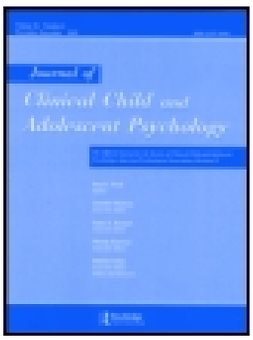

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Impaired Maturation of Cognitive Control in Adolescents Who Develop Major Depressive Disorder

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This study examined whether development of two forms of cognitive control (proactive and reactive) between early and midadolescence was associated with the onset of major depressive disorder (MDD) during the same period and if it prospectively predicted MDD onset between mid- and late adolescence. Adolescents ($N = 165$) completed 3 waves of assessments, at 12 (T1), 16 (T2), and 18 (T3) years of age. Diagnostic interviews were conducted at each time point to identify three groups of adolescents: "early MDD," those who developed MDD between early (T1) and mid- (T2) adolescence ($n = 23$); "late MDD," those who developed MDD between mid- (T2) and late (T3) adolescence ($n = 20$); and "controls," those who did not develop MDD ($n = 122$). A modified Stroop task was completed at T1 and T2 to examine development of proactive and reactive cognitive control. Adolescents with early MDD exhibited significant declines in reactive control, as well as a trend level decline for proactive control, during this period compared to controls. No significant differences in reactive or proactive control were identified in adolescents with late MDD compared to controls, but they did exhibit significant improvements in proactive control compared to those with early MDD. These findings suggest that normative maturation of reactive, and possibly proactive, cognitive control abilities are impaired in adolescents who develop MDD between early and mid-adolescence. This has implications for understanding the mechanisms underlying certain forms of behavioral dysregulation that are commonly seen in MDD.

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INTRODUCTION

Adolescence represents a time of heightened risk for the onset of depressive disorders. The incidence of depressive symptomatology rises sharply during the transition

from childhood to adolescence (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993), with the point prevalence of unipolar depression estimated to be approximately 5.6% in adolescents, compared to 2.8% in children younger than age 13 (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). The onset of depression during this important developmental period has adverse effects on functioning across many domains, including increased academic failure, family problems, substance abuse, and truancy (Kessler, Avenevoli, & Merikangas, 2001).

Adolescence is also a period of significant development of cognitive control (Leon-carrion, García-Orza, & Pérez-Santamaria, 2004; Luna, 2009), which refers to the ability to flexibly adapt and direct cognitive processing, enabling individuals to engage in goal-directed behavior, such as control of impulses, inhibition of unwanted thoughts, and regulation of emotions (Heatherton & Wagner, 2011; Hofmann, Schmeichel, & Baddeley, 2012). It is now widely acknowledged that cognitive control deficits are an important aspect of depression (Snyder, 2013), with patients frequently complaining of attention and concentration difficulties, and diagnostic criteria for major depressive disorder (MDD) including "diminished ability to think or concentrate, or more indecisiveness" (American Psychiatric Association, 2013).

COGNITIVE CONTROL IN DEPRESSION

There is now ample evidence from empirical studies that MDD is related to impairments in cognitive control in adults (see Austin, Mitchell, & Goodwin, 2001; Fossati, Ergis, & Allilaire, 2002; Rogers et al., 2004, for reviews), with a recent meta-analysis of 113 studies finding effect sizes of 0.32–0.97 across different components of cognitive control (Snyder, 2013). The most consistent findings come from studies on patients with current MDD, which suggests that cognitive control deficits may be state dependent (Baune et al., 2010; Gohier et al., 2009; Harvey et al., 2004; Stordal et al., 2004). However, some studies on adults have also identified impairments in patients following the remission of acute episodes (Bhardwaj, Wilkinson, Srivastava, & Sharma, 2010; Clark, Sarna, & Goodwin, 2005; Hammar et al., 2010; Paelecke-Habermann, Pohl, & Leplow, 2005; Preiss et al., 2009; Weiland-Fiedler et al., 2004), suggesting that trait-dependent deficits may also be present. Moreover, it remains unclear whether these findings in remitted adults are reflective of cognitive control deficits as predisposing factors that precede illness onset or if they are purely sequelae of the illness.

Significantly less research on cognitive control deficits has been done in younger MDD populations. Similar to the adult literature, there is some evidence for cognitive control deficits in children and adolescents with current MDD (Cataldo, Nobile, Lorusso, Battaglia, & Molteni, 2005; Micco et al., 2009; Vergara-Lopez, Lopez-Vergara, & Colder, 2013; Wilkinson & Goodyer, 2006), although some studies suggest otherwise (Favre et al., 2008; Hill et al., 2012). Two studies on at-risk children of mothers with depression have also failed to identify any impairments in cognitive control (Klimes-Dougan, Ronsaville, Wiggs, & Martinez, 2006; Micco et al., 2009), which argues against these deficits being vulnerability factors that are observable prior to illness onset (although it should be noted that it is uncertain whether these children went on to develop depression). Further, the only study examining cognitive control in adolescents in remission from depression failed to identify any significant differences from healthy controls (Maalouf et al., 2011), although this needs replication before strong conclusions can be drawn. Given the inconsistencies in the current literature, and the lack of evidence for or against cognitive control deficits representing a vulnerability factor for illness onset, further research is needed to better understand whether cognitive control deficits are present prior to onset and/or following remission of depression. Furthermore, there has been no research to date addressing the relationship between depression and the *development* of cognitive control. As just mentioned, significant maturation of cognitive abilities occurs during adolescence, and it is possible that this developmental process may be more sensitive to the onset of depression than absolute abilities at any one point in time.

DUAL MECHANISMS OF CONTROL

Recent research suggests that cognitive control can be differentiated into a number of component processes. One such model by Braver and colleagues (Braver, 2012; Braver, Gray, & Burgess, 2007) argues for two distinct operating modes of cognitive control—"proactive" and "reactive" control—which are differentially engaged based on situational demands. Proactive control refers to preparatory/anticipatory processes that are aimed at enhancing coping before conflict occurs, and are thus sustained over the duration of a given task. Reactive control refers to transient/corrective processes that are implemented once conflict has occurred. Therefore, these two forms of control are thought to be more efficient at different moments when monitoring and dealing with conflict (i.e., early/sustained selection for proactive control vs. late/transient correction for reactive control; Vanderhasselt et al., 2014). For example, within the context of interference resolution on a cognitive task, such as the Stroop, reactive control

would detect and resolve interference after the onset of incongruent color-word stimuli, whereas proactive control would anticipate and prevent interference prior to the onset of stimuli.

Therefore, an important factor that modulates the extent to which each type of control is engaged is the expectation of error. For example, manipulation of the proportion of incongruent stimuli relative to congruent stimuli within the Stroop task has been found to influence the use of proactive versus reactive strategies. Behavioral and neuroimaging data indicate that proactive control is engaged when a high proportion of incongruent trails are presented in a block of Stroop stimuli, as participants can predict these trials and employ conscious top-down control to adjust the relative influence of word reading on color naming and reduce the amount of response competition for incongruent trials. Comparatively, during conditions of high expectancy of congruent trials, participants cannot develop such cognitive strategy and use reactive/evaluative control for each trial.

Given that task demands may differentially engage proactive and reactive control, it is possible that proactive and reactive control are used to regulate emotions at different points along the timeline of the emotion generative process, as proposed by Gross (1998), Gross and John (2003), and Gross and Thompson (2007). Proactive control may be more likely to be engaged to regulate emotions before an emotion-eliciting event occurs, such as "situation selection" to approach or avoid certain places, people, or things. In comparison, reactive control may be more likely to be engaged to regulate emotions after the emotion-eliciting event has already occurred, and may involve processes such as "cognitive reappraisal" to change the interpretation of a situation in order to alter its emotional impact.

These two types of cognitive control have been hypothesized to be driven by the functioning of distinct neural circuits (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Kerns et al., 2004; van Veen, Holroyd, Cohen, Stenger, & Carter, 2004), and dysfunction in these same neural circuits has been consistently identified in depression (Drevets, Price, & Furey, 2008; Levin, Heller, Mohanty, Herrington, & Miller, 2007; Price & Drevets, 2009; Rogers et al., 2004), suggesting that both proactive and reactive control might be impaired in patients with depression. In adults, there is some support for this hypothesis from neurophysiological data (Vanderhasselt et al., 2014; West, Choi, & Travers, 2010). Previous studies investigating event-related potentials (ERPs) have identified poorer proactive control when preparing for upcoming stimuli, based on reductions in the amplitude of the prestimulus slow waves (West et al., 2010) and the contingent negative variation component (Vanderhasselt et al., 2014). Similarly, abnormal reactive control has been identified in depression based on the amplitude of the medial frontal negativity related to conflict/error detection in the Stroop task (Holmes & Pizzagalli, 2008; West et al., 2010).

In comparison, support from behavioral findings has been inconsistent. For example, Holmes and Pizzagalli (2008) identified reactive control deficits in a clinical sample of current MDD patients, but they did not investigate proactive control abilities. In contrast, others have failed to identify a relationship between reactive (Saunders & Jentzsch, 2013; West et al., 2010) and proactive (Saunders & Jentzsch, 2013) control abilities and depressive symptomatology, although these studies did not examine clinical samples of patients with MDD diagnoses. Consequently, it remains uncertain whether depression is characterized by impairments in proactive and/or reactive control abilities at the behavioral level.

Furthermore, there has been no research to date examining proactive and reactive control abilities in adolescents with depression. Prior research by our group identified different developmental patterns for proactive and reactive control between 12 and 16 years of age, with reactive control improving (in male individuals alone) and proactive control not changing (Vijayakumar et al., 2013). Andrews-Hanna et al.'s (2011) functional neuroimaging study of 14- to 25-year-olds identified earlier development of neural regions implicated in reactive control and later development of those supporting proactive control. Similar differences have been found in structural neuroimaging research, with more protracted and continued development of regions underlying strategy generation and maintenance (i.e., aspects of proactive control) into early adulthood (Gogtay et al., 2004). Therefore, it is possible that global measures of cognitive control used in past research, such as the Wisconsin Card Sorting Test or Tower of London, may have failed to identify significant impairments associated with depression, especially during adolescence, because they were not sensitive to the two distinct maturational patterns of proactive and reactive control.

THE CURRENT STUDY

As previously noted, there is a paucity of prospective and longitudinal research examining cognitive control prior, and subsequent, to the onset of MDD during adolescence. We were able to address this gap in the literature by utilizing an existing data set from a community adolescent sample comprising cognitive control data at early and midadolescence and clinical diagnostic data at early, mid-, and late adolescence. The current study investigated whether the development of proactive and

reactive control between early and midadolescence was associated with MDD onset during the same period, and whether it predicted MDD onset subsequently, between mid- and late adolescence. We chose to examine *development* of (i.e., longitudinal change), as opposed to *absolute* (i.e., cross-sectional), cognitive control abilities, on the basis that it may be a more sensitive measure given the continued neurobiological and behavioral maturation occurring during this developmental period.

It was hypothesized that individuals who developed MDD between early and midadolescence would exhibit compromised development of reactive control compared to those who did not develop MDD, given that the postulated neurobiological substrates of reactive control have been implicated in depression and significant behavioral maturation of reactive control occurs during this period. However, it was not predicted that development of MDD between early and midadolescence would affect proactive control, as past research has failed to identify significant behavioral maturation of proactive control during this period. Furthermore, it was anticipated that the development of cognitive control would not predict the onset of MDD between mid- and late adolescence, given that prior research has failed to identify cognitive control difficulties in nonsymptomatic children at high risk for depression.

METHODOLOGY

Participants

Participants were a community sample of 165 adolescents (M age = 12.67 years, $SD = 0.39$; 86 female) recruited from schools across metropolitan Melbourne, Australia, as part of a larger cohort study, the Orygen Adolescent Development Study (OADS). The primary aim of the OADS was to prospectively examine biopsychosocial risk and resilience factors for emotional and behavioral problems during adolescence. The OADS screened a large number (i.e., 2,453) of early adolescents from primary schools using the Early Adolescent Temperament Questionnaire–Revised (Capaldi & Rothbart, 1992). Based on their scores on this measure, a smaller sample (415 students) was invited to participate in the study, with adolescents at the extreme ends of the temperamental distribution being oversampled to maximize risk and resilience for psychopathology. This sampling procedure has previously been described in detail by Yap et al. (2011).

Of the invited 415 students, 245 agreed to participate in the OADS, with 165[1] of these adolescents agreeing to participate in all three waves (at early [Time 1, or T1], mid- [T2], and late adolescence [T3]) of assessment of Axis 1 disorders, using the Schedule for Affective Disorder and Schizophrenia for School-Aged Children: Present and Lifetime Version (Kaufman & Schweder, 2004). Individuals who met the criteria for current or past MDD, substance-use disorder, or eating disorder at baseline were excluded due to the broader aims of the study. Therefore, data on Axis 1 disorders collected at T2 and T3 provided information about the onset of MDD between early and late adolescence. In the current sample, 23 adolescents (14%) experienced the onset of MDD between T1 and T2 (referred to as "early MDD"), 20 adolescents (12%) experienced the onset of MDD between T2 and T3 (referred to as "late MDD"), and 122 adolescents (74%) did not develop MDD between T1 and T3 (referred to as "controls"). Refer to Figure 1 for an illustration of this information. Two adolescents who had current episodes of MDD during the T2 assessments were excluded from the analyses in order to avoid confounding state effects. Comorbid disorders were common; refer to Tables 1 and 2 for information on the number and type of comorbid diagnoses present in the sample, respectively.

Adolescents completed cognitive assessments at T1 and T2 (see details next). IQ was also assessed at baseline using a short form of the Weschler Intelligence Scale for Children, Fourth Version (Wechsler, 2003) and socioeconomic classification (socioeconomic status [SES]) was assessed based on the Australian National University Four Scale (Jones & McMillan, 2001). Refer to Table 3 for information of IQ, SES, and other demographic characteristics of the sample. Informed consent was obtained from the child and at least one parent/guardian at each time point, consistent with the guidelines of the Human Research Ethics Committee at the University of Melbourne, Australia.

Modified Stroop Paradigm

Participants completed a modified version of the Stroop task at T1 and T2, which has been previously validated by Carter et al. (2000). The original Stroop task has been found to be sensitive to improvement in cognitive control during adolescence (Biederman, Petty, Doyle, et al., 2007; Biederman, Petty, Fried, et al., 2007; Leon-carrion et al., 2004). Furthermore, the modified paradigm used in this study has been shown to be particularly sensitive to individual differences and distinguishing cognitive control in clinical samples (Andrews-Hanna et al., 2011; Braver et al., 2007; Vijayakumar et al., 2013; Yücel et al., 2012). Similar to the original Stroop task, participants were required to respond to the color of written stimuli, which were themselves names of colors. Stimuli were either

[1]The final sample of 165 did not differ from the original screening sample of 2,453 on SES or gender ($p > .05$).

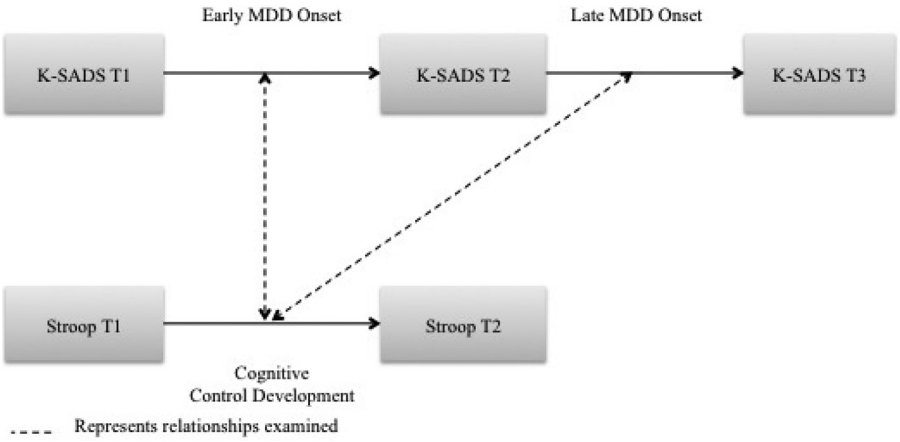

FIGURE 1 Overview of data collected and relationships examined. *Note:* MDD = major depressive disorder; K-SADS = Schedule for Affective Disorder and Schizophrenia for School-Aged Children.

congruous (i.e., the word "blue" written in blue: BLUE) or incongruous (i.e., the word "red" written in blue: RED). Incongruent trials required participants to select a weaker, task-relevant response (naming the color) in the face of a competing stronger but task-irrelevant response (reading the word). The response competition usually increases response time for incongruent trials, resulting in an "interference effect," which is the difference between reaction time for congruent and incongruent trials.

In addition, the paradigm modified the proportion of incongruent trials within task blocks in order to examine proactive versus reactive aspects of cognitive control. Participants completed 48 practice trials, followed by two blocks of 96 experimental trials. One block was manipulated to have a higher proportion of congruent trials ("Mostly Congruent" [MC]: 75% probability; 72 congruent and 24 incongruent trials), whereas the other block had a higher proportion of incongruent trials ("Mostly Incongruent" [MI]: 75% probability; 72 incongruent and 24 congruent trials). It is purported that proactive control is employed during conditions of high probability of incongruent trials in the MI block, as participants can predict these trials and employ conscious top-down control to adjust the relative influence of word reading on color naming and reduce the amount of response competition for incongruent trials. Comparatively, during conditions of high expectancy of congruent trials in the MC block, participants cannot develop such cognitive strategy and use reactive/evaluative control for each trial. This is thought to result in less control over the prepotent word reading tendency, thus causing greater response conflict for incongruent trials. The

TABLE 1
Number of Comorbid Diagnoses

		*Controls**		*Early MDD*		*Late MDD*	
	No. of diagnoses	*N*	%	*N*	%	*N*	%
T1	0	108	88	12	52	15	75
	1	12	10	11	48	4	20
	2 or more	2	2	0	0	1	5
T1–T2	0	101	83	10	44	16	80
	1	16	13	7	30	4	20
	2 or more	5	4	6	26	0	0
T2–T3	0	103	84	14	61	9	45
	1	16	13	8	35	7	35
	2 or more	3	2	1	4	4	20

Note: MDD = major depressive disorder.
*Values refer to total number of diagnoses, as opposed to comorbid diagnoses.

TABLE 2
Type of Comorbid Diagnoses

		Controls		*Early MDD*		*Late MDD*	
		N	%	*N*	%	*N*	%
T1	Depression	0	0	1	4	1	5
	Anxiety	10	8	10	43	5	25
	Behavior	4	3	0	0	0	0
T1–T2	Depression	2	2	0	0	1	5
	Anxiety	8	7	4	17	2	10
	Substance	6	5	5	22	1	5
	Eating	2	2	1	4	0	0
	Adjustment	2	2	3	13	0	0
	Behavior	4	3	5	22	0	0
T2–T3	Depression	2	2	2	9	1	5
	Anxiety	5	4	3	13	7	35
	Substance	11	9	3	13	2	10
	Eating	1	1	0	0	1	5
	Adjustment	2	2	2	9	0	0
	Behavior	1	1	0	0	0	0

Note: MDD = major depressive disorder. Values refer to type of diagnoses, as opposed to type of comorbid diagnoses.

TABLE 3
Sample Characteristics

	Overall[a]	*Controls*[b]	*Early MDD*[c]	*Late MDD*[d]
Age at T1	12.67 (0.39)	12.68 (0.39)	12.64 (0.44)	12.63 (0.32)
Age at T2	16.5 (0.52)	16.51 (0.54)	16.48 (0.49)	16.47 (0.41)
Age at T3	18.90 (0.46)	18.87 (0.46)	18.94 (0.49)	18.94 (0.48)
SES	58.37 (20.85)	61.34 (20.59)	50.31 (19.85)[e]	49.59 (19.50)[f]
IQ	109.08 (16.66)	108.81 (17.29)	107.35 (11.00)	113 (18.46)
Sex (F:M)	86:79	58:64	17:6[e]	11:9

Note: Data for all continuous variables represent mean (standard deviation). MDD = major depressive disorder; SES = socioeconomic status; F = female; M = male.
[a]$n = 165$. [b]$n = 122$. [c]$n = 23$. [d]$n = 20$. [e]Early MDD vs. controls: $p < .05$. [f]Late MDD vs. controls: $p < .05$.

order of administration of MI and MC blocks was counterbalanced across participants. The task was presented using Presentation 0.70 software (Neurobehavioral Systems) on a PC laptop.

Statistical Analysis

All analyses were conducted using SPSS version 20, and results were considered significant at $p < .05$. Analyses were conducted using Stroop reaction time (RT) interference scores, which were calculated by subtracting mean RT for congruent trials from mean RT for incongruent trials, with larger scores indicating poorer response inhibition.

Multinomial logistic regression analyses were performed to investigate group differences in cognitive control development from T1 and T2 between controls (those who did not develop MDD), early MDD (those who developed MDD between T1 and T2), and late MDD (those who developed MDD between T2 and T3) groups. All regression analyses included sex, age at T1, SES at T1, IQ at T1, and Stroop interference at T1 in Step 1 as covariates, followed by the Stroop interference at T2 in Step 2. Controls were set as the reference category, thus allowing comparison with the early MDD and late MDD groups. Separate sets of analyses were performed for the proactive (MI) and reactive (MC) conditions of the Stroop task.

Treatment of Missing Data

Seventy-one participants (43%) in the sample had missing Stroop data at T1 and/or T2, with 19% missing data from both T1 and T3. In addition, one participant's Stroop scores were excluded on the basis of errors being greater than 50%. Participants with and without Stroop data did not differ on sex, onset of MDD, or SES and IQ at baseline ($p > .05$). Little's MCAR test was found to be nonsignificant ($p > .05$), indicating that the data were missing completely at random; thus missing data were imputed using the Expectation Maximization procedure in SPSS version 20.

RESULTS

A final sample of 165 adolescents was used in the analyses. Tables 4 and 5 show the mean RT and percentage errors for the Stroop task at T1 and T2.

TABLE 4
Mean (Standard Deviation) Stroop RT at T1 and T2 Assessments

	Mostly Congruent			*Mostly Incongruent*		
	Incongruent RT	*Congruent RT*	*RT INT*	*Incongruent RT*	*Congruent RT*	*RT INT*
			T1			
Overall	1057.54 (132.87)	849.76 (106.79)	207.77 (85.64)	979.26 (124.87)	865.94 (118.07)	113.32 (74.38)
Controls	1056.70 (130.67)	850.31 (105.62)	206.39 (89.79)	974.60 (131.58)	864.49 (125.26)	110.11 (80.96)
Early MDD	1065.14 (154.47)	863.23 (106.65)	201.91 (84.48)	1006.13 (94.38)	885.76 (90.05)	120.38 (45.43)
Late MDD	822.43 (119.78)	741.54 (98.62)	222.95 (58.32)	976.79 (113.99)	852.01 (101.64)	124.78 (57.94)
			T2			
Overall	899.89 (140.31)	732.24 (106.02)	167.65 (83.46)	846.24 (122.57)	749.19 (110.33)	97.05 (59.23)
Controls	896.82 (144.13)	732.90 (110.34)	163.92 (83.95)	845.04 (125.16)	749.28 (115.50)	95.76 (57.00)
Early MDD	947.24 (142.93)	750.93 (104.70)	196.30 (84.38)	873.33 (110.26)	755.37 (94.17)	117.96 (60.85)
Late MDD	864.19 (99.32)	706.73 (75.36)	157.46 (76.19)	822.43 (119.78)	741.54 (98.62)	80.89 (66.98)

Note: RT = reaction time; INT = interference score; T = Time; MDD = major depressive disorder.

TABLE 5
Mean (Standard Deviation) Stroop Error % at T1 and T2 Assessments

	Mostly Congruent			*Mostly Incongruent*		
	Incongruent Error %	*Congruent Error %*	*Error INT*	*Incongruent Error %*	*Congruent Error %*	*Error INT*
			T1			
Overall	15.23 (8.88)	7.33 (5.78)	9.30 (5.66)	13.21 (9.85)	8.98 (8.49)	4.83 (3.34)
Controls	14.68 (7.95)	6.94 (5.16)	8.88 (4.83)	13.29 (10.39)	9.21 (9.46)	4.61 (3.48)
Early MDD	15.97 (11.32)	9.37 (8.83)	11.02 (8.90)	13.27 (9.39)	8.53 (5.16)	5.61 (2.35)
Late MDD	17.73 (11.00)	7.34 (4.80)	9.94 (5.58)	12.64 (6.86)	8.12 (4.42)	5.33 (3.38)
			T2			
Overall	7.80 (7.80)	3.85 (3.16)	12.15 (9.02)	6.26 (4.26)	4.41 (4.34)	5.80 (3.91)
Controls	7.67 (7.67)	3.58 (3.39)	12.27 (9.63)	6.12 (4.18)	4.29 (4.43)	5.66 (3.83)
Early MDD	8.26 (8.26)	4.73 (2.44)	12.08 (8.06)	6.79 (3.75)	5.51 (4.10)	6.47 (3.70)
Late MDD	8.03 (8.03)	4.42 (2.07)	11.51 (6.03)	6.53 (5.31)	3.88 (4.06)	5.86 (4.70)

Note: INT = interference score; MDD = major depressive disorder.

Logistic regression analyses revealed a significant main effect of sex and SES on early MDD. Females and adolescents with lower levels of SES at baseline predicted membership in the early MDD compared to the controls group, indicating that they were more likely to experience MDD onset between T1 and T2. Lower SES levels at baseline also predicted membership in the late MDD compared to the controls group, but sex was not a significant predictor. IQ and age at baseline did not significantly predict early MDD or late MDD compared to controls. Furthermore, none of these demographic variables predicted membership in early MDD compared to late MDD groups.

In relation to the Stroop development variables, change in the MC interference score (i.e., MC interference score at T2 after controlling for interference score at T1) significantly predicted early MDD ($p = .042$). As illustrated in Figure 2, the controls and early MDD groups exhibited similar Stroop interference scores at T1 for the MC condition. However, controls exhibited a significant decline in interference scores for the MC condition ($p < .001$), whereas no change was identified in the early MDD group. In addition, interference scores were not significantly different between groups at T1 but were trending toward significance at T2 ($p = .092$). Further exploration of RT for the incongruent and congruent trials at T1 and T2 revealed that controls and early MDD groups exhibited similar reductions in speed for congruent trials, but controls had greater reductions in speed for incongruent trails compared to the early MDD group (see Figure 3). There was also trend toward significance for change in MI interference score predicting early MDD ($p = .071$), with less reduction in interference score being related to MDD onset.

Stroop development (i.e., interference score at T2 after controlling for interference score at T1) did not significantly predict late MDD compared to controls for either MI or MC conditions. However, change in the MI interference score significantly predicted early MDD compared to late MDD ($p = .039$). As illustrated in Figure 4, the early MDD and late MDD groups exhibited similar Stroop interference scores at T1 for the MI condition. This was followed by a significant decline in interference scores for the MI condition in the late MDD group ($p = .005$), whereas no change was identified in the early MDD group. In addition, interference scores were not significantly different

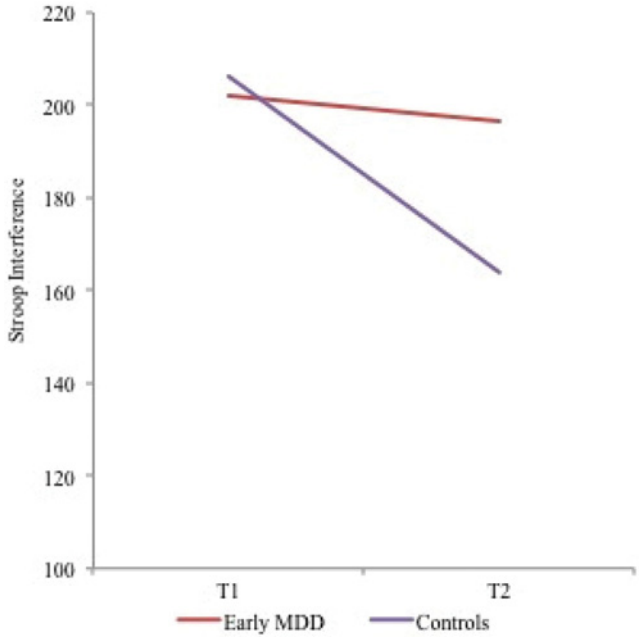

FIGURE 2 Stroop interference scores for controls and early major depressive disorder (MDD) groups for the Mostly Congruent condition.

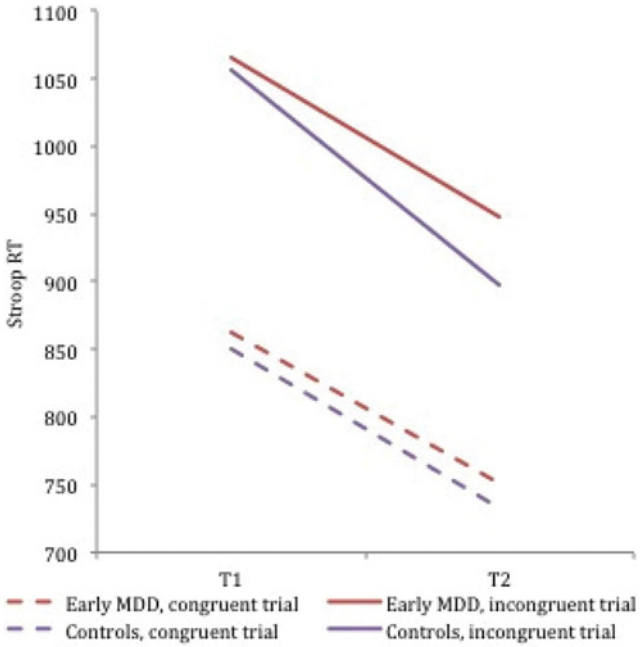

FIGURE 3 Stroop Mostly Congruent reaction time (RT) for incongruent and congruent trials for controls and early MDD groups. *Note:* MDD = major depressive disorder.

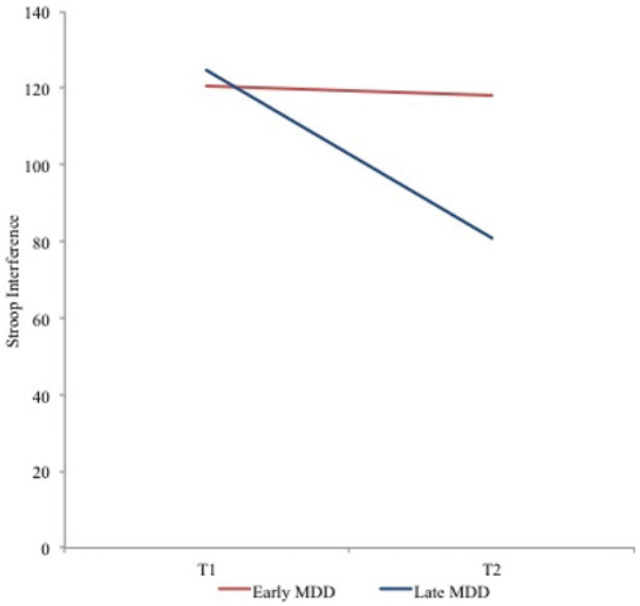

FIGURE 4 Stroop interference scores for early MDD and late MDD groups for the Mostly Incongruent condition. *Note:* MDD = major depressive disorder.

between groups at T1 but were trending toward significance at T2 ($p = .064$).[2,3] Refer to Tables 6 and 7 for further detail on the multinomial logistic regression analyses.

DISCUSSION

The current study investigated the relationship between the development of two forms of cognitive control (proactive and reactive) during adolescence and the onset of MDD. Our findings indicate that development of proactive and reactive forms of cognitive control between early and midadolescence are not predictive of future onset of MDD. However, less improvement in reactive control between early and midadolescence was identified in those adolescents who had experienced the onset of an episode of MDD over the same period but were in remission by midadolescence. Less improvement was also identified in proactive control abilities of adolescents in remission, although this finding was only trending toward significance.

Our findings reveal that adolescents experiencing the onset of MDD between early and midadolescence had poorer development of reactive forms of cognitive control over the same time compared to those who did not develop MDD. This is consistent with past research by Holmes and Pizzagalli (2008), which identified poorer reactive control abilities, based on reaction time on the Stroop task, in adults with MDD. Further, prior research on adults has found depressive symptoms to be associated with decreased amplitude of ERPs that are hypothesized to be related to reactive control (i.e., Vanderhasselt et al., 2014; West et al., 2010), and functional neuroimaging data have identified reduced activation of the anterior cingulate cortex, which is hypothesized to underlie reactive control abilities, in depressed patients (Drevets et al., 2008; Price & Drevets, 2009). Also, prior work with the current sample showed that improvements in reactive control from early to midadolescence were associated with thinning of the anterior cingulate cortex (Vijayakumar et al., 2013). Together, these findings suggest that deficits in the development of reactive control in depression may be partly due to abnormalities in the development of underlying neural circuitry. Based on the current findings and the prolonged maturation of the cortex and cognitive control abilities into early adulthood (Giedd, 2004; Luna, 2009), is it possible that the onset of MDD will continue to impact on reactive control development into the early 20 s.

[2]Similar analyses on error scores failed to identify any significant effects of development of response accuracy on MDD onset between T1 and T2, or between T2 and T3.

[3]Similar analyses using the Center for Epidemiological Studies–Depression scores as the outcome variable also failed to identify any significant effects of development of response accuracy on change in depressive symptomatology between T1 and T2, or between T2 and T3.

TABLE 6
Multinomial Logistic Regression Analyses on Stroop Development for the MC Condition

	Early MDD vs. Controls				*Late MDD vs. Controls*				*Early MDD vs. Late MDD*			
	B	*SE (B)*	*Wald*	*OR*	*B*	*SE (B)*	*Wald*	*OR*	*B*	*SE (B)*	*Wald*	*OR*
Age	−0.45	0.73	0.39	0.64	0.04	0.72	0.00	0.95	−0.41	0.94	0.19	0.67
Sex	−1.06	0.52	4.10**	0.35	−0.28	0.50	0.32	0.75	−0.78	0.67	1.35	0.46
SES	−0.03	0.01	5.71**	0.97	−0.03	0.01	5.92**	0.97	0.00	0.02	0.00	1.00
IQ	0.00	0.02	0.01	1.00	0.02	0.02	1.15	1.02	−0.02	0.02	0.90	0.98
Stroop INT 1	0.00	0.00	0.01	1.00	0.00	0.00	1.21	1.00	0.00	0.00	0.84	1.00
Stroop INT 2	0.01	0.00	4.14**	1.01	0.00	0.00	0.01	1.00	0.01	0.00	2.52	1.01

Note: Reference group is controls. MC = Mostly Congruent; MDD = major depressive disorder; SES = socioeconomic status; INT = interference score.
**$p < .05$.

Although there has been no research to date investigating reactive control abilities in depressed adolescents, there is some evidence for executive function deficits in adolescents with current MDD (Micco et al., 2009; Vergara-Lopez et al., 2013; Wilkinson & Goodyer, 2006). However, our identification of deficits in remitted patients suggests that the effects are independent of current MDD. In comparison, a previous study by Maalouf et al. (2011) failed to identify any executive function impairments in adolescents in remission from MDD. It is possible that the more global measures of cognitive control used in this prior study were not sensitive to the impairments in reactive control identified in the current study. Furthermore, Maalouf et al. investigated cross-sectional abilities, whereas our results indicate that depression impacts the *development* of cognitive abilities during adolescence. These findings suggest that depression may have a continued impact on functioning via enduring effects on reactive cognitive control after the resolution of acute episodes.

Our findings also indicate a trend level effect for poorer proactive control development in individuals who developed MDD between early and midadolescence, compared to those who did not. In addition, individuals who developed MDD between early and midadolescence had significantly poorer proactive control development compared to those who developed MDD at a later period of adolescence (i.e., between mid- and late adolescence). Prior research by Vanderhasselt et al. (2014) identified behavioral (response time) and ERP differences in patients with MDD compared to controls during conditions of proactive control on a cued emotional conflict task, where participants were required to respond to the emotional expression of a target face based on a cue preceding stimulus onset. Similarly, West et al. (2010) found that increased negative affectivity was related to decreased amplitude of an ERP, which is thought to represent preparatory control processes preceding stimulus onset (i.e., involved in proactive control), during a counting Stroop task. However, Saunders and Jentzsch (2013) failed to identify any relationship between proactive control abilities and depressive symptomatology in adults, based on response time and accuracy data from the Stroop task. This inconsistency with our finding may be related to sample characteristics, as Saunders and Jentzsch examined a nonclinical adult sample compared to our clinical adolescent sample.

TABLE 7
Multinomial Logistic Regression Analyses on Stroop Development for the MI Condition

	Early MDD vs. Controls				*Late MDD vs. Controls*				*Early MDD vs. Late MDD*			
	B	*SE (B)*	*Wald*	*OR*	*B*	*SE (B)*	*Wald*	*OR*	*B*	*SE (B)*	*Wald*	*OR*
Age	−0.79	0.74	1.14	0.45	0.17	0.75	0.05	0.818	−0.97	0.97	0.98	0.38
Sex	−0.97	0.53	3.34*	0.38	−0.26	0.51	0.25	0.78	−0.71	0.68	1.10	0.49
SES	−0.03	0.01	4.47**	0.97	−0.03	0.01	4.88**	0.97	0.00	0.02	0.01	1.00
IQ	0.00	0.02	0.00	1.00	0.02	0.02	1.17	1.02	−0.02	0.02	0.73	0.98
Stroop INT 1	0.00	0.00	0.01	1.00	0.00	0.00	0.55	1.00	−0.00	0.01	0.43	1.00
Stroop INT 2	0.01	0.00	3.26*	1.01	0.00	0.01	0.73	1.00	0.01	0.01	4.01**	1.01

Note: Reference group is controls. MC = Mostly Incongruent; MDD = major depressive disorder; SES = socioeconomic status; INT = interference score.
*$p < .10$. **$p < .05$.

The deficits in proactive control indicate that adolescents with depression may exhibit some difficulty with emotion regulation even when they can anticipate upcoming emotion-eliciting situations. For example, they may find it difficult to engage in forward-planning and goal-directed emotion regulatory activities, such as seeking social support, managing stress, or engaging in behavioral activation (i.e., hobbies, physical activity) to help improve their mood. However, these results were present only at a trend level, and as such this and any other interpretation should be considered with caution. By comparison, our significant findings relating to reactive control suggest that adolescents with depression might particularly struggle to regulate their thoughts, behavior, and emotions in conditions when they *cannot* anticipate/prepare for stimuli (i.e., when they are not expecting an emotion-eliciting event and thus have to regulate their emotions following the occurrence of the event). For example, they may have difficulties engaging in cognitive reappraisal and redirect attention away from upsetting stimuli following *unexpected* stressful peer interactions.

It is also possible that the somewhat differing results between reactive and proactive control (i.e., more pronounced MDD-related deficits in reactive compared to proactive cognitive control) may arise from differing developmental patterns for the two forms of cognition. Previous research by our group identified significant improvements in reactive control between early and midadolescence, though no such effect was found for proactive control (Vijayakumar et al., 2013). Furthermore, Andrews-Hanna et al. (2011) identified later development of brain regions underlying proactive control abilities compared to those underlying reactive control based on functional neuroimaging while performing the Stroop task. Similarly, structural neuroimaging research has identified protracted maturation of the dorsolateral prefrontal cortex, which is hypothesized to underlie proactive control, into early adulthood (Gogtay et al., 2004). Therefore, it is possible that reactive control difficulties were more strongly affected by the onset of MDD in the current study as we examined a crucial period for its development, and there remains the possibility that the experience of depression during later adolescence or early adulthood may have a greater impact on proactive control abilities given its comparatively delayed development.

Our findings also indicated that development of cognitive control between early and midadolescence was not predictive of later MDD onset between mid- and late adolescence, which is consistent with past research that has failed to identify cognitive control deficits in at-risk children of depressed mothers (Klimes-Dougan et al., 2006; Micco et al., 2009). Further support comes from our finding of poorer proactive control development in adolescents who had MDD onset between early and midadolescence compared to those who experienced later MDD onset. These finding suggests that cognitive control is unlikely to be a predisposing factor that precedes the development of depression. Rather, cognitive deficits (specifically in reactive control) are more likely to be secondary effects arising from depression rather than vulnerability markers. Supporting this view, residual deficits during remission in adults have been found to correlate with the number and severity of prior episodes, after controlling for residual symptoms (Bhardwaj et al., 2010). Similarly, Vanderhasselt and De Raedt (2009) found that the amplitude of the N450 ERP component, which is related to cognitive control, was inversely related to the number of prior depressive episodes. Nevertheless, the findings highlight the importance of early intervention to minimize the persistence of cognitive deficits, which may continue to impact on functioning after the resolution of depressive episodes and possibly play a role in future recurrence of episodes. Indeed, it has been hypothesized that cognitive control influences emotion regulatory abilities, with impairments causing difficulty with disengaging from negative stimuli, and inhibiting the elaborative processing of such stimuli (Joormann, 2010).

One of the limitations of the current study is that the directionality of the relationship between reactive control development and depression cannot be teased apart given the correlational nature of the data. That is, there remains the possibility that poorer reactive control development between early and midadolescence may be responsible for the onset of depression during the same period, even though it was not related to onset at later periods. Furthermore, other factors that were not examined in the current study, such as genes, might predispose individuals to both MDD and a reduction in cognitive control during adolescence. Other limitations of note include comorbid diagnoses that were common in both our MDD groups. However, given that other diagnoses were also present in the control group, it is unlikely that the current findings were driven solely by comorbid diagnoses. In addition, we did not examine cognitive control abilities at T3 (late adolescence). Future research incorporating these data would provide valuable insight into the development of cognitive control abilities in later adolescence for those with early MDD (i.e., Does their reactive control continue to decline? Is proactive control more strongly implicated in later adolescence?) and those with late MDD (i.e., Do they also exhibit poorer cognitive control development during late adolescence?). Another limitation of this study is statistical power, given the relatively smaller number of participants in the early MDD and late MDD groups compared to the control group. The small clinical samples also meant that sex differences could

not be investigated. It will be important for future studies to investigate these effects given that sex differences have been identified in both the onset of depression during adolescence and the development of cognitive control. Future research incorporating neuroimaging data may also shed light on the proposition that brain development mediates the relationship between depression and reactive control given their common neural underpinnings.

In conclusion, the current study identified poorer development of reactive control abilities in individuals who developed MDD between early and midadolescence, as well as a trend for poorer development of proactive control abilities. This finding highlights the continued impact of the illness on cognitive functioning following the resolution of acute episodes. Furthermore, cognitive development was not predictive of future MDD onset, suggesting that cognitive control deficits were more likely to be secondary consequences of depression rather than vulnerability markers for the illness, thus emphasizing the importance of early intervention.

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