 (177)

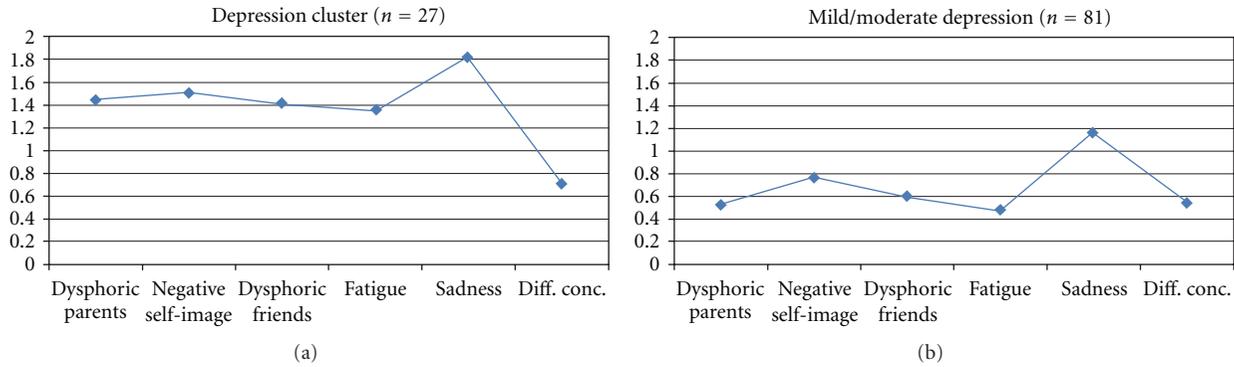


FIGURE 1: Depression-related clusters. Profiles in terms of z-scores (where $z = 0$ corresponds to the whole sample's mean on each subscale).

cluster that combined high scores on Fatigue/somatic problems and Dysphoric relations to friends (called the "Fatigue and problems with friends" cluster, $n = 65$). As seen in Table 3, however, none of these clusters scored especially high on the total depression score.

As seen in Figure 3, three "one-problem clusters" were also identified: one with a highly negative self-image but normal scores on all other indexes ("Negative self-cluster," $n = 71$), another with high scores only on Sadness/loneliness ("Sadness/loneliness cluster," $n = 90$), and a third with high scores on Difficulties in concentration but relatively normal scores on the other indexes ("Concentration difficulties cluster," $n = 97$).

Finally, the analysis identified three relatively large "healthy clusters" which together comprised 49.2% of the total sample: one cluster with low scores ($z < -0.50$) on all depression subscales ("Happy and healthy," $n = 189$), another with relatively low scores on all indexes except Negative self-image where they scored close to average (the "No problems cluster," $n = 154$), and a third with close to average scores on the indexes (the "Average problems cluster," $n = 126$) (see Figure 4).

A one-way ANOVA with the ten-cluster categorization as the independent variable and the total depression index score as the dependent variable showed that the clusters differed on depression, $F(9, 943) = 641.1, P < .0001$. Tukey post-hoc tests showed a categorization of the clusters into eight subsets, which differed significantly in the following order: Depression cluster > Mild/moderate depression cluster > "Fatigue and problems with parents" cluster > Negative self-cluster, "Fatigue and problems with friends" cluster > Sadness/loneliness cluster, and Average problems > Concentration difficulties cluster > No problems > Happy and healthy.

3.4. Gender Comparison between the Clusters. As expected, the girls were overrepresented in the depression-related clusters. The gender distribution in the ten clusters is shown in Table 3. To test the hypothesis that girls would be overrepresented in the depression-related clusters, the observed frequency was compared with the frequency that should be expected by chance alone, and one-tailed probabilities were computed according to the fixed-margins model using EXACON [33]. The results showed that the girls were

overrepresented in both the Depression cluster (observed frequency 20, expected frequency 13.5, $\chi^2 = 3.13, P = .009$) and in the Mild/moderate depression cluster (observed frequency 53, expected frequency 40.5, $\chi^2 = 3.86, P = .003$). Explorative comparisons of the gender distributions in the eight remaining clusters, with the Bonferroni correction ($P < .05/8 = .006$), showed only one significant effect: boys were overrepresented in the Average problems cluster (observed frequency 89, expected frequency 63, $\chi^2 = 10.73, P < .0001$, two tailed).

3.5. Comparison between the Clusters on Self-Harm. As expected, the depression-related clusters were associated with high levels of self-harm, but this was also the case for the "Fatigue and problems with parents" cluster and the Negative self-cluster. Table 3 shows the mean scores on the DSHI-9r for all the ten clusters. A one-way ANOVA with the ten-cluster categorization as the independent variable and the DSHI-9r as dependent variable showed that the clusters differed on self-harm, $F(9, 936) = 38.9, P < .0001$. Tukey post-hoc tests showed that the Depression cluster scored higher than all the other clusters. Further, the "Fatigue and problems with parents" cluster scored higher than six of the remaining clusters, the Mild/moderate depression cluster scored higher than five of the remaining clusters, and the Negative self-cluster scored higher than the three clusters with lowest DSHI-9r scores.

To study the stability of these results, a similar one-way ANOVA was carried out with the DSHI-9r at Time 2 as the dependent variable. As seen in Table 3, the results were highly similar, showing that the Time 1 clusters differed significantly also on Time 2 self-harm, $F(9, 857) = 14.6, P < .0001$. Again, Tukey post-hoc tests showed that the Depression cluster scored higher than all the other clusters and that the "Fatigue and problems with parents" cluster scored higher than six of the remaining clusters. The Mild/moderate depression cluster and the Negative self-cluster also scored higher than the five clusters with the lowest self-reported frequencies of self-harm.

4. Discussion

There are two main findings of the present study. First, there was support for the hypothesis of a bidirectional relationship

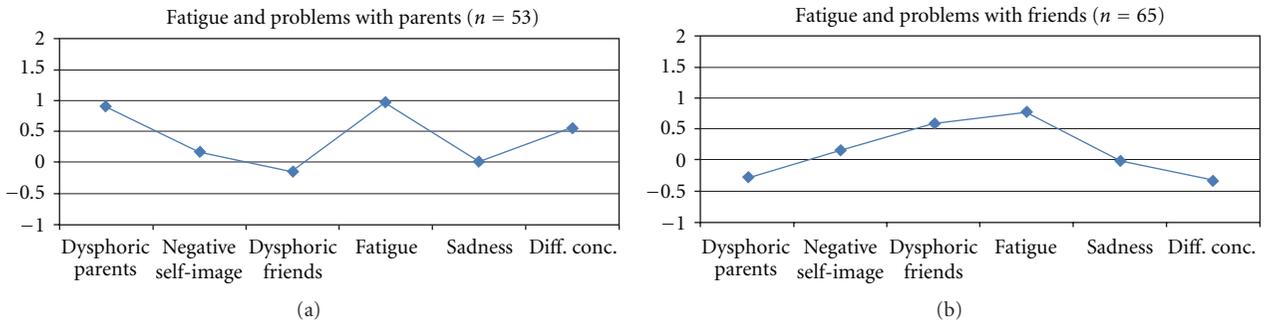


FIGURE 2: Two- and three-problem clusters. Profiles in terms of z-scores (where $z = 0$ corresponds to the whole sample’s mean on each subscale).

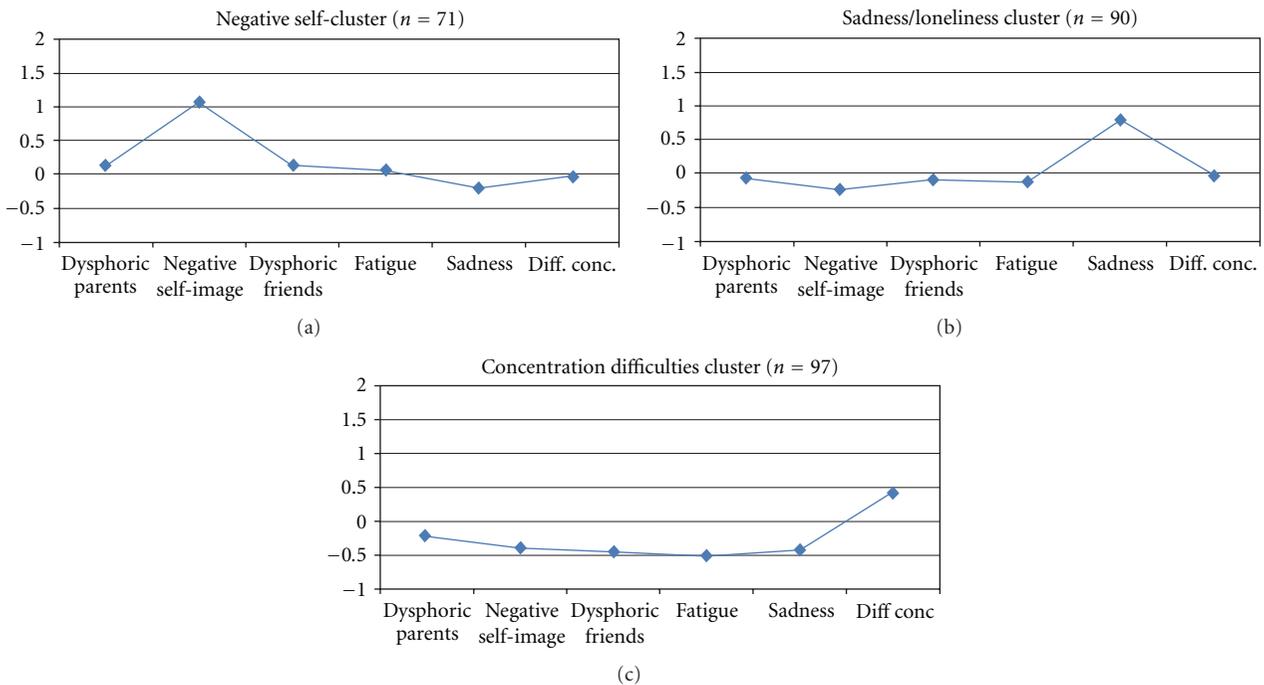


FIGURE 3: One-problem clusters. Profiles in terms of z-scores (where $z = 0$ corresponds to the whole sample’s mean on each subscale).

between depressive symptoms and self-harm in the girls but not in the boys (where there was only support for a unidirectional relationship, depressive symptoms being a predictor of increased self-harm one year later). Second, and in line with expectations, among the ten profiles of depressive symptoms identified in the cluster analysis there were two depression-related clusters with an overrepresentation of girls and significantly increased levels of self-harm and also a single-problem cluster characterized by negative self-image and significantly higher levels of self-harm.

Importantly, the demonstration of a bidirectional prospective relationship between depression and self-harm in girls means that higher levels of one of these variables *at a given time* are associated with increasing levels in the other variable *over time*. Whereas the concurrent analyses showed that *levels* of depression are strongly associated with *levels* of self-harm, the prospective analyses showed that higher levels of depression predispose to *increased* levels of self-harm within the next year and conversely that higher levels of

self-harm predispose to *increased* depression within the next year. Although the effects were not strong, they are of a more dynamic order than the concurrent associations and suggest the possibility that depression and self-harm may enter into a self-generating “vicious cycle” where increases in the one variable lead to increases in the other, and vice versa. This can be described in terms of a dynamic system, where internal feedback processes lead to the emergence and stabilization of pathological patterns of depressive symptoms and self-harm. The absence of evidence for a bidirectional relationship in boys suggests that different developmental dynamics may be involved in girls and boys—or, in other words, that self-harm has a different meaning and function in girls than in boys.

In a previous study [20], we found no support for the hypothesis of emotional problems being a risk factor for the development of self-harm. In that study, emotional problems were measured by the Emotional symptoms subscale of the Strengths and Difficulties Questionnaire (SDQ). The present results corroborate the assumption that the SDQ Emotional

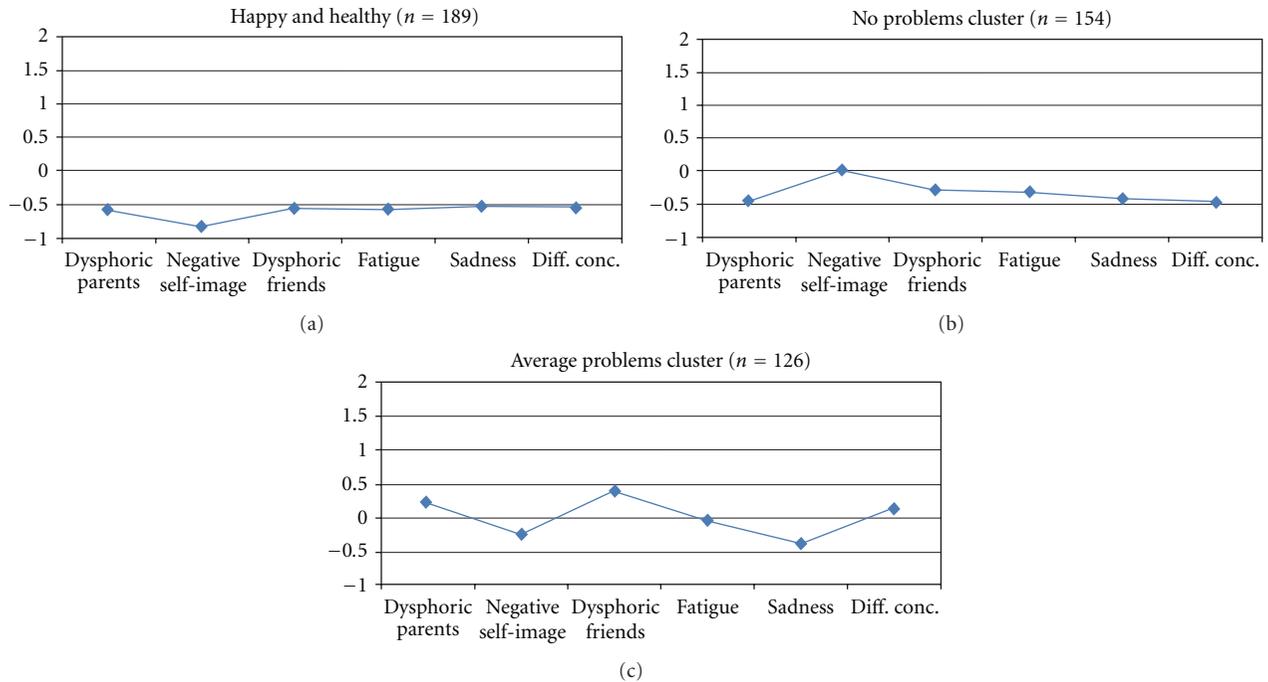


FIGURE 4: Healthy clusters. Profiles in terms of z-scores (where $z = 0$ corresponds to the whole sample's mean on each subscale).

symptoms scale is not sufficiently sensitive to capture the role of depressive symptoms for the development of self-harm, as it contains only one depression-related item.

There are several interesting results from the cluster analytic part of the present study. First, two depression-related clusters were identified, suggesting that 2-3% of the adolescents (27 of 953) in this community sample suffered from depression and that an additional 8-9% (81 of 953) suffered from something that is reminiscent of at least "minor depression." Both of these clusters were characterized by an overrepresentation of girls (74% and 65%, resp.) and were also characterized by significantly higher frequencies of self-harm than most other clusters. The individuals in the depression cluster showed particularly high frequencies of self-harm.

Second, the analysis identified three one-problem clusters of adolescents (the Negative self-cluster, the Sadness/loneliness cluster, and the Concentration difficulties cluster), characterized by high scores on one of the subscales without any elevated scores on the total depression index. In line with expectations, the Negative self-cluster was characterized by significantly increased levels of self-harm, whereas the other two were not. This is consistent with the hypothesis that self-harm is more associated with negative self-related emotions than with feelings of sadness. This finding also suggests the need for more specific theoretical models concerning the emotion-regulating role of self-harm. It is well established that a primary function of deliberate self-harm is the regulation of negative emotions [14]. But are all kinds of negative feelings (fear, sadness, shame, guilt, self-hate, etc.) equivalent in this respect, or are some emotional experiences more likely than others to be handled by self-harm? Because deliberate self-harm involves a direct physical

attack on one's own body, and attack is more associated with anger or aggression than with fear or sadness, it may be hypothesized that self-harm is used primarily to regulate feelings of self-directed anger or aggression. It may be a task for future research to develop more specific theoretical models in this area and to develop instruments for testing these models. It should be noted that the items in the negative self-image subscale of the Depression Index in the present study were not constructed for this purpose and actually deal more with the absence of positive self-feelings than with the presence of negative self-feelings (see the appendix). The fact that this relatively weak index of a negative self-image was still able to produce results in line with the hypothesis suggests that this line of research may be worth pursuing.

A third finding from the cluster analysis is that high scores on fatigue/somatic complaints combined with dysphoric relations to parents and dysphoric relations to friends, respectively, into two separate problem clusters. For both of these, high scores on one of the dysphoric relations factors were combined with completely normal scores on the other. More specifically, the adolescents in the "Fatigue and problems with parents cluster" scored even slightly below average ($z < 0$) on dysphoric relations to friends. And, conversely, the adolescents in the "Fatigue and problems with friends cluster" scored slightly below average ($z < 0$) on dysphoric relations to parents. Whereas the "Fatigue and problems with parents" cluster had elevated scores on self-harm, this was not the case for the "Fatigue and problems with friends" cluster. This suggests that self-harm is more associated with negative relations to parents than with negative relations to friends.

Fourth, the results from the cluster analysis gain further strength by their stability; the clusters defined at Time 1

showed highly similar results on self-harm also at Time 2. Still, it may be asked if the clusters with more circumscribed problems represent stable profiles or if the individuals in these clusters constitute risk groups for developing more depression later on. This could be studied by an analysis of the stability of these clusters from Time 1 to Time 2.

A general comment is that the identification of subgroups like these probably cannot be done by variable-oriented approaches that rely only on the analysis of linear correlations; here, person-oriented methods like cluster analysis may represent an important complement, which make it possible to discover aspects of the data that are hard to detect otherwise.

The present study has several strengths: it uses a large representative sample of adolescents, and there were longitudinal data for over 83% of all adolescents. The study, however, also suffers from several limitations. For example, the study used only two measure points, and it is possible that other results on risk factors would have been obtained if the first measure point had been earlier or if more measure points had been added. Another limitation is that the study relied entirely on self-assessment instruments; a multimethod approach might have made it possible to draw stronger conclusions. Further, the study did not use any established measures of depression but a specially constructed index based on items selected from different parts of a large questionnaire. A content analysis of the items (see the appendix) indicates that most of the criteria of major depression, as defined by the DSM-IV [29], are represented among the items. Two exceptions, however, are the DSM-IV criteria of weight loss or weight gain, and recurrent thoughts of death and suicide. This means that the depression index used in the present study does not do full justice to the psychiatric notion of major depression. On the other hand, the use of the present kind of depression index produced some interesting findings of potential interest to the understanding of adolescent depression, which would probably not have been seen if an established measure of depression had been used. For example, the present results identified dysphoric relations to parents and dysphoric relations to friends as two separate factors, with at least partly different meaning. One limitation with the dysphoric relations to parents subscale, however, is that all the items in this subscale refer to “parents” as the unit and do not allow for the possibility that some adolescents may feel differently towards their mother and father and hence be somewhat confused as to what to respond.

Finally, a possible risk with collecting data in school settings is that insufficient privacy may impact on levels of disclosure and thus threaten the validity and reliability of the results. An alternative possibility would have been to let the participants fill out the questionnaire at home; however, this would enter other ethical concerns and possible threats to response rates, as well as to validity and reliability. Also, the reports from the research assistants who administered the questionnaires gave no reason for concern about negative effects of lack of privacy but indicated that the students in general were very focused on the questionnaire during the lecture hour that was set off for filling it out. Further, the

last page of the 11-page questionnaire included a question with a four-response format asking “how interesting and meaningful” the participants thought it had been to answer the questionnaire; the fact that over 80% of the students responded “very” or “fairly” to this question corroborates the impression that at least a large majority of the students were indeed engaged in the task.

5. Conclusion

To summarize, the present study contributes to the literature in at least two ways. First, it shows evidence of a bidirectional relationship over time between depressive symptoms and self-harm in young girls, although there was only evidence for a unidirectional relationship from depressive symptoms to self-harm among young boys. This suggests the hypothesis that depressive symptoms and self-harm in young girls may form a dynamic system, where feedback processes lead to the emergence and stabilization of self-generating “vicious cycles” of depressive symptoms and repeated self-harm.

Second, the present study uses cluster analysis in a way that gives a partly new perspective on aspects of adolescent depression and depression-related problem patterns. Apart from the identification of two depression-related clusters which were both characterized by an overrepresentation of girls and by elevated levels of self-harm, the analysis also identified a number of clusters with more circumscribed problems, and with different associations with self-harm. For example, the results also suggest that dysphoric relations to parents and to friends represent two separate dimensions that form part of different problem profiles and may be important to differentiate in order to understand the nature of depressive experiences in adolescence. Finally, the results indicate that negative self-image, sadness/loneliness, and difficulties in concentration exist as significant one-problem patterns in relatively large subgroups of adolescents, who do not show evidence of depression; of these, only the Negative self-cluster was associated with elevated levels of self-harm. The latter finding suggests that self-harm may serve to regulate negative self-related emotions rather than feelings of sadness; more research, however, is required on the kind of emotions that are associated with self-harm.

Appendix

Items in the Depression Index and Its Subscales (with Response Format)

Dysphoric Relations to Parents. When I am with my parents (or think about them), I feel:

- (i) sad, disappointed, depressed (Never/Seldom/Often/Very often)
- (ii) bored (Never/Seldom/Often/Very often)
- (iii) angry, irritated (Never/Seldom/Often/Very often)
- (iv) rejected, ignored, badly treated (Never/Seldom/Often/Very often)
- (v) lonely, left out (Never/Seldom/Often/Very often)

- (vi) uneasy, restless (Never/Seldom/Often/Very often)
- (vii) calm, relaxed (Never/Seldom/Often/Very often) REV
- (viii) happy, joyous, glad (Never/Seldom/Often/Very often) REV
- (ix) a sense of belongingness (Never/Seldom/Often/Very often) REV
- (x) liked, loved, cared for (Never/Seldom/Often/Very often) REV.

Negative Self-Image.

- (i) When I am with my parents (or think about them), I feel proud of myself (Never/Seldom/Often/Very often) REV
- (ii) I am ashamed of my looks (Never/Seldom/Often/Very often)
- (iii) I am rather satisfied with my appearance (Never/Seldom/Often/Very often) REV
- (iv) I wish I looked better (Never/Seldom/Often/Very often)
- (v) I am proud of my body (Never/Seldom/Often/Very often) REV
- (vi) How do you think your life will be? (Very good/Rather good/Acceptable/Not very good/Not at all good).

Dysphoric Relations to Friends.

- (i) Other people my age generally like me (Not true/Somewhat true/Certainly true) REV.
- (ii) When I am with my closest friends (or think about them), I feel:
 - (a) happy, joyous, glad (Never/Seldom/Often/Very often) REV
 - (b) proud and sure of myself (Never/Seldom/Often/Very often) REV
 - (c) a sense of belongingness (Never/Seldom/Often/Very often) REV
 - (d) eager and full of energy (Never/Seldom/Often/Very often) REV
 - (e) calm and relaxed (Never/Seldom/Often/Very often) REV.

Fatigue/Somatic Complaints.

- (i) Do you feel alert and energetic during the day? (Always/Most often/Sometimes/Seldom/Never)
- (ii) Do you sleep well? (Always/Most often/Sometimes/Seldom/Never)
- (iii) In general, how would you say your health is? (Very good/Fair/Poor)
- (iv) I get a lot of headaches, stomach aches, or sickness (Not true/Somewhat true/Certainly true)

- (v) When I am with my parents (or think about them), I feel eager and full of energy (Never/Seldom/Often/Very often) REV.

Sadness/Loneliness.

- (i) I am often unhappy, down hearted, or tearful (Not true/Somewhat true/Certainly true).
- (ii) When I am with my closest friends (or think about them), I feel:
 - (a) sad, disappointed, depressed (Never/Seldom/Often/Very often)
 - (b) angry, irritated (Never/Seldom/Often/Very often)
 - (c) lonely, left out (Never/Seldom/Often/Very often).

Difficulties in Concentration.

- (i) I am restless, I cannot stay still for long (Not true/Somewhat true/Certainly true)
- (ii) I am easily distracted, I find it difficult to concentrate (Not true/Somewhat true/Certainly true)
- (iii) I finish the work I am doing. My attention is good (Not true/Somewhat true/Certainly true) REV
- (iv) When I am with my closest friends (or think about them), I feel uneasy, restless (Never/Seldom/Often/Very often).

Acknowledgments

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Research Article

Depression with and without Comorbid Substance Dependence in a Child Welfare Sample of Young Adults

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The objective of this study was to estimate the prevalence of depression with and without substance dependence and examine the effect of risk factors on subsequent disorders among a cohort of young adults in the US Child Welfare System (CWS). We used longitudinal data for 834 young adults age 18–21 from the National Survey of Child and Adolescent Well-being. Depressive symptoms and substance use were measured at baseline (age 11–15); diagnoses of depression and substance dependence were identified at the last wave of data collection (age 18–21). Likelihood of subsequent depression with or without substance dependence was three times higher for those with clinically significant depressive symptoms at baseline. Frequent use of substances at baseline significantly increased the likelihood of subsequent depression with comorbid substance dependence compared to depression alone. These results support screening youth in the CWS at younger ages for both depressive symptoms and substance use with the hope that these disorders can be detected earlier.

1. Introduction

The US Child Welfare System (CWS) sees a large number of youth with mental health problems because they have often experienced maltreatment and other risk factors such as poverty and separation from caregivers [1–3]. A study of youth age 2 to 14 years ($n = 3,803$) in the US CWS estimated that nearly half had significant emotional or behavioral problems upon entering the system [3]. A smaller study of 426 youth age 6 to 18 years in the CWS in San Diego County estimated the prevalence of major depression to be 4.7% [4].

Depression and substance use disorders are known to occur together more often than not [5–8]. In clinical samples, comorbid depression is seen in 70–80% of adolescents with substance use disorder [9]. In studies of depression, substance use is often found to significantly increase the risk of depression. One such study found that among youth in grades 6, 8, and 10 ($n = 9,863$), substance use significantly increased the risk of depression, with relative risks ranging from 2.5 to 3.1 depending on the type of substance used

[10]. Comorbid depression and substance use disorder is a risk factor for poor treatment outcomes, treatment drop-out, and earlier relapse [11]. Youth with depression and comorbid substance use disorder also have an increased risk for suicidal behaviors, including ideation, attempt, and completed suicide [12].

The estimate of major depression prevalence among youth in the San Diego County CWS (4.7%) resulted from a multisector study in San Diego County which randomly selected youth age 6 to 18 years who were active in at least one of the following systems: Child Welfare, Substance Abuse programs, Juvenile Justice, Specialty Mental Health, and Public-School programs for youth with serious emotional disturbance. While the prevalence of major depression among youth in Child Welfare was 4.7%, it was estimated to be nearly double (8.3%) among youth in Substance Abuse programs [4]. Focusing on youth aged 13 to 18 in the San Diego County multisector study, lifetime and past year nontobacco substance use disorder were estimated to be 19.2% and 11.0%, respectively, among youth in

Child Welfare [13]. The prevalence of lifetime and past year substance use disorder among youth in the Specialty Mental Health sector was more than double (40.8% and 22.9%, respectively). Although comorbidity of depression and substance use disorder was not directly reported, these results support a possible co-occurrence in these youth [4, 13].

A recent study used data from the National Survey of Child and Adolescent Well-Being (NSCAW) to describe depressive symptoms and substance use among youth in the CWS [14]. Among a sample of 1,179 NSCAW youth aged 11 to 15 years at baseline, 9% had clinically significant depressive symptoms, 5% had problems with frequent substance use, and 4% had comorbid depressive symptoms and frequent substance use. Depressive symptoms were measured using the Children's Depression Inventory (CDI), and frequent substance use was based on the youth self-report of type and frequency of substances used in the past 30 days. The study found that clinically significant depressive symptoms were more than twice as common in girls as boys (17% versus 7%, $P < .05$), but there were no gender differences in the prevalence of past month substance use. Differences in depression symptoms and substance use severity were found between groups; boys with clinically significant depressive symptoms and frequent substance use had more severe depression symptoms compared to boys with clinically significant depressive symptoms without frequent substance use.

Two main developmental hypotheses attempt to explain the comorbidity between depression and substance use disorders. First, depression develops as a consequence of a substance use disorder; and second, depression precedes the substance use disorder [8]. The latter is often referred to as the self-medication hypothesis and has found less support in the current literature. For example, a study using AddHealth examined the ordering of depression symptoms and tobacco use and found a significant association where tobacco use preceded subsequent depressive symptoms; the reverse association was not supported [15]. A longitudinal study of Australian school-aged youth found that marijuana use strongly predicted later depression; again, the reverse relationship was not supported [16].

Depression and substance use disorder in youth are both more likely in the presence of stressors and share many risk factors [8]. Having a caregiver with a mental health problem has been found to increase the risk of the youth having emotional or substance use problems themselves [17–20]. A prior study using NSCAW found a significant association between caregiver depression and substance use problems and youth internalizing problems [2]. Child maltreatment in general is known to increase the likelihood of both depression and substance use disorder in youth [21–23]. Type of maltreatment has also been found to be associated with these mental health problems. One study in particular found that sexual abuse and neglect significantly increased the risk of substance use disorder among a sample of youth in the CWS in San Diego County [24]. Other risk factors unique to youth in the CWS are placement instability and number of out-of-home placements, which have been found

to be associated with well-being outcomes [25], behavior problems [26], child maladjustment [27], and substance use disorder [24].

NSCAW has paved the way for filling gaps in child welfare mental health research by providing a large, nationally representative sample of youth in the US CWS with a wide range of outcomes and risk factors measured longitudinally for youth and their families. However, until the most recent (fifth) wave of data collection, no diagnostic instrument for obtaining clinical indications of depression or substance use disorder was included in the youth interview. Therefore, all estimates of depression and substance use disorders have relied on instruments such as the Child Behavior Checklist, the CDI, and self-report of substance use. Wave 5 of NSCAW data collection included a young adult interview given to the cohort of NSCAW youth who were age 18 or older by April 20, 2006. This interview included an instrument that resulted in a clinical diagnosis of both depression and substance dependence not previously available for this cohort.

The goal of the current study was to build on prior work of the authors [14] and use the newest data from Wave 5 of NSCAW to fill the following gaps: (1) estimate the prevalence of major depression with and without comorbid substance dependence among young adults in NSCAW, (2) estimate the effect of depressive symptoms and frequent substance use at baseline of NSCAW on subsequent depression with and without substance dependence, and (3) examine the effect of other possible risk factors measured at baseline of NSCAW on subsequent depression with and without comorbid substance dependence. Risk factors were chosen based on those found to be associated with depression and/or substance use disorder in the current literature, and particularly those specific to children in the child welfare system: caregiver mental health and substance use problems, out-of-home placements, and type of maltreatment experienced.

The authors hypothesized that depressive symptoms and patterns of substance use at baseline would be significantly associated with the presence of a subsequent clinical diagnosis of depression and/or substance dependence. Specifically, the authors sought to determine whether baseline depressive symptoms and substance use would increase the likelihood of subsequent comorbid diagnoses among youth with subsequent depression. Results from this study will help focus screening and intervention efforts of children as they enter and navigate the CWS.

2. Materials and Methods

2.1. Study Cohort. Data for this study were drawn from NSCAW, a longitudinal study of youth and their families who came into contact with the US CWS between October 1999 and December 2000. At baseline (Wave 1), 5,501 youth aged birth to 15 years were interviewed, as were their caseworker, primary caregiver, and teacher (if applicable). The youth were then followed for up to four additional waves of data collection: 12-month followup (Wave 2), 18-month followup (Wave 3), 36-month followup (Wave 4), and a final wave of data collection based on age cohort

(Wave 5). Wave 5 included the Young Adult Interview, a special instrument administered to youth who had turned 18 years old by April 2006. Data for the current study were drawn from the youth, caregiver and caseworker interviews at baseline, and the Young Adult Interview at Wave 5.

The current study was built on a previous study for which the focus was the comorbidity of depression symptoms and substance use at baseline among a cohort of 1,179 NSCAW youth age 11 to 15 years [14]. The Young Adult Interview administered at Wave 5 did not include the same measurements of depressive symptoms and substance use as available at baseline but did include measurements of clinical depression and alcohol and drug dependence. From the previous study's cohort of 1,179, a total of 834 youth had aged into the young adult cohort at Wave 5 and were administered the diagnostic instruments; the current study included these 834 youth. The number of months between the baseline interview and the Wave 5 Young Adult Interview ranged from 64 to 93 (mean = 74 months). Compared to the 345 youth excluded, the study cohort ($n = 834$) had significantly more females and significantly higher depressive symptoms at baseline. The groups did not differ with respect to any other baseline characteristics.

2.2. Measures. *Depressive symptoms* were measured at baseline for youth age 7 and older through administration of the Children's Depression Inventory (CDI) [28]. According to a meta-analysis of 310 datasets which included 61,424 youth age 8 to 16 (29,637 boys), the CDI is the most commonly used instrument to assess depression in youth [29]. The CDI includes 27 items addressing a range of depressive symptoms, each answered on a 3-point scale (0 = absence of symptom, 1 = mild symptom, 2 = definite symptom). The sum of all 27 responses was used to create a total raw score, which was converted to a standardized t -score ranging from 0 to 100 using the standardized tables in Kovacs' CDI Profile Form [28]. Internal consistency for the CDI was examined for the NSCAW sample and found to be good, with a mean internal consistency of 0.81 for youth age 7 to 12 years old and 0.87 for 13 to 15 years old [30]. A total score of 66 and greater was used to indicate *clinically significant depressive symptoms*, as done by the experts involved in the development of NSCAW [30].

Substance use (type and frequency) was measured at baseline for youth age 11 years and older through a set of questions regarding lifetime and past month use of alcohol, tobacco, and other substances. In order to assure confidentiality, these items were administered using an Audio Computer-Assisted Self-Interview program, which allowed the youth to answer the substance use items without the interviewer knowing their answers. Youth were first asked if they had used a particular substance in their lifetime. Those who indicated they had used that substance at least once were then asked on how many days they had used that substance in the past 30 days (none, 1 day, 2 days, 3–5 days, 6–11 days, 12–19 days, and 20 or more days). The youth were asked about each of the following substances: tobacco (cigarettes

and chewing tobacco); alcohol; marijuana; inhalants (glue, gasoline, and other liquids and gases); hard drugs (cocaine, crack, and heroin); nonmedical use of prescription medications such as pain killers, tranquilizers, stimulants, and sedatives.

Since a clinical interview that would result in a diagnosis of substance dependence was not administered to the youth at baseline, severity of substance use was instead characterized using the number of days in the past month substances were used. Specifically, *frequent substance use (FSU)* was defined as use of one or more substances, including tobacco, for 20 or more days in the past month.

Depression symptom and FSU comorbidity was identified at baseline using four mutually exclusive groups: neither clinically significant depressive symptoms (CDI score ≥ 66) nor FSU; clinically significant depressive symptoms (CDI score ≥ 66) only; FSU only; clinically significant depressive symptoms (CDI score ≥ 66) comorbid with FSU.

Diagnoses of major depression and substance dependence in the past 12 months were obtained for young adults at Wave 5 through administration of a structured clinical interview using the Composite International Diagnostic Interview—Short Form (CIDI-SF) [31]. The CIDI-SF resulted in diagnoses for major depression, alcohol dependence, and drug dependence, each based on DSM-IV criteria. Diagnoses of alcohol and drug abuse were not assessed with the CIDI-SF. Alcohol dependence and drug dependence were assessed separately; a participant was considered to have substance dependence if either alcohol dependence or drug dependence was present. Drugs included in the dependence diagnosis were inhalants, marijuana, cocaine, heroin, hallucinogens, and prescription medications without a prescription, such as sedatives, tranquilizers, amphetamines/stimulants, and prescription pain killers. Tobacco was not included in the substance dependence diagnosis.

Caregiver mental health and substance use problems were assessed at baseline through both caregiver self-report and caseworker report. Caregivers were administered the CIDI-SF [31] to assess major depression and substance dependence. In addition to these clinical diagnoses, caseworkers indicated whether or not the caregiver had serious emotional problems or substance abuse problems at baseline. These two sources of data (caregiver report and caseworker report) were combined to create a summary indication of caregiver depression or mental health problems and a summary indication of caregiver substance use problems at baseline. For each, if either the caregiver self-report of the caseworker report indicated the problem, the combined summary variable was set to present.

Demographic characteristics of the youth included gender, age (in years at baseline), and race/ethnicity (White, Black, Hispanic, and other (including American Indian and Asian)). Placement of the youth (in-home versus out-of-home) was known at baseline; a count of out-of-home placements for the youth between baseline and Wave 5 was also available. The type(s) of maltreatment experienced by the child and included in the caseworker's initial report to the CWS was indicated by the caseworker at baseline using a modified Maltreatment Classification Scale [32]. Categories

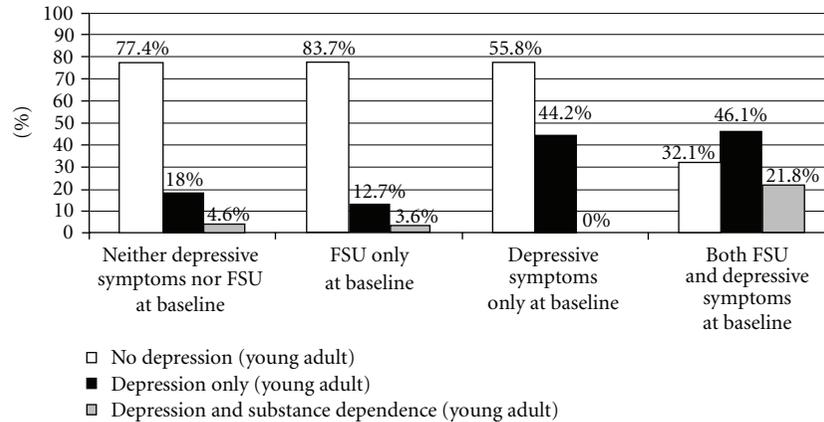


FIGURE 1: Prevalence of depression with or without substance dependence as a young adult by depressive symptoms and substance use at baseline.

included physical abuse, sexual abuse, emotional abuse, physical neglect (i.e., failure to provide), nonphysical neglect (i.e., failure to supervise), and other types of neglect (i.e., abandonment, moral or legal maltreatment, educational maltreatment, and exploitation). More than one type of maltreatment could have been reported for each youth.

2.3. Statistical Analysis. The NSCAW study design included a two-stage, complex sampling process in which the US was first divided into strata, and then each stratum was divided into primary sampling units. In order to adjust for this complex sample design and calculate population-based estimates representative of the youth in the US CWS, analysis weights and other study design variables were incorporated into all analyses, which were completed using STATA 10 [33]. Additional detailed information about the NSCAW study design and weight derivation is published elsewhere [34, 35].

Characteristics of the study cohort were described and compared by depression and substance dependence status at Wave 5 using weighted chi-square tests and t-tests. Separate weighted multivariate logistic regression models were used to estimate the conditional likelihood of subsequent depression (with or without substance dependence) compared to no depression at Wave 5, and the conditional likelihood of depression without substance dependence compared to depression with substance dependence at Wave 5. The final multivariate models reported included demographic variables regardless of significance, and other variables found to be statistically significant at $P < .05$. All percentages, odds ratios, confidence intervals, and statistical tests reported are weighted, while all sample sizes are reported as unweighted.

3. Results and Discussion

3.1. Description of Study Cohort. The study cohort included 834 youth age 11 to 15 years old (mean = 12.8 years) at baseline of NSCAW; ages ranged from 18 to 21 years old at Wave 5 (mean = 19.1 years). Slightly more than half the cohort (58%) was male. The cohort was ethnically diverse, with 50% being White, 28% being Black, 15% being Hispanic, and

7% being of another race/ethnic group. The majority of the cohort was living at home at baseline (87%); 82% had no out-of-home placements between baseline and their young adult survey, which occurred between 64 and 93 months following baseline (mean = 74.5 months). At baseline, the caseworkers reported all types of maltreatment that had been reported in the initial investigation; 41% of the youth had been physically abused, 12% had been sexually abused, 14% had been emotionally abused, 19% had been physically neglected, 38% had experienced nonphysical neglect, and the remaining 16% were reported to have experienced some other form of maltreatment or neglect. The mean number of different types of maltreatment reported per youth was 1.4, indicating some had experienced more than one type of maltreatment prior to the investigation.

3.2. Depression and Substance Dependence. Twenty-six percent ($n = 208$) of the study cohort had major depression at Wave 5 (21% without substance dependence; 5% comorbid with substance dependence). At baseline, depressive symptoms and substance use were assessed through self-report. Based on prior work [14], youth in the current study cohort were identified as being in one of four groups at baseline: clinically significant depressive symptoms only (10.0% at baseline), frequent substance use (FSU) only (5.3% at baseline), both clinically significant depressive symptoms and FSU (4.3% at baseline), or neither clinically significant depressive symptoms nor FSU (80.4% at baseline). For each of these four groups, prevalence of major depression with and without substance dependence at Wave 5 is presented in Figure 1. Prevalence of depression without substance dependence as a young adult was highest in the youth who had clinically significant depressive symptoms with or without FSU at baseline. Prevalence of depression with substance dependence as a young adult was highest in the group with both clinically significant depressive symptoms and FSU at baseline. The overall association between depressive symptoms and FSU at baseline and subsequent depression with or without substance dependence was statistically significant ($\chi^2(6) = 490.53, P < .01$).

3.3. Unadjusted Associations. Unadjusted associations between baseline characteristics of the youth and their caregivers and subsequent depression with and without substance dependence are presented in Table 1. There were significantly more girls with subsequent depression (with or without substance dependence) compared to no subsequent depression ($P < .05$). There was no significant effect of gender, however, on having subsequent depression without substance dependence compared to subsequent depression comorbid with substance dependence. FSU (20 + days in the past 30 days) at baseline was seen significantly more in the group with subsequent depression (with or without substance dependence) for all substances except tobacco and marijuana. Frequent substance use at baseline also significantly differentiated between youth with subsequent depression without substance dependence compared to youth with both depression and substance dependence. Youth with comorbid depression and substance dependence were significantly more likely to have used alcohol, inhalants, hard drugs, and prescription medication for nonmedical purposes at baseline than those with depression only. Baseline depressive symptoms were also significantly associated with subsequent depression. Among the youth with subsequent depression (with or without substance dependence), 28% had clinically significant depressive symptoms at baseline, compared to only 10% of youth without subsequent depression ($P < .01$). Baseline depressive symptoms were not significantly associated with subsequent depression alone versus subsequent depression comorbid with substance dependence.

Type of maltreatment experienced by the youth at baseline was not significantly associated with subsequent depression with or without substance dependence. Being in an out-of-home placement at baseline or any time between baseline and Wave 5 was also not associated with depression (with or without substance dependence) or associated with depression without substance dependence versus depression comorbid with substance dependence (see Table 1). However, the number of out-of-home placements between baseline and Wave 5 was significantly associated with depression without substance dependence versus depression comorbid with substance dependence. Youth with subsequent comorbid depression and substance dependence had significantly fewer out-of-home placements between baseline and Wave 5 (mean = 1.8) compared to those with subsequent depression without substance dependence (mean = 3.2; $P < .05$).

Having a caregiver with a substance use problem at baseline was also significantly associated with comorbid depression and substance dependence at Wave 5. Significantly fewer youth with subsequent depression and substance dependence had a caregiver at baseline with a substance use problem compared to youth with subsequent depression without substance dependence. These unadjusted results were further explored using multivariate logistic regression (see Table 2).

3.4. Adjusted Associations. The likelihood of subsequent depression (with or without substance dependence) and

of subsequent depression comorbid with substance dependence, conditional on baseline depressive symptoms and substance use, was estimated using multivariate logistic regression (see Table 2). Age, gender, and race/ethnicity of the youth were adjusted for in the multivariate models. After adjusting for these baseline characteristics, number of out-of-home placements and caregiver substance use problems at baseline no longer had a significant effect on subsequent depression comorbid with substance dependence and were therefore excluded from the final multivariate model reported in Table 2. However, likelihood of depression with or without substance dependence (compared to no depression) was nearly three times higher for those with clinically significant depressive symptoms at baseline than those without clinically significant depressive symptoms (OR = 2.6, $P < .01$); baseline depressive symptoms did not have a significant effect on a subsequent depression diagnosis comorbid with substance dependence compared to a depression diagnosis only. Frequent use of alcohol at baseline also significantly increased the likelihood of subsequent depression with or without substance dependence (OR = 21.0, $P < .01$). Frequent use of any nontobacco substance at baseline significantly increased the likelihood of subsequent depression with comorbid substance dependence compared to depression alone (OR = 37.2, $P < .01$).

4. Conclusions

Prevalence of major depression among the current cohort of young adults age 18 to 21 years at Wave 5 of NSCAW was 26%; 18% of these youth also had comorbid substance dependence. In the National Comorbidity Study (NCS), past year major depression was seen in 21.2%, 11.7%, and 15.6% of young adults and adults age 17 to 18, 19 to 20, and 21 to 22 years, respectively [36]. Prevalence of major depression in the past 12 months was estimated to be 6.6% for adults age 18 years and older in the NCS-R; 8.5% of those with 12-month major depressive disorder also had comorbid substance dependence in the past 12 months [37]. Prevalence of major depression overall, and prevalence of comorbid substance dependence among those with major depression, was higher in the current study's cohort of young adults from NSCAW. This is consistent with other studies in the literature that have found more depression and emotional problems among youth in the CWS. The only other study known to have estimated the prevalence of major depression among youth in the CWS included youth age 6 to 18 years old in the San Diego County CWS and found a much lower prevalence of depression, only 4.7% [4]. However, this number is difficult to compare to the current study because of the age difference between the two study cohorts.

Results of this study support the idea that some aspects of the CWS may in fact be protective against disorders among youth in the system [38]. Youth with a caregiver with substance use problems at baseline were significantly less likely to have depression with substance dependence, compared to depression only, at Wave 5 of NSCAW. The number of out-of-home placements was also found to be protective, with

TABLE 1: Associations between baseline youth and caregiver characteristics and subsequent depression ($n = 834$).

	Total sample ($n = 834$)		Depression ($n = 208$)	
	No depression ($n = 626$)	Depression ^a ($n = 208$)	Depression only ($n = 173$)	Depression + substance dependence ($n = 35$)
<i>Youth demographic characteristics</i>				
Age in years at baseline (range 11–15)				
Mean (SE)	12.80 (0.08)	12.92 (0.14)	12.85 (0.16)	13.21 (0.32)
Female (%)	54.33	69.75*	71.56	61.72
Race/ethnicity (%)				
White	49.23	53.27	49.56	68.96
Black	30.83	18.17	19.81	11.24*
Hispanic	13.91	17.13	17.77	14.44
Other	6.04	11.43	12.86	5.37
Initial type of maltreatment (%)				
Physical abuse	43.6	35.2	32.43	45.6
Sexual abuse	11.54	14.42	12.06	23.3
Emotional abuse	14.38	14.08	15.14	10.09
Physical neglect	17.85	23.05	25.75	12.93
Neglect	36.46	41.17	43.54	32.25
Out-of-home placement				
At baseline (%)	12.07	13.94	11.75	23.69
Ever (baseline to W5) (%)	16.25	22.16	20.14	30.37
If ever had OOH placement:				
Mean no. placements (SE)	2.75 (0.16)	2.80 (0.41)	3.18 (0.50)	1.78 (0.34)*
Range	1–11	1–18	1–18	1–7
<i>Caregiver characteristics at baseline</i>				
Substance use problem (%)	15.52	9.48	10.96	2.77*
Depression/MH problem (%)	37.02	38.4	41.37	25.13
<i>Youth substance use and depressive symptoms</i>				
Substance use at baseline (% who reported 20+ days in past 30 days)				
Alcohol	0.14	3.86**	0.12	20.55**
Tobacco	8.06	14.08	12.12	22.56
Marijuana	1.09	4.52	0.67	21.06**
Inhalants	0.19	3.61**	0.001	18.87**
Hard drugs	0.13	3.57**	0	18.87**
Nonmedical use of Rx	0.11	3.58**	0	18.87**
Mean no. substances used (SE)	0.09 (0.02)	0.32 (0.20)	0.12 (0.05)	1.20 (0.91)
FSU at baseline (%)	7.91	14.34	12.36	23.13
Clinically significant depressive sx at baseline (%)	9.46	27.62**	29.48	19.29

Abbreviations: SUD: substance use disorder; Rx: prescription; FSU: frequent substance use; sx: symptoms.

** $P < .01$; * $P < .05$.

^a Depression with and without substance dependence.

more out-of-home placements being significantly associated with a decreased risk of depression comorbid with substance dependence compared to depression alone. It's important to note that these significant associations were unadjusted; after other covariates such as age, gender, and baseline depressive symptoms and substance use were included in the model, these associations were no longer significant. However, they

are important to consider when planning future research and interventions. Youth with caregivers who have a substance use problem when they enter the system may be more visible and therefore have a higher chance of receiving preventive services throughout their experience in the system.

A similar argument may be made for number of out-of-home placements. The more often the youth moves in

TABLE 2: Conditional likelihood of subsequent depression with or without substance dependence.

	Depression with or without substance dependence ^a		Depression with comorbid substance dependence ^b	
	OR	(95% CI)	OR	(95% CI)
Model N		793		205
<i>Youth demographic characteristics</i>				
Age in years at baseline	1.01	(0.81, 1.25)	1.1	(0.65, 1.86)
Female	1.64	(0.88, 3.06)	1.22	(0.29, 5.16)
Race/ethnicity				
White (referent group)				
Black	0.7	(0.29, 1.70)	0.53	(0.11, 2.63)
Hispanic	1.26	(0.47, 3.38)	0.75	(0.11, 4.96)
Other	2.02	(0.84, 4.82)	0.19	(0.02, 1.93)
<i>Youth substance use and depressive symptoms</i>				
Substance use at baseline (20+ days in past 30)				
Alcohol	21.03	(2.22, 199.22)**	—	
Any nontobacco substance	—		37.25	(3.33, 416.54)**
Clinically significant depressive sx at baseline	2.63	(1.08, 6.38)**	—	

Abbreviation: sx: symptoms.

— Not included in model.

** OR significantly different from 1.0, $P < .01$.

^aReferent group: no depression.

^bReferent group: depression without substance dependence.

the system, the more visible they are to the caseworkers and the more likely they may be to get preventive or treatment services. The CWS is thought to be a gateway to service for these reasons [38]. However, the association between out-of-home placements and substance use disorders among youth has been somewhat inconsistent in research to date. One study of youth age 13 to 18 years in the San Diego County CWS found a marginal association between number of out-of-home placements and severity of substance use problems [24]. Another study of adolescents in foster care found no association between placements and drug use [39]. These inconsistent results support the need for further research on the impact of out-of-home placements on subsequent disorders in youth.

Multivariate models found that youth with clinically significant depressive symptoms at baseline were significantly more likely to have major depression at Wave 5, but such depressive symptoms did not make those youth more or less likely to have depression and substance dependence compared to depression alone. These results were adjusted for baseline substance use and demographic correlates such as gender, age and race/ethnicity. Frequent alcohol use significantly increased the risk of subsequent depression, while frequent use of any nontobacco substance significantly increased the risk of depression comorbid with substance dependence versus depression alone among those youth with major depression at Wave 5.

This study found that youth who presented to the CWS with clinically significant depressive symptoms were more likely to have a subsequent diagnosis of depression. Other studies have made similar conclusions. One such study found that adolescent depression significantly predicted adult depression, but that the effect was accounted for by other comorbid diagnoses such as substance use disorder [40]. In the current study, youth who presented with frequent use of nontobacco substances were more likely to have subsequent comorbid depression and substance dependence. With both depressive symptoms and substance use at baseline significantly predicting subsequent clinical disorder, the time after the youth enter the CWS presents a window of opportunity for intervention and treatment that may decrease the likelihood of these youth having subsequent disorders.

4.1. Strengths and Limitations. The limitations of this study are tied to the challenges associated with using pre-existing data such as that from NSCAW. The instruments administered to the youth at baseline did not include a diagnostic assessment of depression or substance dependence. Substance use was measured through self-report as type and frequency during lifetime and the past 30 days. Depressive symptoms were measured using a validated instrument, the CDI, which provided a guideline for identifying youth

with clinically significant depressive symptoms but did not provide an actual diagnosis based on DSM criteria. The addition of the CIDI-SF to the young adult interview at Wave 5 provided a subsequent clinical diagnosis of major depression and substance dependence. However, because substance *abuse* was not included in the diagnosis, this study may have underestimated the scope of substance use disorders in this population.

The young adults who received the CIDI-SF at Wave 5 did not also receive the interview section on type and frequency of substances use, or the CDI, so direct correlations between the two types of measurements could not be investigated. We were, however, able to estimate the association between the baseline measurements of depressive symptoms and substance use with Wave 5 diagnoses of major depression and substance dependence, which is not known to have been done before.

Compared to the 345 subjects excluded, the study cohort ($n = 834$) had significantly more females and significantly higher depressive symptoms at baseline. The groups did not differ with respect to any other baseline characteristics. These results indicate that the 834 young adults included in the current study may have been more likely to have depression at Wave 5 because they had higher depressive symptoms scores at baseline and included more females. However, it is unknown how the exclusion of the 345 subjects may have biased the results. Another limitation results from the small sample size among the youth with depression at Wave 5; only 35 youth had comorbid depression and substance dependence, possibly resulting in a lack of power to detect significant associations among that group.

Other limitations of the current study arise from measures not included in analyses. Reports of services received by the youth are collected from the caseworker, caregiver, and youth at each wave of NSCWA data collection. However, these services cannot be tied to specific diagnoses. Therefore, treatment received by the youth was not controlled for in the current analyses. Although an in-depth analysis of the services received by youth with depression and/or substance dependence is possible, it is outside the scope of the current study. Also excluded from this study were measures of external behaviors, such as conduct disorder, which are known to be associated with substance use disorders among young adults [41, 42]. Although outside the scope of the current study, further research that considers externalizing disorders as a possible moderator or mediator of the association between baseline depressive symptoms and substance use and subsequent clinical diagnoses is warranted.

NSCAW is a large, nationally representative sample of youth in the US CWS and provides numerous measurements for youth and their caregivers. The availability of child maltreatment data reported by the caseworker at baseline avoided having to rely on self-report from the youth or their caregiver. The cohort included in this study was old enough at baseline to report their own substance use and depressive symptoms, so the less reliable caregiver reports did not need to be used. Overall, NSCAW provides a unique, large dataset with numerous opportunities to explore mental health among youth in the child welfare system.

4.2. Policy Implications. A recent systematic review for the US Preventive Services Task Force concluded that regular screening of adolescents for depression could lead to increased and earlier diagnosis and greater improvement of symptoms [43]. The results of the current study support that conclusion, finding a significant association between depressive symptoms at baseline of NSCAW and subsequent major depression. Further, youth who reported frequent use of nontobacco substances at baseline were significantly more likely to have both depression and substance dependence at Wave 5 compared to depression alone. Although these results cannot be generalized to youth outside the US Child Welfare System, they lend support to the idea that screening youth at younger ages for both depression symptoms and substance use may lead to prevention or earlier detection of depression and substance disorders. Substance use disorders and depression are often comorbid, and treatment of one may lead to improvement of the other [44, 45]. By screening for both substance use and depression when youth are admitted to the Child Welfare System, the problems with which the youth are struggling at that time can be identified and appropriate help can be made available.

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Research Article

Differences in Characteristics and Treatment Received among Depressed Adolescent Psychiatric Outpatients with and without Co-Occurring Alcohol Misuse: A 1-Year Follow-Up Study

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Objectives. We aimed at examining the differences between depressed psychiatric adolescent outpatients with and without cooccurring alcohol misuse in psychosocial background, clinical characteristics, and treatment received during one-year followup. Furthermore, we investigated factors related to nonattendance at treatment. **Materials and Methods.** Consecutive 156 adolescent (13–19 years) psychiatric outpatients with a unipolar depressive disorder at baseline were interviewed using structured measures at baseline and at 12 months. Alcohol misuse was defined as having an AUDIT score of 8 or more points. The outpatients received “treatment as usual” of clinically defined duration. **Results.** Among depressive outpatients, poor parental support, parental alcohol use and decreased attendance at treatment associated with alcohol misuse. The severity of alcohol use as measured by AUDIT-score was the strongest factor independently predicting nonattendance at treatment in multivariate analysis. **Conclusions.** Alcohol misuse indicates family problems, has a deleterious effect on treatment attendance, and should be taken into account when managing treatment for depressive adolescent outpatients.

1. Introduction

Depressive disorders are common among adolescents with an estimated point prevalence of about 3% to 8%, and with a lifetime prevalence of approximately 20% by the end of adolescence [1–3]. Among adolescents, excessive substance use often co-occurs with depressive illness [4], alcohol being the most common substance used among Finnish adolescent population [5, 6]. Studies have found positive associations of alcohol use frequency and recurrent intoxication among depressed adolescents and of early-onset depressive disorders with elevated levels of later addictive substance use [5]. It has been reported that 10–25% of depressed subjects have cooccurring substance use disorder (SUD) both in general

and clinical populations with clearly higher figures in clinical populations [7, 8].

There is evidence that comorbid conditions increase utilization of psychiatric services, complicate treatment, and have a negative impact on the course and prognosis of both disorders [9–13]. Depression and SUD each, and their cooccurrence in particular, are associated with psychosocial impairment, more severe symptoms, and increased suicidality [4, 14–19]. The growing literature emphasizes the clinical importance of substance use not reaching the diagnostic threshold in young people [10, 20] as it seems to associate with a greater number of other psychiatric symptoms [20].

Despite recognized risks of adolescent substance use among depressed juvenile patients, studies focusing on

treatment of cooccurring depression and SUD are still scarce [21]. The outcome of treating either disorder alone is known to be unsatisfactory [22, 23]. Cornelius et al. [24] suggested that psychological intervention should be considered first-line treatment for this population, with pharmacotherapy offered to those who do not respond to this intervention alone. In the study by Riggs et al. [25], the combination of fluoxetine and cognitive behavioural therapy (CBT) showed superior efficacy to that of placebo and CBT for MDD according to changes of the Childhood Depression Rating Scale-Revised in adolescents with SUD. The relative gap of our knowledge is partly due to historical separation of and lack of coordination between substance and mental health treatment programs in many countries. Furthermore, in controlled treatment studies, patients with comorbid disorders are usually excluded [21, 26]. In psychiatric clinical practise, it seems that substance-related problems are not detected at all or diagnosed too late leading to a lack or delay of treatment, as Couwenbergh et al. have pointed out [27]. For the development of specific treatments for these adolescents with comorbid disorders, more research is needed.

The present study seeks to add to the limited research on treatment of depression in the context of cooccurring alcohol use and on factors related to nonattendance at treatment in depressed adolescents. We analyzed data from a Finnish naturalistic one-year follow-up study of depressed adolescent outpatients. Our aim was to examine the differences in psychosocial background characteristics and treatment received during the one-year follow-up time of depressed psychiatric outpatient adolescents with and without cooccurring alcohol misuse and to investigate factors related to nonattendance at treatment in this population.

2. Materials and Methods

2.1. Subjects and Measures. This study forms part of the Adolescent Depression Study (ADS), a collaboration study between the Department of Adolescent Psychiatry of Peijas Medical Health Care District (PMCD) of Helsinki University Central Hospital and the Department of Mental Health and Substance Abuse Services of the National Institute for Health and Welfare (former National Public Health Institute), Finland. The study protocol was accepted by the ethics committees of Helsinki University Central Hospital and PMCD.

The ADS is a naturalistic clinical research and development project on adolescent depressive mood disorders in a sample of consecutive adolescent psychiatric outpatients. The study population was drawn from the PMCD's two adolescent psychiatric outpatient clinics between February 1, 1998 and December 31, 2001. PMCD covers approximately 210,000 inhabitants (about 15% adolescents) of the Helsinki metropolitan area in southern Finland. The outpatient clinics in the PMCD offer secondary care to all 13- to 19-year-old citizens. Patients are mainly referred from schools, health care centers, and social and family counseling services. Subjects believed to have a predominant and severe substance use disorder (SUD) and the most severe eating disorders are

offered treatment elsewhere in specialized units. Finland has universal access to health care, and adolescent psychiatric care is free of charge for the patient. The outpatient clinics offer eclectic psychiatric treatment including individual supportive therapy, family consultations, and psychotropic medication when appropriate.

The screening and diagnostic procedure and the study population ($n = 218$) of the ADS study have been described in more detail previously [3, 28]. In brief, of the 774 consecutive outpatients, 660 (85.3%) were considered eligible. The exclusion criteria were age below 13 or over 19 years, mental retardation, insufficient knowledge of the Finnish language, or admission including no individual appointments. The patient sampling for ADS involved screening all patients by the Beck Depression Inventory-21 [29] and the General Health Questionnaire-36 self-report measures [30]. Studies have supported the reliability and validity of BDI-21 and GHQ-36 also among adolescent populations [31–34]. The sum scores ≥ 10 on the BDI-21 and ≥ 5 on the GHQ-36 were considered screening positives and were invited to participate. Screening positives were fully informed of the study project, and written informed consent was requested from both participants and their parents from those under 18 years of age. Of the eligible patients, 373 (56.5%) were screen positives. Of the screen positives, 221 (59.2%) agreed to participate in the study and were then interviewed. Almost all of the interviewed subjects ($N = 218$) had an ongoing episode of either unipolar or bipolar depression at baseline evaluation and were recruited to the study. Adolescent who declined to participate were similar to the study subjects in terms of age, sex, and parental socioeconomic status while they tended to have lower BDI-21 (19.0 versus 21.0, $z = -1.93$, $df = 371$, $P = .05$) and lower GHQ-36 (21.0 versus 24.0, $z = -1.98$, $df = 367$, $P = .05$) median sum scores [3, 8].

Data were obtained by interviewing the adolescents themselves and collecting additional background data from the clinical records. In clinical practice, at baseline, parents were offered at least one consultation appointment and data on adolescent's as well as parental problems were collected. Special efforts were made in order to confirm that all data in clinical records were appropriate, right, and timed. In a naturalistic manner, after the comprehensive baseline evaluation (T1), the outpatients received "treatment as usual" of clinically defined duration. The study subjects were reevaluated in 6 months and one year (T2). The median time interval between T1 and T2 was 59.5 weeks (interquartile range (IQR), 57.0–63.0 weeks).

Excluded for the analyses of this study were those subjects (1) who were diagnosed either at the baseline or at later diagnostic interviews as having bipolar disorder ($n = 21$), (2) with missing data of Alcohol Use Disorder Identification Test (AUDIT) [35] at baseline ($n = 12$), and (3) who did not participate in the one-year interview ($n = 29$). Consequently, the final study population of this study comprised of 156 patients with diagnosed unipolar depression. For the analyses, the subjects were classified into two groups according to level of self-reported alcohol use at baseline: (1) nonmisusers ($n = 86$) had AUDIT score of less than 8 and (2) alcohol misusers ($n = 70$) had

AUDIT score of 8 or more. The cutoff point of 8 in AUDIT was chosen based on previous research [35, 36]. AUDIT is a self-report measure to assess alcohol-related problems, which is a commonly used and a clinically meaningful instrument in ordinary clinical practice. The AUDIT has reasonable psychometric properties among adolescents [35, 37–39].

2.2. Sociodemographic, Diagnostic, and Clinical Characteristics at Baseline. Sex and age at baseline were taken directly from the data. The socioeconomic status (SES) of the adolescent's parents was classified as follows: upper middle class, lower middle class, working class, or other (including students, unemployed, retired (pensioner), others not defined) [40]. Parents' divorce, alcohol use, or mental health problems were recorded based on the information received from the subjects and/or the parents. Social support was assessed by the Perceived Social Support Scale-Revised (PSSS-R) [41]. PSSS-R measures persons' subjective perceptions of social support and emotional closeness, not actual number of supportive contacts. It has been shown to be a useful method in assessing perceived social support in Finnish adolescents [42, 43].

The Schedule for Affective Disorders and Schizophrenia for School-Aged Children—Present and Life-time version (K-SADS-PL) [44] was used to assess present and lifetime episodes of DSM-IV Axis I disorders. The DSM-IV Axis II disorders were assessed with the Structural Clinical Interview for DSM-IV Axis II Disorders (SCID-II) interview [45]. For the analyses, Axis II diagnoses were dichotomized (yes/no Axis II diagnose). Axis III diagnoses were dichotomized according to whether the patient had any doctor-diagnosed medical condition or not. Nine researchers, who were expert level clinicians (educated psychiatrists and psychologists), conducted the diagnostic interviews, and all the research diagnoses were confirmed in a subsequent diagnostic meeting. Interrater reliability, assessed using 15 randomly selected videotaped interviews, was good for mood disorder diagnoses (weighted kappa [46, 47] for MDD, other mood disorder, no mood disorder 0.87 (95% CI 0.81, 0.93)) [3].

Current psychosocial functioning (Global Assessment of Functioning, GAF) was assessed according to the DSM-IV Axis V definitions [48], that is, indicating the level of functioning at the time of the interview. For the group comparisons, the GAF score was used as a dichotomous variable, with a cut-point of 60 indicating "at least moderate impairment" [3, 8].

Severity of depression was measured by Beck Depression Inventory (BDI-21) [29] total sum-scores (range 0 to 60). Alcohol misuse was assessed by the AUDIT sum-score (range 0 to 40), severity of anxiety symptoms was measured by using the sum-score (range 0 to 63) of the Beck Anxiety Inventory (BAI) [49] and severity of suicidality by using the sum-score of the Scale for Suicidal Ideation (SSI) [50].

Age of onset of depression was recorded based on the information collected from the clinical records and at the interview.

2.3. Treatment. As the study was naturalistic, the outpatients received "treatment as usual" of clinically defined duration in a general adolescent psychiatric setting of Finnish secondary health care. The treatment team consists of a psychiatrist specialized in adolescent psychiatry, a psychologist, one or more psychiatric nurses, and a social worker. The treatment modalities used at the outpatient clinics consisted of individual supportive therapy, family consultations, and psychotropic medication when appropriate. The treatment always begun with an evaluation phase. The information of treatment received was gathered from the one-year follow-up data. At the one-year follow-up time, 65,4% of the subjects were continuing their treatment.

2.3.1. Psychosocial Treatment. Following data were gathered from the medical records: number of scheduled and kept individual appointments, number of scheduled and kept family/network appointments, *intensity* (kept individual appointments/month), and *attendance at treatment* (proportion of kept to scheduled individual appointments).

2.3.2. Pharmacological Treatment. The information of pharmacological treatment was used as a dichotomous variable (yes/no prescribed medication during the treatment/one-year follow-up time) in the following medication groups: (1) Serotonin reuptake inhibitors (SSRI:s), (2) other antidepressant medication, (3) anxiolytics, (4) antipsychotics, and (5) other psychotropic medication.

Combined treatment was classified as follows: (1) individual psychotherapy, (2) individual psychotherapy and family counseling, (3) individual psychotherapy and medication, and (4) individual psychotherapy, family counseling, and medication. The information of *hospitalization* was used as a dichotomous variable (yes/no hospitalization during the one-year follow-up time). In addition, the number of hospitalization days during the treatment was recorded.

2.4. Diagnostic and Clinical Characteristics at One-Year Followup. At one-year followup, the severity of depression was measured by the BDI-21. Total sum score and the level of psychosocial functioning (Global Assessment of Functioning, GAF) were assessed according to the DSM-IV Axis V definitions. Level of alcohol use was assessed by using the AUDIT sum-score. The diagnostic status of the depressive disorder at the time of the one-year follow-up diagnostic interviews was rated as follows: (1) recovery, (2) persistent depression, and (3) recurrence during the study period. Two months of 1 or no symptoms (no depressed or irritable mood or anhedonia) was defined as recovery. Lack of recovery during the one-year follow-up period was defined as persistent depression. Recurrence was defined as a new depressive episode emerging after the beginning of recovery.

2.5. Statistical Analysis. To analyze differences between alcohol misusers and nonmisusers, we used Chi-square for the categorical variables, *t*-test for normally, and Mann-Whitney *U* tests for non-normally distributed continuous variables. To analyze factors independently associating with treatment

TABLE 1: Sociodemographic, diagnostic, and clinical characteristics of depressed outpatients by alcohol use at baseline ($n = 156$).

Characteristic	Nonmisusers (Audit <8) (86)	Alcohol misusers (Audit ≥8) (70)	<i>P</i> value
Sex, no. (%)			.51
Male	17 (19.8)	11 (15.7)	
Female	69 (80.2)	59 (84.3)	
Age at baseline, mean (SD; median)	16.3 (1.5; 17.0)	16.5 (1.7; 17.0)	.64
SES of parents, no. (%)			.19
Upper middle cl	23 (26.7)	18 (25.7)	
Lower middle cl	38 (44.2)	21 (30.0)	
Working class	19 (22.1)	25 (35.7)	
Other	6 (7.0)	6 (8.6)	
Divorce of parents, no. (%)	36 (41.9)	31 (44.3)	.65
Parents' mental health problems, no. (%)	35 (42.2)	28 (43.1)	.91
Parents' alcohol use problems, no. (%)	31 (36.9)	35 (53.0)	.048
Perceived social support			
Total, mean (SD; median)	44.7 (11.2; 46.5)	43.5 (8.9;44.5)	.17
Close/intimate friend	16.1 (4.9; 18.0)	16.7 (4.1;18.0)	.63
Family	13.8 (4.4; 14.0)	11.6 (4.7;11.5)	.004
Friends	14.9 (5.1; 16.5)	15.2 (4.8;16.0)	.87
Age of 1st mood disorder, mean (SD; median)	13.4 (2.5; 13.5)	13.2 (2.9; 13.0)	.67
Depression diagnosis, no. (%)			.75
MDD single	45 (52.3)	36 (51.4)	
MDD recurrent	21 (24.4)	20 (28.6)	
Dysthymia/Double dep	13 (15.1)	7 (10.0)	
Minor	7 (8.1)	7 (10.0)	
Axis I: any comorbidity, no. (%)	62 (72.1)	52 (74.3)	.76
Any SUD, no. (%)	4 (4.7)	22 (31.4)	< .001
Axis II: any comorbidity, no. (%)	30 (34.9)	28 (41.2)	.42
Axis III: medical comorbidity, no (%)	29 (33.7)	14 (20.0)	.56
Axis V: GAF score <60, no. (%)	64 (74.4)	61 (87.1)	.048
BDI score at baseline, mean (SD; median)	21.0 (8.7; 19.5)	23.8 (8.8; 24.5)	.03
AUDIT score at baseline, mean (SD; median)	2.7 (2.4; 2.0)	14.4 (5.9; 12.0)	< .001
BAI score at baseline, mean (SD; median)	20.8 (11.4; 19.0)	22.9 (12.2; 20.0)	.38
SSI score at baseline, mean (SD; median)	4.1 (7.1; 0)	5.8 (7.2; 3.0)	.12

attendance, a logistic regression model was conducted with response variable (=attendance) equal to the binomial proportion of kept to scheduled individual appointments. Possibly compliance-related clinical and treatment factors were the explanatory variables including AUDIT and BDI scores at baseline, perceived social support from the family, psychosocial functioning (GAF score), comorbid axis I and axis II diagnoses at baseline, and prescribed use of psychotropic medication. In this model, the AUDIT, BDI, GAF, and PSSS-R scores were treated as continuous variables. The model was adjusted for age and sex. *P* values < .05 were considered statistically significant. Statistical analyses were carried out using SPSS 14.0 software package [51] and PASW 18.0 [52].

3. Results

3.1. Characteristics at Baseline. The adolescents with and without alcohol misuse did not differ significantly in terms of gender, age, or parental socioeconomic status. Those with alcohol misuse had perceived less social support from their families (11.6 versus 13.8, $P < .01$), had worse psychosocial functioning as measured by GAF score (GAF score < 60, 87.1% versus 74.4%, $P < .05$), higher mean BDI scores (23.8 versus 21, $P = .03$), and their parents had more often problems with alcohol use (53.0% versus 36.9%, $P < .05$), (Table 1). The mean of the AUDIT score for the alcohol misusers was 14.4 compared to 2.7 of nonmisusers. Altogether, six subjects had other than alcohol related-substance

TABLE 2: Treatment received of depressed outpatients by alcohol use during the one-year follow-up period ($n = 156$).

Characteristic	Nonmisusers (Audit < 8) (86)	Alcohol misusers (Audit ≥ 8) (70)	P value
<i>Psychosocial treatment</i>			
(i) Kept individual appointments, no. (%)			.35
<10	15 (17.4)	17 (24.3)	
10–25	42 (48.8)	36 (51.4)	
>25	29 (33.7)	17 (24.3)	
(ii) Kept family/network appoint, mean (SD; median)	1.4 (1.9; 1.0)	1.2 (2.1; 0.0)	.34
(iii) Intensity (individual appoint/month), mean (SD; median)	1.9 (1.1; 1.8)	1.5 (0.8; 1.3)	.04
(iv) Attendance % (individual appoint), mean (SD; median)	76.5 (15.6; 76.0)	68.8 (15.8; 68.5)	.004
<i>Psychotropic medications</i>			
Any, no (%)	42 (48.8)	48 (68.6)	.01
(i) Antidepressants	39 (45.3)	41 (58.6)	.10
SSRI	38 (44.2)	39 (55.7)	.15
other antidepressants	8 (9.3)	13 (18.6)	.17
(ii) anxiolytics/ sedatives	20 (23.3)	26 (37.1)	.06
(iii) antipsychotics	8 (9.3)	11 (15.7)	.22
(iv) other	4 (4.7)	3 (4.3)	.66
<i>Combined treatment, no. (%)</i>			
Individual psychotherapy	20 (23.3)	17 (24.3)	
+ family counselling	27 (31.4)	12 (17.1)	
+ medication	19 (22.1)	21 (30.0)	
+ medic + family	20 (23.3)	20 (28.6)	
<i>Hospitalization, no. (%)</i>	13 (15.1)	10 (14.3)	.88
<i>Number of hospitalization days, mean (SD; median)</i>	13.3 (48.5; 0.0)	10.4 (32.9; 0.0)	.89
<i>Treatment status at the one-year followup, no. (%)</i>			
(i) No treatment	30 (34.9)	24 (34.3)	
(ii) Psychiatric outpatient	55 (64.0)	43 (61.4)	
(iii) Psychiatric inpatient	1 (1.2)	3 (4.3)	

TABLE 3: Diagnostic and clinical characteristics of depressed outpatients at the one-year followup (by the alcohol use at baseline) $n = 156$.

Characteristic	Nonmisusers (Audit < 8) (86)	Alcohol misusers (Audit ≥ 8) (70)	P value
BDI score, mean (SD; median)	7.2 (7.8; 6.0)	9.7 (9.5; 7.0)	.045
Axis V: GAF score < 60, no. (%)	32 (37.2)	38 (55.1)	.03
AUDIT score, mean (SD; median)	4.2 (4.5; 3.0)	10.8 (6.4; 10.0)	< .001
<i>Diagnostic status, no. (%)</i>			
recovery	32 (37.2)	26 (37.1)	
persistent depression	42 (48.8)	37 (52.9)	
recurrence	12 (14.0)	7 (10.0)	
Any SUD, no. (%)	1 (1.2)	13 (18.6)	< .001

use at diagnosed level, two in the alcohol nonmisusers group (both amphetamine-related disorder NOS), and four in the alcohol misusers group (three cannabis abuse, and one amphetamine-related disorder NOS).

3.2. *Treatment Characteristics during the Follow-Up Period.* By the one-year follow-up time point, those with alcohol misuse had lower treatment intensity scores (mean 1.5 versus

1.9, $P = .04$) and a poorer attendance measure (68.8% versus 76.5%, $P < .01$) than the nonmisusers. They had also more often received psychotropic medication (68.6% versus 48.8%, $P = .01$; Table 2).

3.3. *Diagnostic and Clinical Characteristics at One-Year Followup.* Those with alcohol misuse had higher BDI score (mean 9.7 versus 7.2, $P < .05$) and higher AUDIT score

TABLE 4: Logistic regression analysis of treatment attendance¹ during one-year followup on baseline clinical and treatment-related characteristics.

	Wald χ^2	P	OR [95% CI]
Age at baseline	0.960	.327	1.03 [0.98–1.08]
Sex	0.626	.429	0.92 [0.74–1.14]
BDI score	0.245	.621	1.00 [1.00–1.01]
AUDIT score	56.009	< .001	0.97 [0.96–0.97]
Axis I: any comorbidity	2.251	.134	1.14 [0.96–1.36]
Axis II: any comorbidity	17.143	< .001	1.44 [1.21–1.71]
Axis V: GAF score	2.259	.133	0.13 [1.0–1.02]
Perceived social support from the family	0.142	.706	1.00 [0.99–1.02]
Psychotropic medication, any	0.017	.895	0.99 [0.83–1.18]

¹Proportion of kept to scheduled individual appointments.

(mean 10.8 versus 4.2, $P < .001$), and 55.1% of them had GAF score below 60 (versus 37.2%, $P = .03$) compared with the nonmisusers (Table 3).

3.4. The Logistic Regression Model for Treatment Attendance.

When age and sex were adjusted, and selected depression severity and comorbidity variables entered into the model, the strongest clinical- or treatment-related factor significantly associating with attendance (proportion of kept to scheduled individual appointments) was the baseline AUDIT score (Wald $\chi^2 = 56.009$, $df = 1$, $P < .001$, OR 0.97; 95% CI 0.96, 0.97; Table 4).

4. Discussion

The comparisons between depressed adolescent outpatients with and without alcohol misuse yielded some important significant differences. Specifically, those with alcohol misuse had perceived less social support from their families. In addition, their parents often had more problems with alcohol use. These results are in line with previous findings indicating a number of family-related factors, such as parental substance use or abuse, poor parent-child relationships, low perceived parental support, poor communication, and poor parent supervision and management of the adolescent's behaviour, as risk factors for the development of substance abuse among adolescents in general [53].

Further, those with alcohol misuse had more depressive symptoms and poorer psychosocial functioning both at the baseline and still after a one-year follow-up period time, which is in line with the findings by Goldstein et al. [11]. Our results were expected as Meririnne et al. [12] recently reported from the ADS study that alcohol use negatively affects the course of adolescent depression and psychosocial functioning. It is also important to notice, that even after a one-year follow-up period the AUDIT scores were still very high for the alcohol misuse group, and indicated an increasing trend for the nonmisusers. Further, our study supports the previous findings that not only boys but also depressed girls appear to be at a risk for substance use involvement during adolescence [5]. This is particularly

interesting, as recent observations of increasing drinking among girls have raised public concern.

The differences in treatment received between the two groups were, as a whole, only minor: those with alcohol misuse had more often received psychotropic medication altogether compared to the nonmisusers. These minor differences in treatment may reflect the fact that substance-related problems were either not detected at all or not taken into account when planning the treatment. This may lead to insufficient treatment, as Couwenbergh et al. [27] have pointed out. On the other hand, as the outpatients with alcohol misuse had more severe symptoms of depression, they were probably more often prescribed psychotropic medication. The possible problems recognizing alcohol use and not taken into account when formulating treatment for depressed adolescents may at least reflect (1) the lack of sufficient knowledge and expertise to assess and treat substance use problems, (2) the way the clinicians conceptualize their work (treat mental disorders versus SUDs), (3) the way the clinicians interpret the level of adolescents substance use (part of normative development versus disturbed development), (4) level of trust and therapeutic alliance between the adolescent and clinician, and (5) adolescent's own thoughts or feelings about his/her substance use leading to underreported or nondisclosure of substance use.

Compared to, for example, practice parameters by the American Academy of Child and Adolescent Psychiatry (2007) [23], one may see at least one significant shortcoming in treatment: the exiguity of family and school involvement in treatment. According to the practise parameter for the assessment and treatment of children and adolescents with substance use disorders [53], family therapy or significant family/parental involvement are critical to the success of any treatment approach for adolescents with SUDs. Our findings on significant parental problems with alcohol among adolescents with alcohol misuse may indicate that the parents themselves were so severely distressed that the adolescents were left too alone in the family in their efforts to get help for their problems. In addition, from the adolescent developmental perspective, growing efforts to independence from the parents are commonly seen as an age-related behaviour,

and the parents may have misinterpreted adolescents' alcohol use as such as a "normal" behavior. Although some degree of risk-taking may be normal in adolescence, repeated engagement in high-risk activities, persistent disregard for attempts at limit setting by authority figures, and aggressive behavior may be signs of a more serious problem [54]. Thus, both the parents and the clinicians should not interpret too easily adolescent's alcohol use as normative experimentation.

Interestingly, findings from this naturalistic clinical one-year follow-up study of depressed psychiatric outpatient adolescents indicate that an adolescent's severity of alcohol use, even at subdiagnostic level, has an independent negative effect on attendance at individual treatment appointments, which, for its part, most likely has a negative effect on outcome. Haw et al. [55] have concluded that both comorbidity with alcohol abuse and poor compliance with treatment may be important factors complicating therapy in many depressed patients with deliberate self-harm. As outpatient nonattendance is a serious problem in clinical and economic terms, we must at least try to speculate the reasons for poorer attendance. A number of family factors, such as parental involvement in treatment and family cohesion, have been identified as factors relating to treatment compliance among adolescent suicide attempters [56]. As mentioned above, this could be the case also in this study as depressive adolescents with alcohol misuse had perceived less social support from their families and their parents often had more problems with alcohol use. The adolescents may miss their treatment appointments also because of the direct negative impact of alcohol use on functioning (intoxication, hangover, or guilt), and the absence of subjective need for help (not seeing current pattern of alcohol use as a problem). In addition, regular alcohol use may complicate or even hinder recovery, leading to subjective disappointments in treatment.

Interestingly, in the logistic model, an association was noted between an axis II comorbidity and treatment attendance. This may suggest that special emphasis was placed on the treatment of those depressed individuals with comorbid personality disorders. This finding is to be studied in more detail in the near future.

In developing adolescent treatment settings and services, more specific treatment options are needed regarding alcohol use problems, for instance. This would require identification of different clinical presentations of disorders and sub-threshold disorders, and subsequent education of personnel accordingly. As an example, failures to keep appointments should alert for potential alcohol or other substance use problem.

4.1. Study Limitations. The present one-year follow-up study included consequently referred depressed adolescent outpatients, whose psychiatric diagnoses and clinical characteristics were comprehensively assessed using a reliable interview instrument and self-report scales. Alcohol use was assessed by a self-report scale of AUDIT using a cutoff point of 8 points [35, 36]. Lower cutoff points have also been suggested for use among adolescents [38]; however, we aimed at studying alcohol use "severe enough" but not necessarily meeting diagnostic criteria for actual substance

use disorders. It is critically important to assess the predictive meaning of alcohol misuse among depressed adolescents in order to find out effective treatments in ordinary clinical practice.

There is a possibility that the study findings are not exclusive to alcohol misuse, being perhaps influenced by other substances. However, this is unlikely as the number of other substance-related diagnoses was small.

Generalization of the finding to other cultures should be made with the understanding of possible differences between health care systems. Due to the Finnish system, adolescents believed to have a predominant and severe substance use disorders were treated elsewhere in specialized units.

This is a naturalistic clinical follow-up study, so any conclusions regarding the actual effect of the treatment on the outcome are precluded. On the other hand, naturalistic follow-up data are useful in assessing other questions, for example, factors associating with treatment adherence among "real-life" outpatients.

Due to the descriptive nature of the data, we reported the values for all the collected treatment-related variables. As these were numerous, a possibility for significant findings by chance arises. In the context of a relatively small study population, we wanted to avoid missing possible significant findings (a type II error) and decided to use basic significance tests without adjustments for the significance level, while recognizing that in so doing some of the statistically significant effects may be spurious.

5. Conclusions

Alcohol misuse was related to lower perceived parental support and greater parental alcohol use problems among depressed adolescent outpatients. Further, those with alcohol misuse had more depressive symptoms and poorer psychosocial functioning both at the baseline and still after one-year-follow-up period time. An adolescent's severity of alcohol use, even at subdiagnostic level as measured by self-report questionnaire and had an independent negative effect on attendance at individual treatment appointments, which, for its part, most likely had a negative effect on outcome. Early recognition of alcohol misuse is most important among adolescents in primary level treatment and other service settings. It should not be overlooked either in specialized settings focusing on the assessment and treatment of predominantly psychiatric disorders like depressive disorders. This view emphasizes the need for integrated services, having potential for simultaneous interventions and tolerance for both substance-related and other psychiatric problems. Among adolescents, the effect of family to attrition from treatment may be greater than thought. This should perhaps be taken into account by favouring family-related psychosocial methods, when possible, particularly in substance-related psychiatric problems. It seems that for depressed adolescents with alcohol and other substance-related problems specific multimodal treatment programs, accepted by adolescents and their parents, are needed.

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Research Article

Hopelessness and Excessive Drinking among Aboriginal Adolescents: The Mediating Roles of Depressive Symptoms and Drinking to Cope

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Canadian Aboriginal youth show high rates of excessive drinking, hopelessness, and depressive symptoms. We propose that Aboriginal adolescents with higher levels of hopelessness are more susceptible to depressive symptoms, which in turn predispose them to drinking to cope—which ultimately puts them at risk for excessive drinking. Adolescent drinkers ($n = 551$; 52% boys; mean age = 15.9 years) from 10 Canadian schools completed a survey consisting of the substance use risk profile scale (hopelessness), the brief symptom inventory (depressive symptoms), the drinking motives questionnaire—revised (drinking to cope), and quantity, frequency, and binge measures of excessive drinking. Structural equation modeling demonstrated the excellent fit of a model linking hopelessness to excessive drinking indirectly via depressive symptoms and drinking to cope. Bootstrapping indicated that this indirect effect was significant. Both depressive symptoms and drinking to cope should be intervention targets to prevent/decrease excessive drinking among Aboriginal youth high in hopelessness.

1. Introduction

Alcohol misuse is a serious problem among many North American Indigenous communities [1]. In Canada, the Aboriginal Peoples Survey [2] showed that 73% of First Nations respondents reported that alcohol was a problem in their communities. A recent review concluded that rates of alcohol misuse are higher among American Indians than among those in the general U.S. population, and that this is true for both adults and adolescents [3]. For example, rates of past month drunkenness are about twice as high among Native American adolescents as among nonnative American adolescents [3]. The 2002-03 First Nations Regional Longitudinal Health Survey [4] suggests this is also true for Canadian Aboriginal people, for example, the proportion of Aboriginals who reported weekly heavy drinking (5+ drinks

on a single occasion) was more than double that of those in the general Canadian population (16.0% versus 7.9%, resp.), despite the fact that when compared to the general Canadian population, Aboriginals are less likely to be current drinkers [5]. Some research suggests that Aboriginal youth may be particularly susceptible to excessive drinking [6].

These high rates of excessive drinking have many negative consequences for Aboriginal communities. For example, death related to alcohol use disorders is higher for Aboriginal people than for other ethnic groups [7]. In fact, alcohol has been identified as a leading cause of adolescent morbidity and mortality consequent to violence, falls, suicide, drowning, motorvehicle accidents, and risky sexual behavior [8]. For these reasons excessive drinking among Aboriginal youth must be considered an important public health concern [9].

In addition to elevated rates of excessive drinking, other problems faced by Aboriginal youth include high levels of depressive symptoms (e.g., extreme sadness, loss of interest, suicidality, and fatigue) and hopelessness (i.e., feelings of worthlessness and pessimism about the future). Profound changes brought upon Aboriginal peoples through colonialism have been linked to high rates of depressive symptoms and suicide in many communities [10]. Examples of such profound changes include geographic dislocation and disruptions to their connections to the land and to their traditional patterns of subsistence [1]. Depressive symptoms and suicide appear to be particular problems for Aboriginal youth [11]. Discrimination has been associated with depressive symptoms in both American Indians adults [12] and adolescents [13]. Abject poverty created by colonial policies has resulted in a lack of control for Canadian Aboriginal peoples, which has contributed to feelings of hopelessness [6, 14].

Cognitive theories of depression posit a key role to hopelessness as an individual differences factor that sets the stage for the development of depressive symptoms (e.g., [15, 16]). While it is well established that hopelessness is a risk factor for depression in nonAboriginal groups (e.g., [17–19]), there is little data examining the impact of hopelessness on depressive symptoms among Aboriginal peoples. One cannot simply assume that hopelessness leads to a given mental health outcome in the same way in Aboriginal communities as it does in nonAboriginal groups (e.g., see [20]).

It is well established in nonAboriginal people, that depression and alcohol abuse/dependence commonly cooccur (see reviews in [21–23]). American epidemiologic surveys in the adult general population [24, 25] show significant odds ratios for depression comorbid with alcohol use disorders indicating that the two disorders cooccur at rates that far exceed chance. A number of studies have confirmed a cooccurrence of depression and alcohol misuse in nonAboriginal adolescents as well (see review in [23]). For example, it has been shown that depression rates increase from about 5% in American youth who abstain from alcohol to about 24% in those who use alcohol at least weekly [26].

In nonAboriginals, hopelessness has been linked to alcohol use and abuse in both adults and adolescents (e.g., [18, 19]), for example, a cross-sectional study found that hopelessness in adolescents was significantly positively related to lifetime alcohol use [27]. It was also shown that hopelessness was associated longitudinally with alcohol use and drunkenness over a one-year interval in a group of Canadian adolescents [28]. In sum, there are clearly established relations between hopelessness and depression with excessive drinking in nonAboriginal youth. Data that directly examines and tests the relationships between hopelessness, depressive symptoms, and excessive drinking is lacking, however, among Aboriginal adolescents.

A number of theoretical models have been proposed to explain the relation between depression and alcohol misuse (see reviews in [23, 29]). One popular model is *self-medication* [30], which posits that depressed individuals drink to reduce negative emotions, and are thus at risk for

heavier drinking and more alcohol-related problems as a consequence. Consistent with this theory, elevated depressive symptoms in adolescence have been shown to predict future alcohol use disorders (e.g., [31]), alcohol-related problems (e.g., [32]), and alcohol use levels (e.g., [33]), for example, in a study of 1545 Finnish twins, it was found that early onset depressive disorder (at age 14 years) predicted later alcohol use and recurrent intoxication (at age 17.5 years), even after the effects of other substance use and other psychiatric disorders were controlled [34].

Few studies, however, have tested the underlying mechanism posited in self-medication theory—namely, drinking-to-cope with negative emotions. In two separate groups of nonAboriginal Canadian adolescents, it was shown that consistent with self-medication theory predictions, drinking to cope mediated the relations of both hopelessness and depressive symptoms with alcohol-related problems [35]. It was concluded that both hopelessness and depressive symptoms play a role in adolescent motivation for alcohol use, with both reflecting a desire to diminish negative affect [35]. However, the study did not test the possibility that hopelessness might be related to drinking to cope indirectly via its effects on depressive symptoms.

The purpose of the present study was to examine the relations among four variables (i.e., hopelessness, depressive symptoms, drinking to cope, and excessive drinking) assessed cross-sectionally in a large group of Canadian Aboriginal adolescent drinkers. We propose that Aboriginal adolescents with higher levels of hopelessness are more susceptible to depressive symptoms, which in turn predispose them to drinking to cope—a drinking motivation that puts them at greater risk for excessive drinking. Thus, we specified a structural equation model in which we hypothesized (a) hopelessness would be directly linked to depressive symptoms, (b) depressive symptoms would be directly linked to drinking to cope, and (c) drinking to cope would be directly linked to excessive drinking. We also specified three mediational hypotheses. We expected (d) hopelessness to be indirectly related to drinking to cope through depressive symptoms, (e) depressive symptoms to be indirectly related to excessive drinking through drinking to cope, and (f) hopelessness to be indirectly related to excessive drinking through depressive symptoms and drinking to cope. The hypothesized structural model is depicted in Figure 1.

2. Materials and Methods

2.1. Participants. Participants were drawn from among 837 student respondents enrolled in any one of 10 participating schools in the Canadian provinces of Saskatchewan, Manitoba, and Quebec. All participating schools contained a large representation of Aboriginal students. Six schools were from the Canadian province of Saskatchewan, two from Manitoba, and two from Quebec (one from a Cree and one from an Inuit community). Both urban ($n=2$) and rural ($n=8$) schools were represented. Of the total 837 students, 286 (34%) indicated that they had not consumed any alcohol in the last four months, leaving 551 participants (66%) to be classified as “drinkers” (52% boys; mean age

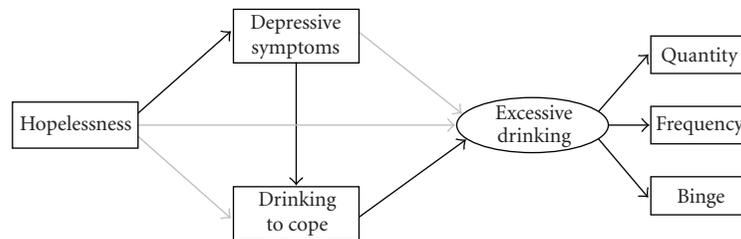


FIGURE 1: The hypothesized structural model. Rectangles represent manifest variables; ovals represent latent variables. Black arrows represent hypothesized direct effects; grey arrows represent paths hypothesized to be explained by indirect effects. Quantity = drinking quantity; Frequency = drinking frequency; Binge = binge drinking.

15.9 yrs., $SD = 1.3$, range = 14–18; mean grade = 9.4, $SD = 1.2$, range = 7–12). Of the 551 drinkers, 178 (32%) self-identified as Cree, 91 (16%) as Ojibway, 57 (10%) as Metis, 32 (6%) as Oji-Cree, and 25 (5%) as Dakota. A further 29 (5%) were classified as “Other Aboriginal”.² Also included among the drinkers, due to their attendance at the participating schools, were 63 (12%) students who self-identified as Caucasian or Black.³ A further 76 (14%) drinkers skipped the race/ethnicity question altogether.⁴ Of the 551 drinkers, 326 (59%) were from schools in Saskatchewan, 168 (31%) from schools in Manitoba, and 57 (10%) from schools in Quebec. Only the data provided by the 551 drinkers were employed in all subsequent analyses. Abstainers were excluded from completing the measure of drinking to cope because the measure used requires that respondents have at least some relatively recent drinking experience to answer items enquiring about why they drink. The inclusion of even infrequent, light drinkers (e.g., those having consumed only one drink in the last four months) ensured that the results would be applicable to a wide range of adolescent drinkers from participating schools (see [36]).

2.2. Measures

2.2.1. Substance Use Risk Profile Scale (SURPS). The SURPS [19] is a 23-item self-report scale designed to measure four individual difference factors, including hopelessness, each of which has been shown to be related to risk for alcohol use/misuse. Participants are asked to rate the degree to which they agree with each item on a scale ranging from 1 = strongly disagree to 4 = strongly agree. A sample hopelessness scale item is “I feel that I am a failure.” The SURPS hopelessness score is calculated by summing across all 7 hopelessness items following reverse scoring of several inverselykeyed items. The SURPS has been shown to have a stable four factor structure and the four scales have been found to have good internal consistency and good construct validity in nonclinical adolescents recruited through Canadian schools [19]. In particular, the hopelessness scale shows significant bivariate correlations with depressive symptoms, drinking to cope, drinking quantity, drinking frequency, binge drinking, and alcohol-related problems in nonAboriginal Canadian youth [19]. The SURPS hopelessness scale has also been shown to correlate significantly with lifetime alcohol involvement in a group of American adolescents [27]. In the present study, the

internal consistency of the hopelessness scale was acceptable at $\alpha = .78$.

2.2.2. Depression Subscale of the Brief Symptom Inventory (BSI-DEP). The BSI-DEP [37] is a 7-item measure that assesses depressive symptoms. The full BSI was developed as a brief version of its longer parent instrument, the 90-item Symptom Check List—Revised (SCL-90-R; [38]). Each item is rated on a scale ranging from 0 = not at all to 4 = extremely. A sample BSI-DEP item is “Feeling no interest in things.” The BSI-DEP total score was calculated by summing across all seven depression items. Factor analytic studies of the BSI suggest good structural validity (e.g., all BSI-DEP items show salient loading on a single depression factor). Both test-retest reliability and internal consistency are high for the BSI-DEP scale and it correlates well with the depression scale from the original SCL-90-R. Moreover, the BSI-DEP scale shows high convergence with other established depression scales [37]. While there are many validated measures of depression for use in adolescents, we chose the BSI-DEP scale mainly for its brief length (i.e., 7 items) in order to reduce participant burden. In the present study, the internal consistency of the BSI-DEP was good at $\alpha = .86$.

2.2.3. Drinking Motives Questionnaire—Revised (DMQ-R). The DMQ-R [39] is a 20-item self-report measure that taps four distinct motivations for alcohol use among adolescents, including drinking to cope. Respondents indicate their relative frequency of alcohol use in each of the indicated circumstances, when they drink. Each subscale consists of five items which are rated on a scale ranging from 1 = almost never/never to 5 = almost always/always. Subscale scores are computed as the mean of the relative frequency ratings for each of the five items on each subscale [39–41]. The drinking to cope scale measures drinking to reduce or avoid a range of negative affective states and consists of items such as “Because it helps you when you feel depressed or nervous.” Prior work shows that the drinking to cope scale is associated both with excessive drinking and with adverse consequences of drinking among groups of American, Swiss, and Canadian adolescents [39, 42]. The drinking to cope scale has also been shown to have good internal consistency and structural validity among Canadian Aboriginal adolescents [43]. In the present study, the internal consistency of the drinking to cope scale was good at $\alpha = .81$.

2.2.4. Excessive Drinking. Excessive drinking was indexed with three items assessing degree of alcohol use in the last four months (see also [19]). First, participants indicated the number of alcoholic beverages they normally consumed per drinking day; this index was referred to as drinking *quantity*. Participants were informed with visual and verbal cues that one drink equals one bottle/can of beer, one small glass of wine, one shot of hard liquor, or one cooler. Next, participants reported how often they normally consumed alcohol; this index was referred to as drinking *frequency*. Finally, participants indicated how often they normally consumed 5+ drinks in a single sitting (4+ drinks for girls); this index was referred to as *binge* drinking. Each item was rated on a scale of 1–5, such that high scores on each of the three measures index higher levels of *excessive* (i.e., more frequent, heavy, or intense) drinking. Response options for the quantity item were 1 = 1 or 2, 2 = 3 or 4, 3 = 5 or 6, 4 = 7 to 9, and 5 = 10 or more. Response options for the frequency item were 1 = less than monthly, 2 = once per month, 3 = 2 to 3 times per month, 4 = weekly, and 5 = daily or almost daily. Response options for the binge item were 1 = never, 2 = less than monthly, 3 = monthly, 4 = weekly, and 5 = daily or almost daily. Previous research has found adequate reliability of self-reported alcohol consumption measures across a broad range of response formats [44]. Nonetheless, recommended methods were used to enhance the accuracy of participants' self-reports [45]. Specifically, drinking behavior items were embedded within other questions on demographics to minimize their salience. Moreover, since extensive evidence supports the validity of self-reported alcohol use when participants are assured confidentiality [46], students were verbally assured confidentiality prior to survey completion. In the present study, this three item measure showed acceptable internal consistency ($\alpha = .79$).

2.3. Procedure. This study was part of a larger project on alcohol abuse prevention in the 10 participating schools. The project received approval from the Dalhousie Health Sciences Human Research Ethics Board (protocol no.: 2007-1628) and Health Canada's Research Ethics Board (protocol no.: 2007-0026). Recruitment occurred through the active process of relationship- and partnership-building with the communities involved. Policing partners and other community members (e.g., Elders) approached the investigators upon learning of our previous alcohol abuse prevention work in Aboriginal (i.e., Mi'kmaq) communities in Nova Scotia [47]. Essentially, community partners self-identified for inclusion in the larger project. Community partners then identified schools that would be interested in being involved. This study engaged Aboriginal youth (grades 7–12) through its grounding in the school system of the Aboriginal community. Reflecting the deep value of Elders' knowledge of the participating communities, the project was arranged to encourage meaningful participation among school partners, policing partners, and study investigators.

School administration partners advised as to the method of distributing information about the study to parents/guardians of students in grades 7–12 in participating schools, prior to administration of the survey. An

information sheet describing the study was sent in a mail-out directly to parents/guardians. Parents/guardians were encouraged to contact the researchers or school principal for any further information they desired about the study. Parents/guardians were asked to let the researchers/school principal know if they did not consent to having their child participate (i.e., a negative consent procedure was used). Parents/guardians were provided with a toll-free number to contact the researchers for additional information, if they so desired.

School administration partners at each site also advised as to whether announcements describing the study should be delivered school-wide through the loud-speaker system along with regular morning announcements and/or delivered by individual classroom teachers. Prior to survey administration, students were informed about the nature of the study, and willing students provided written informed consent at the time of the survey. Consent forms were maintained separately from the completed questionnaires to ensure confidentiality and anonymity. Students were informed that the purpose of the survey was to investigate individual differences in reasons for alcohol use.

The student consent form provided potential participants with information about the purpose of the survey, as well as the voluntary and confidential nature of the questions. Students were told that they were free to decline to participate and free to withdraw at any time. Those who declined participation were invited to the school library (under the librarian's supervision) or were asked to remain seated and read while their classmates were completing the survey. All students in grades 7–12 in participating schools were invited to take part in the survey. Approximately 20 students in total declined study participation resulting in a very high response rate (approximately 98% from among eligible students who were attending school on the day of survey administration). Data collection was conducted on a grade-by-grade basis during class time, with the permission and input of the school principal. Data collection was led by one of the coauthors (MNC). Following survey administration, the researcher leading the data collection offered a brief presentation on psychology research. No feedback was given to parents, teachers, or students regarding individual students' scores. Teachers had the option of remaining in the classroom at the time of the survey. Measures were administered in a standard order as follows: demographics, excessive drinking indices, BSI-DEP, DMQ-R, and SURPS. During questionnaire completion, students were permitted to ask questions of the researchers. The small minority of students who had difficulties with reading were offered assistance in reading the survey questions by trusted teachers or classroom aides. Translation into French was provided for the students in the Cree school in Quebec.

In order to protect any student from being singled out and labeled, the survey was anonymous. To maintain anonymity and confidentiality, students were asked not to write their names on the forms. The toll-free number mentioned above that was established for parent communication was also offered to students in case they had

TABLE 1: Descriptive statistics and bivariate correlations between study measures.

	(1)	(2)	(3)	(4)	(5)	(6)	Mean (SD)
(1) Hopelessness	—						14.1 (4.0)
(2) Depressive symptoms	.42**	—					7.4 (5.9)
(3) Drinking to cope	.26**	.39**	—				2.1 (0.9)
(4) Drinking quantity	.12**	.02	.25**	—			3.4 (1.4)
(5) Drinking frequency	.14**	.13**	.30**	.49**	—		2.7 (1.1)
(6) Binge drinking	.13**	.10*	.30**	.56**	.73**	—	2.8 (1.0)

Note. Sample sizes vary from 486 to 531 due to missing data on various study measures. * $P < .05$. ** $P < .01$.

had any questions about the survey forms in particular, or about the research project more generally. The privacy of each call was ensured because one of the coauthors (M. N. Comeau) was the only person who took the calls. Although participants were not compensated financially, survey administrations were concluded with a snack or meal. By integrating an educational component into data collection and engaging the students, they had the opportunity to participate in a project where the ultimate goal was to develop future culturally relevant alcohol abuse prevention efforts that are more meaningful to the lives of youth in their communities.

3. Results

3.1. Descriptive Statistics and Bivariate Correlations. Mean (and SD) scores on each of the study measures for the total group of 551 drinkers are displayed in the right hand column of Table 1. The descriptive statistics for the three excessive drinking indices suggest that the average student was drinking relatively frequently, heavily, and intensely (i.e., drinking 2 to 3 times per month, consuming 5 or 6 standard drinks on each drinking occasion, and binge drinking monthly).

Bivariate correlations between the various study measures are also shown in Table 1. All study measures were significantly intercorrelated with one exception: depressive symptoms were not significantly correlated with drinking quantity. Several of the correlations among study variables were moderate to strong. However, multicollinearity and redundancy of variables are only a concern when correlations exceed 90 [48]. While the data in Table 1 suggests some expected overlap between several of the study variables (e.g., 24%–53% shared variance between the three indices of excessive drinking), none of the variables should be considered redundant. It is important to note that the correlation between hopelessness and depressive symptoms was significant ($r = .42$, $P < .01$) but did not approach the strength at which there would be concern about multicollinearity or redundancy. This result lends support to our conceptualization of hopelessness and depressive symptoms as distinct constructs.⁵

3.2. Structural Equation Modeling (SEM). SEM was conducted using AMOS 7.0 [49]. Full information maximum likelihood estimation was utilized to deal with missing data [50]. For the structural model, fit was evaluated via multiple

indices [51]. Adequate fit is indicated by a chi-square/degrees of freedom ratio (χ^2/df) around 2, a comparative fit index (CFI) and an incremental fit index (IFI) around .95, and a root-mean-square error of approximation (RMSEA) around .06 [52]. We report the RMSEA value along with 90% confidence intervals (90% CI).

Three manifest variables were selected to represent the excessive drinking latent variable: drinking quantity, drinking frequency, and binge drinking. Each observed variable showed substantial and significant loadings (ranging from .63 to .89) on the excessive drinking latent variable. Fit indices also suggested the structural model fit the data well: $\chi^2(6, N = 551) = 12.93$, $P < .05$; $\chi^2/df = 2.16$; CFI = .99; IFI = .99; RMSEA = .05 (90% CI: .01, .08). The final model is depicted in Figure 2 with significant paths indicated with black arrows and nonsignificant paths indicated with grey arrows. As hypothesized, (a) hopelessness was directly linked to depressive symptoms, (b) depressive symptoms were directly linked to drinking to cope, and (c) drinking to cope was directly linked to excessive drinking (see Figure 2). These three above-mentioned direct paths were significant, substantial, and consistent with the hypothesized structural model (see Figure 1). Congruent with two of the mediational hypotheses, depressive symptoms were unrelated to excessive drinking, and hopelessness was unrelated to excessive drinking (see Figure 2). Unexpectedly though, hopelessness was directly linked to drinking to cope ($P < .05$; see Figure 2). It should be noted, however, that while the direct relationship between hopelessness and drinking to cope was significant, it was not much different in magnitude than the nonsignificant relationship between hopelessness and excessive drinking (see Figure 2).

A significant indirect effect indicates that mediation has taken place [53]. We used bootstrap analyses to test the significance level of the three hypothesized indirect effects (see Figure 1). For each test of indirect effects, we used random sampling replacement to create 20,000 ($n = 551$) bootstrap samples. These samples were then utilized to estimate bias-corrected standard errors for each hypothesized indirect effect in question. In the case of the indirect path from hopelessness to excessive drinking, the indirect effect was based on all paths and was computed by multiplying (a) path coefficients from the predictor to mediators and (b) path coefficients from mediators to the criterion. In addition, CIs were computed. An indirect effect may be described as significant ($P < .05$) when the 95% CI for this indirect effect does not include zero.

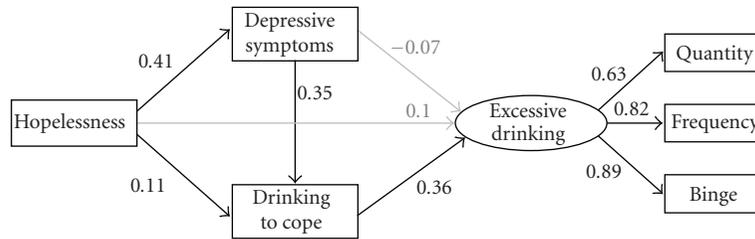


FIGURE 2: The hypothesized structural model. Rectangles represent manifest variables; ovals represent latent variables. Black arrows represent significant paths (i.e., $P < .05$). Grey arrows represent nonsignificant paths (i.e., $P > .05$). Path coefficients are standardized. Quantity = drinking quantity; Frequency = drinking frequency; Binge = binge drinking.

TABLE 2: Bootstrap analyses of hypothesized indirect effects.

Hypothesized Indirect effect	Bootstrap estimates			
	Unstandardized indirect effect	Standardized indirect effect	SE for standardized indirect effect	95% confidence interval for standardized indirect effect (lower and upper)
Hopelessness to drinking to cope through depressive symptoms	.167	.143	.025	.094, .192*
Depressive symptoms to excessive drinking through drinking to cope	.019	.123	.025	.074, .173*
Hopelessness to excessive drinking ^a	.013	.059	.029	.002, .116*

Note. SE = bias-corrected standard error. ^aindirect effect is based on all indirect paths. *Confidence intervals excluding zero are significant at $P < .05$.

First, we tested the indirect effect of hopelessness on drinking to cope. Bootstrap estimates indicated this hypothesized indirect effect was significant: $\beta = .143$, $B = .167$, (95% CI: .094, .192), and $SE = .025$. That is, the indirect effect of hopelessness on drinking to cope through depressive symptoms was significant (see Table 2). Next, we tested the indirect effect of depressive symptoms on excessive drinking. Bootstrap estimates indicated this hypothesized indirect effect was also significant: $\beta = .123$, $B = .019$, (95% CI: .074, .173), and $SE = .025$. Put differently, the indirect effect of depressive symptoms on excessive drinking through drinking to cope was significant (see Table 2). Finally, we tested the indirect effect of hopelessness on excessive drinking. Bootstrap estimates indicated this hypothesized indirect effect was again significant: $\beta = .059$, $B = .013$, (95% CI: .002, .116), and $SE = .029$. That is, the indirect effect of hopelessness on excessive drinking through depressive symptoms and drinking to cope was significant (see Table 2).

In sum, results suggest the hypothesized structural model is a well-fitting model that is consistent with the expected pattern of direct and indirect effects in this group of Canadian Aboriginal adolescent drinkers (see also Endnote 5).

4. Discussion

Consistent with hypotheses, hopelessness was directly linked to depressive symptoms. This finding replicates, in an Aboriginal adolescent group, much previous work linking hopelessness with depression in nonAboriginal groups [17–19]. This is an important finding since hopelessness has not

always been linked to mental health outcomes in Aboriginal groups in the same way it has been in nonAboriginal groups (e.g., [20]). The finding of a direct path from hopelessness to depressive symptoms within the structural model is consistent with predictions of models positing a key role for hopelessness as a cognitive risk factor for depression (e.g., [15, 16]). The discrimination, disruptions to family connections, geographic dislocation, and abject poverty arising from colonial policies have resulted in social and economic circumstances which are often objectively bleak for Canadian Aboriginal people, setting the stage for the development of hopelessness [6, 14]. But even Beck's model of depression acknowledges that such negative cognitions are not always distorted or inaccurate—merely that they are maladaptive in terms of increasing risk for depressive symptoms [54].

The present finding linking depressive symptoms directly to drinking to cope replicates, in Canadian Aboriginal adolescents, previous findings from nonAboriginal adolescents showing that depressive symptoms were related to drinking to cope with negative emotions [35]. The structural model also pointed to two ways in which hopelessness is linked to drinking to cope: the hypothesized indirect relation through depressive symptoms and an additional (unexpected) direct relation. This suggests that Aboriginal youth with higher levels of hopelessness are at increased risk of drinking to cope for two reasons. First, they are at risk of developing depressive symptoms which may motivate them to drink to eliminate or numb those unpleasant feelings. Second, hopeless individuals may attempt to block their pessimistic thoughts through drinking. It can be concluded, consistent

with findings in nonAboriginal Canadian adolescents, that both hopelessness and depressive symptoms play a role in Aboriginal adolescents' motivations for alcohol use [35], with both reflecting a desire to diminish unpleasant cognitions or emotional states.

The third hypothesized direct effect in the current study was a path from drinking to cope to excessive drinking. Cooper's model of adolescent drinking motives contends that drinking to cope is a particularly risky motivation for drinking that sets teens up for greater rates of excessive drinking and drinking-related problems [39]. The present finding showing a significant and substantial direct path between drinking to cope and excessive drinking in a Canadian Aboriginal group adds to the growing literature suggesting that the link between drinking to cope and excessive drinking persists cross-culturally [42].

As hypothesized, the relation of depressive symptoms to excessive drinking was indirect—an effect mediated through drinking to cope. This finding contributes to the understanding of the significant overlap between depression and alcohol use disorders in adults and adolescents alike [23] by suggesting one possible mechanism underlying this relationship. Specifically, the present findings suggest that adolescents with higher levels of depressive symptoms drink to excess more so than other adolescents because they are drinking to alleviate or numb negative emotions. This finding is consistent with the negative reinforcement mechanism postulated in self-medication theory [30] to explain the overlap of depressive symptoms and excessive drinking. The lack of a relation between depressive symptoms and drinking quantity in the bivariate correlations is, however, inconsistent with some previous research which has demonstrated such a link [55]. Nonetheless, this previous research was conducted with nonAboriginal adults rather than Aboriginal adolescents.

Previous cross-sectional and longitudinal research with nonAboriginal youth suggests a relationship between hopelessness and excessive drinking [19, 27, 28]. Consistent with hypothesis, the path from hopelessness to excessive drinking in the present study was indirect—mediated through depressive symptoms and drinking to cope. It is interesting to consider this finding in relation to another model (alternative to the mediational model tested herein) that has been posited to account for the high overlap of depression and alcohol disorders—namely the common factors model [23, 29]. Specifically, it has been suggested that a third factor or common underlying vulnerability (such as hopelessness) contributes to the apparent association between depression and excessive drinking ([e.g., [17]). In other words, hopelessness is thought to independently and directly contribute to the development of both depressive symptoms and excessive drinking creating an apparent association between the latter two variables. The present findings are inconsistent with the common factors model given that we did not observe any direct relation between hopelessness and excessive drinking. Instead, the association between hopelessness and excessive drinking was indirect, and mediated through depressive symptoms and drinking to cope.

Two comments should be made on the composition of our study group. Although data were collected across three Canadian provinces, from both urban and rural communities, and included distinct Aboriginal groups (i.e., First Nation, Inuit, and Métis), national representation and ability to generalize across all Canadian Aboriginal groups were nonetheless limited. While initially this may appear a limitation of the present study, this criticism would be misguided. For example, in Canada, there are 11 major Aboriginal language families and 65 distinct dialects [56]. To expect that there is a singular, representative, and general Aboriginal group in Canada, to which all results would apply, only serves to perpetuate biases that all Aboriginal groups are the same. While there are significant similarities that might be related to alcohol misuse (e.g., discrimination, disruptions to family connections, geographic dislocation, and abject poverty), all Aboriginal groups have rich cultures and histories that are unique.

Second, participants in the present study did not entirely consist of Aboriginal youth, and included at least 12% of student drinkers who were nonAboriginal (see Endnotes 3 and 4). However, the 10 participating schools were schools with high proportions of Aboriginal students, and the large majority of the study participants were Aboriginal. The decision to include all student drinkers regardless of ethnicity/race was consistent with the wishes of our community partners (see Endnote 3) and enhanced our ability to generalize the findings to a wide variety of students attending such schools in Canada.

Several potential limitations to the present study should be acknowledged, each of which suggests important avenues for future research. The study was cross-sectional in nature and lies in contrast with the time frame of the theoretical model, which implies unfolding of relations between the study variables over time (i.e., hopelessness leading to the later development of depression, which leads to the eventual development of drinking to cope, which in turn results in excessive drinking). While this cross-sectional analysis is a first step in testing the utility of the proposed structural model, the model still requires further investigation within a multiwave longitudinal design [57]. Second, the proposed model is linear and unidirectional and does not acknowledge the possible reciprocal relations between study variables over time. For example, it is possible that excessive drinking actually increases depressive symptoms and/or hopelessness in the longer term either through the physiological or psychological consequences of heavy drinking [58]. It is also possible that depressive symptoms cause increases in hopelessness [59]. Such more complex reciprocal relations between study variables over time could be tested using longitudinal methods and multiwave data (e.g., see [60]), consistent with calls for examination of more complex models in Aboriginal alcohol research [61].

A third possible limitation is that study measures were developed for use with nonAboriginal adolescents and not all have been investigated in terms of their psychometric properties when used with Aboriginal youth. Nonetheless, all showed good internal consistency in the present study and some (e.g., DMQ-R coping motives subscale; [43]) have

been previously validated in Aboriginal adolescents. Fourth, all study variables were assessed via retrospective self-report which may be subject to various biases including memory distortions and social desirability. Nevertheless, we did use methods for increasing the accuracy of participants' reports (e.g., [45]) and studies using other methodologies have shown similar results (e.g., sad mood induction leading to increased drinking in the lab among female young people who drink to cope; [62]). A fourth potential limitation was that drinking to cope was assessed with a "generic" coping motives scale [39]. More recently, a measure has been developed and validated that distinguishes drinking to cope with *depression* from drinking to cope with *anxiety* [63]; this refined measure might be useful for future studies in this area.

Fifth, the present study did not consider potential moderators. For example, given known gender differences in depressive symptoms (greater in females [64]), drinking to cope with depression (greater in females [65]), and excessive drinking (greater in males [66]), future research should examine whether the hypothesized model is moderated by gender. Moreover, given recent evidence that resilience (individual, family, and community; [67]) buffers the effects of violence exposure on symptoms of posttraumatic stress disorder in Aboriginal youth [68], resilience might prove a useful variable to examine in future as a moderator in our proposed model. In particular, the construct of resilience might prove useful in further research to understand how some Aboriginal youth fare so well in terms of their emotional and behavioral health in spite of the gross social inequities they face daily in their environments. Given such a complex and multidimensional issue as excessive drinking among Aboriginal youth, it is likely that many environmental, interpersonal, and individual risk and protective factors will be found to play moderating roles in the preliminary model tested herein. Finally, the present study only focused on one possible pathway to excessive drinking in Aboriginal youth. While the present results do suggest that hopelessness may be one risk factor for excessive drinking in Aboriginal adolescents, other studies support additional risk pathways. For example, a recent study showed that exposure to violence was related to excessive drinking in Aboriginal youth and that this relation was mediated by symptoms of posttraumatic stress disorder rather than depressive symptoms [69].

The present findings of an indirect relation between hopelessness and excessive drinking suggest that targeted interventions for Aboriginal youth who are high in hopelessness are needed to prevent or decrease excessive drinking. The mediational results can be helpful for informing the content of such preventative or early interventions [70]. Mediational findings from the present study suggest the need to focus on depressive symptoms and maladaptive drinking to cope in targeted interventions for Aboriginal youth with high levels of hopelessness. Additionally, the unexpected direct path from hopelessness to risky drinking to cope suggests that hopeless cognitions may need to be a direct target in such preventative interventions as well. Cognitive practitioners would need to be particularly mindful of the objectively difficult circumstances facing

many Canadian Aboriginal youth which set the stage for maladaptive, but not necessarily irrational, hopeless thinking styles. The direct effect of hopelessness on risky drinking to cope also points to the importance of primary prevention efforts (e.g., improving schools, developing sustainable local economies grounded in natural resources, and providing better education and employment opportunities) to deter the development of hopelessness among Aboriginal youth. A comprehensive approach to the problem of excessive drinking would pair community-wide primary prevention with school-based secondary prevention targeted toward high risk (e.g., high hopelessness) youth.

A cognitive-behavioral secondary prevention program focusing on hopelessness, depressive symptoms, and drinking to cope has been tested in the form of a school-based intervention among nonAboriginal youth via two randomized controlled trials (RCTs). The intervention targeting adolescents with high levels of hopelessness was shown to increase alcohol abstinence, decrease problem drinking [71], and reduce depressive symptoms [72]. More recently, this approach has been culturally adapted and has shown promise for reducing excessive and problematic drinking in a group of Canadian Aboriginal youth in an open trial [47]. The culturally adapted intervention still needs to be tested in an RCT. Such a trial could also test if intervention-induced changes in depressive symptoms, drinking to cope, and/or hopelessness mediate intervention-induced changes in excessive drinking among youth high in hopelessness at baseline. This would prove an even more stringent test of the theoretical model supported in the present study.

5. Conclusions

In sum, we used structural equation modeling to demonstrate the excellent fit of a model which links hopelessness to excessive drinking indirectly via depressive symptoms and drinking to cope in Canadian Aboriginal youth. Bootstrapping indicated that this indirect effect of hopelessness on excessive drinking was significant. Both depressive symptoms and drinking to cope should be intervention targets in school-based programs designed to prevent or decrease excessive drinking among Aboriginal youth with high levels of hopelessness.

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Endnotes

1. Aboriginal people can include First Nations, Inuit, and Métis peoples as recognized by the Constitution of Canada [73]. In this paper, we use the term Aboriginal to refer collectively to people from all three groups. Otherwise, we specify the particular Aboriginal/Indigenous group in question.
2. The “Other” Aboriginal category included youth who self-identified as belonging to the following Aboriginal groups: Nakota, Inuit, Nakota-Cree, Cree-Saulteaux, Saulteaux, Assiniboine, Sioux, Dene, Nakota-Assiniboine, Cree-Metis, and Ojibway-Black.
3. All students at the six participating Saskatchewan schools were invited to be involved in the research including a number of Caucasian youth and a few Black youth. Elders and school partners stressed the importance of including all students in the study in order to demonstrate a cultural value of connectedness and collaboration rather than risk marginalization and stigmatizing of certain groups by exclusion from the study. Three of the six Saskatchewan schools included students from First Nations communities encompassed within the provincial government education division; the three other Saskatchewan schools included students from First Nations communities governed within the educational jurisdiction of the Tribal Council. The respectful cooperation among school governance partners to implement this study is noteworthy.
4. This relatively high rate of missing data for the ethnicity/race item is likely due to the manner in which the question was structured. Students were provided five options which consisted of the five groups we expected to be most represented in the total group of students based on consultation with Elders and school partners (i.e., Cree, Ojibway, Metis, Oji-Cree, and Dakota). If students did not self-identify with one of these five groups, they were asked to specify their ethnicity/race in an open-ended item placed on the survey itself. It appears that many students either did not understand the instructions for this item or did not wish to provide this information to the investigators.
5. The first and second authors, both of whom are registered clinical psychologists, independently rated the BSI-DEP items for potential overlap with the hopelessness construct. Both raters agreed that there was only one BSI-DEP item that could be considered redundant with hopelessness (i.e., BSI-DEP item 5 “I am hopeless about the future”). Our central analysis

(i.e., the hypothesized structural model depicted in Figure 1) was rerun after rescoring the BSI-DEP with this overlapping item removed. We found our results were virtually unaltered when using a version of the BSI-DEP that dropped this overlapping (redundant) item. Detailed results of this supplementary analysis are available from the first author upon request.

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Review Article

Can Basic Risk Research Help in the Prevention of Childhood and Adolescent Depression? Examining a Cognitive and Emotional Regulation Approach

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This paper aims to highlight ways in which basic research findings in the field of childhood and adolescent depression can help to inform and refine preventive intervention efforts. We selectively review basic research evidence from community, clinical, and high-risk populations that identifies cognitive mechanisms (thought processes and reactions to negative affect) and emotional regulation as key processes involved in the onset and maintenance of depression. We focus on cognitive and emotional mechanisms in order to allow comparability with the majority of current preventive interventions. A range of basic research strategies and studies are then suggested that could be employed to help the development and refinement of prevention strategies. These include the need for prospective longitudinal studies to identify causal risk and protective factors, an integration of research approaches and methods, and a focus on understanding potential aetiological heterogeneity between childhood and adolescent depression.

1. Introduction

Depression is one of the leading causes of disability worldwide and the leading cause of nonfatal burden [1]. In childhood, estimates of the 12-month prevalence of depressive disorder range from 0.5% to 3% [2, 3], and an equal proportion of girls and boys are affected. Adolescence is associated with increases in the prevalence of depressive disorder and symptoms. Estimates of the 12-month prevalence of depressive disorder in adolescence range from 2%–8%, and the figure for lifetime adolescent depression is 20% [2–4]. The ratio of affected females to males is 2:1 in adolescence mirroring the pattern seen in adult life [2, 3]. Thus, adolescence is a key vulnerability period for depression with first onsets often occurring during this time and subthreshold symptoms increasing markedly [5–7]. There is strong continuity between adolescent depression with depression in adult life [8]. Both depressive disorder and symptoms in young people are associated with a range of negative outcomes. They deleteriously impact on current social, academic, and behavioural functioning and

substantially increase the risk for completed suicide [3, 9–11]. In the long term, depression also reduces the ability of young people to meet their academic and social potential, and a significant proportion of depressed adolescents continue to have mental health problems and poor outcomes in adult life [9, 12]. Thus, depression in young people, both in terms of symptoms and clinical disorder, is an area of significant public health concern.

2. Prevention as an Aim

Although there are a number of theoretical models of depression in young people [13–15], it is widely accepted that depression has a complex, multifactorial aetiology with multiple risk and protective factors acting together. Recent research strategy has emphasised the importance of prevention and early identification of risk for childhood and adolescent depression [16–18]. Efforts at prevention are warranted given the often chronic and relapsing course of depression, especially when onset is early [19,

20] and the limited treatment options for depression in young people following concerns about the safety and efficacy of antidepressants in this age group [21, 22]. The observation that adolescence is an important vulnerability period for the onset of depression suggests that prevention efforts may be usefully targeted during this period. Preventing the onset of depression rather than treating episodes when they occur has the potential to alter developmental trajectories and individual suffering across a lifetime.

This paper will selectively summarise what is currently known about cognitive risk factors and processes involved in the development of depressive symptoms and disorder in children and adolescents. A broad and inclusive definition of cognitive processes is taken that includes negative cognitions (e.g., pessimistic explanatory styles, negative beliefs about the self, world, and future), emotional regulation strategies, and characteristic information processing styles. We include research from community, clinical, and high-risk groups. We then identify some remaining questions that could be addressed in basic research and highlight how and why these might be informative for the continued development and refinement of preventive interventions. Thus we aim to link this selective review to prevention research. By way of introduction, we therefore first briefly mention current preventive interventions, give a rationale for prevention focusing on high-risk groups, and discuss the conclusions about risk processes that can be made from different types of research design.

2.1. Cognitive Processes as a Focus in Preventive Interventions. There is potential to prevent depression in young people, in particular, when psychological interventions are targeted at a variety of high-risk groups [23]. To date, the majority of these interventions have been based on current cognitive-behavioural treatment protocols. For instance, the preventive intervention showing the largest effect size in recent meta-analyses [24, 25] focuses on teaching cognitive restructuring techniques and coping strategies for stressful situations [26–28]. This emphasis on cognitive restructuring in prevention is in line with evidence showing the efficacy of cognitive behavioural therapy (CBT) for treating episodes of adolescent depression when they occur [29, 30] as well as with basic research evidence suggesting the importance of a range of cognitive products (e.g., dysfunctional attitudes, a pessimistic attributional style) in the aetiology of depression in adults and young people (reviewed later). Nevertheless, the effect sizes for CBT as a treatment for depression in young people are modest suggesting the potential for improving treatment efficacy [29]. Similarly, a recent meta-analytical review evaluated the effects of thirty-two depression prevention programmes for young people and concluded that 59% of programmes did not reduce depressive symptoms and 77% of evaluated programmes did not significantly reduce the risk for onset of major depression [24]. Generally, prevention effect sizes tended to be small, and intervention content was unrelated to effect sizes. This highlights the need for closer inspection of current prevention strategies: basic research can help to identify specific processes and active

ingredients with greater precision, and disentangling these from less crucial components is likely to allow for refinement of programme content and the development of more effective preventive approaches. Finally, the mechanisms that bring about change in effective interventions are not well understood, and a recent study indicated that prevention programmes that are generally effective can be ineffective in certain subgroups [28]. Thus there is still a place for basic research to test hypotheses of relevance to prevention efforts.

2.2. The Rationale for Focusing on High-Risk Groups for Prevention. There are various approaches to prevention the respective advantages and disadvantages of each are described in detail elsewhere [23, 31–35]. Briefly, prevention can be universal (involving all individuals within a particular population such as a school), selective (involving individuals who possess a risk factor for the disorder), or indicated (involving individuals with subthreshold symptoms of the disorder). To date, selective or indicated interventions (or a combination of both) show the most promising results for the prevention of depression in young people [23–25, 34]. For the purpose of brevity, we therefore focus on these types of prevention in this paper but note that universal interventions are reviewed in detail elsewhere [23, 34]. Epidemiological research and family studies have identified groups of young people at increased risk of developing depression. Risk factors precede and increase the probability of an outcome. However, they do not explain the causal pathways through which risk is increased. Risk factors can be fixed and unchangeable (e.g., gender) or potentially modifiable (e.g., pessimistic explanatory style). Although a variety of risk factors for depression in young people have been identified, the three groups that have been targeted in prevention efforts with some success are those with subthreshold depressive symptoms, previous depressive episodes, and the offspring of depressed parents [23]. Nevertheless, the majority of prevention efforts have selected participants on the basis of adolescent depressive symptoms (indicated prevention) rather than family history of depression (selective prevention). Children and adolescents with high levels of subthreshold depressive symptoms are more likely to develop depressive disorder in the future [36–38]. For instance, in a community cohort of adolescents aged between 9–18 years, symptoms in adolescence that were two standard deviations above the mean were associated with a 2-fold increase in the risk of depressive disorder in adult life [36]. Children and adolescents with a depressed parent are three to four times more likely to develop depressive disorder [39–41]. The course of depression is especially severe, recurrent, and impairing in the offspring of depressed parents [19]. Thus, basic research has identified groups that merit special consideration for early intervention [21] and prevention programmes [42]. However, it is important to understand the mechanisms or pathways through which depressive disorder develops in these high-risk groups because altering these processes will change the likelihood of the outcome [35]. This is not a straightforward task; for example, there is considerable heterogeneity in

outcome for the offspring of depressed parents [40], and the paths underlying these differences in children exposed to a similar risk factor are not well understood. A greater understanding of the causal mechanisms involved can help to refine preventive interventions [17, 35]. It is also possible that there are other high-risk groups where prevention has not yet been well examined but that may show benefit from preventive interventions. Children and adolescents with high levels of anxiety may be one such group given observations that anxiety often precedes depression and shows high levels of comorbidity with depression [17].

3. Cognition, Affect, and Depression in Young People

There is a huge literature on the role of cognitions in depression in children and adolescents, and recent reviews effectively summarise research findings for a range of cognitive constructs including rumination [43], attributional style, and dysfunctional attitudes [44]. A thorough review is therefore beyond the scope of this paper. Instead, we aim to briefly summarise key findings and put forward a series of suggestions of remaining research questions to be addressed that could be helpful for prevention research. We therefore particularly focus on newer areas of research where findings have not yet been incorporated into existing prevention strategies. Our suggestions are as follows. (1) Prospective longitudinal high-risk studies will be informative for identifying mechanisms and modifiers of risk that may be helpful for selective prevention efforts. (2) Innovations in measurement to assess “hot cognition” in the context of emotional challenge will be a useful adjunct to findings using self-report cognitive measures. (3) A greater understanding of potential heterogeneity between childhood and adolescent depression may help in refining prevention strategies for younger adolescents and children. (4) An integration of different research approaches may help in the identification of novel processes that could be targeted in prevention efforts. For instance, one potentially interesting area not yet considered in preventive interventions for young people is the role of cognitive control processes and mood repair strategies involved in the downregulation of negative affect.

3.1. Cognitive Studies: Research Design and Identifying Risk Processes. A variety of research designs have been used to assess the role of cognitive processing in child and adolescent depression including prospective community studies, cross-sectional studies of vulnerable groups (in particular the offspring of depressed parents), and case-control studies of clinically depressed children and adolescents. As highlighted elsewhere, modification of causal risk and protective processes is the aim of intervention as this will result in change in the outcome variable [35]. Nevertheless, different study designs are needed to distinguish risk factors and causal risk mechanisms. Different study types also tend to measure cognition in different ways: in the main, community studies have tended to use self-report questionnaire measures of negative cognitions (e.g., attributional style, dysfunctional attitudes)

whereas most case-control studies of clinically depressed groups have used information processing measures (e.g., decision making tasks). The majority of high-risk studies have used self-report measures although a few make use of observational or information processing measures. Mean level differences between high and low risk groups show that there are differences and suggest that these differences may be risk factors, some of which could be modifiable, but they are not informative about the risk *processes* involved in the onset of a condition (although prospective high-risk studies are an exception to this rule). Differences between clinical and control groups can be informative for identifying factors that might be risk factors; however it is not possible to tell whether these differences precede or follow the onset of disorder—temporal precedence being a necessary condition for defining a risk factor [45]. Identifying temporal precedence is a particular challenge for cognitive studies as studies of depressed adults show that depressed individuals show a range of characteristic information processing styles including selective attention and memory for negative emotional information as well as dysfunctional attitudes and rumination in response to low mood [46, 47]. It is important to establish whether these cognitive variables simply reflect the presence of depressive symptoms or whether they predict the onset and clinical course of depression. Studies that track clinical groups over time can be useful for identifying cognitive variables that influence the course of the disorder as well as distinguishing between variables that are mood state dependent and independent. Although widely examined in adults, this kind of research has not yet been carried out in clinically depressed young people. Causal hypotheses about risk mechanisms can only be tested using alternative research designs, in particular, treatment trials but prospective longitudinal studies are also informative about mechanisms provided a repeated measures design is used and assessment of the putative risk mechanisms can occur prior to the onset of disorder. High-risk or long term community followup studies provide an excellent opportunity for basic research to identify risk mechanisms because, crucially, cognition can be assessed prior to the development of depression. In summary, difference research designs allow different conclusions to be made regarding the role of cognitions as risk and protective factors.

3.2. Negative Cognitions and Self-Perceptions. Cognitive vulnerability models of depression have been a prominent theoretical approach in child and adolescent depression research, and attributional style and dysfunctional attitudes have been examined as predictors of depression in children and adolescents. In essence, classic cognitive vulnerability models posit a diathesis stress relationship between a stressful experience and an individual’s interpretation of the reasons underlying the experience whereby those individuals with negative beliefs about the self, world, and future (dysfunctional attitudes) and ascribe the causes of negative events to internal, global, and stable factors (negative attributional style) are more likely to develop depression following exposure to stressors [13, 48]. However, the precise role of cognitive reactivity in the onset of depression as compared

to the maintenance and recurrence of depression is less clear [13, 48].

On the whole, evidence shows that a negative attributional style is associated with depressive symptoms in prospective longitudinal studies in adolescents although the evidence is less consistent in children [44]. Given that episodes of depression involve symptoms such as a pessimistic outlook and a negative evaluative style, it seems likely that there may be a bidirectional relationship between measures of negative cognitions and depression in young people, and this pattern of results has been supported in recent studies [49]. It is also possible that past episodes of depression can influence explanatory style [50]. There is some evidence that prior depression negatively influences attitudes and estimates of personal competencies in young people [50, 51]. High-risk studies of the offspring of depressed parents also show that, in comparison to healthy control groups, these young adolescents have a more negative attributional style, lower self worth, and increased self-criticism [52, 53] with a suggestion that this could be related to severity of maternal depression [53]. Some studies report results which suggest that attributional style and rumination become increasingly more stable from childhood to adolescence [14, 43, 54]. An increase in the coherence of explanatory style at adolescence would be in line with developments seen in the complexity and coherence of self-representations from middle childhood throughout adolescence which are perhaps supported by improvements in more basic cognitive operations and reasoning skills [55–57]. In terms of attributional style and dysfunctional attitudes, there is better evidence for a prospective relationship with adolescent depression than childhood depression, and many studies do not find a consistent relationship with depression in childhood. The evidence for rumination seems similar, the effect is greater for adolescent depression than childhood depression, and there is not good evidence for a prospective relationship between rumination and depression over time once prior depression has been statistically controlled [43]. This developmental difference in the relationship between self-reported cognitive measures and depression may be due to at least some of the following factors: issues of measurement in childhood, general cognitive developments that underpin the development of individual's self-representations and aetiological heterogeneity between childhood and adolescent depression.

3.3. Emotionally Challenging or Stressful Situations. Research using observational measures has shown that high-risk children exhibit negative cognitions and show maladaptive emotional regulation strategies in emotionally challenging situations. For instance, an observational study of high-risk 5-year olds whose mothers had experienced postnatal depression showed that these children made more negative, stable, and internal statements in the context of losing (but not winning) a rigged game [58]. Another study illustrated that when forced to wait for a desired outcome, high-risk children of depressed mothers (aged 4–7 years) did not use strategies like distraction to regulate emotion but instead focused their attention on the desired object [59]. Moreover,

for girls, use of this less effective, more passive strategy was correlated with the number of previous maternal depressive episodes. Recent evidence from two small studies has also shown differences between high- and low-risk groups using information processing measures with an emotional stressor. The first found that high-risk adolescent girls (aged 9–14 years) showed selective attention for negative faces in an emotional dot probe task (following a mood induction) [60] although group differences have not been found on a range of standard nonemotional tasks of memory and attention [61]. The second used event related potentials and suggested that the children of depressed parents aged 6–9 years needed to use greater cognitive control processes in order to perform at the same level as healthy comparison children on an emotional (but not a neutral) attention task [62]. Taken together, these observational and information processing studies of high-risk groups suggest the importance of measurement and context. Two of the observational studies included young high-risk children suggesting that these observational measures may have been more appropriate for assessing cognitive-affective vulnerabilities in younger children (as opposed to the self-reports requiring introspection which are frequently used in studies of adolescents). The results also highlight the importance of “triggering” events or the possibility of “latent” cognitive styles that are activated following emotional challenge. Indeed, it is increasingly understood that cognitive representations exist in forms available to conscious introspection as well as forms that are not [63]. This underscores the importance of measures that require “online” processing of emotional material—this can be achieved in a number of ways, for instance, by using imagery or imaginative based formats, memory or attention tasks, the experience sampling method (ESM), or observational paradigms. Silk and colleagues used an ESM approach where participants aged 12–17 years reported on mood and regulation strategies on multiple occasions per day for one week [64]. They found that the use of involuntary engagement (e.g., rumination) and disengagement (e.g., avoidance, denial) was not effective in regulating anger and sadness, and these strategies were related to problem behaviours and depression, respectively. One final possibility for assessing “latent” cognition is the use of cognitive load manipulations based on the theory that individuals who are vulnerable to depression actively suppress negative cognitions and images [63, 65]. Thus, Rude and colleagues [66] used the scrambled sentences task [67] which is carried out both in a cognitive load and a nonload condition and found that the difference in negativity between the load and nonload conditions predicted the onset of depressive disorder in a group of female students. In summary, the use of tasks that assess cognitions in the context of cognitive load or emotional challenge may help in understanding the cognitive mechanisms involved in childhood and adolescent depression.

3.4. Neurocognition in Adolescent Depression. Depressed adults show impairments relative to healthy controls in a range of cognitive domains including attention, executive function, and memory [68, 69]. Evidence to date suggests

similar neurocognitive processing profiles in adolescent depression for tasks that involve processing emotional information [70, 71] and for overgeneral autobiographical memory [72] although adolescent depression does not seem to be associated with the more wide ranging impairments in cognitive functioning often observed in depressed adults [68, 73]. Autobiographical memory has been of interest to depression researchers in part because it is believed to be important in the development of self-perceptions [74], and evidence from adult studies suggests that overgeneral autobiographical memory could be a vulnerability marker for depression [75] (see below). Kyte and colleagues [70] used the Cambridge Automated Neurocognition Battery (CANTAB) [76] in a sample of adolescents with first episode depression. They reported similar affective biases as found in depressed adults [77, 78] although no impairments were found on a measure of attentional flexibility [69, 79]. Moreover, there was evidence of developmental effects on a decision making task with adolescent depressed cases showing more impulsive strategies than controls while depressed adults showed more conservative strategies [80]. Studies of neurocognition in adolescent depression are few and are invariably cross-sectional meaning it is difficult to separate causal precursors from correlates of current depression. However, some evidence from studies of adults suggests that these characteristics are not purely correlates of current mood state and could be vulnerability markers of depression in that cognitive variables like overgeneral autobiographical memory have been found to predict the course of depression, to remain following remission, and to be ameliorated by mindfulness CBT [81] which focuses on improving self-awareness. To summarise, there are few studies of neurocognition in child and adolescent depression. Longitudinal studies of clinical groups may be helpful in identifying cognitive factors that predict the course and prognosis of clinical disorder as well as potential markers that increase vulnerability for depression.

3.5. Response to Low Mood in Parents. Garber and colleagues [28] found that current parental depression was a moderator of the effectiveness of a combined selective and indicated trial of a CBT-based preventive intervention. That study reported effectiveness in preventing new onsets of major depressive disorder in young people compared to treatment as usual (overall hazard ratio = 0.63) but the intervention was not more effective than usual care in preventing depression for adolescents with a parent who was currently depressed (hazard ratio for adolescents with nondepressed parent = 0.24 versus 1.43 for adolescents with a depressed parent). A wide range of research has shown that current parental depression has deleterious effects for offspring, and a range of potential processes have been proposed including genetic and neuroendocrine factors, family dysfunction, and exposure to negative cognitions; for reviews see [41, 82–84]. Several lines of evidence now suggest a direct environmental link between maternal depression and offspring mental health. Firstly, a naturalistic trial of adult depression showed that remission of maternal depression was accompanied by improvement in offspring mental health [85–87]. Similarly,

genetically sensitive study designs show that there is a direct environmental link between maternal depressive disorder and symptoms with offspring depression [88–90]. A number of processes have been examined as potential candidates through which parental depression adversely affects offspring including impaired family functioning and parenting quality [41, 83, 84]. One possible factor that has not been well examined is the manner in which children respond to low mood in their parents. A small number of studies [91–93] indicate that emotional overinvolvement or feelings of responsibility for parental low mood are associated with emotional and behavioural problems in offspring as well as high levels of discrepancy between parental and child reports of child distress and parenting style. Emotionally overinvolved responses to parental low mood are more typical of girls than boys. Beardslee and colleagues have highlighted the likelihood of communication problems in families where a parent is depressed and have suggested the importance of self-understanding, and the ability to separate one's own feelings from those of significant others in resilient outcomes for these children [94–96]. Indeed, a clinician led psychoeducational programme that provided information on parental depression and sought to promote greater family understanding and communication has shown positive effects on depressive symptoms and other outcomes [97, 98].

3.6. Mood Repair and Metacognition. The proposal that self understanding and self-awareness are fundamental to adaptive outcomes in the offspring of depressed parents [94] has parallels with developmental research on emotional regulation as well as therapeutic approaches used in adult depression such as attention training [99] and mindfulness CBT which emphasise self-awareness, self-observation, and metacognition and show initial evidence as effective treatments for depression [100].

Mood Repair and Metacognition: Evidence from Young Children. Emotional regulation strategies show a prolonged developmental sequence with marked individual variation. Both individual child characteristics and parental influences are involved in the development of young people's characteristic emotional regulation styles [101–104]. Basic research in the area of temperament and emotion has long acknowledged the importance of higher-order cognitive processes in regulating and inhibiting prepotent responses including emotional responses. A number of studies have shown a relationship between inhibitory control with emotional regulation and emotional understanding in young children [105–107], and one randomised trial shows that it may be possible to improve inhibitory control processes through teacher-led classroom based initiatives [108]. Taken together, this evidence suggests that perhaps training in cognitive control processes and executive functioning might be useful in promoting effective emotional regulation strategies which in turn could have knock-on effects on depression in young people. Kovacs and Lopez-Duran [38] highlight the importance of mood repair in the development of depression in young people. They focus on the offspring

of depressed parents and propose that parental depression impairs children's ability to show positive affect relatively early in life which later interferes with the ability to effectively repair low mood. Effective mood repair involves activities such as finding meaning to negative experience, engaging in pleasant activities, thinking about pleasant experiences, and evoking happy memories. Thus, it follows that low positive affect may well interfere with effective mood repair. This is an interesting line of research that has not been well examined in the offspring of depressed parents and may provide a useful avenue for selective prevention.

Mood Repair and Metacognition: Evidence from Adult Psychotherapy. Conceptualisations of depression from an emotional regulation perspective propose that depressed mood is the result of an impaired ability to regulate negative emotions and an inability to switch off emotional systems once activated [47, 109]. This therefore has parallels with the developmental research mentioned above which highlights the importance of cognitive control processes in the development of effective emotion regulation strategies. Studies of depressed adults and adolescents indicate compromised cognitive control over emotion and particularly alterations in the processing and regulation of negative emotional information [110, 111], suggesting perseverative efforts to regulate emotion without appropriate relief.

A goal of emotion regulation strategies is not to prevent initial affective reactions from occurring but instead to experience them in ways that are more functional [47]. One therapeutic approach is to develop metacognitive skills for tolerating negative affect and approaching negative affect from a wider attentional frame [112]. Although various definitions of the term exist, fundamentally, metacognition involves active cognitive control over the process of thinking. In adult studies of current and remitted depression, increased metacognitive awareness of emotions is associated with reduced endorsement of dysfunctional cognitions following a negative mood induction [113], as well as reduced cognitive processing of negative material and increased willingness to tolerate negative affect in studies with nonclinical adult samples [114, 115]. One approach for increasing meta-awareness is mindfulness CBT which seeks to teach individuals that thoughts and feelings can be held in awareness without reacting to them as if they were true and to recognise the transience of these mental events [116]. As such, there is no emphasis on change in the degree of beliefs or attitudes but rather a development of decentered perspective to mood states and a view that although thoughts can be problematic, they do not have to be as appropriate responses can render them harmless. Drawing this further out may be useful in the development of more efficacious prevention approaches for young people. In summary, drawing on basic developmental research and adult treatment research, we speculate that cognitive control processes could be novel targets for boosting resilience against the onset of depressive disorder. This speculation

should be further examined using the relevant basic research designs.

4. Suggestions of Basic Research That May Help Refine Preventive Interventions

The results of recent randomised controlled trials herald the potential for the prevention of depression in young people [24, 25, 28]. Given the often chronic and relapsing course of depression and the huge amount of associated individual and societal burden, the potential of prevention holds great promise. There are good reasons to expect early life, in particular adolescence, to be crucial developmental periods for targeting prevention efforts, especially, observations that adolescence is a key period for first onsets of depressive disorder and that the course of depression is especially severe when onset is early [19]. With a few notable exceptions [117, 118], preventive initiatives have nearly exclusively been based on CBT treatment protocols. Meta-analytical reviews have shown that the effect sizes from prevention studies tend to be small and that somewhat surprisingly intervention content was unrelated to prevention effect sizes. However, this null second finding may in part be due to lack of variability in intervention content. Effect sizes have also been reported to be larger for older adolescents [24]. Nonetheless, small effect sizes are not a characteristic that is exclusive of preventive interventions with similarly small to medium effects reported for therapeutic interventions of adolescent depression [29].

We have argued that the findings from prevention programmes suggest that basic research studies can be used to generate hypotheses about putative causal risk and protective processes with greater precision and that this can help in the refinement of the content of preventive programmes. Here, we specify a number of particular suggestions for basic research that we believe could be helpful in refining the content of prevention programmes, identifying potential effect modifiers, and delivering interventions that are developmentally appropriate for the participating individuals.

- (1) Prospective longitudinal studies that track the development of high-risk groups prior to the development of depressive disorder will be informative for identifying novel targets and modifiers of effects for intervention in high-risk groups [38]. The study of high-risk groups may be particularly informative since prevention approaches appear to be most effective in a range of high-risk groups [23]. We suggest that the offspring of depressed parents are a high-risk group of particular interest given the increased risk of depressive disorder and poor prognosis in this group [19, 39–41] and the relative lack of focus on this group in prevention efforts in comparison to indicated interventions. Whilst there are many long-term studies of the offspring of depressed parents, few have used a prospective repeated measures approach. Important questions that remain to be addressed include the following. (a) Why and how does current parental depression have particularly

adverse effects on young people? (b) Are the same processes involved in the onset and recurrence of depressive disorder in young people? The first question is important for understanding a crucial effect modifier in prevention programmes [28] and the second for the selection of groups to be targeted in prevention studies (which to date have been most effective when focused on a combination of the following groups: adolescents with a prior depressive episode, those with subthreshold symptoms, and those with a depressed parent). Refining the selection of high-risk groups could be one way of improving effect sizes for prevention [38], and understanding general and specific processes involved in onset and recurrence, respectively, could be useful in addressing this issue. A study that prospectively examines a high-risk group over time and traces the development of depression *within* that group provides a rigorous method for identifying factors that predict depression and that could be targeted in selective interventions. This approach involves tracking individuals over time and examining whether changes in hypothesised vulnerability factors predict changes in depression whilst controlling for the mutual influences within and between vulnerability factors and depression.

- (2) Innovations in measurement are needed to assess “hot cognition.” That is, rather than relying on self-reports of how an individual thinks they would behave, contextual assessment of emotional regulation and/or cognitive processing especially in the context of an emotional challenge or stressor would be extremely useful. Possibilities include the experience sampling method, “online” information processing assessments, cognitive load manipulations, and observational tasks.
- (3) Basic epidemiological, family, and twin research indicates the likelihood of developmental differences between childhood and adolescent depression in terms of prevalence, risk factor profile, genetic aetiology, and rates of continuity with adult depression. In brief, depressive symptoms and disorder with an onset in childhood are more strongly associated with general family adversity [12, 119], have an environmental rather than genetic aetiology [39, 120, 121], and rates of continuity with depression in adult life are lower in comparison to adolescent depression [122]. The likelihood of aetiological heterogeneity between childhood and adolescent depression suggested by a combination of basic research findings suggests the possibility that it may be necessary to use different prevention strategies according to children’s age or cognitive development. A greater understanding of developmental differences is needed, in particular as one meta-analysis reports that preventive interventions are most effective in older adolescents [24]. Intuitively, it seems that older adolescents may be more developmentally ready for

cognitive therapeutic approaches which require skills such as problem solving and self-reflection, and this may reflect the reported correlation between intervention effectiveness and participant age. However, a more fundamental possibility is that developmental differences in the aetiology of childhood and adolescent depression exist which suggests different processes may need to be targeted in prevention at different ages. Longitudinal studies that begin in middle childhood are likely to be helpful in addressing these issues.

- (4) An integration of different research approaches to include basic research on temperament, emotional regulation, and neuroscience as well as therapeutic evidence from the adult depression literature could be helpful in creating modified interventions. One area that seems potentially fruitful is the role of cognitive control, and it remains to be seen whether interventions seeking to improve cognitive control and higher-order executive processes could be useful in preventing depression in young people.

5. Conclusion

In this selective review, we have examined the role of cognitive processes in childhood and adolescent depression. There is good evidence that a range of cognitive factors (e.g., dysfunctional attitudes, emotional regulation strategies) are associated with the onset and maintenance of depression in young people. However, the precise role of different cognitive factors—be they risk factors, causal risk factors, or correlates of depression is not always clear. Basic research that can more clearly elucidate these types of associations will be informative for preventive interventions. We have made a number of suggestions for basic research that may help address issues identified by reviews and meta-analyses of prevention interventions for childhood and adolescent depression including developmental differences and greater integration of findings from different research areas [24, 38]. We have particularly focused on prevention that targets high-risk groups. The development of preventive interventions is an incredible challenge and existing interventions show promise. The possibility of basic research informing prevention and vice versa relies on open channels of communication between researchers and clinicians. As suggested by others [123], the hope is that an iterative process can develop where basic research can help to modify and adjust interventions in an effort to maximise effectiveness, but that innovation in intervention also informs research knowledge.

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Research Article

Feasibility of the Positive Thoughts and Actions Prevention Program for Middle Schoolers at Risk for Depression

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Despite the importance of adolescent depression, few school-based prevention programs have been developed and tested in the United States with middle school populations. This study examined the acceptability and changes in targeted outcomes for a new preventative program, Positive Thoughts and Actions (PTA). Sixty-seven 7th grade students with elevated depressive symptoms were recruited from public schools and randomized to the 12-week PTA program with a parent-component or to a school-as-usual control group. The PTA prevention program was well received by students and parents, yielding high rates of participation and satisfaction among those randomized to receive the intervention. However, analyses of the efficacy of the program in changing depressive symptoms were not significant. In terms of our proximal program targets, most differences were not statistically significant, though effect sizes suggested advantage of PTA over control group in coping, cognitive style, and parent-child communication. This preliminary research highlights a need for further testing of programs for school-based prevention of depression and promotion of positive emotional health.

1. Introduction

Elevated levels of depressive symptoms can be detrimental for adolescents because they may interfere with important developmental processes and lead to a cascade of adjustment difficulties [1]. Youth with depressive symptoms are at significant risk for meeting diagnostic criteria for a depressive disorder later in adolescence [2] and in adulthood [3]. Depression and depressive symptoms are a primary risk for suicide, a leading cause of death for adolescents [4]. Longitudinal research has shown substantial continuity of youth depression into adulthood, with impaired functioning in work, social, and family life, and elevated risk of adult suicide attempts and completed suicide [5, 6]. A recent report by the National Research Council and the Institute of Medicine concludes that it is critical to shift the focus to advancing health and preventing disorders from occurring in the first place, rather than waiting until a disorder is well established and has done considerable harm [7]. The goal

of the current paper is to describe the development of a prevention model that addresses the needs of middle/junior high school students at risk for depression.

Schools play an increasingly important role in providing mental health services to children [8]. For the majority of children and adolescents, the school system provides the only source of mental health service [9]. Despite the importance of addressing depression and mental health as an overall component of youth health, only a handful of preventative programs targeting depression have been developed for middle schools, most of which have been tested in Australia [10–12]. In this country, the Penn Resiliency Program and the Coping and Support Training Program are the only two preventative interventions targeting depression that have been specifically developed for and empirically tested in middle/junior high schools [13, 14]. Thus overall, there is still a dearth of available programs targeted to meet the particular developmental needs of adolescents in this formative transition.

In response to a scarcity of curricula to address middle school stress and depression, the Positive Thoughts and Actions (PTA) program was developed and tested for feasibility in the current pilot study. The PTA program falls under the spectrum of mental health interventions as an indicated school-based prevention, while schools that operate under a positive behavioral support framework would classify the PTA program as a selected/targeted intervention [15]. The middle/junior high school time-frame was chosen because it marks a time of change and transition when youth adapt to numerous psychological, physical, cognitive, and social changes that are associated with an increase in psychopathology [16–18]. Our PTA curriculum was designed to address depressive symptoms through intervention on three proximal intervention targets. These intervention targets—coping, cognitive style, and parent-child communication—were chosen as indicators of outcome based upon their theoretical links to risk for depression. Addressing some of the risk factors that contribute to the escalation of depressive symptoms at this age may be important in preventing long-term adjustment difficulties that can arise from subclinical depressive symptoms. This developmentally based prevention program is unique and innovative in addressing key factors that contribute to and perpetuate depressive symptoms during the early adolescent years.

First, evidence suggests that both youth and adults with depression have less adaptive and more limited coping repertoires compared to youth without depression [19, 20]. For example, youth with depression may use less primary control (efforts to cope by making objective conditions conform to one's wishes) and secondary control (efforts to cope by adjusting oneself to fit objective conditions). Second, certain cognitive styles, including excessive negative thoughts and low levels of perceived control are characteristics unique to the development of depression [21, 22]. Third, poor parent-child relationships and family communication difficulties serve as risk factors for the development of depression among youth [23].

Most tested intervention programs for adolescent depression, including those that are not school-based, have been delivered exclusively to the youth, without any parental involvement [23]. There are a number of reasons why the inclusion of parents in the intervention process may be particularly important for younger adolescents, including that reactions to difficult events or circumstances within the family can precipitate depressive symptoms, youth cannot change many aspects of their environment, and interventions can be more effective when they are implemented consistently across situations and persons. Providing psychoeducation to parents of depressed youth has been found to be beneficial by improving parents' coping skills and the family climate [24]. Moreover, several studies suggest that adolescents learn cognitive and coping styles from their parents and caregivers [25]. Thus far, only two other school-based depression prevention programs published in the literature have included an active parent component [12, 26].

This pilot trial of the PTA program was conducted to determine if the contextual focus of the PTA program

was (1) acceptable to children and families, as indicated by their participation and satisfaction with intervention components, and (2) associated with improvements in youth's depressive symptoms, coping, cognitive style, and parent-child communication.

2. Methods

2.1. Subjects. A total of 67 7th grade students were recruited from 4 Seattle Public Middle schools after school wide screening for depression was conducted on a larger sample ($n = 684$) in Fall of 2005 and Fall of 2006. Students who scored higher than 14 (top 25%) on the Mood and Feelings Questionnaire (described below) after screening were invited to participate in the study. Exclusion criteria for students included (1) clinically elevated externalizing problems, (2) the presence of suicidal ideation, (3) probable diagnoses of Major Depressive Episode on the Patient Health Questionnaire—Adolescent Form, (4) plans to move to a nonparticipating school, and (5) parents who did not speak English. The first three exclusion categories were designed to ensure we were identifying youth who were appropriate for prevention and were not showing clinical levels of depression and related problem-behavior. Demographic characteristics of participating students and their families are provided in Table 1. We were able to retain 58 of the original 67 students (86.5%) for all followup assessments, as well as 60 of their parents (89.5%).

2.2. Instruments

2.2.1. Mood and Feelings Questionnaire (MFQ). The MFQ was designed for children ranging in age from 8 to 18 and was written in parallel versions for parent and child, which both were administered in this study. The MFQ comprises both the full range of items assessing the DSM diagnostic criteria for depressive disorders as well as additional items reflecting common affective, cognitive, and vegetative aspects of childhood depression [27]. It has shown both high content validity and criterion validity [28]. Internal consistency (Cronbach's Alpha) was reported at .90 in both parent and child samples. Parent-report items predict psychiatric versus pediatric patient status and depressed versus nondepressed status in clinical groups. The MFQ correlates highly with depression diagnoses and the Child Depression Inventory [29]. For this study, internal consistency (Cronbach's alpha) ranged from .89 to .94 across the four time points.

2.2.2. Children's Depression Rating Scale-Revised (CDRS). The CDRS-R was administered to youth to assess the severity of depressive symptomatology. The CDRS-R is a clinician-rated scale used as a screening and diagnostic tool, consisting of 17 items scored from 1 to 5 or 1 to 7 [30]. The total score of the CDRS-R has been shown to be sensitive to change in severity of symptoms in treatment studies [31]. For this study, internal consistency (Cronbach alpha) ranged from .71 to .84 across the four time points.

TABLE 1: Demographic characteristics of participants.

Characteristics	PTA group <i>n</i> = 36		Control group <i>n</i> = 31	
Mean Age (SD)	12.97	(0.36)	13	(.40)
Sex, <i>n</i> (%)				
Female	20	(55.6)	14	(45.2)
Male	16	(44.4)	17	(54.8)
Race, <i>n</i> (%)				
White	24	(66.7)	19	(61.3)
African American	1	(2.8)	3	(9.7)
Asian	2	(5.6)	3	(9.7)
Native American	2	(5.6)	—	—
Other	7	(19.4)	6	(19.4)
Ethnicity, <i>n</i> (%)				
Hispanic	1	(2.8)	6	(19.4)
Non-Hispanic	35	(97.2)	25	(80.6)
Parental Education, <i>n</i> (%)				
HS Diploma/GED/Some College	13	(36)	12	(39)
Associates/Bachelor's Degree	18	(50)	15	(48)
Masters/Professional/Doctoral Degree	5	(14)	4	(13)
Family Constellation, <i>n</i> (%)				
Single (1 parent family)	15	(42)	9	(29)
Married (or 2 cohabitating parent)	21	(58)	22	(71)

2.2.3. *Responses to Stress Questionnaire (RSQ)*. The RSQ [32] measures a range of responses to stress, including voluntary or controlled coping responses and involuntary or automatic reactions. Students are asked to rate how much they used specific coping techniques when faced with specific recent stressors. Scores from two scales were used for the current study: (1) *primary control engagement coping (PCEC)*, encompassing problem solving, emotion regulation, and emotional expression, and (2) *secondary control engagement coping (SCEC)*, encompassing positive thinking, cognitive restructuring, acceptance, and distraction. Convergent and discriminant validity of the RSQ has previously been established [33]. In the current sample, internal consistency (Cronbach alpha) for primary control coping ranged from .79 to .86, and secondary control coping ranged from .68 to .84.

2.2.4. *Personal Control Scale*. The personal control scale is a 5-item scale assessing the degree to which the youth feels a sense of control over their mood, problems, and life in general. It has previously shown good internal consistency ($\alpha = .77$) among high-school students [34]. For this study, internal consistency (Cronbach alpha) ranged from .82 to .85.

2.2.5. *Children's Automatic Thoughts Scale (CATS)*. CATS measures the frequency of negative thoughts, and has been validated on children aged 7–16 [35]. A 5-point rating scale ranging from 0 “not at all” to 4 “all the time” is used to rate 40 different automatic negative thoughts, including thoughts related to physical threat, social threat, personal

failure, and hostility. Internal consistency of the subscales is high, with test-retest reliability adequate. In prior research, the CATS measure clearly discriminated clinically depressed youth from those with anxious and oppositional problems [35]. Internal consistency (Cronbach alpha) for this study was high, ranging from .91 to .96.

2.2.6. *Parent-Child Communication Scale (PCC)*. The PCC Scale includes both parent and child-report forms, and was adapted from the Revised Parent-Adolescent Communication Form of the Pittsburgh Youth Study [36, 37]. The child measure assesses children's perceptions of their primary caregiver's openness to communication (10 items), and the parent measure (20 items) assesses both parent and child communication skills. Other studies have reported alpha coefficients for communication subscales ranging from .66 to .81 in 7th grade samples [38]. For this study, internal consistency (Cronbach alpha) for parent ratings of communication ranged from .51 to .81 across the four time points, whereas child ratings of communication ranged from .76 to .84.

2.3. *Procedures*. All students who scored 14 or above on the MFQ were individually evaluated for clinical needs using a brief clinical evaluation protocol. Their parent or guardian was called and provided with feedback about the child's needs and referrals for resources, if indicated. Students and parents who met inclusion criteria and consented to participate were randomly assigned to the intervention group, Positive Thoughts and Actions (PTA) or the control group.

TABLE 2: Outline of the Positive Thoughts and Actions prevention program content.

(a)

Student content		
Session no.	Title	Content
Week 1	Introduction and Purpose	To convey the purpose of the program, build a positive peer group, and to practice being positive with others
Week 2	Setting Goals	To motivate students to identify areas of their lives they would like to change and to select targeted goals and specific steps to take within each program area
Week 3	Start with Action	To teach the importance of getting active to improve mood and persist in goals
Week 4	Positive Thoughts, Positive Feelings	To understand the link between thoughts and actions and to increase positive thinking
Week 5	Changing the Way We Think and Feel	To identify negative and irrational thoughts and change them to be more positive and realistic
Week 6	STOP before Responding	To help students recognize when they are having an emotional reaction and to regulate their affect
Week 7	Making Decisions & Problem-Solving	To teach a 5-step approach to making decisions and solving problems
Week 8	Managing Conflict & Anger	To help students manage moods by controlling anger and resolving conflicts more productively
Week 9	Learning	To apply the skills learned to identified school goals
Week 10	Relationships	To practice applying Positive Thoughts and Actions to relationships
Week 11	Making Healthy Decisions	To adopt more healthy behaviors
Week 12	Staying on Track & Celebration	To recognize progress and identify areas of continued effort

(b)

Parent content		
Session type/no.	Title	Content
Home Visit no. 1	Getting to Know Each Other	To build rapport, to assess strengths and needs, and to help clarify the parents' supportive role
Parent Workshop no. 1	Positive Thoughts & Actions for Parents	To teach parents perspective-taking, and to provide an overview to the parent of emotion regulation strategies
Parent Workshop no. 2	Communicating with Your Teen	To give parents an opportunity to learn and practice different ways to communicate about feelings and/or problems
Home Visit no. 2	Staying Successful	To provide an opportunity for the student to summarize key concepts and identify how the parent can support them

PTA took place at school, consisting of 12 weekly (once per week) group-administered sessions, two home visits with parents and student together, and two group-based parent workshops, conducted in the evenings at the students' school. The PTA program included aspects of behavioral, cognitive, interpersonal, and family-systems interventions, the content of which is detailed in Table 2. PTA taught three major skills: thinking positively, taking positive action, and problem solving. Students applied these skills to self-identified problems/goals, and parents were given communication and problem-solving tools to help support their children.

The control group participants received usual care in the school, meaning they were free to seek school-based

(e.g., counseling) or other services (e.g., community mental health), but they were not provided with systematic intervention. Control group students attended their regular academic classes during the PTA student group time. Thirty-one students were assigned to the control group and 36 to the PTA intervention group.

Trained graduate level interviewers conducted structured research interviews in the family home. All instruments were administered to students and parents by separate interviewers after explaining the instructions and answering their questions. Participants were interviewed using all study measures at four time points: Winter of 7th grade, prior to the start of intervention (Baseline), Spring of 7th grade, in the weeks following intervention (Postintervention), Winter

of 8th grade, (6-months followup), and Fall/Winter of 9th grade, (18-months followup).

2.4. Data Analysis. Descriptive statistics were used to summarize demographic data. To determine the effects of the intervention, general linear model (GLM) repeated-measures analyses were conducted for each dependent variable with group (PTA versus control group) as the between-subjects variable and time as the within-subject variable. All analyses were conducted controlling for baseline levels depressive symptoms (CDRS). When significant time or group effects were found, posthoc contrasts were analyzed to determine the source of the individual differences. The statistical package used to run all analyses was SPSS (version 17.0), with statistical significance set at $P < .05$. Effect sizes (ESs) were also computed for all variables in order to examine the magnitude and direction of effects, using the procedures for Cohen's d with adjusted means (difference between the adjusted means of the treatment and control group, divided by a pooled standard deviation) [39]. All ESs were calculated such that positive values implied an advantage for intervention over control group.

3. Results

3.1. Participation and Satisfaction. Of the students randomized to the intervention group, 35 of 36 completed the prevention program, with an average attendance rate of 11 of 12 sessions for completers. One hundred percent of the parents of PTA youth received at least some of the parent intervention, and 94% received at least three of the four sessions. Twenty-six of the parent participants were mothers only (72%), 6 were fathers only (17%), 3 families had both parents participate (8%), and 1 "other caregiver" was the primary respondent (3%).

Parent satisfaction ratings were obtained following the initial parent-child home visit and the parent workshops. Of the 36 PTA parents, 72% of the parents found the initial home visit to be "very helpful", 25% found it to be "somewhat helpful", while one parent found the session to be "a little helpful". Overall, the parent workshop components were rated to be "very helpful" (45%) or "somewhat helpful" (55%) by those who participated.

Student satisfaction with their group membership and feelings about PTA class were gathered at week 11 of the intervention. Of the 36 PTA students, 48% liked the group they were in "very much", 36% liked the group "pretty much", and 13% felt the group was "all right". One student (3%) disliked the group a little and felt "embarrassed" about being in the class. Twenty percent reported feeling neutral about being in the class. The remaining students felt "comfortable" (30%) or "very comfortable" (47%) about being in the class.

3.2. Depressive Outcomes. Total scores on the Children's Depression Rating Scale-Revised showed a significant main effect across time $F_{2,53} = 4.89, P = .01$. Followup analyses revealed CDRS-R scores varied for the control group only

($F_{2,20} = 8.67, P = .002$) with higher CDRS-R scores at post-intervention and 18-month followup compared to 6-month followup. No significant interaction effect between time \times group ($F_{2,53} = .73, P = .49$) was found. Overall, parents reported fewer depressive symptoms than youth, as shown in Table 4. Parent ratings of depressive symptoms (MFQP) varied significantly across time, $F_{3,49} = 4.10, P = .01$. Parents in the control group reported significantly fewer depressive symptoms at 6-month and 18-month followup than baseline, $F_{3,18} = 3.62, P = .03$. For parents in the intervention group, ratings of their child's depression were lower at 6-month followup compared to baseline ($P = .03$), though overall mean differences were nonsignificant, $F_{3,28} = 1.71, P = .19$. No significant effects were found for depressive symptoms on the child-report MFQ ($P = .95$).

Examination of effect sizes for depression outcomes yielded a mixed picture, depending on time and informant (see Table 5). Youth PTA participants reported slightly higher mean levels of depressive symptoms after the intervention, compared to control youth, as indicated by the negative effect size value ($ES = -.16$). By 18 month followup, when participants were in the 9th grade, the pattern of results reversed, showing a slight advantage for PTA participants ($ES = .18$). Effect sizes based upon parental report suggested that parents of youth in the intervention group reported more depressive symptoms among their children at postintervention and 18-month followup. The CDRS yielded neutral to medium *negative* effect size values, although the two groups were not well matched in their CDRS scores at baseline.

3.3. Coping, Cognitive Style, and Parent-Child Communication. As for the three proximal intervention targets, primary control coping showed a significant main effect for group, $F_{1,53} = 7.22, P = .01$. PTA participants were found to have significantly higher mean levels of primary control engagement coping than the control group. Follow-up repeated-measures for group showed the PTA participants demonstrated significant improvements in coping at post-intervention compared to baseline ($F_{3,29} = 7.43, P = .01$). No significant interaction effect between time \times group for primary coping (PCEC) were found ($P = .86$), nor were differences in secondary coping (SCEC) on the RSQ significant ($P = .20$). In terms of our two measures of cognition, differences across time and group were found for one of these outcomes. Personal control scale showed a significant time \times group interaction, $F_{3,52} = 3.61, P = .02$. Followup analyses revealed differences for both groups, with mean differences shown in Table 3. PTA participants had significantly higher mean levels of perceived control at 18-month follow-up compared to baseline ($F_{3,30} = 2.92, P = .05$), while control group youth had significantly higher mean levels at 6-month follow up compared to post-intervention ($F_{3,19} = 3.70, P = .03$). No significant effects between groups were found for automatic negative thoughts ($P = .92$), though effect sizes suggested small advantages for the treatment group over all time points (see Table 4).

Finally, for parent-child communication, no significant differences were found for communication on the PCC parent or child versions, $P = .61$ and $P = .31$, respectively.

TABLE 3: Adjusted mean scores on depressive measures by group and time.

Construct Measure	Depressive Symptoms		
	MFQ-C	MFQ-P	CDRS
PTA			
Baseline	14.42 (9.85)	10.51 (10.17)	26.17 (7.50)
Post-intervention	15.91 (10.24)	9.11 (11.27)	27.45 (7.43)
6-month followup	10.86 (10.59)	7.37 (7.64)	25.67 (7.77)
18-month followup	16.17 (10.83)	9.28 (8.42)	27.75 (9.04)
Controls			
Baseline	14.87 (10.41)	10.67 (7.22)	23.95 (6.17)
Post-intervention	14.50 (7.41)	8.05 (6.44)	27.30 (6.78)
6-month followup	11.67 (6.83)	7.57 (5.65)	22.96 (4.01)
18-month followup	18.10 (10.96)	6.01 (5.26)	27.01 (9.61)

Standard deviations are in parentheses. MFQ-C: Mood and Feelings Questionnaire-Child; MFQ-P: Mood and Feelings Questionnaire-Parent; CDRS: Children's Depression Rating Scale.

TABLE 4: Effect sizes (Cohen's D) for outcomes across time.

Construct Measure	Coping		Cognition		Parent-child communication		Depressive symptoms		
	PCEC	SCEC	PC	CATS	PCCC	PCCP	MFQ-C	MFQ-P	CDRS
Postintervention									
	.67	.51	.64	.17	.35	.39	-.16	-.12	-.02
6-month followup									
	.52	-.05	-.32	.2	-.04	.15	.09	.03	-.44
18-month followup									
	.5	.16	.2	.24	.04	-.01	.18	-.47	-.08

Effect sizes were calculated using adjusted means from GLM (reported in Table 3). (+) = Treatment > Control (better than); (-) = Treatment < Control (worse than).

Effect sizes suggested that both parent and youth participants in the intervention condition reported better parent-child communication following intervention, but that these improvements relative to the control group did not sustain over time.

4. Discussion

The Positive Thoughts and Actions prevention program was well received by students and parents, yielding high rates of participation and satisfaction among those randomized to receive the intervention. Nearly 84% of adolescent participants reported liking the group. While indicated prevention programs that target individuals who display some early signs or symptoms have been criticized for the potential for increased labeling and stigma, we found that students' perceived embarrassment as a result of participation was low, with 77% reporting feeling "comfortable" or "very comfortable" participating, and another 20% reporting "neutral" feelings. These low levels of stigma are consistent with those reported among participants another school-based depression prevention, the Adolescents Coping with Emotions program [40]. Overall, the program was acceptable to students and families, and the structure of the intervention was conducive to participation. The conceptual framework for the program, intervention targets, and inclusion of

developmentally salient applications, such as learning, relationships, and making healthy decisions, were well received by our partner schools.

Analyses of the efficacy of the PTA program relative to the control group in changing depressive symptoms were not significant. Effect-size patterns were inconsistent across time and informant, with some negative effect-size values which could indicate iatrogenic effects of the PTA group. However, given the degree of scatter in mean values for youth-report measures, the fact that the PTA participants had higher CDRS scores at baseline, and the decrease in parent-reported depressive symptoms across time for both groups, it is difficult to discern the overall impact of PTA with these data. Moreover, effect size estimates using small sample sizes are prone to bias [41], and this can be further exacerbated when measuring episodic phenomena such as depressive symptoms. The PTA program developers are currently revising program materials based on input from consumers and two depression prevention expert consultants, and will conduct a larger trial as a next step. Increased sample size and power will allow for stronger conclusions about intervention effects to be made, but does not replace the importance of publishing these pilot data [42].

In terms of our proximal program targets, effect sizes suggested an advantage of the PTA over control group in each area, though most differences (aside from primary control

TABLE 5: Adjusted mean scores on proximal outcome measures by group and time.

Construct Measure	Coping		Cognition		Parent-Child Communication	
	PCEC	SCEC	PC	CATS	PCCC	PCCP
PTA						
Baseline	25.88 (4.37)	30.93 (4.97)	20.78 (5.65)	25.06 (18.75)	19.93 (3.15)	25.56 (3.28)
Postintervention	26.15 (5.74)	32.42 (7.54)	21.79 (5.39)	19.92 (14.84)	19.29 (4.61)	25.64 (2.86)
6-month followup	26.04 (5.14)	21.61 (6.03)	21.61 (6.03)	19.61 (19.55)	19.60 (2.77)	25.24 (2.90)
18-month followup	26.10 (6.17)	22.93 (5.22)	22.93 (5.22)	19.67 (19.84)	19.31 (3.93)	24.58 (3.73)
Controls						
Baseline	22.81 (5.17)	30.06 (6.43)	19.63 (6.13)	26.15 (20.68)	19.76 (4.07)	24.93 (3.61)
Post-intervention	22.48 (5.23)	28.85 (6.33)	18.27 (5.59)	22.55 (15.92)	17.83 (3.56)	24.51 (2.88)
6-month followup	23.42 (5.03)	23.31 (4.48)	23.31 (4.48)	23.77 (22.06)	19.72 (3.35)	24.80 (3.04)
18-month followup	23.61 (3.28)	21.89 (5.08)	21.89 (5.08)	24.48 (19.06)	19.15 (4.24)	24.62 (3.75)

Standard deviations are in parentheses. PCEC: Primary Control Engagement Coping; SCEC: Secondary Control Engagement Coping; PC: Personal Control; CATS: Children's Automatic Thoughts Questionnaire; PCCC: Parent Child Communication—Child; PCCP: Parent Child Communication—Parent.

engagement coping) were not large enough to achieve statistical significance. The largest and most robust increases for PTA participants were in areas of personal control and primary control engagement coping over time. Personal control and coping are common targets of preventative programs targeted to youth with internalizing problems, such as depression or suicidal ideation [34]. Gains in self-efficacy and control are associated with decreased vulnerability to suicide [43, 44] and moreover these skills are potentially important in and of themselves because they confer a sense of autonomy and increased personal resources for handling stress. Knowledge of coping skills can reduce and prevent negative consequences of stress in adolescence, thereby having far-reaching benefits that extend beyond a specific disorder. Resiliency research has linked positive adjustment to successful coping with developmental challenges. In terms of magnitude, differences in primary control engagement coping between PTA and control groups were “medium” in size.

However, it is important to note that despite these changes in primary control engagement coping, none of the changes in depressive symptoms were significant over time for PTA participants, showing a disconnect between outcomes. While we had conceptualized coping, cognitive style, and parent-child communication to be potential mechanisms for change in depressive symptoms, our results were not consistent with a mediational model because mediation is predicated on observing a clear intervention effect. Few other studies have examined mechanisms of change in depression intervention [45], and those that have tested for mediation have not found evidence that cognition necessarily mediates outcome in CBT outcome [46].

PTA stands out as one of the first programs to successfully implement a school-based intervention focused on depression with high levels of parent engagement. We were able to successfully engage at least one parent from all families through our outreach efforts to meet with them at their home or another convenient location. Meetings were scheduled in the evenings or on the weekends, at a time that worked best for the family. The same intervention specialist

who would be leading student groups set up a meeting with the family prior to the first group. Surveys of parent availability were conducted prior to scheduling all parent workshops to maximize attendance. Intervention specialists made personalized reminder calls prior to each workshop and family meeting to engage parents. In the final student group, students practiced giving a short speech about what they gained from the experience. Then, in the final family session, students presented this synopsis to their parent(s), as well as demonstrating some of the concepts learned from the PTA curriculum. All in all, the close tie between student groups and the parent component was believed to help foster parents' engagement and interest in participating.

This participation rate is higher than that found by other depression prevention programs based in schools. For example, in the Australian test of the resourceful adolescent program (RAP), only 36% of adolescents had a parent attend any session and 10% attended all 3 sessions. Our rates of parent participation in any intervention were similar to those achieved by the Penn Resiliency Program (PRP), where parents of 91% of students attended at least one of the parent intervention component sessions. However, our study yielded a much larger uptake when considering the percentage of parents participating in the majority of the intervention, with 94% of our parents completing at least three of the four parent sessions. The PRP parent intervention consisted of six 90-minute sessions targeted to parents' cognitions and coping skills, with 41% of parents attending at least five of the six sessions [26]. Thus, the results of our study, conducted in urban Seattle, and those of the PRP, conducted in suburban Pennsylvania, together demonstrate that it is possible to achieve high rates of parental involvement for school-based prevention programs when adequate resources for outreach are provided, such as meeting with parents at their home and at their convenience.

5. Conclusion

Schools are faced with increasing pressure to address the emotional wellbeing of students to promote learning and

healthy development. Building programs to address mental health is a critical component to improving overall school health. This preliminary research highlights the feasibility of school-based prevention programs addressing depression and promotion of positive emotional health. The most significant limitation of this research is the small sample size that was used in this feasibility trial. While it is difficult to draw conclusions about the outcome of the intervention given the small sample size, the intervention was well received, and parents were particularly engaged. However, it possible that identification and attention alone contributed to parent and student satisfaction, and both conditions were provided with a brief clinical evaluation and feedback, which may have had some effect on outcomes. Study information contributes to the meager understanding of the feasibility and uptake of school-based preventative interventions addressing depressive symptoms during middle/junior high school, and lays the foundation for further research examining the effectiveness and value of such programs for consumers, including students, parents, and school personnel. Future examination of the PTA program in additional schools with a larger sample will determine the effectiveness as a depression prevention curriculum.

Human Subjects Approval Statement

All study procedures were approved by the University of Washington Institutional Review Board.

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