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ORIGINAL ARTICLE

Investigating the Link Between Depression, Cognition, and Motivation in Late Childhood

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Abstract

Research has revealed broad cognitive deficits (e.g., memory, learning) in depression, and that motivation may account for this link. We tested the *state* (i.e., only present during depression), *trait* (i.e., underlying vulnerability) and *scar* (i.e., lasting corollary) hypotheses of cognitive dysfunction in depression. We additionally tested subjective motivation as a mediator of the concurrent depression-cognition link. In a longitudinal sample of 11,878 children ages 9–11, we found no evidence of a concurrent *state* or longitudinal *trait* or *scar* relationship between depression and cognition. The pattern of depression-cognition relationships—which precluded a mediator analysis—in our childhood sample is a departure from previous studies. Our findings indicate that cognitive deficits are not strongly associated with depression in childhood, in contrast with the impairment commonly seen in older individuals with depression.

Keywords Motivation · Depression · Cognitive function · Children · Longitudinal analysis

Introduction

There is a large body of literature suggesting that individuals with depression may experience cognitive impairment [1–4]. But which factors drive these associations, particularly in school-age children, remains unknown. Negative emotions can inhibit cognitive performance, while positive emotions can enhance cognition (e.g., [5]). One potential mechanism of such a relation of negative mood states (e.g., depression) to cognitive function is through disrupted motivation. School children of all ages typically experience a positive effect of motivation on cognition. Indeed, meta-analytic research has shown that motivational factors, such as the motivation to achieve, predict a student's academic achievement and cognitive ability [6]. The nonclinical child research hints at an explanatory role of motivation in the relationship between mood and cognition. Yet only a handful of studies examining mood, cognition, and motivation have specifically assessed *depressed* mood (e.g., [7–10]); to our knowledge, none have done so in a child population. Instead, research has primarily investigated the separate relations between depression and cognition (e.g., [11–13]), depression and motivation (e.g., [14, 15]), and motivation and cognition (e.g., [16–18]).

Given the evidence of pairwise relations, despite the limited research on interrelations between the three variables, the present study examined the role of motivation in the concurrent relation of depression to cognitive deficits among school-age children. Motivation is defined as the effect of a potential reward or punishment on one's behavior and cognitive functioning [19]. One prominent theory of motivation proposes two orthogonal motivational systems: the behavioral inhibition (or avoidance) system and the behavioral activation (or approach) system, known respectively as the BIS and BAS [20, 21]. The BIS governs aversive motivation, being preferentially sensitive to cues that indicate a negative or undesired outcome (e.g., answering incorrectly on a test). The BAS manages approach-worthy motivation, being preferentially sensitive to rewarding cues (e.g., answering correctly on a test).

In addition to the sparse literature on the relationship of motivation to depression-related cognitive dysfunction, questions remain about the *nature* (i.e., directionality) of the relationships between depression and cognition. Some

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research suggests that impaired cognition is a *state feature* of depression, such that it is a direct consequence of a depressive episode that disappears when symptoms remit (e.g., [22]). Other researchers find a lasting *scar effect* of depression on cognition (e.g., [11, 23]). There is also support for a possible *trait nature* of cognitive dysfunction, such that it represents a premorbid risk-factor for depression (e.g., [12, 24]). We addressed this research gap by investigating the concurrent and prospective relations between depression and cognition.

Theories of Cognitive Dysfunction in Depression

Systematic and meta-analytic reviews have consistently found broad cognitive deficits in adult depression [1–4]. Wagner et al. [25] extended these meta-analytic findings to youth, revealing impairments among depressed children and adolescents in executive functions (e.g., inhibition, attention), memory, and learning. While the childhood depression research is notably sparse, there is growing evidence of cognitive deficits in childhood samples not including adolescents (e.g., [26]). Accordingly, cognitive impairment is a potential target for early remedial intervention, in light of evidence that such deficits impede a patient's response to pharmacological (e.g., [27]) and cognitive-behavioral (e.g., [28]) treatment. There are several prevailing theories that attempt to explain a link between depression and cognition.

State Model

Some researchers posit that cognitive dysfunction is a *state feature* of depression, in that it only appears in currently depressed individuals. For example, the cognitive effort hypothesis suggests that depressed mood reduces capacity to allocate cognitive resources toward effortful—but not automatic—processing, resulting in cognitive impairment [29, 30]. In support of this hypothesis, Hammar et al. [22] found impaired cognition in depressed individuals on cognitively effortful tasks, despite intact ability for tasks reliant on automatic processing.

Other studies have challenged the cognitive effort model. Rose and Ebmeier [13] found MDD-related working memory (WM) deficits that were unaffected by task difficulty. Another study similarly found WM impairment in MDD patients relative to healthy controls that were unaffected by task complexity [31]. All the above-mentioned studies utilized MDD samples ranging between young and middle adulthood; to our knowledge, no studies have examined the mediational effect of cognitive effort—in our case, achievement motivation—in the link between childhood depression and cognitive dysfunction.

Scar Model

An alternative to state models is the scar framework, which holds that depression has lasting effects on cognition. A leading example is the neurotoxicity theory, which claims that the neurobiological mechanisms underlying depression impair cognitive ability via abnormal neurotransmitter activity and damage to relevant brain regions [32, 33]. Strong support for the neurotoxicity theory has been the finding of volumetric reductions in the hippocampus—a temporal lobe structure vital for learning and memory—amongst depressed individuals [33]. Furthermore, there is some evidence of chronic hypersecretion of the stress hormone glucocorticoid which may elevate vulnerability of the hippocampal neurons to damage [33].

In support of the scarring effects of depression on cognition, multiple adolescent studies have found worse depression to prospectively relate to worse cognitive ability (e.g., [11, 23]). Barch et al. [34] studied this link in a younger cohort and found that preschool-onset MDD was linked to later deficits in adolescent episodic memory (EM), but not in other facets of cognition such as executive function or processing speed. They also found cumulative depression severity (i.e., from preschool to adolescence) to relate to adolescent EM deficits, when controlling for cumulative psychiatric comorbidities like anxiety. It is important to note that the scar effect is not consistently supported. For instance, Schaefer et al. [35] did not find lasting cognitive deficits in MDD adults. To our knowledge, no studies have directly examined the prospective relation of depression to cognitive ability in a late childhood sample. Thus, it is uncertain whether depression relates to worse subsequent cognitive ability during late childhood.

Trait Model

The vulnerability hypothesis holds that certain characteristics of depression, such as cognitive deficits, are present *prior* to diagnosis and therefore represent risk factors for the disorder [25]. Research has been mixed about a trait role of cognition in depression. Studies of adolescent depression have found evidence for (e.g., [12, 36]) and against (e.g., [37]) premorbid executive dysfunction. Evans et al. [24] found that lower executive functioning in late childhood and early adolescence related to worse depressive symptoms 4 and 8 months later. This finding supports the trait theory that early cognitive impairment may be a premorbid factor of depression in late childhood. In contrast, Schaefer et al. [35] did not find childhood cognition to predict later MDD. Crucially, Evans et al. did not control for psychiatric comorbidities (e.g., anxiety), which may have influenced cognitive differences between more and less depressed youth. Schaefer et al. found evidence that cognitive deficits prior

to the onset of depression may only be present in individuals with comorbid mental illness.

All in all, very few studies have examined the prospective relations between depression and cognition specifically within late childhood. Therefore, it is unclear whether depression has a temporary *state* and/or lingering *scar* effect on cognition. Even more conflicting is the evidence as to whether cognitive dysfunction is an underlying trait of depression.

Depression and Motivation

In addition to cognition, motivation forms a key association with depression. Most importantly, the DSM currently designates anhedonia—substantially reduced interest or pleasure in enjoyable activities—as a core symptom of a MDE [38]. Adult depression research has consistently found decreased reward-response and effort expenditure (i.e., lower approach motivation) on various effortful cognitive tasks (e.g., [39–42]); additionally, such reward processing dysfunction has been linked to abnormal neural activation in reward-related brain regions among depressed adults (for a review: see [43]).

The child literature is less substantial. There is some evidence that depressed children exhibit diminished effort expenditure to reward (e.g., [14]) and demonstrate atypical reward-related brain activity (e.g., [44, 45]). Much of this research focuses on extrinsic motivation, whereas the current study is concerned with intrinsic motivational deficits. There is, in fact, limited evidence of achievement motivational deficits in late childhood depression [15]. To summarize, the research on motivation in depression suggests that depressed children may have deficits in effort expenditure on effortful tasks and in intrinsic achievement motivation.

Motivation and Cognitive Function

A large body of research has found motivation to strongly relate to cognitive function (for reviews: see [18, 19]) and general performance (for a review: see [46]). Research in this domain has typically examined the role of motivation in executive functions [18, 47]. For instance, Padmala and Pessoa [16] found that participants' selective attention improved as a function of greater monetary rewards (i.e., extrinsic motivation). The more central question in the current study, however, concerned intangible rewards, such as being correct (i.e., achievement motivation). To that end, there is some correlational [17, 18] and experimental (e.g., [48, 49]) evidence of a positive link between intrinsic motivation and learning and cognitive performance. The above-reviewed research calls for a closer investigation of the behavioral effect of intrinsic motivation on cognitive performance; to underscore the implication, achievement motivation (i.e., being correct) likely boosts cognitive performance and thus generally represents a confounding variable in a study of cognitive ability.

Motivation as a Mediator of the Depression-Cognition Link

Consistent with the above-reviewed evidence of dyadic relations between depression, cognition, and motivation, it has been theorized that motivation underlies cognitive dysfunction in depression [47]. Advocates of this mediational hypothesis suggest that depressed individuals do not experience dysfunctional cognition but rather *motivational deficits* that inhibit cognition. As an example, Bowie et al. [7] found that participants with more severe depressive symptoms more frequently skipped WM test items of higher difficulty than did participants with lower levels of symptoms. Further, more depressed participants did not differ from less depressed ones on WM during the low difficulty trials. These findings highlight that depression may not be characterized by cognitive deficits but rather impaired performance due to cognitive disengagement (i.e., low motivation to achieve).

While this work provides some evidence that motivation may explain worsened cognition in dimensional depression, the question remains whether motivation can account for the more severe cognitive impairment in clinical depression. Moritz et al. [9] addressed this and found MDD-related cognitive deficits, and that subjective task-related (i.e., state) motivation mediated this depression-cognition link. This mediational finding is especially relevant to the current study, as it signals that depressed individuals may be less motivated to expend mental and/or physical effort, leading to worsened task-related cognitive performance.

Hypotheses

A primary goal of the present study was to replicate the findings of depression-related cognitive dysfunction and to specifically extend this link to late childhood. We hypothesized that depression diagnosis/severity would relate concurrently and prospectively to lower cognitive ability, in line with the state and scar frameworks, respectively. However, we did not predict a significant relation of baseline cognition to later depression diagnosis/severity, as we expected that the prospective relation of cognition to later depression could be explained by psychiatric comorbidities, like anxiety, as submitted by Schaefer et al. [35]. An additional goal of the present study was to determine the potential mechanistic role of (impaired) motivation in the cognitive dysfunction commonly seen in depression. We hypothesized that motivation would mediate the link between depression and cognition.

Methods

Participants

We examined multi-site longitudinal data from the ongoing Adolescent Brain Cognitive Development (ABCD) study, which tracks 11,878 children from 21 research sites across the United States. All parents signed informed consent, and all youth participants completed written and verbal assent. All procedures in the current study were approved by either the centralized IRB at Washington University or by the IRB of a separate study site. Characteristics of the analyzed sample at baseline ($N = 10{,}337$) and Year 2 ($N = 5686$) can be found in Table 1.

Table 1 Sample characteristics

	Baseline				Year 2			
	Mean	SD	Min	Max	Mean	SD	Min	Max
Age (years)	9.91	0.62	8.92	11.08	11.96	0.64	10.58	13.58
CBCL depression t-score	53.57	5.71	50.00	89.00	53.70	5.87	50.00	89.00
CBCL anxiety t-score	53.49	6.12	50.00	97.00	53.36	5.85	50.00	100.00
Fluid composite t-score								
Sample	96.90	12.25	53.00	162.00	103.80	12.28	64.00	143.67
Depressed[a]	93.00	12.00	62.70	126.00	102.00	12.00	78.70	122.00
Not depressed[a]	96.90	12.20	53.00	162.00	104.00	12.30	64.00	144.00
Crystallized composite t-score								
Sample	106.20	18.29	33.00	197.00	103.60	16.69	55.00	169.00
Depressed[a]	99.40	17.20	52.00	158.00	103.00	18.60	70.00	146.00
Not depressed[a]	106.00	18.30	33.00	197.00	104.00	16.70	55.00	169.00
BIS sum score								
Sample	5.53	2.83	0.00	12.00	5.02	2.79	0.00	12.00
Depressed[a]	6.96	3.35	0.00	12.00	7.50	3.26	1.00	12.00
Not depressed[a]	5.51	2.82	0.00	12.00	5.00	2.78	0.00	12.00
BAS sum score								
Sample	12.86	4.64	0.00	24.00	11.71	4.54	0.00	24.00
Depressed[a]	14.60	5.45	0.00	24.00	13.10	5.05	5.00	22.00
Not depressed[a]	12.80	4.62	0.00	24.00	11.70	4.54	0.00	24.00
				%				%
Sex								
Male				52.2				52.1
Race/Ethnicity								
White				54.3				58.7
Black				13.9				11.1
Hispanic				19.2				18.3
Asian				2.0				2.0
Other				10.6				9.8
Household income (SES)								
< $25,000				15.0				12.2
$25,000 to $49,999				14.6				14.7
$50,000 to $99,999				28.4				29.6
> $100,000				42.0				43.5
Child-report depression Dx				0.95				0.70
Parent-report depression Dx				0.45				0.62
Child-report anxiety Dx				0.61				1.10
Parent-report anxiety Dx				1.97				1.74

[a]As assessed by Child-reported KSADS

Measures

Mental Health

We analyzed categorical and dimensional youth depression, given strong evidence that the relationship between depression and cognition is stronger in a continuous than a categorical assessment [50]. Categorical youth depression was measured at baseline and Year 2 via self- and parent informant-report with the computerized Kiddie Schedule for Affective Disorders and Schizophrenia for DSM-5 (KSADS-5; [51, 52]). Depression scores were assigned a "1" if the child met for a diagnosis of MDD or Depressive Disorder Not Otherwise Specified; scores were assigned a "0" if no such diagnoses applied. We also used self- and informant-report KSADS anxiety scores as a covariate in all models of depression. Anxiety scores were given a "1" if the child met for a diagnosis of Social Anxiety Disorder, Generalized Anxiety Disorder, or Specific Phobia; scores were given a "0" if no such diagnoses applied.

Baseline and Year 2 dimensional youth depression and anxiety were reported by parent informants using the Child Behavior Checklist (CBCL) DSM-5-oriented scales for Depressive Problems and Anxiety Problems [51, 53]. CBCL questions utilized a 3-point Likert scale from 0 ("not true") to 2 ("very/often true"), thereby measuring the severity of depressive and anxiety symptoms. We converted raw CBCL scores into T-scores and then to Z-scores for analysis. A detailed description of the diagnostic process can be found in the Supplementary Text.

Cognition

Cognitive function was measured at baseline and Year 2 via Fluid and Crystallized scales from the NIH Toolbox in the ABCD neurocognitive battery [54]. The three Fluid cognitive tasks tap into more flexible and adaptive problem-solving abilities that engage psychomotor function, memory, and cognitive control. The two Crystallized cognitive tasks measure language abilities that are reliant on knowledge/experience (e.g., vocabulary and reading). We further describe the NIH Toolbox Cognition battery in the Supplements. Two Fluid subscales were not administered at Year 2, which prompted us to manually compute the Fluid composite; we describe exploratory analyses of the missing subscales in the Supplements. We converted raw Fluid and Crystallized composite scores into age-corrected T-scores and then to Z-scores for analysis.

Motivation

We measured baseline and Year 2 subjective motivation via the 24-item self-report Behavioral Inhibition System/Behavioral Approach System (BIS/BAS) scales [20]. The BIS/BAS is treated in the literature as a stable, trait marker of motivation [20]. However, experimental research has found that sad mood predicts changes in one's scores on a personality questionnaire (e.g., [55]). Thus, the present study analyzed the BIS and BAS as state components of motivation. The BIS/BAS measures aspects of approach (i.e., response to reward cues) and inhibitory (i.e., response to punishment cues) behavior. Detailed descriptions of BIS/BAS subscales can be obtained from Barch et al. [51]. BIS and BAS sum scores (Drive and Reward Responsiveness) were used for analyses.

Statistical Methods

We carried out analyses in R of linear mixed effect models with standardized predictors, such that beta weights represented effect sizes. We additionally computed R^2 for all analyses as a measure of effect size [56, 57]. We designated a significance level of $\alpha = 0.05$ and corrected for multiple comparisons using the False Discovery Rate (FDR; [58, 59]). All significant results described below (and in the Supplementary Text) survived FDR correction, unless otherwise noted. All regression models included *age*, *gender*, *race/ethnicity*, *and socioeconomic status (SES)* as standard covariates. Models with depression controlled for concurrent anxiety. All analyses excluded participants with incomplete data; cross sectional analyses had a sample size of 10,337 at baseline and 5686 at Year 2, and longitudinal analyses had a sample size of 5391. We controlled for random effects caused by the inclusion of (approximately 800) twins and the multi-site setup of the ABCD study by nesting within family and study site. As we describe in our results, the mediation analysis was not performed due to insignificant mediation pathways. We describe the data analysis at length in the Supplementary Text.

Results

In our exploratory analyses, we computed the bivariate correlations of each of the demographic variables to the primary variables of interest, to assess the potential influence of demographics on our models' significance (see Table S6). We also ran post-hoc Mann–Whitney U tests to assess for clinical group differences in the NIH Toolbox Cognition composite and BIS/BAS motivation scores (see Table S8). These group difference tests are described in the Supplementary Text.

Concurrent and Prospective Relations Between Depression and Cognition

Group Fluid and Crystallized means for depressed and non-depressed children (according to Child KSADS) can be found in Table 1. As seen in Table 2, we did not find a significant concurrent relation at either baseline or Year 2 of self-reported KSADS Depression to Fluid or Crystallized cognition, when controlling for demographics and anxiety. Likewise, the concurrent relation of parent informant-reported KSADS Depression to concurrent Fluid or Crystallized cognition was not significant at either baseline and Year 2 (Table 2). We found greater CBCL Depression symptom scores to relate to worse Fluid scores at baseline, above and beyond age, sex, race, SES, and anxiety. This relationship was not significant at Year 2. In contrast, we found greater CBCL Depression scores to relate to better Crystallized cognition at Year 2, though not at baseline. Yet, neither of these significant depression-cognition findings passed FDR correction. To the extent that depression did relate to cognition, we wondered whether depression-related cognitive deficits do not appear in a linear fashion. We did not discover non-linearity in scatterplots (Figures S7–S10) of the correlations of depression severity to cognition.

Table 3 shows longitudinal relations of baseline depression to Year 2 cognition, when controlling for demographics, baseline cognition, and Year 2 anxiety. There were no significant relationships between any of the depression measures and Year 2 Fluid or Crystallized cognition. Table 3 also shows the absence of significant prospective relations of baseline Fluid scores to Year 2 KSADS or CBCL Depression scores, when controlling for demographics, baseline

Table 2 Concurrent relations of depression to cognition

Predictor	Fluid ability				Crystallized ability			
	B	t-value	Sig	R^2	B	t-value	Sig	R^2
Baseline child-report depression Dx	−0.163	−1.675	0.094	0.050	−0.153	−1.728	0.084	0.153
Baseline parent-report depression Dx	−0.055	−0.392	0.695	0.051	0.228	1.760	0.079	0.153
Baseline depression severity	**−0.027**	**−2.183**	**0.029**[a]	**0.053**	0.013	1.111	0.267	0.153
Year 2 child-report depression Dx	−0.154	−1.017	0.309	0.054	−0.004	−0.025	0.980	0.130
Year 2 parent-report depression Dx	0.217	1.324	0.185	0.055	0.165	1.093	0.275	0.131
Year 2 depression severity	−0.019	−1.132	0.258	0.055	**0.031**	**2.024**	**0.043**[a]	**0.130**

Models controlling for age, sex, race/ethnicity, SES, anxiety

Bold values indicate $p < .05$

[a]Model did not survive FDR correction

Table 3 Prospective relations between depression and cognition

Baseline predictor	Year 2 outcome	B	t-value	Sig	R^2
Child-report depression Dx[a]	Fluid ability	0.033	0.291	0.771	0.303
	Crystallized ability	0.057	0.661	0.509	0.592
Parent-report depression Dx[a]	Fluid ability	−0.262	−1.553	0.121	0.303
	Crystallized ability	0.188	1.468	0.142	0.593
Depression severity[a]	Fluid ability	−0.011	−0.729	0.466	0.303
	Crystallized ability	0.014	1.293	0.196	0.592
Baseline predictor	Year 2 outcome	B	z-value	Sig	R^2
Fluid[b]	Child-report depression Dx	0.073	0.069	0.945	0.001
	Parent-report depression Dx	0.359	0.220	0.826	0.009
	Depression severity	0.011	1.088	0.277	0.488
Cryst.[b]	Child-report depression Dx	**0.127**	**91.26**	**< 0.001**	**0.007**
	Parent-report depression Dx	0.752	0.516	0.606	0.014
	Depression severity	**0.037**	**3.541**	**< 0.001**	**0.490**

Bold values indicate $p < .05$

[a]In models controlling for age, sex, race/ethnicity, SES, baseline cognition, baseline anxiety

[b]In models controlling for age, sex, race/ethnicity, SES, baseline depression, Year 2 cognition, Year 2 anxiety

depression, and Year 2 Fluid and anxiety scores. Higher Crystallized scores at baseline did significantly predict a greater likelihood of child-reported Depression diagnosis at Year 2 when accounting for anxiety, though not parent-reported Depression diagnosis. There was also a significant positive relation of baseline Crystallized scores to Year 2 CBCL Depression scores (Figure S1), such that higher Crystallized cognitive performance was prospectively associated with more severe depressive symptom scores, even when controlling for baseline depression symptoms. Although, this prospective depression-cognition relationship was not significant for parent-report KSADS Depression. All significant concurrent and prospective depression-cognition relations were small effects according to their R^2 effect sizes, with the exception of the medium effect of baseline Crystallized scores predicting Year 2 CBCL Depression severity (Tables 1, 2, 3).

Of relevance, all depression-cognition analyses included anxiety as a covariate to establish specificity, and a high depression-anxiety comorbidity may impact our ability to see relationships with depression. We also considered that controlling for SES, unlike most other research in this field, may have influenced the null findings. We measured the effect of these correlations on Hypotheses I-III in Tables S1-S3, the details of which are described in the Supplementary Text. Ultimately, we found that controlling for anxiety and SES did mask some relations to depression. With that said, we additionally conducted Bayesian model comparison analyses to attempt to confirm our null results, and we found depression to generally be a weak predictor in our models with cognition. We describe the results of these post-hoc tests (Table S7) in the Supplementary Text.

Concurrent Relations of Depression to Motivation

Group BIS/BAS means for depressed and non-depressed children (according to Child KSADS) can be found in Table 1. Consistent with our hypotheses, we found that KSADS Depression was positively associated with BIS at baseline (Table 4), but not at Year 2. Contrary to our predictions, we also saw a positive relationship between KSADS Depression and BAS at baseline, rather than a negative one, though again this relationship did not hold at Year 2. We did not find any significant relations of CBCL Depression to BIS or BAS at either time point when controlling for all selected covariates (i.e., demographics and CBCL Anxiety) and nesting in family and site. All significant depression-BIS/BAS relations were small effects according to their R^2 effect sizes (Table 4). In light of the unexpected positive direction of the relationship between depression and approach motivation (BAS), we speculated that children may have misunderstood the BAS questions as reflecting externalizing behaviors. As a result, we carried out exploratory analyses involving externalizing mental health behaviors and attention-deficit hyperactivity issues. The details of these additional analyses are described in the Supplementary Text.

Concurrent Relations of Motivation to Cognition

Table 4 also depicts the concurrent relation of BIS and BAS scores to Fluid and Crystallized scores. There was a significant positive relation of BIS scores with cognitive performance at Year 2 (Figures S2–S3), but not at baseline. In contrast, we found a significant negative relation of BAS scores to cognitive performance at baseline (Figures S4-S5), such that higher approach motivation was associated with lower Fluid and Crystallized scores. The negative relation

Table 4 Concurrent relations of depression to motivation, and motivation to cognition

Predictor	Outcome	Baseline				Year 2			
		B	t-value	Sig	R^2	B	t-value	Sig	R^2
Child-report depression Dx[a]	BIS	**0.452**	**4.502**	**< 0.001**	**0.013**	**0.662**	**4.306**	**< 0.001**	**0.054**
	BAS	**0.258**	**2.618**	**0.009**	**0.028**	0.292	1.869	0.062	0.012
Parent-report depression Dx[a]	BIS	**0.480**	**3.287**	**0.001**	**0.008**	0.234	1.400	0.162	0.041
	BAS	**0.306**	**2.154**	**0.031**	**0.027**	−0.142	−0.841	0.400	0.012
Depression Severity*	BIS	0.001	0.078	0.938	0.018	0.021	1.232	0.218	0.062
	BAS	0.001	0.100	0.920	0.027	−0.011	−0.667	0.505	0.012
BIS[b]	Fluid	−0.001	−0.132	0.895	0.051	**0.034**	**2.604**	**0.009**	**0.055**
	Cryst	0.016	1.828	0.068	0.153	**0.111**	**9.335**	**< 0.001**	**0.142**
BAS[b]	Fluid	**−0.046**	**−4.693**	**< 0.001**	**0.053**	−0.013	−1.005	0.315	0.055
	Cryst	**−0.078**	**−8.705**	**< 0.001**	**0.160**	**−0.042**	**−3.494**	**< 0.001**	**0.132**

Bold values indicate $p < .05$

[a]In models controlling for age, sex, race/ethnicity, SES, anxiety

[b]In models controlling for age, sex, race/ethnicity, SES

of BAS to Crystallized performance was present at Year 2 (Figure S6), but that of BAS to Fluid was not significant above and beyond demographic covariates. All significant BIS/BAS-cognition relations were small effects according to their R^2 effect sizes (Table 4). Further, the baseline BIS-Crystallized relation became significant and positive in an exploratory analysis that controlled for ADHD symptoms (Table S4). We still saw a negative relation of BAS to Fluid at baseline (Year 2 was not significant) and to Crystallized ability at baseline and Year 2, when controlling for externalizing behaviors and for ADHD symptoms (Table S4). Of relevance, ADHD and Externalizing were each significant predictors of concurrent BAS scores at baseline and Year 2, above and beyond demographics (Table S5).

Mediation Model of Depression-Cognition Link

Concurrent relations of depression to motivation ("*a* path"), motivation to cognition ("*b* path"), and depression o cognition ("*c* path"), were not consistently significant at baseline and Year 2. This does not provide support for the hypothesis that motivation mediates the relationship between depression and cognitive performance. Therefore, a mediation model was not performed. Likewise, given these primary results, we did not perform any secondary analyses with the Fluid and Crystallized and BIS/BAS subscale scores.

Discussion

The present study sought to parse out the role of motivation in the relationship between depression and cognition. It also had the goal of clarifying the state, trait, and scar nature of cognitive deficits in depression, in a late childhood sample. There was no support for our predictions that depression would be negatively related to concurrent or later cognition. In line with our prediction, we did not find strong evidence of a prospective relation of cognitive function to the onset of depression or depressive symptoms, when accounting for comorbid anxiety. We also did not find evidence consistent with the hypotheses that disruptions in motivation might be a mediator of the relationship between depression and cognition. In general, the significant relations between depression, cognition, and motivation were small effects; the positive prospective link between Crystallized ability and later informant-reported depression severity was a medium effect. It is possible that future analysis of these variables with more follow-up waves of the ABCD study will generate larger magnitude effects. We discuss below the possible explanations for the findings that are consistent with the extant literature, as well as those that appear to contradict prior findings.

The first set of findings—that depression was not related to worse cognition with any measure at either time point—is a departure from previous meta-analytic findings of broad cognitive deficits in early-onset depression [25]. The present study may have had more comprehensive models of covariates, such as anxiety. In fact, only a *handful* of key studies in this domain of childhood research have controlled for anxiety (e.g., [60–62]). Even so, excluding anxiety in our post-hoc analyses changed only one relationship—that of baseline depression severity to concurrent Fluid ability. That depression severity relates to worse Fluid ability is consistent with existing research on the depression-cognition link (e.g., [13]). Of the few studies that took anxiety into account, even fewer controlled for race (e.g., [60]), and none controlled for socioeconomic circumstances, whereas the present study assessed both. There is strong evidence of a relationship between SES and depression (e.g., [63]) and cognition (e.g., [64]), and thus SES may have been a significant confound in prior studies. Yet, our exploratory analyses did not reveal a noteworthy impact of SES on our null findings. Our study also differs from prior studies in that our sample did not include adolescents. We discuss this below in "Limitations" section.

We found no significant relationships with either composite score of cognition at Year 2 across the three baseline reports of depression (categorical child-report and informant-report and dimensional informant-report). These results are not consistent with research supporting scar frameworks of cognitive dysfunction in depression, such as the neurotoxicity theory (e.g., [11, 23, 65]), but are consistent with studies that have not found a scarring effect of depression on cognitive ability (e.g., [36]). It is possible that the low rate of depression in our sample affected the analytical power of this prospective relation, as the average parent reported few depressive symptoms on the KSADS and CBCL for their child. We expect parents to report more depressive symptoms as their children age, given evidence that depressive symptoms become more significant during adolescence (e.g., [66]). Additionally, previous research may have detected a scar relation due to a less comprehensive model of covariates or to sample age heterogeneity.

The third hypothesis—that cognition does not significantly predict later depression, when controlling for anxiety—was largely borne out by the data. This finding is consistent with prior research that has also found a lack of evidence for an underlying trait role of impaired cognition on later depression (e.g., [35, 37]). However, there were two analyses where higher baseline Crystallized ability predicted Year 2 depression (i.e., child-report KSADS, CBCL). While we did not expect cognition to predict later depression, these select cases of significance do not support the conventional trait vulnerability hypothesis of the depression-cognition relationship. It is unlikely that *high* Crystallized ability

represents a trait marker of depression, and this positive relationship is not consistent with prior research that has instead found a negative relationship (e.g., [23, 67, 68]). That said, an earlier cross-sectional study found a similarly positive relationship between internalizing mental health behaviors (e.g., depressed mood and related somatic issues) and Crystallized cognition in the ABCD dataset [69]. An alternative explanation for this positive relation is that children high in certain Crystallized abilities pertaining to language (e.g., vocabulary) are better at grasping their mood disturbances and putting words to their emotional challenges, relative to peers lower in this cognitive domain. As a result, we speculate that children high in Crystallized ability may be better equipped to identify and rate their depressive symptoms on questionnaires such as the KSADS and the CBCL. The fact that prior studies have not uncovered this particular finding may be attributed to less comprehensive models (i.e., with fewer demographic covariates) and smaller sample sizes.

Our fourth hypothesis generally showed mixed support. The positive depression-BIS relationship was anticipated. On the other hand, the positive depression-BAS relationship was not consistent with existing research on subjective achievement motivation in relation to depression (e.g., [15]). Our post-hoc findings with CBCL Externalizing and ADHD, however, indicate that participants may have misinterpreted BAS questions as reflecting less adaptive externalizing qualities (e.g., impulsivity) rather than more adaptive reward-driven motivation.

The fifth hypothesis—that BIS and BAS, respectively, relate negatively and positively to cognitive performance—was not supported. Instead, we found BIS to associate positively, and BAS to associate negatively, with cognition. These findings are not consistent with prior research on achievement motivation and cognitive function (e.g., [48, 49]). Even our post-hoc analyses with CBCL Externalizing did not reverse the direction of the relation, as it did with some of our depression-motivation relations. Ultimately, we were unable to examine BIS/BAS motivation as meditators of the concurrent depression-cognition link. It is possible that our findings suggest a lack of these motivation associations to depression and cognition in childhood. It is worth noting that, during adolescence and puberty, the reward-related corticostriatal regions in the brain (e.g., basal ganglia, thalamus, amygdala) experience significant developmental changes (e.g., [70]). Perhaps, then, as children undergo puberty and mature into adolescents, motivation becomes an explanatory factor in mood-related cognitive dysfunction.

Limitations

Though the current study had many critical strengths, such as its sample size and geographic diversity, there were also several key methodological limitations. Principally, our analyses of the mediation model pathways involving motivation were limited by our measure. The BIS/BAS is treated in the literature as a trait index of approach/avoidance motivation, and we are not aware of any research utilizing the BIS/BAS as a state measure of subjective motivation. Furthermore, we recognize that the BIS/BAS is an index of general approach and avoidance motivation, and that a more specific definition and measure of subjective achievement motivation may be necessary in a similar study. This study was somewhat restricted in its scope in the sense that we had no direct assessments of subjective achievement motivation. An interesting example of this test of motivation can be found in the methodology of Moritz et al. [9]. Secondly, given that we had to delete two subscales from our baseline Fluid Composite due to those measures not being collected at Year 2, it is possible that we were less able to detect significant concurrent and longitudinal relations at that time point (i.e., Hypothesis I–III, V). However, exploratory analyses of the baseline data with the complete Fluid Composite scores (i.e., all five subscales) did not reveal any discrepant results. Lastly, the current study was limited by having a single point of longitudinal analysis. Ideally, a similar study would feature multiple points of analysis to examine the depression-cognition relationship throughout development. As such, it is possible that our hypotheses will become progressively more accurate in the ABCD sample, as the child participants age and their brains develop.

Future Directions

Future studies into the role of motivation in the depression-cognition link might benefit from using a validated state measure of subjective motivation. As we mention above, a study by Moritz and colleagues developed such a measure, the *Momentary Influences, Attitudes and Motivation Impact on Cognitive Performance Scale* (*MIAMI*; [9]). Whereas the BIS/BAS asks participants about *general* tendencies to approach rewarding, or avoid aversive, outcomes, the MIAMI *directly* probes distinct elements of participants' achievement motivation in relation to the cognitive tasks used in the study. The MIAMI uses a 4-point Likert scale to measure such aspects as participants' subjective motivation to exert oneself on a cognitive task and fear of cognitive task difficulty (see Moritz et al. for example questions on the MIAMI).

We hypothesize that a stronger depression-cognition link may emerge over time. Furthermore, we found modest evidence of a prospective Crystallized cognition-depression link but not in the typically negative direction. Researchers should closely scrutinize the nature of this relationship to determine whether better language and reading skills in youth make it easier to *detect* early depressive symptoms. If

true, this would suggest a need to develop additional methods of detecting depression in younger children that are less reliant on language abilities. In general, there are methodological considerations that may account for our null findings. Notably, much of the prior research on depression-related cognitive deficits in childhood utilized a wide range of age. Many researchers express the concern that depressive samples that span from childhood to adolescence may obscure the developing nature of cognitive dysfunction in youth depression [71, 72]. In addition, while the present study sought to identify depression-specific deficits in cognition by controlling for anxiety, a recent meta-analysis suggests that cognitive dysfunction—the *c factor*—may be a transdiagnostic element across internalizing and externalizing disorders [73]. Thus, future longitudinal youth research (e.g., ABCD) can control for a greater diversity of psychiatric disorders to determine which cognitive deficits are specific to depression and which are potentially transdiagnostic.

Summary

The present study was novel in its ability to test various theories of cognitive dysfunction in depression in a large, longitudinal sample with a strong model of demographic and mental health covariates. We failed to replicate previous findings of broad cognitive deficits in youth depression. Our results indicate that prior findings of cognitive deficits in youth depression may be partly attributed to demographic factors, comorbid psychopathology, and age heterogeneity. We also did not find that subjective motivation mediates the depression-cognition link. Our study suggests that cognitive dysfunction may not strongly typify youth depression, and it highlights the developmental question of when and why cognitive dysfunction appears in depression.

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Declarations

Conflict of interest The authors declare that they have no conflicts of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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