Executive Function in Pediatric Bipolar Disorder and Attention-Deficit Hyperactivity Disorder: In Search of Distinct Phenotypic Profiles

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Abstract Often, there is diagnostic confusion between bipolar disorder (BD) and attention-deficit hyperactivity disorder (ADHD) in youth due to similar behavioral presentations. Both disorders have been implicated as having abnormal functioning in the prefrontal cortex; however, there may be subtle differences in the manner in which the prefrontal cortex functions in each disorder that could assist in their differentiation. Executive function is a construct thought to be a behavioral analogy to prefrontal cortex functioning. We provide a qualitative review of the literature on performance on executive function tasks for BD and ADHD in order to determine differences in task performance and neurocognitive profile. Our review found primary differences in executive function in the areas of interference control, working memory, planning, cognitive flexibility, and fluency. These differences may begin to establish a pediatric BD profile that provides a more objective means of differential diagnosis between BD and ADHD when they are not reliably distinguished by clinical diagnostic methods.

Keywords Bipolar disorder · Attention-deficit hyperactivity disorder · Executive function · Prefrontal cortex · Neurocognitive

Introduction

In 2001, the National Institute of Mental Health (NIMH) Roundtable on Prepubertal Bipolar Disorder proposed the diagnosis of BD in children (NIMH Roundtable 2001). The diagnosis, however, still remains somewhat controversial and often misused because of a lack of research in the heterogeneity of the disorder in youth (e.g., Geller et al. 2000, 2002; Wozniak et al. 1995). The clinical presentation of BD in adults often has an episodic course, with individuals switching from distinct affective episodes of depression, mania or hypomania, and euthymia. In children, however, agreement on the clinical presentation of BD is less clear, with some studies indicating a more chronic course of symptoms with ultra-rapid shifting of mood states from euphoria to irritability (Geller et al. 2004) or a more irritable and violent presentation during manic states (Wozniak et al. 1995). Perhaps unsurprisingly, more recent studies have indicated that narrowly defining the presentation to adult DSM-IV criteria yields different results in cognitive deficits and brain activation patterns than when the criteria are more loosely defined (Leibenluft et al. 2007; Rich et al. 2007). Despite the heterogeneity of the clinical presentation in children, BD is still considered a valid diagnosis in pediatric populations (see Youngstrom et al. 2008, for a review).

An added complication in the diagnosis of bipolar disorder in pediatric populations is the considerable overlap of symptoms with Attention-Deficit Hyperactivity Disorder (ADHD). Hypomania and mania in BD share with ADHD the symptoms of excessive talking, increased activity, inappropriate actions and verbal responses in social situations, lack of inhibition, and distractibility (Geller et al. 2002; see Kent and Craddock 2003). Additionally, the hallmark feature of chronic irritability in childhood mania...
can often mimic the chronic low frustration tolerance and emotional lability of ADHD (Geller et al. 2002; Wozniak et al. 1995). It is estimated that the comorbidity between BD and ADHD in youth ranges from 60–93% (Axelson et al. 2006; Farone et al. 1997; Geller et al. 2000, 2004; Wozniak et al. 1995). Although it has been described as a distinct diagnostic category (Geller et al. 2002, 2004; Youngstrom et al. 2008), only a limited number of studies have examined BD in youth without this comorbidity or compared the two disorders directly (see Kent and Craddock 2003; Rucklidge 2006). If pediatric BD is to be considered a distinct diagnostic entity, further work must examine its presentation in the absence of comorbidity with ADHD. Given the limited understanding of BD in youth, the differential diagnosis between BD and ADHD remains a controversial issue.

The primary aim of this review is to directly examine the quantitative similarities and differences in executive functioning between BD and ADHD in order to determine if a distinct neurocognitive profile exists for each. Both disorders are currently diagnosed using a clinical interview and self-report checklists completed by the individual, a parent, or teacher. Unfortunately, the diagnostician must rely on self-report evidence and clinical judgment to arrive at a diagnosis, as there is no valid biological or behavioral test for arriving at the diagnosis. It is tenuous, however, to rely solely on self-reported characteristics to form a differential diagnosis for these disorders given the behaviorally similar presentation in childhood. Much of the controversy regarding the diagnosis of pediatric BD lies in the Bipolar Disorder Not Otherwise Specified (BDNOS) category. As this is a more broad-reaching category including symptoms of BD that do not reach the threshold for full diagnostic criteria, there is often confusion on whether BD or a disruptive behavior disorder, such as ADHD, should be diagnosed when children present as chronically irritable and emotionally labile (Galantar and Leibenluft 2008). While several researchers have made attempts to define the BD phenotype as more narrowly conforming to the adult criteria for BD (Leibenluft and Rich 2008; Stringaris et al. 2009), there remains no clear-cut answer for the definition of BDNOS in pediatric samples.

The inherent problems in differential diagnosis from BD warrant the use of more objective supporting evidence, such as neuroimaging and cognitive testing, to assist in diagnosis in children and adolescents, although some have questioned this approach in ADHD (Barkley and Grodzinsky 1994). This objective data, however, would provide supporting evidence for differing neural networks that occur in pediatric BD and ADHD, despite similar behavioral presentations. Research in magnetic resonance imaging (MRI) and diffusion tensor imaging (DTI), indicate differences in structural abnormalities in BD versus ADHD, with volumetric abnormalities of the limbic system and smaller volume of the ventrolateral prefrontal cortex (VLPFC) and rostral prefrontal cortex (PFC) implicated in pediatric BD and smaller volume of striatal structures implicated in ADHD but not BD (Blumberg et al. 2005; Kalmar et al. 2009; Lopez-Larson et al. 2009). Disruption in white matter appears more diffuse across the brain regions in ADHD as compared to BD, whereas white matter abnormalities are more restricted to cortico-limbic connections in the PFC for BD (Pavuluri et al. 2009b). Evidence from functional imaging studies reveal relatively greater activation in the bilateral VLPFC and right dorsolateral PFC (DLPFC) for pediatric BD relative to the ADHD during response inhibition tasks (Passarotti et al. 2009). Other tasks of executive function indicate greater activation in left DLPFC, bilateral ACC, and left putamen and thalamus for children with BD (Blumberg et al. 2003; Chang et al. 2004; Nelson et al. 2007), whereas children with ADHD exhibit lower activation in right DLPFC and ACC, and striatal areas (Casey et al. 1997; Konrad et al. 2007; Smith et al. 2008). Although there is evidence that individuals with ADHD and BD differ on neuroanatomical structure and function, such neurobiological measurements (e.g., MRI, SPECT) remain impractical and inefficient in the typical clinical setting. Thus, developing a neurocognitive profile for each disorder would aid in differential diagnosis and avoid the pitfalls of misdiagnosis in youth.

Executive Function as a Model

The PFC and its related neural circuitry are critically involved in executing many of the components underlying executive function. Pennington’s (1997) concept of the “frontal metaphor” applies to the term “executive function”, in that executive function is a latent construct born from theory and, thus, is not an exact representation of the activity in PFC. Measurement of executive function thus proves difficult, as it is a latent construct composed of several components, which are not clearly defined. Factor analyses of test batteries of executive function do not result in a single factor, but at least five factors: 1) inhibition, 2) working memory, 3) planning, 4) set-shifting, and 5) fluency. (Pennington 1997; Pennington et al. 1996; Welsh et al. 1991) A recent review of a highly similar latent construct, cognitive control, suggests similar dimensions based on literature mining (Sabb et al. 2008). Although five distinct dimensions, these components of executive function are interrelated processes that depend on interactions with the others to execute control over behavior. Thus, here we use the term ‘executive function’ to refer to the overarching representation of PFC function, recognizing that this latent construct is multifactorial and remains ill-defined.
Executive Function, BD, and ADHD

The present review examines BD and ADHD as disorders of the PFC, although it is clear that many areas of the brain are implicated. Although both BD and ADHD show both abnormal PFC functioning and deficits in executive function, there may be distinct signatures in the patterns of deficits that suggest two different disorders. Pennington (1997) notes that, in general, while two separate disorders may both involve dysfunction of the PFC and have similar behavioral presentations, discriminant validity may arise from deficits in different areas of executive function. For example, individuals with schizophrenia have been shown to have greater impairment in some aspects of problem-solving than individuals with BD, but not in the areas of set-shifting and attention (see Bearden et al. 2001 for review).

Here we review five facets of executive function through neurocognitive tasks found in both the pediatric BD and ADHD literatures (see Table 1 for a summary of domains of executive functioning and tasks examined in this review). To the best of our knowledge, this is the first review comparing the neurocognitive function of individuals with pediatric BD and ADHD, and while several studies have emerged to provide support for a neurocognitive profile of pediatric BD research in this new field remains scant. We end our review with a discussion of the executive functioning differences in BD and ADHD and their implications for differential diagnosis of these two disorders.

Method

The current review was conducted following an extensive search through the research databases PsychInfo and Medline. Key terms were selected based on previous factor analyses on executive function and review papers in the areas of BD and ADHD. Terms included, ‘bipolar’, ‘mania’, ‘manic’, ‘ euthymic’, ‘ADHD’, ‘attention-deficit disorder’, and ‘hyperactivity’, and were cross-referenced with the terms ‘cognitive’, ‘neuropsychological’, ‘executive’, ‘prefrontal’, ‘inhibition’, ‘working memory’, ‘planning’, ‘fluency’, and ‘set-shifting’. Articles were selected based on the following criteria: use of English language, conducted between the years of 1989 and 2009, clear presentation of results for variables of interest, and use of children or adolescents. Additionally, bibliographies of the articles selected were used to supplement citations that had not been acquired through the database literature search. For the purposes of consistency in measurement, we only review tasks that have been validated in the literature and have been studied in both pediatric BD and ADHD samples.

Sixty-eight studies in ADHD and sixteen studies in pediatric BD were included in the review. Given the paucity of research conducted on pediatric BD to date with most studies having small sample sizes themselves, we chose to present quantitative data from these studies noting their sample sizes and effect sizes, but not conduct a formal meta-analytic review, which may be biased by small numbers of studies with small sample sizes. A majority of these studies are new, suggesting a burgeoning and important field, which will hopefully allow for more strict meta-analyses to be conducted in the future.

All studies selected utilized diagnostic criteria from either DSM-III-R or DSM-IV in their methodology. Due to the nascentness of research in pediatric BD, all but one of the BD studies used DSM-IV criteria. In the ADHD literature, 24 studies used DSM-III-R criteria and 44 studies used DSM-IV criteria. Studies on BD were included if subjects met criteria for bipolar I disorder, bipolar II disorder, and bipolar disorder not otherwise specified (BDNOS). Not all studies examined all executive function variables in this included review; 23 studies were single measure studies and 59 used more than one task or a battery. One study included examined cognitive performance in at-risk adolescents who later developed bipolar disorder (Meyer et al. 2004). Eleven of the 16 BD studies included statistics on comorbid ADHD, the average rate of which was 54%. Effects of comorbidity are noted where appropriate in the review. As in most studies of BD, pediatric samples were taking one or more medications at the time of testing. Where examined, effects of medication were found not to produce differences in performance in four of those five studies (Del Bello et al. 2004; Leibenluft et al. 2007; Pavuluri et al. 2006). The remaining study found that psychotropic medication was associated with a decrease in verbal fluency score (Bearden et al. 2007).

Although approximately a third of studies (21 total) reviewed from the ADHD literature distinguished subtypes of ADHD or included analyses on comorbid learning disabilities (LD; 21 total), the majority did not and these factors were combined in the overall findings. Subtype and comorbid LD findings are noted in this review where appropriate, but because the ADHD literature does not always distinguish them, this is noted as a limitation to the conclusions of the present review. For all ADHD studies reviewed, children and adolescents were either off of medication for >24 hours or medication naïve. For both BD and ADHD literature, studies included were sampled from a variety of populations, but included mostly outpatient or community samples (97%).

To provide a more comprehensive review in comparing these two groups, we have calculated average effect size (ES), weighted by sample size, for each of the tasks.
for each group (ADHD and BD) using Cohen’s $d$ (Cohen 1988).

$$ES = \frac{\sum_{i=1}^{n_i} d_i n_i}{\sum_{i=1}^{n_i} n_i}$$

In order to reduce bias from a particular sample, only a single contribution from each independent sample was used; thus, if a study examined two tasks from the same domain of executive functioning, only the larger of the two effect sizes was used in the average weighted effect size calculation presented in Table 2. Descriptors for effect size are provided for relative comparison and are as defined by Cohen (1988): small = 0.2, medium = 0.5, large > 0.8. An absolute difference score was calculated between the average weighted effect sizes of the two groups. This was used to create a visual presentation of these findings in Fig. 1, which illustrates the areas of executive function showing the largest difference on the left to the smallest difference on the right. Weighted effect sizes for each study are provided in Table 3 for ADHD and Table 4 for BD. As there are only two studies to date that has directly compared executive function in pure pediatric BD and pure ADHD (Passarotti et al. 2009; Rucklidge 2006), all effect sizes presented are each population as compared to control groups. It should be noted that effect sizes for non-significant findings were included and all findings were indicative of either worse performance or no difference from control group.

Results

Inhibition

Inhibition is a component of executive function that can be broken into several subsystems (see Barkley 1997; Nigg 2000; Tannock 1998, for reviews; Quay 1988, 1997; Schachar and Logan 1990). Barkley (1997) proposes a comprehensive theory of inhibition that consists of three interrelated processes based on both timing and situation in which stimuli are presented including inhibition in regard to an initial response to a stimulus or an ongoing response (Response Inhibition), and inhibition of interfering stimuli (Interference Control).

Response Inhibition

Response inhibition is the process of physically inhibiting a response to a stimulus. Several behavioral tasks have been developed to measure response inhibition; two of which have been examined in the pediatric BD and ADHD literatures: go/no-go style variants of the Continuous Performance Test

Table 1 Tasks of executive function across pediatric BD and ADHD literature

| Executive function domain | Tasks | Description | Key scores |
|---------------------------|-------|-------------|------------|
| Inhibition                |       |             |            |
| Response inhibition       | CPT   | Inhibit response when target stimulus shown; rapid response when other stimuli are shown | Errors of commission on CPT (CPT-EC) or Stop-signal reaction time (SSRT) |
|                           | SST   | Inhibit prepotent response when tone is heard | Interference score |
| Interference control      | Stroop task | Rapidly naming colors (e.g., “green”) of ink in which color words (e.g., “red”) are printed |            |
| Working memory            | Digit-Span Total (DS-T) | Repeat a series of numbers in forward or reverse order from which they are presented; Total score includes both forward and backward presentations | # of series of digits correct |
|                           | DS-B  | | |
| Spatial working memory    | CANTAB SWM Task | Maintain spatial memory of already selected material | Between search errors |
|                           | SSP   | Mentally rearrange spatial information and output a behavioral response | # of series of blocks/squares correct |
| Planning                  | ToL   | Move 3+ rings/balls to match a particular arrangement, while adhering to specific rules of how they can be moved | Total score (ToL-T) |
| Set-shifting              | Wisconsin Card Sort Task | Sort cards according to shifting rules | Perseverative errors (WCST-PE) |
| Fluency                   | F-A-S Test | Rapidly name words that begin with ‘F’ (or ‘A’ or ‘S’) | # of words |
|                           | Categories | Rapidly name words that belong to a category | # of words |
(CPT; Conners 1985, 1992; Gordon 1983; Rosvold et al. 1956), and the Stop-Signal Task (SST; Logan et al. 1984). These tasks may putatively engage two separate processes activated either by inhibition of an initial response or deactivation of an ongoing response to a stimulus. The primary response inhibition variables (or indicators—as we define the independent variables of interest) for these tasks are errors of commission (EC) and stop-signal reaction time (SSRT), respectively.

CPTs are widely used measures of response inhibition in the ADHD literature. Of the 68 child ADHD studies reviewed here, 21 examined EC on the CPT. Overall, ADHD samples showed ES = .56 on CPT-EC as compared to controls, indicating a moderate effect for ADHD to exhibit a more impulsive response style on the CPT (see Table 2). In comparison, thirteen studies found no deficits in inhibition in children with ADHD, with effect sizes ranging from .07 to 1.27 for EC. Only two studies examined the effects of ADHD subtype on CPT-EC. Tsal et al. (2005) found that inattentive-type was associated with more EC than combined-type, while Willcutt et al. (2005) found no differences between subtypes in EC.

**Table 2** Average weighted effect size by task

| Executive function domain | Variable | BD weighted ES | ADHD weighted ES |
|---------------------------|----------|----------------|------------------|
| **Inhibition**            |          |                |                  |
| Response inhibition       | CPT Errors of Commission (CPT-EC) | .40 | .56 |
|                           | Stop-Signal Reaction Time (SSRT) | .31 | .63 |
| Interference control      | Stroop interference (Stroop) | .60 | .38 |
| **Working memory**        |          |                |                  |
| Verbal working memory     | Digit-Span Total (DS-T) | .67 | .66 |
|                           | Digit-Span Backward (DS-B) | .38a | .63 |
| Spatial working memory    | CANTAB SWM – Between Search Errors (SWM-BSE) | .35a | .77 |
|                           | Spatial Span (SSp) | .80 | .94 |
| **Planning**              |          |                |                  |
|                         | Tower of London – Total (ToL) | .96 | .38 |
| **Set-shifting**          |          |                |                  |
|                         | WSCT – Perseverative Errors (WCST-PE) | .73 | .37 |
| **Fluency**               |          |                |                  |
| Phonemic fluency          | F-A-S | .34 | .68 |
| Semantic fluency          | Categories | .38 | .38 |

*CPT* Continuous Performance Test, *CANTAB* Cambridge Neuropsychological Test Automated Battery

*a These ES’s are the result of only one study and are thus not weighted averages

Fig. 1 Executive function domains for bipolar disorder (BD) and attention-deficit hyperactivity disorder (ADHD) organized from largest to smallest absolute differences in average weighted effect sizes.
| Reference                  | n   | CPT-EC | SSRT | Stroop | DS-T | DS-B | SWM-BSE | Stp | ToL | WCST | FAS | Categories |
|----------------------------|-----|--------|------|--------|------|------|---------|-----|-----|------|-----|------------|
| Alloway et al. (2009)      | ADHD=46 C=20 | .14<sup>b</sup> | -    | -      | -    | -    | -       | -   | -   | -    | -   | -         |
| Assesmany et al. (2001)    | ADHD=40  C=40 | -    | -    | -      | 1.41 | -    | -       | -   | -   | -    | -   | -         |
| Barkley et al. (2001)      | ADHD=101 C=39  | .07<sup>a</sup> | -    | -      | -    | .22  | -       | -   | -   | -    | -   | -         |
| Barkley et al. (1992)      | ADHD=24 C=12  | 1.29<sup>b</sup> | -    | 1.49   | -    | -    | -       | -   | .46 | .86  | .51 | -         |
| Barnett et al. (2001)      | ADHD=27 C=26  | -    | -    | -      | -    | 1.48 | -       | -   | -   | -    | -   | -         |
| Bedard et al. (2003)       | ADHD=59 C=59  | -    | -    | -      | -    | -    | -       | -   | -   | -    | -   | -         |
| Berlin et al. (2004)       | ADHD=21 C=42  | -    | .57  | -      | -    | -    | -       | -   | -   | -    | -   | -         |
| Borger et al. (1999)       | ADHD=21 C=16  | .65<sup>b</sup> | -    | -      | -    | -    | -       | -   | -   | -    | -   | -         |
| Brewer et al. (2001)       | ADHD=26 C=22  | .38<sup>b</sup> | -    | -      | -    | -    | -       | -   | -   | -    | -   | -         |
| Cairney et al. (2001)      | ADHD=13 C=15  | -    | -    | -      | -    | 1.13 | -       | -   | -   | -    | -   | -         |
| Collings (2003)            | ADHD=46 C=24  | .28<sup>b</sup> | -    | -      | -    | -    | -       | -   | -   | -    | -   | -         |
| Corbett et al. (2009)      | ADHD=18 C=18  | -    | -    | .31    | -    | -    | .21     | .75 | -   | .03  | .07 | -         |
| Dimoska et al. (2003)      | ADHD=13 C=13  | -    | .137 | -      | -    | -    | -       | -   | -   | -    | -   | -         |
| Epstein et al. (2003)      | ADHD=21 C=795 | .46<sup>a</sup> | -    | -      | -    | -    | -       | -   | -   | -    | -   | -         |
| Geurts et al. (2004)       | ADHD=54 C=41  | -    | .98  | -      | -    | -    | -       | .36 | -   | .87  | .58 | -         |
| Goldberg et al. (2005)     | ADHD=12 C=32  | -    | -    | .12    | -    | -    | .57     | -   | -   | -    | -   | -         |
| Grodzinsky and Diamond (1992) | ADHD=66 C=64 | .76<sup>b</sup> | -    | 1.11   | -    | -    | -       | -   | -   | .16  | -   | -         |
| Happe et al. (2006)        | ADHD=30 C=32  | -    | -    | -      | -    | -    | 1.06    | -   | -   | -    | .63 | .07       |
| Henin et al. (2007)        | ADHD=102 C=120 | -    | -    | .2     | .2   | -    | -       | -   | -   | .27  | -   | -         |
| Horn et al. (1989)         | ADHD=54 C=31  | .83<sup>b</sup> | -    | -      | -    | -    | -       | -   | -   | -    | -   | -         |
| Houghton et al. (1999)     | ADHD=94 C=28  | -    | -    | .17    | -    | -    | -       | .15 | .31 | -    | -   | -         |
| Jennings et al. (1997)     | ADHD=40 C=26  | -    | .53  | -      | -    | -    | -       | -   | -   | -    | -   | -         |
| Kempton et al. (1999)      | ADHD=15 C=15  | -    | -    | -      | -    | -    | 1.34    | 1.26 | .48 | -    | -   | -         |
| Kerns et al. (2001)        | ADHD=21 C=21  | .23<sup>a</sup> | -    | .45    | -    | -    | -       | -   | -   | -    | -   | -         |
| Klorman et al. (1999)      | ADHD=299 C=28 | -    | -    | -      | -    | -    | -       | -   | .07 | -    | -   | -         |
| Konrad et al. (2000)       | ADHD=16 C=21  | -    | 1.15 | -      | -    | -    | -       | -   | -   | -    | -   | -         |
| Lawrence et al. (2004)     | ADHD=22 C=22  | -    | -    | .12    | -    | -    | -       | -   | -   | 1.09 | -   | -         |
| Loge et al. (1990)         | ADHD=20 C=20  | .85<sup>b</sup> | -    | -      | -    | -    | -       | -   | .16 | .17  | .42 | -         |
| Luft et al. (1990)         | ADHD=29 C=20  | -    | -    | .7     | 1.23 | -    | -       | -   | -   | -    | -   | -         |
| Reference                      | n   | CPT-EC | SSRT | Stroop | DS-T | DS-B | SWM-BSE | SSρ | ToL | WCST | FAS | Categories |
|--------------------------------|-----|--------|------|--------|------|------|---------|-----|-----|------|-----|------------|
| Manassis et al. (2000)        | ADHD=30 | .17 |      |       |      |      |         |     |     |      |     |            |
| Mariani and Barkley (1997)    | ADHD=34 | .46  |      |       |      |      |      |     |     |      |     |            |
| Marzoochi et al. (2008)       | ADHD=35 | .19  |      |       |      |      |      |     |     |      |     |            |
| Mataró et al. (1997)          | ADHD=11 | .39  |      |       |      |      |      |     |     |      |     |            |
| McNeary and Kerns (2003)      | ADHD=30 | .86  |      |       |      |      |      |     |     |      |     |            |
| Melnes et al. (2003)          | ADHD=39 |      |      |       |      |      |      |     |     |      |     |            |
| Nigg (1999)                   | ADHD=25 | .88  |      |       |      |      |      |     |     |      |     |            |
| Nigg et al. (2002)            | ADHD=64 | .8   |      |       |      |      |      |     |     |      |     |            |
| Overtoom et al. (2002)        | ADHD=16 | .92  |      |       |      |      |      |     |     |      |     |            |
| Passarotti et al. (2009)      | ADHD=15 | .43  |      |       |      |      |      |     |     |      |     |            |
| Pennington et al. (1993)      | ADHD=32 | 1.16 |      |       |      |      |      |     |     |      |     |            |
| Pineda et al. (1999)          | ADHD=62 | .65  |      |       |      |      |      |     |     |      |     |            |
| Pliszka (1992)                | ADHD=92 | .87a |      |       |      |      |      |     |     |      |     |            |
| Pliszka et al. (1997)         | ADHD=13 | .136 |      |       |      |      |      |     |     |      |     |            |
| Pliszka et al. (2000)         | ADHD=10 | .75  |      |       |      |      |      |     |     |      |     |            |
| Purvis and Tannock (2000)     | ADHD=34 | .48a |      |       |      |      |      |     |     |      |     |            |
| Rubia et al. (2001)           | ADHD=16 | .61  |      |       |      |      |      |     |     |      |     |            |
| Rucklidge and Tannock (2002)  | ADHD=59 | .72  |      |       |      |      |      |     |     |      |     |            |
| Rucklidge (2006)              | ADHD=30 | .32a |      |       |      |      |      |     |     |      |     |            |
| Sartory et al. (2002)         | ADHD=20 | 1.01a|      |       |      |      |      |     |     |      |     |            |
| Schachar et al. (2000)        | ADHD=72 | .58  |      |       |      |      |      |     |     |      |     |            |
| Schachar and Tannock (1995)   | ADHD=40 | .64  |      |       |      |      |      |     |     |      |     |            |
| Schachar et al. (1995)        | ADHD=14 | .6   |      |       |      |      |      |     |     |      |     |            |
| Scheres et al. (2001)         | ADHD=24 | .39  |      |       |      |      |      |     |     |      |     |            |
| Schmitz et al. (2002)         | ADHD=30 | .14  |      |       |      |      |      |     |     |      |     |            |
| Seidman et al. (1997a)        | ADHD=43 | .15  |      |       |      |      |      |     |     |      |     |            |
| Seidman et al. (1997b)        | ADHD=118 | .31 |      |       |      |      |      |     |     |      |     |            |
| Shue and Douglas (1992)        | ADHD=24 | .88  |      |       |      |      |      |     |     |      |     |            |
| Solanto et al. (2001)         | ADHD=77 | .69  |      |       |      |      |      |     |     |      |     |            |
Similarly, four studies in the pediatric BD literature have explored EC on the CPT with an overall ES=.40, which is somewhat less than in the ADHD literature, but also indicates a moderate effect for response inhibition difficulties. One of these studies found significant differences from control groups (d= .69; Pavuluri et al. 2006), while the other three did not (DelBello et al. 2004; Robertson et al. 2003; Rucklidge 2006). Two of these studies examined medication effects on EC and found no associated changes in performance (DelBello et al. 2004; Pavuluri et al. 2006).

One issue noted when reviewing the literature in both groups for the CPT, however, is that there is a difference in effect size for deficits on EC depending on whether the CPT involves inhibiting a prepotent response (i.e., inhibiting to a target stimulus; e.g., Conners 1985, 1992) or responding to a target (i.e., inhibiting to non-target stimuli; e.g., Gordon 1983; Rosvold et al. 1956), the latter of which some might classify as more a task of vigilance and sustained attention. In the ADHD literature, the average weighted effect size is much larger (ES=.39 versus .67) for inhibiting to target versus non-target. The same holds true for the BD literature (ES=.23 versus .60). Thus, differences observed in these two diagnostic groups depend on CPT version (see Tables 3 and 4 for clarification on which CPT version was used in each study). Unfortunately, these versions are often combined in reviews.

The SST is also consistently associated with deficits in response inhibition in ADHD across the lifespan (Nigg 1999; Nigg et al. 2002; Rucklidge and Tannock 2002; Schachar et al. 2000). The average weighted effect size for studies reviewed was moderate to large (ES=.63). Nineteen out of 25 studies reviewed (76%) found deficits in SSRT for children and adolescents with ADHD as compared to control groups. Those studies that did examine subtypes separately did not find effects of subtype on SSRT performance (Bedard et al. 2003; Geurts et al. 2004; Willcutt et al. 2005).

In the pediatric BD literature, three studies have examined deficits on SSRT and indicate a small effect (ES=.31). None of these studies found their BD groups to be significantly different than control groups (Leibenluft et al. 2007; McClure et al. 2005; Passarotti et al. 2009). The Passarotti study (2009), however, also directly compared

### Table 3 (continued)

| Reference                  | n   | CPT-EC | SSRT | Stroop | DS-T | DS-B | SWM-BSE | SSp | ToL | WCST | FAS | Categories |
|---------------------------|-----|--------|------|--------|------|------|---------|-----|-----|------|-----|------------|
| Stevens et al. (2002)     | ADHD=76 C=76 | - | .34  | - | - | - | - | - | - | - | - | - |
| Toplak et al. (2003)       | ADHD=59 C=39 | - | - | - | - | .7 | - | - | - | - | - | - |
| Toplak et al. (2009)       | ADHD=45 C=42 | - | - | - | - | .58 | - | - | - | - | - | - |
| Tripp and Alsop (1999)     | ADHD=43 C=19 | .48<sup>b</sup> | - | - | - | - | - | - | - | - | - | - |
| Tripp et al. (2002)        | ADHD=28 C=28 | .15<sup>b</sup> | - | - | - | - | .9 | - | .69 | - | - | - |
| Tsai et al. (2005)         | ADHD=27 C=15 | .7<sup>b</sup> | - | - | - | - | - | - | - | - | - | - |
| van Leeuwen et al. (1998)  | ADHD=20 C=20 | 1.27<sup>b</sup> | - | - | - | - | - | - | - | - | - | - |
| Wiers et al. (1998)        | ADHD=28 C=34 | - | - | - | - | - | - | - | .33 | - | - | - |
| Willcutt et al. (2005)     | ADHD=177 C=151 | .49<sup>b</sup> | .57 | .07 | - | .59 | .55 | - | - | .43 | - | - |
| Wu et al. (2002)           | ADHD=83 C=29 | - | - | - | - | .55 | - | - | .13 | - | - | - |

<sup>a</sup> ADHD Attention Deficit Hyperactivity Disorder, C Control group

<sup>b</sup>CPT – Inhibit to target

<sup>c</sup>CPT – Inhibit to non-target

<sup>d</sup>All effect sizes presented as Cohen’s d (Cohen 1988), with positive effect sizes indicating ADHD performed worse on the task. Unbolded effect sizes = ADHD group was not significantly different from controls. Bolded effect sizes = ADHD group performed significantly worse than control.
the BD group to an ADHD group and found the ADHD group to have a faster SSRT, but this difference was not significant \((d = .37)\). Thus, while some research focuses on the variable of errors of commission on the CPT as a neuropsychological representation of deficits in response inhibition in each of these disorders, performance on the SST is more consistently different from normal on the SST for ADHD. The two studies that directly compared pediatric BD to ADHD, however, found no significant differences between groups for CPT-EC (Rucklidge 2006) or SST (Passarotti et al. 2009). In sum, the evidence indicates the deficit in response inhibition to be present to some degree in both the ADHD and pediatric BD neurocognitive profiles.

### Interference Control

The Stroop task (Stroop 1935) is a widely used measure of interference control that has been studied in both pediatric BD and ADHD samples, with the primary indicator being the interference score. Overall, Stroop interference in individuals with ADHD produces an overall small weighted effect size of \(.35\). Ten out of 15 studies found no significant difference in Stroop interference performance for ADHD.

### Table 4: BD study effect sizes \((d)\)

| Reference            | \(n\) | CPT-EC | SSRT | Stroop | DS-T | DS-B | SWM-BSE | SSp | ToL | WCST | FAS | Categories |
|----------------------|------|--------|------|--------|------|------|---------|-----|-----|------|-----|-----------|
| Bearden et al. (2007)| BD=31| -      | -    | -      | -    | .38  | -       | -   | -   | -    | .34 | .46       |
| Blumberg et al. (2003)| BD=10| -      | -    | .7     | -    | -    | -       | -   | -   | -    | -   | -         |
| Castillo et al. (2000)| BD=10| -      | -    | -      | -    | -    | -       | -   | .89 | -    | -   | -         |
| DelfBello et al. (2004)| BD=20|       |      |        |      |      |         | .48 | -   | -    | -   | -         |
| Dickstein et al. (2004)| BD=18| -      | -    | -      |      | -    | .35     | -   | -   | -    | -   | -         |
| Doyle et al. (2005)   | BD=57| -      |      | .57    | .74  | -    | -       | -   | -   | .7   | -   | -         |
| Leibenluft et al. (2007)| BD=26| -      |      | .28    | -    | -    | -       | -   | -   | -    | -   | -         |
| McClure et al. (2005) | BD=38| -      |      | .49    | -    | -    | -       | -   | -   | -    | -   | -         |
| Meyer et al. (2004)   | BD=9 | -      |      | -      | -    | -    | -       | -   | -   | .6   | -   | -         |
| Olvera et al. (2005)  | BD=28| -      |      | -      |      | -    | -       | -   | .98 | .89  | -   | -         |
| Passarotti et al. (2009)| BD=15| -      |      | .11    | -    | -    | -       | -   | -   | -    | -   | -         |
| Pavuluri et al. (2006)| BD=26| -      |      | .69    | .43  | -    | -       | .91 | -   | -    | -   | -         |
| Pavuluri et al. (2009a)| BD=26| -      |      | -      |      | .67  | -       | .69 | -   | -    | -   | -         |
| Robertson et al. (2003)| BD=44| -      |      | .23   |      | -    | -       | -   | -   | -    | -   | -         |
| Rucklidge (2006)      | BD=12| -      |      | .35   | .84  | -    | -       | -   | -   | -    | -   | -         |
| Voelbel et al. (2006) | BD=12| -      |      | .69   | -    | -    | -       | -   | .58 | .34  | .16 | -         |

**Mean weighted effect size**: \(d = .40\) \(d = .31\) \(d = .60\) \(d = .67\) \(d = .38\) \(d = .35\) \(d = .80\) \(d = .96\) \(d = .73\) \(d = .34\) \(d = .38\)

**BD**: Bipolar Disorder, **C**: Control group

**CPT**: Continuous Performance Test, **SSRT**: Stop Signal Reaction Time, **DS-T**: Digit Span Total score, **DS-B**: Digit Span Backwards, **SWM-BSE**: Between-Search Errors on the CANTAB spatial working memory task, **SSp**: Spatial Span, **ToL**: Tower of London total score, **WCST**: Wisconsin Card Sorting Task - perseverative errors, **FAS**: F-A-S fluency task

\(a\) CPT - Inhibit to target
\(b\) CPT – Inhibit to non-target
\(c\) All effect sizes presented as Cohen’s \(d\) (Cohen 1988), with positive effect sizes indicating BD performed worse on the task. Unbolded effect sizes = BD group was not significantly different from controls. Bolded effect sizes = BD group performed significantly worse than control.
groups, with effect sizes ranging from .07 to .45. The five studies that did find a significant difference in Stroop interference revealed medium to large effect sizes (ES=.31 to 1.49; Barkley et al. 1992; Berlin et al. 2004; Grodzinsky and Diamond 1992; Lufi et al.1990; Seidman et al. 1997b).

Three studies of pediatric BD literature have examined performance on the Stroop task, with a weighted effect size of .60. One study found children with BD trended towards difference on interference (p=.07, ES=.57; t-scores = 49.0 for BD and 52.9 for NC; Doyle et al. 2005). The remaining two studies in adolescents with BD, however, did not find significant differences between BD and control groups on interference, although effect sizes were .7 and .69 (Blumberg et al. 2003; Voelbel et al. 2006).

Based on the data available to date, there is stronger evidence for interference control deficits on the Stroop task for individuals with BD than for those with ADHD. This may seem surprising given anecdotal evidence that individuals with ADHD are less able to inhibit distraction from external stimuli; however, these studies suggest that individuals with BD may have more difficulty ignoring unimportant stimuli during a task.

Overall, it appears that it is important to examine the subcomponents of inhibition when comparing BD and ADHD. These disorders show a similar pattern of deficits in response inhibition, which are either minor for CPTs requiring inhibition of a prepotent response or extensive for the SST. Difficulties with interference control, however, appear to be specific to BD.

Working Memory

Verbal Working Memory

Verbal working memory (VWM) is a component of executive function that involves the ability to manipulate incoming verbal information into organized output. The most widely used test of VWM is the Digit Span (DS) subtest of the Wechsler intelligence scales (WAIS, WISC; Wechsler 1991, 1994). It has two presentations, DS-Forward and DS-Backward, the latter of which is a robust indicator of executive function, requiring the manipulation of information in memory.

There is support for impairment on the DS-Backward for individuals with ADHD, with an average weighted effect size of .63. Five of 7 studies reporting performance on this subtest indicate that children with ADHD perform significantly worse than controls, with effect sizes ranging from .59 to 1.73. Only two studies did not find deficits in performance on the DS-Backward task for children with ADHD (Barkley et al. 2001; Wu et al. 2002). Literature for DS-Backward is limited, however, in the pediatric BD area. Only one study has been conducted using this subtest, with results reflecting a small effect size of .38 with nonsignificant differences from the control sample (Bearden et al. 2007).

Spatial Working Memory

Spatial working memory (SWM) also involves the mental organization and manipulation of material but with nonverbal information. Several tasks are good representations of SWM, such as the Wechsler Spatial Span tasks and the SWM task from the Cambridge Neuropsychological Test Automated Battery (CANTAB; Luciana and Nelson 2002). As is the problem with DS, results of the Spatial Span task often combine forward and backward presentations, thus making it difficult to tease apart components of working memory (see Table 2 and Fig. 1).

Indications of deficits on the CANTAB SWM task for children with ADHD, however, are consistent with those on VWM tasks (average weighted ES=.77). Five out of 7 studies found significant deficits in SWM on the CANTAB for children with ADHD. Two studies did not find impairment in ADHD on this task, with effect sizes ranging from .2 to .55 (Corbett et al. 2009; Willcutt et al. 2005). Again in pediatric BD only one study has examined SWM (CANTAB), and consistent with the VWM literature in this group, shows no deficits for pediatric BD (ES=.35; Dickstein et al. 2004).

Thus, overall, it appears that working memory impairments in both verbal and spatial domains may be emerging as a marker that differentiates ADHD from BD. Further research must be done to examine working memory specifically as its own domain and in further studies in pediatric BD.

As mentioned above, Wechsler digit and spatial span subtests are often presented as a composite score for the entire DS subtest. This is often the result of earlier versions of the WISC not separating the two, which can obscure important information regarding different components of executive control. As indicated in Table 2, the composite DS score (DS-Total) confounds the difference in working memory function between the two disorders, seen in DS-Backwards. Several of the studies reviewed used the composite score, including 4 in the ADHD literature and 4 in the pediatric BD literature. Despite the confound result reporting, there is emerging evidence for deficits in working memory to be specific to ADHD.

Planning

Planning involves the ability to manipulate information into a reliable sequence that will achieve an end goal. It requires an individual not only to think in terms of the next step, but also to think of the future consequences of such a step. The task typically used to examine planning abilities across the pediatric BD and ADHD literature is the Tower of London (ToL; Shallice 1982).
Results for the ToL indicate a small weighted effect size of .38 for deficits in ADHD. Four out of seven studies found no significant difference in ToL total score from control groups (ES ranging from .13 to .36). Nigg et al. (2002) found children with ADHD-Combined type to have a lower total score on the ToL than controls. Similar findings have been reported in 2 other studies, although not classified by subtype (Kempton et al. 1999; Marzoochi et al. 2008).

Findings in the pediatric BD literature, although limited in number, more strongly indicate a planning deficit with a large weighted effect size of .96. Olvera et al. (2005) found youth with BD to perform significantly below the control group (ES=.89). With a younger sample, Castillo et al. (2000) found children with BD to perform below average on the NEPSY Tower subtest (mean scaled score = 7.17).

While still preliminary based on the small number of studies in this domain, there is evidence for deficits in planning in pediatric BD as measured by the ToL. Furthermore, this dysfunction may be specific to the pediatric BD phenotype, but more research to directly examine this hypothesis needs to be executed.

Set-Shifting

Set-shifting is a process of working memory that involves attention to a current stimulus and the ability to maintain that attention while shifting between stimuli. The ability to adapt and change a response to new incoming stimuli in the environment has long been considered a hallmark of executive function. A measure of this dimension of executive function that spans the pediatric BD and ADHD literature is the perseverative errors score on the Wisconsin Card Sorting Test (WCST; Heaton 1981).

The findings for performance on the WCST for ADHD primarily indicate that children with ADHD do not show a deficit in set-shifting, with a small average weighted effect size of .36. Thirteen of 18 studies found no differences in perseverative errors and categories achieved between children with ADHD and controls (ES=.07 to .6). Five studies found children with ADHD made significantly more perseverative errors than controls, however, two studies reported a greater number of perseverative errors in their ADHD group, but noted that the difference became non-significant when they controlled for IQ, indicating that IQ may play a role in performance on the WCST (Tripp et al. 2002; Wijlcutt et al. 2005).

There is greater evidence for impairment on the WCST in pediatric BD, with a large average weighted effect size of .73. Two out of 4 studies showed significant differences on the WCST from control groups (ES=.6 and .89), while 2 did not (ES=.58 and .7). Meyer et al. (2004) reported that young adults diagnosed with BD showed a trend toward more perseverative errors when the WCST was administered during their adolescence. Results of this study are important because early attentional problems in conjunction with disturbances in executive function on the WCST predicted BD onset, but not unipolar depression or no mood disorder in young adulthood. Thus, like deficits in planning, deficits in set-shifting appear to be specific to the pediatric BD phenotype.

Verbal Fluency

Fluency is a measure not only of vocabulary breadth and semantic memory, but also processing speed, working memory, inhibition, and set maintenance. Verbal fluency is often measured in two conditions: letters (phonemic) and categories (semantic). Perhaps the most widely used test of phonemic verbal fluency is the Controlled Oral Word Association Test (COWAT; Benton and Hamsher 1978), where individuals are required to generate words beginning with a particular letter (‘F’, ‘A’, or ‘S’). For semantic fluency, individuals are required to generate particular items in a category within a given time.

Individuals with ADHD appear to have greater deficits with regard to phonemic fluency (weighted ES=.68) than semantic fluency (weighted ES=.38). Deficits in phonemic fluency discriminate children with ADHD from controls better than semantic categories or designs (Barkley et al. 1992; Marzoochi et al. 2008; Mataró et al. 1997; Pineda et al. 1999). One study has reported deficits in both phonemic and semantic categories for children with ADHD (Geurts et al. 2004), whereas 3 report no difference from control groups on either domain of verbal fluency.

Importantly, the common comorbidity of ADHD and reading disability (RD) has been shown to have additive effects for deficits in verbal fluency (Felton et al. 1987). Of the studies reviewed here, 3 excluded or accounted for RD in their analyses (Barkley et al. 1992; Loge et al. 1990; Marzoochi et al. 2008). The average weighted effect size for these studies continued to be large at 1.03, providing evidence that deficits in phonemic fluency in children with ADHD are not limited to the comorbidity with RD.

Review of the pediatric BD literature indicates a small effect for dysfunction in either phonemic (weighted ES=.34) or semantic fluency (weighted ES=.38). Of the two studies that examined fluency thus far, one did not find deficits in either domain of verbal fluency for children with BD versus controls (Voelbel et al. 2006). The other study found significant differences in both domains, but further analysis indicated that medication accounted for the deficits in fluency (Bearden et al. 2007).

Thus, deficits in phonemic verbal fluency appear to be specific to ADHD and may be present over and above comorbidity with RD. Semantic fluency, however, does not appear to be associated with either ADHD or pediatric BD in any strong regard.
Discussion

Although there appear to be some similarities between BD and ADHD in their neuropsychological function, subtle differences in various areas of executive function emerge as specific to each disorder. Overall, evidence exists for differences between neurocognitive profiles of pediatric BD and ADHD in several areas of executive function. The primary differences implicate impairments in interference control, planning, and set-shifting that are specific to BD, and impairments in verbal and spatial working memory and phonemic verbal fluency that are specific to ADHD. Response inhibition does not appear to be distinctive in discriminating BD from ADHD at this time. Thus, evidence for distinctive profiles of executive function in these disorders exists.

There is evidence for a specific pattern of deficits in pediatric BD. While perhaps unexpected, similar to our findings, a meta-analysis conducted by van Mourik et al. (2005) concluded similarly that the Stroop task does not discriminate between neurocognitive profiles of pediatric BD and ADHD in several areas of executive function. The primary differences implicate impairments in interference control, planning, and set-shifting that are specific to BD, and impairments in verbal and spatial working memory and phonemic verbal fluency that are specific to ADHD. Response inhibition does not appear to be distinctive in discriminating BD from ADHD at this time. Thus, evidence for distinctive profiles of executive function in these disorders exists.

The differences in executive functioning in BD and ADHD may worsen during the course of the illness. Findings from the adult ADHD literature indicate that, similar to children, adults with ADHD show difficulty with working memory and phonemic fluency (Jenkins et al. 2004; Thompson et al. 2005), indicating that performance in these facets of executive function may worsen during the course of the illness. Findings from the adult ADHD literature indicate that, similar to children, adults with ADHD show difficulty with working memory and phonemic fluency (Jenkins et al. 2004; Thompson et al. 2005).

The Search for Potential Biomarkers

With the advancement in neuroimaging techniques, evidence now exists to support the hypothesis that the tasks reviewed here elicit activity in the PFC. Interestingly, in areas of executive function for which differences between the two disorders exist, neurophysiological evidence supports the hypothesis that although the PFC is implicated in both BD and ADHD, it functions differently in each disorder. On tasks of interference control and set-shifting, which are impaired in BD but not ADHD, neuroimaging studies have revealed increases in activation of the DLPFC for children with BD (Nelson et al. 2007), which is confirmed in adult studies of BD (Frey et al. 2005; Gruber et al. 2004; Michael et al. 2003). During working memory tasks, which are impaired in ADHD but not BD, the DLPFC and ventral PFC show blunted activation (Kobel et al. 2008). Interestingly, however, during a SWM task, Chang et al. (2004) found that children with BD showed greater activation of the left DLPFC, despite no significant difference in behavioral response. This follows the pattern of over-activation of the DLPFC and other areas of the PFC in BD and underactivation in ADHD during tasks of executive function. As noted earlier in this review, this pattern is found during response inhibition tasks (Passarotti et al. 2009). Thus, despite similar performance deficits on tasks of response inhibition, there are differing abnormalities in underlying neural networks. Thus, differences between BD and ADHD appear both behaviorally and physiologically during tasks of executive function, indicating that they are indeed separate disorders and can be measured objectively as such.

Implications

The differences in executive functioning in BD and ADHD may help to provide discriminant validity between these
two disorders when they appear behaviorally similar in childhood by creating distinct neurocognitive profiles. Despite the encouraging results of this review, more research is needed in neurocognitive function in pediatric BD to confirm the findings. While the understanding of nosology is important, implications for correct differential diagnosis are imperative for clinical reasons. Treatments for these disorders differ radically, with ADHD utilizing stimulant medication (Greenhill et al. 2002) and behavioral parent training (Barkley 2002), and BD utilizing mood-stabilizing medications (McClellan et al. 2007). Improper diagnosis can result in ADHD children being given medications, such as lithium, which are ineffective at treating symptoms of ADHD and have the potential for potent side effects (Giedd 2000). Additionally, a diagnosis of BD at a young age can have stigmatizing results, as it is a lifetime diagnosis with grave implications for functioning across the lifespan, such as hospitalizations, inability to retain employment, increased risk of substance abuse and suicide, and use of multiple medications throughout the lifespan (Birmaher and Axelson 2006; Chen and Dilsaver 1996; Goodwin and Jamison 1990). With additional research in pediatric BD, a future goal would be to bring these findings into the clinical realm to improve quality of care.

Limitations

One limitation of the bipolar literature on executive function is that the majority of studies do not follow individuals through the course of their illness. The adult BD literature indicates the cognitive abilities of individuals with BD can fluctuate with the presence or absence of an affective episode. For instance, impairments in sustained attention and impulsivity can be amplified during manic episodes (e.g., Sax et al. 1995), whereas Wilder-Willis et al. (2001) found that fine motor skills and reaction time remained impaired in a euthymic mood state. A longitudinal study found that individuals had impairment in serial learning on a verbal task while manic, but not when euthymic (Henry et al. 1973). In this same study, however, individuals showed no differences in impairment on short-term free recall in various affective states. No recent studies, however, have been completed using a within-subject design across mood states.

Rather than being viewed as a complication in assessing neurocognitive abilities in BD, these variations in cognitive abilities across mood states may prove to be a marker for differentiation between BD and ADHD. Cognition in ADHD appears to be relatively stable across the lifespan, with the exception of verbal working memory, which may improve later in life (Biederman et al. 1993). Given the number of studies conducted with youth of different ages, it appears that over a short period of time, the neurocognitive profile of ADHD is relatively stable. Thus, a potential method of differentiating between BD and ADHD may be to re-test individuals over a period of time, during which various mood states have the potential to occur and alter the testing results.

With the high rate of comorbidity reported between BD and ADHD in children, it is surprising that only one study was found directly comparing the neurocognitive aspects of these disorders in the absence of comorbidity. BD is often compared with schizophrenia or other severe psychopathology on cognitive tasks, whereas ADHD is most often compared with learning disabilities, oppositional defiant disorder, and conduct disorder. Clearly, if there is extensive comorbidity between these two disorders in youth as some of the literature has reported (Faraone et al. 1997; Geller et al. 2000; Geller et al. 2004; Wozniak et al. 1995), there is a need to examine not only the behavioral symptom presentations of each, but also the neurocognitive aspects as well.

Another limitation of this literature is the paucity of research on cognitive function in pediatric BD (see Tannock 1998). This field is only in its nascent, however, and it is encouraging to see numerous studies that have been published in the past 3 years examining neurocognition in pediatric BD samples. This paper is a preliminary examination of profiles of pediatric BD and further research is needed to confirm the findings here.

Additionally, there is the complicating factor of reading disorder (RD) comorbidity in ADHD. In a study conducted by Pennington et al. (1993), children with only ADHD exhibited deficits in executive function, but not in phonological processing, whereas children with only RD exhibited the opposite profile (deficits in phonological processing, but not executive function). One limitation of the current literature is that many studies did not control for comorbid RD. Several studies reviewed here examined phonemic fluency in ADHD in the absence of RD, with continued evidence for phonemic fluency dysfunction. Thus, while future studies will need to examine the F-A-S task in ADHD while controlling for the effects of a comorbid RD diagnosis, there is rising evidence that this deficit may not be solely explained by RD and may be particular to the ADHD phenotype.

Conclusions

Here we reviewed the literature and propose evidence for distinct neurocognitive profiles for BD and ADHD. Reports of comorbidity of childhood BD with ADHD range from 65–93% (Faraone et al. 1997; Geller et al. 2000, 2004; Wozniak et al. 1995) in some studies, to 6–10% in others.
(Carlson 1998; Duffy et al. 2001; Kutcher et al. 1998; Robertson et al. 2003). Given the similar presentation of BD and ADHD, it is often difficult to distinguish them from a purely clinical standpoint. Although more research needs to be conducted in the area of executive function, especially with regard to pediatric BD, objective cognitive tasks in the context of a distinctive neurocognitive profile may provide insight into subtle differences between BD and ADHD when they are clinically indistinguishable.

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