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Parent Depressive Symptoms and Offspring Executive Functioning

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Abstract

Introduction: Previous research has found mixed results when assessing the association between parent's history of depressive symptoms and child's abilities on measures of executive functioning (Klimes-Dougan et al., 2006; Micco et al., 1990, 2009). The purpose of this study was to replicate and expand upon these findings by evaluating the influence of a parent's depressive symptoms on youth's executive functioning.

Method: As part of a larger study, 135 children (54.8% female, aged 8–12) and one biological parent completed diagnostic screening interviews. Children then completed a brief executive functioning battery. Symptoms endorsed under the past major depressive episode module of the MINI International Neuropsychiatric Interview was used to measure depressive symptoms of parents.

Results: While controlling for parent alcohol status and age, gender, intelligence, and current depressive symptoms of the child, linear regression models revealed parent's depressive symptoms significantly predicted deficits in Letter-Number Sequencing ($b = -0.15$ (0.07), $p < 0.05$) and Motor Speed ($b = -0.17$ (0.05), $p < 0.005$) on the D-KEFS Trails Test. Parent depressive symptoms had no relationship with inhibition on the D-KEFS Color-Word Interference Test ($b = -0.04$ (0.14), $p = 0.74$) or the Verbal Working Memory subtest of the Stanford-Binet ($b = .14$ (0.12) $p = 0.43$). Greater depressive symptoms in parents were associated with fewer perseverative errors on the Wisconsin Card Sorting Task (WCST) ($b = 0.73$ (0.32), $p < 0.05$).

Conclusion: In sum, parent's depressive symptomatology were differentially associated with youth's neurocognitive abilities. Clinical implications were discussed.

Keywords

parent depression; child executive functioning; cognitive flexibility; inhibition; working memory

Introduction

Executive functioning (EF) is a broad construct that refers to the general integration of cognitive processes to support sustained, goal-oriented behavior (Miyake & Friedman, 2012;

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Miyake et al., 2000). There may be up to 33 different ways to define and categorize executive functioning processes (Sergeant, Geurts, & Oosterlaan, 2002), including planning, attention management, self-monitoring, and self-awareness (Loring, 1999). Of the variety of processes that constitute executive functions, inhibiting prepotent responses (inhibition), shifting between tasks or mental tasks (set-shifting), and updating working memory representations (updating) are three well-studied processes that predict distinct aspects of cognitive control and behavior (Miyake et al., 2000). Deficits in EFs have been associated with a myriad of serious difficulties, such as increased substance use and related problems (Nigg et al., 2004, 2006; Tarter et al., 2003; Wong, Brower, Nigg, & Zucker, 2010), crime (Brower & Price 2001; Meijers, Harte, Jonker, & Meynen, 2015), and severe psychopathology (e.g., anti-social personality disorder; Pennington & Ozonoff, 1996; Morgan & Lilienfeld, 2000). Not limited to severe dysfunction, EFs have also been related to more commonplace difficulties, such as mood disorders, and in particular, depressive symptoms (e.g., Fossati, Ergis, & Allilaire, 2001).

A growing body of literature has highlighted how those with significant depressive symptoms have a greater risk for weaknesses in a variety of cognitive domains including working memory, verbal fluency, set-shifting, planning, attention, inhibition (see review Rogers et al., 2004). While the presence of these deficits is relatively well established, it is unknown whether deficits in cognitive abilities are secondary to a current depressive state or whether they represent potential etiological markers for the later onset of a major depressive episode. One way to assess for this potential mechanism is to evaluate children who are at elevated risk for the development of a depressive episode, or rather, children with a parental history of depression or depressive symptoms. Some estimates suggest that children with a family history of depression are three to four times more likely to develop the disorder later on in life (Garber, 2006). EF deficits that precede an episode of MDD may occur through either genetic or environmental mechanisms. That is, if poor EF is a risk factor for depression, and given the individual differences in EF are almost entirely genetic in origin (Friedman et al., 2008; Miyake & Friedman, 2012), then parents with a history of depression may have passed on genetic variants associated with poorer EF to their children. Conversely, a parent suffering from MDD may inadvertently have deleterious effects on the youth's environment, altering the child's development. Specifically, parent's depressive symptoms may adversely affect the development of the prefrontal cortex (Belsky & de Haan, 2011), an area thought to play a vital role in EFs (Miller & Cohen, 2001).

The prefrontal cortex is thought to be highly susceptible to environmental factors (e.g., Noble, Norman, & Farah, 2005). Studies have demonstrated that exposure to considerable difficulties such as maltreatment or neglect can have long-term, negative consequences for adaptive functioning and EFs (Belsky & de Haan, 2011). While this represents an extreme example, other research is now investigating whether less severe chronic stressors may have an impact on youth's cognitive development (Odgers & Jaffee, 2013). High cortisol levels, a biological marker for stress, may be a potential mechanism for these reduced cognitive performances, as higher cortisol levels have been correlated with deficits in cognitive functions across the lifespan (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007). These deficits were measured with both acute administration (Lupien & McEwen, 1997) as well as at higher basal levels of the hormone (Lupien, Lecours, Lussier, Schwartz, Nair & Meaney,

1994). It is plausible that a parent's depressive symptoms create a stressful environment for children through reduced parent-child interaction, high parental emotional reactivity, and decreased parental modeling of effective coping strategies. This chronic stress and subsequent increase in cortisol levels can impact development of the prefrontal cortex in youth (Wellman, 2001), and, consequently, the youth's expression of a variety of cognitive abilities. Indeed, higher cortisol levels in children were correlated with both parental depressive symptoms and deficits in EF and working memory in the youth (da Rosa Riccolo, de Salles, Garcia Falceto, Luiza Fernandes, & Grassi-Oliveira, 2016). In sum, it appears that evaluating children with a family history of depression represents a possible avenue to better understand potential etiological factors in the development of a major depressive disorder.

The research evaluating this relationship among at-risk children and their parents is limited, and cross sectional research has yielded conflicted results. In a sample of 147 youth (age 6–17 years), Micco et al. (2009) failed to find significant associations in the expected direction (i.e., deficits) between parental depressive symptoms and children's cognitive abilities. Instead they found that parents' depressive symptoms significantly predicted to fewer perseverative errors on the Wisconsin Card Sorting Task. Wagner, Abramson, and Alloy (2015) sampled 493 youth and their parents, and they did not find associations between maternal lifetime history of depression and children's performance on measures of working memory and attention. Conversely, Belleau and her colleagues (2013) compared youth with ($n=29$) and without ($n=29$) a history of mood disorders, both bipolar and major depression, on measures of altering, orienting, and executive attention. Their findings revealed youth with a family history of mood disorders demonstrated significantly slower reaction times on executive attention, but not on alerting or orienting, suggesting neurocognitive deficits may precede mood disorders beyond MDD. Finally, Davidovich and colleagues (2016) assessed whether child's EFs could mediate the relationship between parent and youth's depressive symptoms. This model yielded a significant association in which those children who demonstrated better inhibitory control and cognitive flexibility reported fewer depressive symptoms.

A growing body of longitudinal research highlights a strong association between parental depressive symptoms and youth's later expression of EF abilities, suggesting parental internalizing symptoms do not need to be current to have a detrimental impact on youth's cognitive functioning (Hughes, Roman, Hart, & Ensor, 2013; Roman, Ensor, & Hughes, 2016). The effect of parental psychopathology on youth's cognitive functioning may be present even before birth. Mean levels of maternal anxiety over the course of gestation was associated with lower inhibitory control and visual-spatial working memory abilities in children between the ages of 6 and 9 years old (Buss, Davis, Hobel, & Sandman, 2011). While these studies are useful in identifying potentially familial relationships between cognition and depression, they are not able to parse apart the influence genetics and the environment may contribute to this association. To better understand these influences, some studies have begun to use twin participants with and without one twin diagnosed with MDD.

Indeed, Christensen and colleagues (2006) assessed neurocognitive performance of unaffected twins (mean aged 28–48 years), where their co-twin was either affected by depression (i.e., higher risk) or was not affected (i.e., low risk for disorder development).

Non-affected twins with an affected co-twin evidenced more deficits in areas of cognition such as attention, working memory, executive functioning, language processing, and memory than those who did not have an affected co-twin. These results were replicated in a more recent study (Hsu et al., 2014), where twins (mean age=45 years) with affected co-twins demonstrated poorer performance in attention and general knowledge than those who did not have an affected sibling. Overall, these studies lend support to a genetic basis for the relationship between EF and depression. However, for a recent analysis of the potential mechanisms behind the association between depression and EF, please refer to Allot, Fisher, Amminger, Goodall, and Hetrick's (2016) review.

In sum, there is a developing body of literature assessing how parental depressive symptoms may influence children's executive functioning abilities. This research is still in its nascent stages and has revealed conflicting results. As such, the purpose of the current study was to add to this body of knowledge by assessing the relationship between parent's depressive symptomatology and children's performance on measures of executive functioning. Specifically, this study aimed to assess executive functioning in the realms of set-shifting, inhibition, and working memory, in lines with Miyake et al.'s (2000) description of EFs. Motor ability will also be assessed as some of the cognitive tasks administered had simple motor requirements.

Methods

Participants

One hundred and thirty-four children ages 8–12 (54.8% girls; $M_{age}=10.79$, $SD_{age}=1.60$) participated in a study designed to understand the relationship between parental psychopathology and child neurocognitive functioning. Data presented here are from a larger longitudinal study that assesses parent alcohol status and youth's development, of which, data collection is still ongoing. The ethnic break down of the participants were 76.9% Caucasian, 9% Hispanics and 14.1% were from other ethnic groups (i.e., African-American, Asian, Native American or biracial).

Children and their biological parents were recruited through community flyers and advertisements in local newspapers, radio stations and Facebook. All participants and their parents received payment as compensation. Children with the following characteristics were included: (i) between the ages of 8 to 12; (ii) normal intelligence, i.e., an IQ of 80 or above using the Slosson Intelligence Test (SIT-R3); (iii) able and willing to provide informed assent (child) and consent (parent). The larger longitudinal study from which this data was used assesses the relationship between parent alcohol status (i.e., alcoholic or control) and youth development. Therefore, some of our parent participants met criteria for a history of alcohol use disorder. Importantly, most of the parents who met criteria for a history of alcohol use disorder were the biological fathers of the child participant, whereas most of the parents participating in the psychodiagnostic interviews were biological mothers. All analyses were conducted while controlling for parent alcohol status. Alcohol status of the parent had no significant relationships with any of the dependent variables of interest (see below).

Procedures

While this study was part of a larger investigation, only procedures pertinent to the current study are presented here. Data for this study were collected across two sessions. Families who expressed an interest to participate in the study were asked to schedule an appointment for screening. During the screening interview (Session 1), both the participating parent and the child were administered the MINI International Neuropsychiatric Interview in addition to a number of other questionnaires (data not presented here). In the last session, children were asked to complete several neuropsychological tasks and an intelligence test, including the Wisconsin Card Sorting Task, two subtest of the Delis-Kaplan Executive Functioning System, one subtest of Stanford-Binet Fifth Edition, and the Slosson Intelligence Test-Revised.

Materials

Mini International Neuropsychiatric Interview 6.0. (MINI; Sheehan & Lecrubier, 2010).—The MINI is a valid and reliable short, structure diagnostic interview that enables researchers and clinicians to make diagnoses of psychiatric disorders according to the DSM-IV or ICD-10 (Sheehan et al., 1997; Sheehan et al., 1998). For the purpose of the present study, the Major Depressive Episode module was used. Questions for all modules are listed in a yes/no format. Symptom count was measured through the summation of symptom endorsements during a two-week time period in the parent's life where they felt depressed or down nearly everyday (i.e., lifetime depressive symptoms). Current parental depressive symptoms were also calculated but were not correlated with any of the child neuropsychological variables and was therefore not included in further analyses. Children were administered the child version of the MINI to control for any depressive symptoms the youth was currently experiencing. Current child depressive symptoms were not associated with any of the neuropsychological variables (see below). The range for both the adult and child MINI was from 0 (no symptoms were endorsed) to 8 (all symptoms were endorsed).

Wisconsin Card Sorting Task (WCST; Heaton, 1981).—For the purpose of this study, the computerized WCST was utilized as a gauge for an individual's set-shifting abilities through their perseverative error responses. Respondents were presented with four stimulus cards that incorporate three stimulus parameters (i.e., color, form, and number) and were required to sort numbered response cards according to different principles and to alter their approach during test administration. The WCST has been demonstrated to be both a valid and reliable measure of neurocognitive functioning for clinical (Axelrod, Goldman, & Woodard, 1992; Romine, Lee, Wolfe, Homack, George, & Riccio, 2004; Welsch, Pennington, & Groisser, 1991) and normal populations of children (Welsch, Pennington, & Groisser, 1991), although the specificity of the measure in identifying frontal lesions has recently come under review (Nyhus, & Barceló, 2009). The participant's standard score of their perseverative errors was used for the present study.

Delis-Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001).—The D-KEFS Trails Test involves a series of 5 conditions: Visual Scanning, Number Sequencing, Letter Sequencing, Number-Letter Switching, and Motor Speed. In the Visual Scanning condition, participants cross out all the 3's that appear on the response

sheet. In the Number Sequencing condition, participants draw a line connecting the numbers 1–16 in order; distractor letters appear on the same page. The Letter Sequencing condition requires examinees to connect the letters A through P, with distractor numbers present on the page. The Number-Letter Switching condition, considered the “executive task”, requires participants to switch back and forth between connecting numbers and letters in consecutive order (i.e., 1, A, 2, B, 3, C, etc.). Last, a Motor Speed condition is administered in which the participants trace over a dotted line connecting circles on a page as quickly as possible, in order to gauge their motor drawing speed. For the present study, completion times in the form of scales scores from the Number-Letter Switching condition and Motor Speed condition were used. Secondary analyses from the Number-Letter Switching condition were also used to assess for set-loss errors committed, as completion time offers just one capture of participant’s abilities. This variable is presented as a percentile.

The D-KEFS Color-Word Interference Test is a variation of the classic measure of inhibition, the Stroop Test (Stroop, 1935). The D-KEFS test includes two baseline conditions for evaluating key component skills of the higher-level tasks: basic naming of color patches (Condition 1) and basic reading of words that denote color printed in black ink (Condition 2). On the traditional inference task (Condition 3), the participant must inhibit reading the words denoting colors in order to name the dissonant ink colors in which those words are printed. Finally, in Condition 4, participants are required to switch back and forth between naming the dissonant ink colors and reading the conflicting words. Therefore, this condition measures both inhibition and cognitive flexibility. The D-KEFS Color-Word Interference Test provides normative data for completion times for each condition, contrast measure for parceling baseline scores from scores on high-level tasks, and uncorrected and self-corrected error measures. For the purpose the present study, completion times as scaled scores for Condition 3 were used.

Overall, the D-KEFS has been used to differentiate clinical populations and controls in children with fetal alcohol exposure (Mattson, Goodman, Caine, Delis, & Riley, 1999), childhood stroke (Nicholas, Waller, Trauner, & Delis, 2003), as well as attention-deficit disorder (Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009). The D-KEFS has also been used with children to assess for differences in the normal range of development (i.e., outside of the clinical population; Delis, Kramer, Kaplan, & Holdnack, 2004). Moreover, the D-KEFS has demonstrated reliability for both adults as well as children (Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009; Delis, Kaplan, Kramer, 2001).

Slosson Intelligence Test – Revised (Slosson, 1998).—The Slosson Intelligence Test, Revised (SIT-R) serves as a brief approximation of general verbal cognitive ability or index of crystallized intelligence. The test contains 187 untimed items assessing the cognitive domains of vocabulary, general information, similarities and differences, comprehension quantitative ability, and auditory memory. All items are presented in question and answer format. The raw score is computed by adding the highest item in the basal to the number of correct responses after the basal. The standard score of the full scale IQ was used as a covariate, as intellectual ability has been shown to influence some aspects of executive functioning (Friedman, Miyake, Corley, Young DeFries, & Hewitt, 2006). The Slosson has been demonstrated to be both a reliable and valid measurement of crystallized

intelligence in both clinical (Martin, Blair, Stokes, & Lester, 1977; Reid, Maag, Vasa, & Wright, 1994) and normal populations of children (Bell, Rucker, Finch, & Alexander, 2002).

Stanford-Binet Intelligence Scales – Fifth Edition (Roid, 2003).—The Stanford-Binet Intelligence Scales (or more commonly the Stanford-Binet) is an individually administered cognitive ability and intelligence test. The test includes ten subtests of both verbal and nonverbal domains. To measure the executive functioning ability of working memory, only the standard score from Verbal Working Memory subtest was used. In this subtest a series of questions are read to the participant. The participant is required to answer each of the questions while simultaneously remembering the last word in each question. After the set of questions are read, the participant must repeat back in the correct order the last word in each question of the set. Sets become progressively larger as the participant proceeds through the subtest. The Stanford-Binet has been shown to be a reliable and valid measure of cognitive ability (Bain & Allin, 2005) and working memory (Pomplun & Cluster, 2005) for clinical populations (Silverman et al., 2010) and as a measure of typical development (Rothlisberg, 1987). The standard score of the youth's verbal working memory performance was used for the present analyses.

Results

Descriptive statistics and correlations of all variables are presented in Table 1. Parents endorsed a moderate amount of lifetime depressive symptoms ($M = 4$, $SD = 4$). Parent lifetime depressive symptoms (PDS-Lifetime) were significantly correlated with youth's D-KEFS Letter-Number Sequencing and motor speed scaled score of the D-KEFS Trails Test. Child estimated intelligence quotient was correlated with all neuropsychological variables except for Letter-Number Sequencing set-loss errors. Independent sample T-tests revealed no significant group differences for gender (male versus female), ethnicity (Caucasian and not Caucasian), and target status (alcohol parent and nonalcoholic parent) across neurocognitive measures.

Next, bivariate correlations were employed to assess if there were significant associations between demographic and neuropsychological variables. Child current depressive symptoms (CDS-Current) were significantly correlated with intelligence quotient and motor speed (see Table 1), but was not ultimately a significant predictor in any of the regression analyses (see Table 2). CDS-Current was also not significantly correlated with parent depressive symptoms. Child age was significantly correlated with perseverative errors on the Wisconsin Card Sorting Test, but not with PDS-Lifetime or other neuropsychological variables. Parent alcohol status was not correlated with any of the neuropsychological variables.

To ensure participant age did not influence the findings, means for scaled scores of the “lower-order” subtests were calculated. This included the Color Naming condition and Word Reading condition of the Color-Word Interference as well as the Number-Sequencing and Letter-Sequencing condition on the Trails Test. Mean scaled scores for the Color Naming condition and Word Reading condition of the Color-Word Interference was 10.03 and 11.13, respectively. For Number-Sequencing condition the mean scaled score was 10.76, whereas the mean scaled score for the Letter-Sequencing condition was 9.82.

Five independent multiple regression models tested the relationship between PDS-Lifetime and neurocognitive functioning, while controlling for parent alcohol status and age, gender, ethnicity, current depressive symptoms, and crystallized intelligence of the child (see Table 2). The models used a count of parent lifetime depressive symptoms endorsed on the MINI to predict to perseverative errors of WCST, Verbal Working Memory subtest of the Stanford-Binet, inhibition of the D-KEFS Color-Word Interference Test, Letter-Number Sequencing of the D-KEFS Trails Test, and Motor Speed of the D-KEFS Trails Test, respectively. Results revealed discrepant findings. More specifically, greater PDS-Lifetime predicted slower performance on the Letter-Number Sequencing of the D-KEFS Trails Test ($b = -0.15$ (0.07); $F(6, 117) = 4.25, p = .01, R^2 = 0.09$) and simple Motor Speed of the D-KEFS Trails Test ($b = -0.17$ (0.05); $F(6, 117) = 5.39, p = .002, R^2 = 0.15$). Further analyses indicated that PDS-Lifetime predicted higher number of set-loss errors ($b = -2.31$ (0.82), $F(7, 114) = 2.12, p = .02, R^2 = 0.12$) on the Letter-Number Sequencing subtest of the D-KEFS Trails Test. This suggests that children of parents with more depressive symptoms were likely to take more time to complete the subtest as well as commit more set-loss errors when engaging in cognitive flexibility (i.e., accurately switching between the numbers and letter). Higher PDS-Lifetime predicted fewer perseverative errors (i.e., better performance) on the WCST ($b = 0.73$ (0.32), $F(7, 115) = 4.06, p = .02, R^2 = 0.20$). PDS-Lifetime did not predict completion time of the Inhibition subtest of the D-KEFS Color-Word Interference ($b = -0.03$ (0.09), $F(7, 114) = 2.88, p = .17, R^2 = .15$) or the Verbal Working Memory subtest of the Stanford-Binet ($b = -.05$ (0.06), $F(7, 100) = 2.75, p = 0.35, R^2 = .16$).

Secondary Analyses

To account for the potential of other child psychological disorders driving this association, secondary analyses were conducted. First, children from depressed parents endorsed more symptoms for childhood panic disorder, agoraphobia, social anxiety disorder, separation anxiety disorder, obsessive-compulsive disorder, posttraumatic stress disorder, and generalized anxiety disorder. However, none of the child anxiety disorders were significantly correlated with any of the measured neuropsychological variables, except social anxiety disorder symptoms were negatively correlated with motor speed on the Trails Test for children with depressed parents. This relationship did not maintain when social anxiety disorder symptoms were used to predict motor speed in a regression model ($b = 0.27$ (0.35), $p = .51$).

Second, other childhood depressive disorders were correlated with neuropsychological variables of interest. Namely, endorsed symptoms of childhood dysthymia, past depressive episode (CDS-Lifetime), and current depressive episode (CDS-Current) were correlated with neuropsychological variables used. CDS-Current was correlated with motor speed of the Trails Test. When put into the regression model with PDS-Lifetime, CDS-Current was not a significant predictor ($b = -0.10$ (0.10), $p = 0.29$), whereas PDS-Lifetime was ($b = -0.16$ (0.05), $p = 0.003$). Childhood dysthymia symptoms were associated with set-loss errors on the Trails Test. However, when put into a regression model with PDS-Lifetime, dysthymia symptoms were not a significant predictor ($b = -7.67$ (4.49), $p = 0.11$), whereas PDS-Lifetime was ($b = -2.11$ (0.84), $p = 0.02$). Therefore the findings presenting in the current study reveal associations between PDS-Lifetime and child neurocognitive functioning while

controlling for children's current depressive and anxiety symptoms, suggesting that our results were not a reflection of the child's psychopathology.

Discussion

Depression is one of the most common and costly psychological disorders. It is estimated that the economic burden of depression is approximately \$77.4 billion each year (Greenberg et al., 2003) and significantly raises the mortality rate, even in its subclinical forms (Cuijpers & Smit, 2002). To mitigate the negative outcomes associated with this disorder, researchers have been assessing depression's etiological factors and identifying populations at heightened risk for developing depression. Previous research has hypothesized that diminished executive functioning capacities may play a role in the onset of depression for some individuals (Hughes, Roman, Hart, & Ensor, 2013; Micco et al., 2009). To assess this relationship, researchers have previously investigated the relationship between parental history of depression and executive capacities of the offspring, but research has yielded conflicting results heretofore (Hughes, Roman, Hart, & Ensor, 2013; Micco et al., 2009; Roman, Ensor, & Hughes, 2016; Wagner, Abramson, & Alloy 2015), with some studies reporting inverse relationships between parental depressive symptoms and offspring's EF, and other studies failing to demonstrate significant relationships between the two factors.

The goal of this study was to assess whether PDS-Lifetime predicted poorer neurocognitive functioning of youth, while controlling for child's current depressive symptoms. PDS-Lifetime was associated with some aspects of youth's EF. Specifically, depressive symptoms were associated with youth's cognitive flexibility and accuracy in set-shifting abilities after controlling for the effects of child crystallized intelligence, age, CDS-Current and gender as well as the parent's alcohol status. However, PDS-Lifetime was not related to youth's inhibition or working memory abilities. In addition, depressive symptoms were associated with abilities outside of EF, namely motor speed. Some of our models yielded small effect sizes, thus the relationship between PDS-Lifetime and child neurocognitive functioning are quite modest. For instance, a one-unit increase in parental depressive symptoms is associated with a decrease of .15 scaled scores in the DKFES Letter-Number Sequencing task and a decrease of 2.31 percentile points in set-loss errors. However, our study highlights the association between parental depressive symptoms and several aspects of child neurocognitive functioning not investigated in previous research. In sum, our study lends itself to the growing literature of parental internalizing disorders and youth's cognitive functioning and aligns itself with previous research that has revealed cognitive deficits in offspring of parents with MDD (Davidovich, Collishaw, Thapar, Harold, Thapar, & Rice, 2016; Hughes, Roman, Hart, & Ensor, 2013; Roman, Ensor, & Hughes, 2016). Moreover, this study highlights the critical need to further assess potential etiological factors in the development of a major depressive disorder.

This study also assessed the differential influence of PDS-Lifetime on specific EFs and motor function in youth. Our results demonstrated that PDS-Lifetime had a negative association with youth's ability to cognitively switch between mental tasks (i.e., set-shifting) and speed of motor functions (i.e., motor speed). Moreover, subsequent analyses indicated that not only did PDS-Lifetime inhibit the speed at which youth engage in cognitive

flexibility, it also influenced the accuracy of the mental switching. This implies that offspring of parent's suffering from symptoms of depression may have a more difficult time accurately and quickly processing dual sources of information. These deficits may reveal themselves in a variety of situations both inside and outside the classroom. For example, children who display shortfalls in cognitive flexibility may have a more difficult time adapting to novel situations, sudden shifts in plans or routines, and accessing crystallized knowledge.

Moreover, our findings that children neuropsychological deficits were associated with the presence of PDS-Lifetime is convergent with previous studies showing that youth with depressed parents were more likely to have impairments in cognitive flexibility, inhibitory control, and working memory (Davidovich, Collishaw, Thapar, Harold, Thapar, & Rice, 2016; Hughes, Roman, Hart, & Ensor, 2013). Taken together, our findings in conjunction with previous studies, suggest that neuropsychological impairments associated with depression are not dependent on current symptomatology. Future research should continue to expand upon these results. Notably, research should continue to longitudinally assess at-risk youth's cognitive abilities and the progression of their psychological health.

There were some unexpected findings of PDS-Lifetime being associated with better performance on selected neuropsychological measures. Specifically, presence of lifetime parental MDD symptoms resulted in fewer perseverative errors on the WCST. The discrepant neuropsychological performance between the Letter-Number Sequencing subtest of the Trails Test and the WCST may at first appear contradictory, as one measure appears to suggest a deficit and the other a strength in cognitive flexibility in the context of parental depression. We would argue that these measures are potentially capturing different aspects of cognitive functioning. The Letter-Number Sequencing subtest of the Trail Test requires participants to correctly alternate between connecting number and letters in numerical and alphabetical order. This test measures flexibility in thinking while engaging in a visual-motor sequencing task. In contrast, WCST requires participants to problem solve, using corrective feedback, to formulate the concept being tested (i.e., color, form, or number). After ten consecutive matches, the concept or sorting rule changes, the participant must utilize the new corrective feedback to properly identify the next concept being assessed. It is plausible that the Trails Test and WCST, while generally viewed as measures of the same cognitive function (i.e., cognitive flexibility), are measuring different aspects of neuropsychological functioning. The Trails Test may be measuring the ability and speed of one's cognitive flexibility, whereas the WCST may be capturing more of one's ability in problem solving and concept formation.

Our results of reduced number of perseverative errors on the WCST among children with a parental history of depression mirror previous studies that have assessed this relationship as well (Micco et al., 2009). Micco and colleagues (2009) assessed current presence of parental MDD whereas our study gathered information regarding lifetime depressive symptoms. This suggests that improvements in youth's neuropsychological functioning, particularly in the realms of set shifting, concept formation, and problem solving, may be present for children with a parental history of depression, whether lifetime or current. If these results are to be taken at face value, our results, in conjunction with previous studies, suggest that the

exposure to previous and current symptoms of parental depression may have an unexpected benefit. One potential explanation is that children with parents who have increasing number of depressive symptoms may be more sensitive to feedback, as they have learned to adapt to reduced interpersonal parent interaction, and are therefore more reactive when hearing the corrective feedback during the WCST. However, given our modest sample size and cross-sectional research design, additional research is needed to better understand this relationship.

Our study also revealed no association between PDS-Lifetime and working memory and inhibition. These findings are divergent from previous research that found a relationship with parental depressive symptoms and inhibition and working memory (Davidovich, Collishaw, Thapar, Harold, Thapar, & Rice, 2016; Hughes, Roman, Hart, & Ensor, 2013). However, Davidovich et al.'s study assessed current parental depressive symptoms, whereas the present study measured lifetime symptoms of depression. Child EFs deficits revealed in the Davidovich's study may be expressed only during the presence of a current parental depressive episode. Hughes and colleagues (2013) also found significant relationships between parental depressive symptoms and child EFs, however Hughes measured executive functioning in early childhood, whereas the present study measured EFs in late childhood and early adolescence. Perhaps deficits revealed in early childhood are ameliorated in later development. Future research should measure both current and past depressive symptoms over the course of offspring's development to better parse apart this relationship.

There were some shortcomings to the present study. First the data are limited by the retrospective and self-report nature of the instruments employed to measure parental depression history. Second, the cross-sectional correlational design employed does not allow us to examine any temporal or causal relationships among the variables of interest. Third, our sample participants were primarily from a European-American decent. While this may reflect the local demographic of the data collection site, our results may not be generalizable to others from a more diverse background. Finally, the Wisconsin Card Sorting Task was originally designed for detecting prefrontal damage and other serious neurological problems, and as such may not be ideally suited for detecting more subtle EF deficits (Snyder, Kaiser, Warren & Heller, 2015). Despite these limitations, this study furthers the understanding of the role of executive functioning may play in the in the onset of major depressive disorder

In summary, this study demonstrated significant associations between PDS-Lifetime and several offspring EF abilities. Our results revealed differential relationships between parental depression and different aspects of youth's cognitive abilities. These divergent results highlight the need to further understand the nature of these relationships as well as the role that EF plays in the development of depressive symptoms and a major depressive disorder in children and adolescents.

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Table 1.

Mean, Standard Deviation, and Correlations Among Variables of Interest

	Mean (SD)	Parent Dep. Sym.	Child Dep. Sym	Child Age	Child IQ	Verb.WM SB	Persev Errors	Inhib Scaled Score	LNS SL Errors	LNS Scaled Score	MS SS
Parent Depressive Symptoms	4.30 (4.02)	-									
Child Current Depressive Symptoms	.69 (2.90)	0.15	-								
Child Age	10.79 (1.60)	.12	.03	-							
Intelligence Quotient	97.09 (18.26)	.05	-.21*	.40**	-						
Verbal Working Memory SB	9.86 (2.34)	0.08	.004	-.07	0.35**	-					
Persev. Errors	112 (13)	.09	.09	.16	.20**	.18	-				
Inhibition CWI Scaled Score	10.25 (3.46)	-.09	.003	.004	0.23**	.32**	.24**	-			
LNS Scaled Score	9 (2.30)	-.18*	.002	.01	.17	.15	.17*	.16	-		
LNS Set-Loss Errors	68 (38)	-.24**	-.11	.01	.06	.15	-.01	.04	.25**	-	
Motor Speed Scaled Score	10 (2.30)	-.27**	-.18*	-.08	.20*	.06	-.03	.16	.35**	.04	-

* = $p<.05$

** = $p<.001$

Table 2.
Relations Among Parent Depressive Symptoms, Covariates, and Child Neurocognitive Functioning

	Perseverative Errors (WCST)	Inhibition (CWI)	Letter-Number Sequencing (TMT)	Set-Loss Errors on LNS (TMT)	Motor Speed (TMT)	Verbal WM (SB)
	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)
Parent Depressive Symptoms	.73 (.32) *	-.03 (.09)	-.15 (.07) *	-2.31 (0.82) **	-.17 (0.05) **	-.05 (.06)
I.Q.	.24 (.07) **	.05 (.02) *	.05 (.02) **	-.08 (.22)	.04 (.01) *	.06 (.01)
Target Status	-2.90 (2.38)	.84 (.56)	-.09 (.54)	-8.80 (7.07)	.02 (.41)	.14 (.45)
Child Age	1.99 (.74) *	.02 (.18)	-.14 (.17)	-.80 (2.20)	-.03 (.14)	-.15 (.15)
Child Ethnicity	1.00 (.77)	-.15 (.18)	.01 (.17)	-.51 (2.29)	.22 (.13)	-.08 (.15)
Child Gender	2.48 (2.33)	1.70 (.55) **	1.32 (.53) *	12.46 (6.94)	.19 (.40)	.27 (.45)
Child Current Dep. Symptoms	.72 (.60)	.04 (.14)	.05 (.14)	-2.47 (1.79)	-.11 (.10)	.14 (.12)

* $p < 0.05$

** $p < 0.005$