Is Deprivation-Related ADHD Different from ADHD Among Children Without Histories of Deprivation?

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Abstract

Children reared in the deprived environments of institutions (e.g., orphanages) are at increased risk for problems with inattention and hyperactivity. Although these children have been reported to have higher rates of Attention-Deficit/Hyperactivity Disorder (ADHD) diagnoses than would be expected in the general population, it is unclear if ADHD among post-institutionalized (PI) children is different from that among never institutionalized children. This study directly compared the clinical and symptom profile, executive function task and delay aversion task performances, and event-related potentials of 11- to 15-year old internationally adopted PI children with ADHD (PI-A) to PI children without ADHD (PI-N) and non-adopted children with ADHD (NA-A). PI-As were found to be adopted primarily from Eastern European countries following longer periods of institutionalization than PI-Ns. They performed more poorly on most of the behavioral measures of executive function than PI-Ns; although the performance generally did not differ from NA-As. Relative to NA-As specifically, PI-As reported elevated disinhibited social behavior and, among males, demonstrated more aversion to delay, and had smaller overall N2 amplitude associated with a Go/No-go task. The results indicate that deprivation-related ADHD has an overlapping clinical profile with standard ADHD in addition to a few unique features. This profile associated with early deprived care could be considered a phenocopy of standard ADHD. A description of the deprivation-related ADHD profile has potential implications for identification and treatment of ADHD behaviors among PI youth.

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Many children struggle with difficulties regulating their emotions and behavior as evidenced by the prevalence of children diagnosed with some form of psychopathology. Difficulties related to the self-regulation of attention, hyperactivity, and impulsivity are particularly prevalent as 4.5 million children in the United States ages 3 to17 years carry diagnoses of attention-deficit hyperactivity disorder (ADHD; Bloom & Cohen, 2007). ADHD is considered to be the most prevalent form of psychopathology diagnosed among children, with estimates in the U.S. converging around 6 to 7% (Nigg & Nikolas, 2008) and worldwide of 5.3% (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). Currently, there are three subtypes of ADHD recognized: a Predominantly Inattentive Type, a Predominantly Hyperactive-Impulsive Type, and a Combined Type (American Psychiatric Association, 2000). Inattention involves difficulty maintaining concentration on tasks, difficulty recalling and following through on directions, distractibility, and disorganization (Barkley, 2003; Sagvolden, Johansen, Aase, & Russell, 2005). Hyperactivity/impulsivity includes excessive activity, such as running about or talking excessively, hasty responding, and having difficulty waiting a turn (Biederman, 2005; Sagvolden et al., 2005). The combined type of ADHD includes both of these types of behaviors. This disorder is associated with large social and economic costs (Leibson, Katusic, Barbaresi, Ransom, & O'Brien, 2001) and stress for families (Biederman, 2005). ADHD interferes with many areas of functioning and can impact multiple domains of development. For example, children with ADHD are at risk for academic difficulties (Barbaresi, Katusic, Colligan, Weaver, & Jacobsen, 2007; McGee, Prior, Williams,

Smart, & Sanson, 2002) and difficulties with peer relations (DuPaul, McGoey, Eckert, & VanBrakle, 2001; for review, see Mrug, Hoza, & Gerdes, 2001). ADHD is a multifaceted neurodevelopmental disorder and researchers are just starting to understand the phenotypes and complex etiology of the disorder.

ADHD is considered somewhat of a controversial disorder because, as described by Nigg and Nikolas (2008), appropriate delineation of the corresponding phenotype has been a long-standing concern. For example, motor control problems have been moved in and out of diagnostic criteria despite data continuing to support these deficits in ADHD (e.g., Piek, Pitcher, & Hay, 1999). ADHD has also been found to co-occur with both other externalizing and internalizing disorders (Biederman, 2005). Likewise, there are multiple factors thought to be related to the development of ADHD. In addition to strong genetic underpinnings (Faraone et al., 2005) and environmental risk factors such as exposure to teratogens and perinatal complications (Fryer, Crocker, & Mattson, 2008; Sprich-Buckminster, Biederman, Milberger, Faraone, & Lehman, 1993), early experience, such as that tied to early caregiving environments, has been shown to play a role in the emergence of ADHD and ADHD-like behaviors. An early study by Jacobvitz and Sroufe (1987), for example, demonstrated that within a low socioeconomic sample, maternal intrusion at 6 months of age and maternal overstimulation at 42 months of age predicted hyperactive behaviors in kindergarten. Similar findings were reported within this sample when assessed at older ages, with results noting that maternal insensitivity and overstimulation during infancy predicted both distractible and hyperactive behaviors in the child (Carlson, Jacobvitz, & Sroufe, 1995).

ADHD behaviors have also been linked to the broader context of adversity, such as within studies examining the additive effects of multiple family adversity factors including low socio-economic status, parental psychopathology, marital conflict, and stressful life events (e.g., parent loss of job). A study by Nigg, Nikolas, Friderici, Park, and Zucker (2007), for example, found that higher levels of adversity were related to higher levels of hyperactive/impulsive and inattentive behaviors. Notably, within their study, inattentive behaviors were not independently related to the adversity index when considered along with hyperactive/impulsive behaviors. Beyond this, support for consideration of the caregiving environment as a risk factor in the development of ADHD comes from the emerging literature outlining links between adverse care, like that associated with maltreatment and neglect, and ADHD-related behaviors. For example, a population-based study within the United States suggested that adolescents and young adults who experienced maltreatment were at increased risk of developing ADHD (Ouyang, Fang, Mercy, Perou, & Grosse, 2008). This was true for both abuse and neglect when considering all the subtypes of ADHD. These neglect-related findings closely align with rodent research noting that early maternal separation is associated with increased hyperactivity and impulsivity in later life (e.g., Colorado, Schumake, Conejo, Gonzalez-Pardo, & Gonzalez-Lima, 2006).

Early Deprivation

Consistent with the research outlining the role of adverse caregiving environments in the development of ADHD, children with a history of early deprivation, such as those internationally adopted from institutions (i.e., orphanages overseas) are at increased risk of ADHD symptoms. Indeed, Rutter and colleagues (e.g., Kreppner, O'Connor, & Rutter,

2001) have suggested that inattentive and hyperactive behaviors are part of a "Deprivation Specific Syndrome." While institutionalized, children experience varying degrees of privation which may include malnutrition, receiving inadequate medical care, and the quality of the children's interactions with caregivers is frequently poor (Gunnar, 2001). Because the institutions (e.g., orphanages) are often staffed by caregivers on rotating schedules (Zeanah, Smyke, & Settles, 2006), the children likely fail to form enduring and supportive relationships. Additionally, the institutionalized child may spend much of his time unoccupied and may be left in a crib or placed on the floor to play alone, thus resulting in inadequate motor and cognitive stimulation (D. E. Johnson, 2001). While the quality of care provided varies within institutions around the world, Gunnar (2001) described that institutions failing to provide basic needs of health and nutrition tend to fail on other levels as well, including cognitive, language, and sensorimotor stimulation, and formation of relationships with emotional attachments. Following varying lengths of time, the post-institutionalized (PI) children are adopted out of the depriving conditions of the institutions and enter into middle- to upper-class families; a change in environment that involves significantly more social and physical stimulation (Hellerstedt et al., 2008). Such a significant transition allows for critical examination of the effects of the timing and duration of early deprivation on human development. Research focused on examining this "natural experiment" is indicative of developmental delays across many domains at the time of adoption, with greater time in the institution often associated with poorer outcomes. For example, PI children have been reported to have stunted growth (e.g., D. E. Johnson, 2001; Van Ijzendoorn, Bakermans-Kranenburg, & Juffer, 2007), delayed cognitive and language abilities (e.g.,

Frank & Klass, 1996; Miller, Kiernan, Mathers, & Klein-Gitelman, 1995), and deficient social-emotional functioning (e.g., Rutter et al., 2007; Vorria, Wolkind, Rutter, Pickles, & Hobsbaum, 1998; Zeanah et al., 2009; Zeanah, Smyke, & Dumitrescu, 2002). Following time in the enriching environment of the adoptive home, PI children tend to demonstrate some catch-up across many of these developmental domains (e.g., cognitive: Ames, 1997; Beckett et al., 2006; growth: Van Ijzendoorn et al., 2007); however, difficulties with inattention and hyperactive/impulsive behaviors, such as those related to ADHD, persist for PI children.

Reports of ADHD Behaviors among Children with Histories of Institutionalization

Starting with early studies of children living within institutions in the United States in the 1930s through 1940s, Goldfarb (1943, 1945) observed that institutionalized children demonstrated "restlessness, hyperactivity, and inability to concentrate" (p.19). Children within these institutions likely received basic medical and nutritional care, but were deprived of motor, cognitive, and social stimulation (Provence & Lipton, 1962). However, even children reared within institutions believed to provide higher quality care due to serving as training institutes for nurses were reported to demonstrate poor concentration compared to adopted and community children (Tizard & Rees, 1974). Indeed, it was estimated that these children had been cared for by more than 50 different caregivers when these observations were made (Tizard & Hodges, 1978). These findings are consistent with more recent research suggesting increased levels of hyperactive and inattentive behaviors among children, particularly boys, living within institutions in Greece and the United Kingdom (Roy, Rutter, & Pickles, 2000, 2004; Vorria et al., 1998).

Consistent with these reports of children living within institutions, previously institutionalized children placed into family-based care, including those internationally adopted, have also been reported to demonstrate ADHD-related symptoms of inattention, impulsivity, and hyperactivity (e.g., Goldfarb, 1945; Kreppner et al., 2001; Provence & Lipton, 1962). Indeed, parent, and teacher reports of internationally-adopted post-institutionalized children's behavior indicate that these ADHD-related symptoms are present at the time of adoption and appear to remain years after adoption from institutional care (Maclean, 2003; Stevens et al., 2008). The amount of time spent within the institution prior to adoption is often found to postively correlate with ratings of inattention, distractibility, and hyperactivity/impulsivity (Audet & Le Mare, 2011; Kreppner et al., 2001; Sonuga-Barke et al., 2009). Notably, the English and Romanian Adoption study found that children adopted before 6 months of age from institutional settings exhibited no increase in ADHD symptoms relative to non-institutionalized adopted children, while those adopted later did (Rutter, Kreppner, & O'Connor, 2001). Because age at adoption in this study was largely related to geopolitical events (i.e., fall of communism in Romania) and not child characteristics, this study supports the idea that institutional deprivation induces increases in attention regulatory problems over and above the child's genetic predisposition for such problems. There is some suggestion that adoption at older ages (i.e., over 24 months), irrespective of specific overseas pre-adoptive caregiving environment (e.g., institution, foster care), may be also be a risk factor for developing attention problems (Gunnar, Van Dulmen, & The International Adoption Project Team, 2007). As suggested by Gunnar et al., (2007), it is possible that older age at adoption and PI status have an additive effect in developing

psychopathology. This idea warrants further investigation as others have not found age of placement out of the institution to be related to psychiatric diagnoses even though PI status was associated with presence of disruptive behavioral disorders (Zeanah et al., 2009).

Although the presence of problematic behaviors does not necessarily mean that the children have been diagnosed or demonstrate enough behaviors to warrant a diagnosis, previous research also documents increased prevalence rates of ADHD diagnoses among PI children. Zeanah et al., (2009), for example, reported that 4½- year-old children currently living within a Romanian institution, and those with a history of institutionalization living in foster care, were more likely to meet criteria for a diagnosis of ADHD than never institutionalized children (23%, 19%, 3% respectively). Within samples of PI children from Romania, 15% of 8-year-olds adopted into homes in the Netherlands (Hoksbergen, ter Laak, van Dijkum, Rijk, & Stoutjesdijk, 2003) and 29% of children adopted into Canada 8 or more years prior (as reviewed in Maclean, 2003) met clinical criteria for a diagnosis of ADHD. Similarly, Beverly, McGuinness, and Blanton (2008) reported that 42% of their sample of 9- to 13-year-olds adopted from the former Soviet Union into the United States had an ADHD diagnosis. Among a sample of 8- to 10-year-old PI children adopted from Eastern Europe, a similar rate of ADHD diagnoses (46%) was also reported (Miller, Chan, Tirella, & Perrin, 2009). Although the specific rates of ADHD diagnoses vary across these studies, the prevalence of ADHD among PI children and adolescents is clearly elevated, especially when considered against rates of ADHD diagnoses in the United States (6 to 7%; Nigg & Nikolas, 2008) and worldwide (5.3%; Polanczyk et al., 2007).

Understanding ADHD Behaviors in Post-Institutionalized Children

While ADHD-related behaviors are clearly prevalent among PI children and adolescents, what is not clear is whether the ADHD symptoms in PI children are of a similar nature to ADHD diagnosed in children without histories of deprivation. ADHD among typically developing children is a heterogeneous neurodevelopmental disorder. The symptom presentation and/or specific deficit(s) associated with the diagnosis may vary from one child to the next. This is true even within a group of children diagnosed with the same DSM-IV-TR subtype (i.e., hyperactive/impulsive vs. inattentive vs. combined). Interestingly, few studies exploring ADHD-symptomatology among PI children explicitly examined the different subtypes of ADHD in their analyses (e.g., Ames, 1997; Mainemer, Gilman, & Ames, 1998), although Roy and Rutter (2006) suggest that inattentive behaviors may make up the majority of the inattentive/overactive profile documented among their sample of children adopted from Romania. As reported by Sonuga-Barke (2003), the refined description of the diagnostic criteria included with the DSM-IV spurred research regarding the underlying neurobiological and neuropsychological processes corresponding to ADHD. There are currently two well-documented models regarding the primary deficit or "neuropsychological marker" (Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008, p. 367) within ADHD: a deficit in executive function and a deficit in reward sensitivity.

ADHD: Deficit in Executive Function

A common understanding of ADHD is one which includes a description of it as a disorder of executive functions (EF), often specific to a deficit in inhibition. Inhibition is thought to reflect a primary EF, defined as "one's ability to deliberately inhibit dominant,

automatic, or prepotent responses when necessary" (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000, p. 57). Barkley (1997, 2003) defined the primary deficit in ADHD as behavioral inhibition. In this model, behavioral inhibition refers to the interrelated processes of inhibiting a prepotent response, stopping an ongoing response, and interference control. A deficiency in behavioral inhibition is then associated with secondary impairments in working memory, self-regulation, internalization of speech (i.e., reflection, self-questioning), and reconstitution (i.e., analysis, synthesis, creative thinking) as behavioral inhibition "provides the delay necessary for them to occur" (Barkley, 1997, p. 68). Similarly, Pennington and Ozonoff (1996) described a cognitive model linking EF deficits to ADHD based on historical research noting that prefrontal lesion patients demonstrated the behavioral symptoms of the disorder. Their meta-analysis examining EF task performance found that participants with ADHD consistently demonstrated poorer performance on vigilance/perceptual speed tasks and measures related to motor inhibition, planning (e.g., Tower of Hanoi), and conflict (i.e., Stroop; Pennington & Ozonoff, 1996). They concluded that children with ADHD may have a mix of deficits including a core deficit in EF related to motor inhibition. An updated meta-analysis of studies comparing groups with and without ADHD diagnoses yielded the strongest effect sizes for measures of response inhibition (i.e., stop-signal task, continuous performance test commission errors), vigilance (i.e., continuous performance task omission errors), spatial working memory (i.e., CANTAB spatial working memory, self-ordered pointing) and some aspects of planning (e.g., Tower of Hanoi, Tower of London) thus drawing further support for an EF deficit within ADHD (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). The EF weaknesses demonstrated by the ADHD

groups were not accounted for differences in intelligence, academic achievement, or symptoms of comorbid disorders thus emphasizing that EF difficulties may be a specific component of ADHD (Willcutt et al., 2005). Indeed, as Willcutt et al., point out, although EF weaknesses are an important component of ADHD, they "are neither necessary nor sufficient to cause all cases of ADHD" (p. 1336).

The models outlining ADHD as arising from EF deficits are rooted in the understanding of the prefrontal cortex, the functioning of which is often linked to the symptoms of ADHD. Support for the description of frontal lobe dysfunction in ADHD was originally based on lesion studies noting that frontal lobe lesions sometimes elicit hyperactivity, distractibility, and/or impulsivity, and deficits on EF tasks (Pennington & Ozonoff, 1996; Willcutt et al., 2005). Correspondingly, the prefrontal cortex is part of the neural circuitry that supports EF. Damage to the dorsolateral prefrontal cortex (DL-PFC) has been specifically linked to deficits in the more purely cognitive, "cool" aspects of EF such as those demonstrated in ADHD (as opposed to affective or "hot" EF; Zelazo & Muller, 2002). Neuroimaging research utilizing metabolic procedures (e.g., MRI, positron emission tomography) similarly supports the involvement of prefrontal regions in the circuitry associated with the disorder. For example, a meta-analysis of neuroimaging studies (i.e., functional magnetic imaging and positron emission tomography) comparing participants of all ages with and without ADHD diagnoses yielded hypoactivity in frontostriatal and frontoparietal circuits (Dickstein, Bannon, Castellanos, & Milham, 2006). Functional magnetic resonance imaging (fMRI) studies specifically involving children and adolescents similarly noted reduced frontostriatal activity and poorer behavioral performance on cognitive control (i.e., inhibitory control

[Go/No-go]) tasks (Durston et al., 2003; Schulz et al., 2004; Vaidya et al., 1998). Compatibly, structural MRI studies point to cerebral reductions in volume particularly within frontostriatal and cerebellar regions associated with ADHD (see reviews Durston, 2003; Valera, Faraone, Murray, & Seidman, 2007).

Other types of neuroimaging methods including electrophysiological procedures, such as recording of electroencephalogram (EEG) and the event-related potential (ERP), have also been utilized to distinguish ADHD samples from controls and to assist in understanding the EF deficits within ADHD. Both EEG and ERP provide information about electrical activity of the brain. EEG is thought to reflect the background state of the brain and ERP is the activity time-locked to a presented event (Nelson & Bloom, 1997). Typically developing children demonstrate a developmental change in EEG power including a decrease in relative low frequency slow wave activity (theta) and a corresponding increase in higher frequency (alpha) activity (Marshall, Bar-Haim, & Fox, 2002) whereas children with ADHD demonstrate resting EEG patterns characterized by increased relative theta power, particularly in frontal regions, and decreased relative alpha power (Barry, Clarke, & Johnstone, 2003). ERP studies involving individuals with ADHD have often focused on collecting data during tasks assessing inhibitory control in order to examine the N2 component which is associated with cognitive control, including inhibitory control, stimulus discrimination, and categorization (Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003) and the P300, which is thought to reflect stimulus relevance and cognitive processes including inhibitory control and stimulus evaluation (Tekok-Kilic, Shucard, & Shucard, 2001). Results from studies utilizing a variant of a continuous performance task known as the Go/No-go task (Bezdjian, Baker,

Lozano, & Raine, 2009) provide some evidence for decreased a N2 component in children with ADHD (Johnstone, Barry, Markovska, Dimoska, & Clarke, 2009; Johnstone & Clarke, 2009; Yong-Liang et al., 2000). Differences in amplitude for the P300 collected during continuous performance tasks (i.e., Go/No-go) have also been noted within ADHD samples (Fallgatter et al., 2004; Overtoom et al., 1998; Spronk, Jonkman, & Kemner, 2008). Similar results suggesting atypical activation of inhibition-related ERP components of the N2 and P300 have been noted for the stop-signal task as well (Dimoska, Johnstone, Barry, & Clarke, 2003; Johnstone, Barry, & Clarke, 2007; Kenemans et al., 2005; Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005; Overtoom et al., 2002). Taken together, the EEG and ERP studies provide further evidence for differential neural processing for children with ADHD. The ERP studies, when combined with evidence from early lesion studies and other neuroimaging studies (e.g., fMRI), continue to suggest a deficit in the prefrontal inhibitory control mechanism in children and adolescents with ADHD.

Evidence for EF Deficits Related to Early Deprivation

While the prevalence of ADHD symptoms has been examined in PI children, researchers have only recently begun to use behavioral neuroscience methods to explore EF, executive attention, and other regulatory behaviors in relation to early deprivation. For example, PI children adopted as toddlers from Russian institutions were more likely to have scores suggestive of executive functioning deficits in the home environment on a standardized parent-report measure than were children adopted earlier (Merz & McCall, 2010). Colvert and colleagues (2008) found that 11-year-old PI children adopted from Romanian institutions into the UK made significantly more errors on the Stroop

(color/word) task than both non-institutionalized Romanian adopted children and within-UK adoptees. Additionally, the number of Stroop task errors was positively correlated with duration of institutionalization among the PI group. Among all of the Romanian adoptees, those adopted later (older than 6 months of age) performed more poorly than within-UK adoptees. As noted above, Pennington and Ozonoff (1996) found that participants with ADHD consistently performed poorly on the Stroop. Within the same Romanian-born PI sample, Stevens et al., (2008) reported a significant association between parent and teacher report of inattention/overactivity and deficient Stroop task performance. Based on their results, Stevens and colleagues (2008) concluded that deprivation related inattention/overactivity shares many of the same features of standard ADHD and hypothesized further that similarities may also exist at the neural level perhaps due to common dopamine-modulated networks (Stevens et al., 2008).

Within a more diverse sample of internationally-adopted 8- to 10-year-olds, Pollak et al., (2010) found that PI children performed more poorly than both a group of children internationally-adopted early and a group of non-adopted community children on the Cambridge Neuropsychological Test and Automated Battery (CANTAB) Spatial Working Memory task. Consistent with this, Bos, Fox, Zeanah and Nelson (2009) found that Romanian children with histories of institutionalization performed more poorly than Romanian community controls on the same task. The CANTAB Spatial Working task is considered a sensitive measure of frontal lobe dysfunction (Owen, Downes, Sahakian, Polkey, & Robbins, 1990; Owen, Morris, Sahakian, Polkey, & Robbins, 1996; Owen, Sahakian, Semple, Polkey, & Robbins, 1995) and similar spatial working memory tasks have been found to distinguish groups with and without ADHD diagnoses (Willcutt et al.,

2005). Notably, both Pollak et al. (2010) and Bos et al. (2009) did not find group differences on the CANTAB Stockings of Cambridge task, which is a version of the Tower of London planning task, another task found to have strong effect sizes in studies of ADHD (Willcutt et al., 2005). Therefore, these studies suggest PI children demonstrate similar, although not identical, deficits as non-institutionalized children with ADHD.

The behavioral performance of PI children has also been investigated on tasks related to two other factors related to ADHD: response inhibition and vigilance. Bruce, Tarullo, and Gunnar (2009), for example, evaluated the performance of 6- and 7-year-old PI children on a letter Go/No-go task, in addition to forced choice visual discrimination and delay of gratification tasks. When scores from these tasks were averaged into a composite with the temperament constructs of effortful control and surgency, the results revealed that PI children scored significantly lower on this composite than both children internationally adopted from foster care and community controls (Bruce, Tarullo et al., 2009). These results provide preliminary evidence for deficits in response inhibition and sustained attention in PI children similar to what is noted for ADHD samples; however, interpretation is limited due to the use of the composite variable. More recently, a study of attention and inhibitory control by McDermott, Westerlund, Zeanah, Nelson, and Fox (2012) examined 8-year-old Romanian institutionalized children placed in foster care versus those who remained in institutional "care as usual," and those never institutionalized (i.e., the Bucharest Early Intervention Project or BEIP). The children with histories of institutionalization placed into foster care did not differ from the never institutionalized group in their behavioral performance on a Go/No-go task. Instead,

those who remained in institutional care as usual were found to demonstrate more "go" errors (omission errors) and to have slower reaction times than the other two groups. Typically, ADHD samples demonstrate increased levels of both omission and commission errors (e.g., Sartory, Heine, Müller, & Elvermann-Hallner, 2002); therefore, there is some evidence to suggest that continuous institutionalization may be associated with some, but not all of the behavioral deficits seen with ADHD samples. When McDermott et al. (2012) evaluated differences in the brain activity during the Go/No-go task, both groups of children with histories of institutionalization were found to have smaller P300 ERP components than the never institutionalized group. As noted above, there is some evidence supporting differences in P300 amplitude for ADHD samples (e.g., Fallgatter et al., 2004). Thus, although the children with histories of institutionalization did not demonstrate behavioral differences specifically suggesting deficits in inhibitory control, their brain activity is suggestive of differential processing that may be similar to that seen in ADHD. There is also emerging evidence of different neural processing for PI children as assessed by the other electrophysiological procedure, EEG. Marshall, Fox, and the BEIP Core Group (2004), for example reported that Romanian children with histories of institutionalization showed increased relative theta power in frontal, parietal, and occipital regions and decreased relative alpha power overall compared to community controls. Notably, this pattern of relative power for both theta and alpha is the same as that demonstrated for children with ADHD (Barry et al., 2003). Similar findings were reported in a sample of PI children assessed at 18-months-old (Tarullo, 2008); however, when assessed at age 5.5 years, only increased relative theta power was found as alpha power did not vary relative to non-adopted children

(Wiik, 2009). Notably, a recent BEIP study (Vanderwert, Marshall, Nelson, Zeanah, & Fox, 2010) similarly reported that 8-year-olds removed from institutions before 24-months of age and placed into foster care demonstrated alpha activity similar to never institutionalized children.

In terms of metabolic neuroimaging measures, there are few studies involving MRI and positron emission tomography (PET) among PI samples, and even fewer that correspond to the ADHD literature related to EF deficits. For example, a structural imaging study by Bauer, Hanson, Pierson, Davidson, & Pollak (2009) found that similar to the reductions in cerebellar volume reported among ADHD samples, PI children adopted from orphanages around the world had smaller volume of the superior-posterior cerebellar lobes than similarly-aged non-adopted children. Other neuroimaging studies are only suggestive of differential development of frontal regions among PI children similar to that found in ADHD samples. A small PET study of Romanian PI children revealed that when PI children were compared to healthy adults and children with chronic epilepsy, PI children had decreased glucose metabolism in the prefrontal cortex, including the orbital frontal cortex in addition to medial temporal structures, including amygdala and hippocampus, and the lateral temporal cortex (Chugani et al., 2001). Results from a diffusion tensor imaging study indicated that relative to healthy control children, PI children adopted from Eastern Europe had reduced white matter tracts in the pathway between the anterior temporal lobe and the frontal lobe (Eluvathingal et al., 2006). While these results suggest that PI children have different brain development relative to their non-adopted peers, clearly more research focusing on understanding the neurobiological development of PI children is needed.

ADHD: Deficit in Reward Processing

Despite the prevalent description of ADHD as a disorder characterized by deficits in executive functioning, complementary accounts of ADHD describe the disorder as a deficit in reward processing and motivation. Within these models, the core deficit of ADHD is believed to be an unusual sensitivity to reinforcement (Luman, Oosterlann, & Sergeant, 2005) or correspondingly, suboptimal reward processing (Sonuga-Barke, 2005). Douglas and Parry (1983) were some of the first to identify an altered response to delayed outcomes as being a relevant factor in ADHD. In their study, children with ADHD symptoms performed more poorly on tasks when inconsistent, as opposed to continuous, rewards were given and were less likely to maintain their improved performance relative to baseline once rewards were removed (Douglas & Parry, 1983). More recently, Sonuga-Barke (1994, 2002, 2005) proposed a model for ADHD known as Delay Aversion hypothesizing that a child with ADHD fails to engage effectively in delay-rich environments and thereby has a negative emotional reaction to delay. ADHD behaviors are therefore explained by the core function of the escape or avoidance of delay (Sonuga-Barke, 1994). This avoidance comes from the pattern of negative affect established during development as the already impulsive child learns to associate delay settings with failure (Sonuga-Barke, 2005). When placed in situations when delay is imposed, particularly when it is unexpected, the child will then express inattention and increased activity (Sonuga-Barke, 2005; Sonuga-Barke et al., 2008). Similarly, Sagvolden and colleagues (Sagvolden, Aase, Zeiner, & Berger, 1998; Sagvolden et al., 2005) emphasized that behavioral symptoms of impulsiveness, hyperactivity in novel situations, and delay aversion develop due to mesolimbic dopaminergic dysfunction

producing altered reinforcement of behavior and deficient extinction of previously reinforced behavior. For children with ADHD, the time available for associating behavior with its consequences is shorter than it is for those without ADHD (Sagvolden et al., 2005). The model proposed by Sagvolden and colleagues (2005) places more weight on underlying neural signaling in order to understand the behaviors seen in ADHD. Attentional, executive, and motor deficiencies, for example, are viewed as products of the interaction with other hypofunctioning dopaminergic systems (mesocortical and nigrostriatal, respectively; (Johansen, Aase, Meyer, & Sagvolden, 2002). Indeed, as this model suggests, dopamine is a key modulator in sending signals regarding incentive value and in regulating related behavioral processes within the neural circuitry associated with reward (Wightman & Robinson, 2002).

Experimental evidence in support of this reward-related account of ADHD comes from multiple studies noting that when given the choice, children with ADHD consistently demonstrate a preference for smaller, sooner rewards over larger, later rewards (Antrop et al., 2006; Bitsakou, Psychogiou, Thompson, & Sonuga-Barke, 2009; L. Dalen, Sonuga-Barke, Hall, & Remington, 2004; Luman et al., 2005; Marco et al., 2009; Solanto et al., 2001). The tasks often used in these studies were a Choice-Delay Task (Sonuga-Barke, Taylor, Sembi, & Smith, 1992) or the Maudsley Index of Delay Aversion (Kuntsi, Oosterlaan, & Stevenson, 2001), both of which require deciding between receiving 1 point after a very short (e.g., 2 second) delay or 2 points after a longer delay (e.g., 30 seconds). These tasks have been found to satisfactorily distinguish individuals with ADHD from those without (Sonuga-Barke et al., 2008). Additionally, teacher ratings of hyperactivity and impulsivity correlated with delay aversion as

assessed by the Choice-Delay Task (Solanto et al., 2001). Accordingly, the children demonstrate an unconditional preference for immediate over delayed rewards, even when the sooner reward is smaller (Sonuga-Barke, 2005; Sonuga-Barke et al., 2008). Children with ADHD have also been found to demonstrate negative performance in the face of imposed delay (Sonuga-Barke, Bitsakou, & Thompson, 2010). Accordingly, children with ADHD have been found to demonstrate slower reaction times on trials with longer time windows between stimuli on the Delay Reaction Time task (Bitsakou et al., 2009).

These reward-related accounts of ADHD are grounded in the understanding of frontostriatal reward circuitry, particularly the ventral striatum (e.g., nucleus accumbens) and the orbitofrontal cortex, (Cardinal, 2001; see review Sonuga-Barke et al., 2008) which are supported by neuroimaging research. In an fMRI study, for example, Scheres, Milham, Knutson, and Castellanos (2007) found that adolescents with ADHD had reduced ventral striatal activation during reward anticipation relative to controls. The amygdala is also involved in this reward system. Within a study of adults, Plichta et al. (2009) found that those with ADHD demonstrated hyperactivation of the amygdala and dorsal caudate nucleus to delayed rewards in addition to hypoactivation in the ventral striatum for immediate and delayed rewards. Castellanos (2009) commented that these results are in line with delay aversion because the negative reaction associated with delayed rewards would be reflected by the amygdala. A recent study by Lemiere et al. (2012) directly investigating the neurobiology of delay aversion confirmed hyperactivation of the amygdala, insula, ventral striatum, and orbitofrontal cortex in ADHD participants on trials when delay was unavoidable relative to those when delay could be avoided. In terms of brain structure, children with ADHD have been shown to

have increased hippocampal volume and less of a correlation between amygdala and orbitofrontal cortex volume thus suggesting the connectivity between these regions is disrupted (Plessen et al., 2006). A speculative interpretation of the larger hippocampal volume in ADHD samples is that it is perhaps related to the strategy of increased stimulus-seeking behaviors ADHD children use while experiencing a delay (Plessen et al., 2006).

Finally, evidence from ERP collected in children with ADHD on a reward-related task is also suggestive of differential processing. Holroyd, Baker, Kerns, and Muller (2008) compared the feedback Error-Related Negativity (ERN) recorded from 8- to 13-year-old boys with and without ADHD. Feedback ERN is described as being generated when there is a failure to receive an expected reward. Results indicated that the feedback ERN was larger for ADHD children than control children, but only after they received a bonus payment midway through the experiment. In line with the delay aversion model, the results were interpreted as children with ADHD being more sensitive to temporal proximity of reward and requiring the salience of an actual reward (Holroyd et al., 2008). Van Meel, Oosterlaan, Heslenfeld, and Sergeant (2005) found similar results of a larger feedback ERN for negative feedback and consequent losses for similarly aged children with ADHD. Interestingly, more recently van Meel, Heslenfeld, Oosterlaan, Luman, and Sergeant (2011) found that ADHD children did not demonstrate a feedback ERN when receiving performance related reward or punishment whereas the control children did. The authors propose that the seemingly contradictory results are likely reflective of differing task demands across the studies (van Meel et al., 2011). Overall, the results

across these studies are supportive of differential processing with regard to rewards in children with ADHD relative to controls.

Evidence for Differential Reward Processing Related to Early Deprivation

Although no studies to date have directly looked at the performance of PI children on delay aversion tasks, there is some initial evidence and suggestion of differential reward processing as a result of early deprivation. Gatzke-Kopp (2011), for example, proposed that the mesolimbic dopamine system may be particularly sensitive to environmental adversity during development. More directly, Fries (2008) evaluated 6-year-old PI children's performance on an associative reward learning task wherein the children received a reward after correct performance. The children completed two versions of the task on separate days: one with specific reward schedules including visual cues indicating whether the reward was one or several trials away and a random version wherein cues no longer signified when the reward would be given. Results indicated that the reaction times of the PI children were unrelated to distance to the reward, unlike those of the comparison children, which decreased as the reward become closer. This indicated that the PI children failed to learn the relationship between the visual cues and their motivational significance. Interestingly, unlike comparison children, PI children were slower on trials within the random condition when cues were not predictive of reward. This finding is reminiscent of Douglas and Parry's (1983) finding of poorer performance among ADHD children when rewards were administered inconsistently.

A few neuroimaging studies have been completed to date exploring brain function and structure related to reward processing. When PI adolescents completed a task wherein cues signal the possibility of winning a reward, they did not differ in behavioral

performance or response time from non-adopted, community adolescents; however, the PI adolescents failed to demonstrate activation of ventral striatum during reward anticipation suggesting a lack of sensitivity to reward (Mehta et al., 2010). In a separate neuroimaging study, this same group examined structural differences of other brain regions implicated in the delay aversion model of ADHD: the amygdala and hippocampus (Mehta et al., 2009). After correcting for differences in total grey and white matter, the PI adolescents had larger amygdala volumes, although no differences were found for hippocampal volume. As suggested by Mehta et al. (2009), larger amygdala volume is likely related to disturbed affective processing for the PI adolescents. Interpreting these findings of a lack of ventral striatal activation and a larger amygdala with regard to delay aversion is difficult given the neural underpinnings are just starting to be explored in ADHD populations. However, taken together with evidence of involvement of both the amygdala and ventral striatum in reward circuitry, these results are at least suggestive of altered reward-related behavior in children and adolescents with a history of early deprivation.

Summary

Overall, research to date is suggestive of similar patterns of behavior in PI children as are found in non-institutionalized children with ADHD although the exact pattern of deficits in EF, reward processing, and the supporting neural circuitry may be slightly different. Importantly, however, these studies have reached these conclusions by comparing PI children to non-adopted children. Therefore, direct comparison of PI children both with and without ADHD diagnoses to never institutionalized, non-adopted children with ADHD is needed to understand whether deprivation-related ADHD in PI

children is different from ADHD in children without histories of deprivation. Indeed, only one study to date has included such a direct comparison (e.g., Sonuga-Barke & Rubia, 2008). Speficially, a small sample of Romanian PI children with a history of inattentive and overactive symptoms (PI-IO), Romanian PI comparison children, and children with a standard clinical diagnosis of ADHD completed three executive function tasks, which contributed to a combined inhibition score. Their parents completed ratings of ADHD symptoms. The results indicated that both male and female PI-IO children demonstrated more neuropsychological impairment than the comparison groups, although the symptom profile varied by gender. Males displayed similar symptom profiles to non-institutionalized children with ADHD whereas females were more similar to the PI comparison children. These results suggest that ADHD in PI-IO children may in fact be dissimilar to the symptom and behavioral profile demonstrated by non-institutionalized children with ADHD (Sonuga-Barke & Rubia, 2008).

Not all studies of PI children and adolescents have documented higher instances of inattention and hyperactivity in males. In fact, when it was evaluated within individual studies, the results across the literature were quite mixed. Some studies of PI children found that the ADHD behaviors were more prevalent in males (Roy et al., 2004; Vorria et al., 1998; Wiik et al., 2011); whereas others did not find differences in the behaviors between males and females (Gunnar et al., 2007; Kreppner et al., 2001; Stevens et al., 2008). Assessment of gender differences in deprivation-related ADHD relative to ADHD among non-adopted children warrants further investigation, particularly as prevalence studies report that ADHD in non-adopted children is generally more common in males than females (Biederman, 2005). Notably, research examining the biological

and neuropsychological correlates of ADHD suggests that the same construct is present in males and females, despite the reported gender differences in prevalence (Nigg & Nikolas, 2008). Whether or not deprivation-related ADHD is associated with similar gender distributions in symptom prevalence and neuropsychological functioning would further contribute to understanding ADHD among PI children and adolescents and help identify whether certain PI children are at higher risk for demonstrating the increased inattentive and hyperactive/impulsive behaviors.

Replicating previous PI findings and expanding upon the study by Sonuga-Barke and Rubia (2008) will assist in developing an understanding of how and if deprivation-related ADHD in PI children differs from ADHD displayed in children without histories of deprivation. Such findings would ultimately inform best clinical practices, both for identification and treatment. Additionally, one cannot consider deprivation-related ADHD symptoms as completed isolated from other developmental patterns PI children exhibit. For example, in addition to consistently demonstrating difficulty with hyperactivity and inattention, children with histories of institutional rearing show disinhibited social behavior years after adoption. These behavioral patterns in PI adolescents are often highly correlated in studies of PI children (e.g., Bruce, Tarullo et al., 2009; Kreppner et al., 2001; Rutter et al., 2001; Stevens et al., 2008) and are hypothesized to be interrelated, although the etiology and the neural underpinnings remain to be fully understood. Therefore, it is possible that the disinhibited social behavior may be a distinct feature of the ADHD symptom presentation demonstrated by PI children. Moreover, professionals implementing interventions for deprivation-related

ADHD will benefit from understanding a more complete profile of post-institutionalized children including their unique needs and behaviors.

Previous Empirical Studies

Behavioral and Emotional Functioning in Internationally-Adopted, Post-Institutionalized Children

Our group has previously examined behavioral and emotional symptoms in post-institutionalized children during middle childhood, a developmental period not well represented in the PI literature (Wiik et al., 2011). The goal was to confirm the presence of ADHD-related symptoms (i.e., inattention and hyperactivity) and to explore internalizing and externalizing symptoms within a sample of children adopted from a range of countries as the majority of the literature has focused on children internationally adopted from Eastern Europe. The middle childhood developmental period was not well represented in the PI literature at the time (e.g., M. Dalen & Rygvold, 2006; Hoksbergen et al., 2003) and focusing on this age allowed for consideration of whether symptoms remain years after adoption into enriching environments. Because adoptees as a group have been found to be at higher risk for parent-reported mental health risks (Keyes, Sharma, Elkins, Iacono, & McGue, 2008), most prior research on mental health symptoms in PI children relied on parent report (Juffer & van Ijzendoorn, 2005), and proper assessments of emotional and behavioral functioning are best appraised by self-report (Rey, Schrader, & Morris-Yates, 1992), this study included self-report of symptoms in addition to parent-report. Problem behaviors in PI children may not only be due to institutional care, but could be related to risk factors associated with international adoption in general (e.g., poor prenatal care, prenatal exposure to substances, and mental

health problems among parents who relinquish or abandon their children; D. E. Johnson, 2000). Few studies to date have included the necessary group to identify effects specific to histories of institutionalization. Therefore, we included a second group of children internationally adopted primarily from foster care in order to account for risk factors associated with international adoption in general. To this end, we expected that PI children were expected to exhibit greater ADHD symptoms than other internationally adopted and non-adopted children. Externalizing problems were hypothesized to be elevated for both internationally adopted groups and no firm predictions were made for internalizing concerns beyond expecting that the self-report would be more sensitive to these symptoms.

Methods. Post-institutionalized (PI) internationally adopted children were compared to children internationally adopted early from foster care (FC) and non-adopted children residing with their families of origin. PI children were adopted from Eastern Europe, Asia, and South America at a mean age of 28.6 months (*SD* = 18.1) and had been with their families an average of 7.3 years (*SD* = 2) at the time of assessment. FC children were adopted from Asia and South America at a mean age of 5.1 months (*SD* = 1.6) and had been with their families an average of 9.3 years (*SD* = 1.1years) at the time of assessment. Children were on average 9-years-old (range 8-11 years) at the time of testing. The mental health symptomatology section of the HBQ (Boyce et al., 2002; Essex et al., 2002) was completed by the child participants (HBQ-C). Children were asked to choose which of two opposing statements is most like them (e.g., 'I'm not a sad kid' vs. 'I am a sad kid') and then indicate whether that statement is 'sort of', 'mostly' or 'really' like them. Responses were coded on a 6-point scale based on which statement

(positive or negative) is endorsed and to what degree, with 1 representing the most positive and 6 representing the most negative. One parent report (HBQ-P) was obtained for each child, with the majority completed by mothers. The measure was administered in questionnaire format and assessed symptoms on a 0 ('never or not true') to 2 ('often or very true') scale. In addition to mean symptom level, the percentage of children above clinical cutoffs was examined for the domains of ADHD, externalizing, and internalizing

Results. PI children were reported by their parents to demonstrate a higher level of ADHD symptoms than both the FC and non-adopted children; however, according to child self-report, the PI children and FC children had higher symptom levels than NA children. Notably, both parent-report and self-report were indicative of a higher percentage of PI children above the clinical cutoff for ADHD symptoms than the other two groups. See Table 1 for a summary of group means. Both groups of internationally adopted children had higher levels of externalizing symptoms relative to non-adopted children, with parent report indicating higher numbers of internationally adopted children above the externalizing clinical threshold. Although the pattern was similar for child report of externalizing symptoms, there were no differences in group percentages over the clinical cut-off. With regard to internalizing symptoms, both internationally adopted groups had increased internalizing symptom levels and greater numbers above the clinical threshold relative to the non-adopted children based on parent-report; however, the child report was indicative of this being true only for the PI group.

Discussion. Consistent with prior research, 8- to 11-year-old PI children with prolonged institutional histories were at particular risk for demonstrating ADHD symptoms. While approximately 23% of PI children by parent report and 20% by child

report experienced clinically significant ADHD symptoms, the percentage of EA/FC children above clinical cutoff for ADHD was similar to that of NA children, suggesting that increased ADHD symptoms are specifically associated with a history of institutionalization. In contrast, risks for externalizing problems do not appear to be sensitive to early institutional history, but are instead shared with children internationally adopted early from foster care. For internalizing problems, whether or not PI children were particularly vulnerable depended on reporter. These mixed findings argue for enhanced attention to PI children's views of their struggles to manage their emotional concerns. The results overall are supportive of previous PI literature suggesting that ADHD symptoms specifically, in addition to externalizing and internalizing problems, are not specific to the time immediately following the move to an enriched environment and instead persist for years following adoption from institutions.

Executive Attention and Brain Activity in Post-Institutionalized Children

A second study was designed to explore the specific nature of attention and regulatory problems in PI children and assist in understanding the neurobiological correlates of attention problems that follow early institutional deprivation (Loman et al., in press). Because results from other PI studies (e.g., Bruce, Tarullo, et al., 2009; Pollak et al., 2010) suggested that the PI attentional difficulties may be related to difficulties with inhibitory control and selective attention, we chose to focus on executive attention, a multi-faceted construct involving inhibitory control, response monitoring, and conflict resolution (Rueda, Posner, & Rothbart, 2005). Specifically, we examined behavioral performance and event-related potentials (ERPs) during Go/No-go and Flanker tasks. Go/No-go assesses inhibitory control and is associated with activation of the ventrolateral

prefrontal cortex and anterior cingulate cortex (e.g., Durston et al., 2002; Schulz et al., 2004). Flanker, a measure of selective attention and conflict monitoring, is associated with activation of the dorsolateral prefrontal cortex and anterior cingulate cortex (e.g., Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Casey et al., 2000). Our ERP analyses focused on five components: P2, N2, P300, error-related negativity (ERN), and Pe. As described previously, N2 is associated with inhibitory control and stimulus discrimination (Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003). P300 is associated with stimulus relevance, inhibitory control, and stimulus evaluation (Tekok-Kilic, Shucard, & Shucard, 2001). P2 is thought to reflect a sensory-driven response to visual stimuli and the matching of visual perception to cognitive expectation (Fabiani, Gratton, & Coles, 2000). ERN has been described to reflect initial response monitoring immediately following an incorrect response (van Veen & Carter, 2002) while Pe has been reported to represent further error processing and awareness (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005).

We hypothesized that PI children would be more likely to demonstrate poorer inhibitory control and conflict monitoring than peers without extensive histories of institutionalization. Two comparison groups were included: non-adopted children raised in similarly-resourced families as those who adopt internationally (Loman, Wiik, Frenn, Pollak, & Gunnar, 2009) and children internationally adopted at earlier ages primarily from foster care to account for factors associated with international adoption (e.g., poverty, poor prenatal care; D. E. Johnson, 2000). Although this was the first study to explore ERPs associated with two types of attentional control tasks within the same sample of post-institutionalized children, previous ERP research (e.g., face processing,

Moulson, Westerlund, Fox, Zeanah, & Nelson, 2009; inhibitory control, McDermott et al., 2012) has suggested that post-institutionalized children may have smaller amplitudes and slower latencies for ERP waveforms.

Methods. Post-institutionalized children (PI) internationally adopted at 12-78 months of age having spent at least 75% of their pre-adoptive lives in institutional care were compared to post-foster care (PF) internationally adopted at 2-8 months of age predominantly from foster care and non-adopted children (NA) born and raised in their biological families in the United States. The children were approximately 10 years old at the time of testing. Go/No-go was a computerized task requiring participants to press a button as quickly and accurately as possible for each letter presented (Go trials) except the letter X (No-go trials). Flanker was a computerized task during which the children were presented with a row of five arrows and they had to indicate the direction of the middle arrow (right or left) as quickly and accurately as possible. The middle arrow was flanked by arrows pointing in either the same direction (congruent, <<<<<) or the opposite direction (incongruent, >><>>). ERPs were recorded during the two tasks using a 32-channel Electro-Cap (Electro-Cap International, Inc., Eaton, Ohio).

Results. Results from behavioral measures suggested problems with sustained attention. Contrary to hypotheses, PIs did not demonstrate increased false alarms on No-go trials, but instead made more omission errors (i.e., less accurate on Go trials) on the Go/No-go task than NAs (see Figure 1). Similarly, PIs did not demonstrate deficits only on incongruent trials on the Flanker task, but had lower *overall* accuracy than NAs. PFs and NAs did not differ on either task. Results from ERPs suggested differences in inhibitory control and error monitoring. PIs demonstrated a smaller N2 (less negative)

amplitude than NAs and PFs did not differ from NAs. There was an interaction found for N2 latency such that for females, PIs and PFs did not show the difference in latency by condition (Go later than No-go) demonstrated by NAs. For the P2 component, PIs and PFs both had smaller amplitudes for Go trials than for No-go trials whereas the amplitudes did not differ for NAs. PIs also had a smaller ERN amplitude (less negative) associated with the flanker task compared to NAs.

Discussion. These findings suggested that attention regulatory problems consistently reported for PI children may reflect overall difficulties sustaining attention, rather than specifically inhibitory control or selective attention. PI children demonstrated more omission errors on Go/No-go than NA children, but there was no group difference in commission errors. Omission errors reflect deficits in sustained attention whereas false alarm or commission errors reflect response inhibition (e.g., Willcutt et al., 2005). Group differences noted in P2, a stimulus identification component, augmented the behavioral findings suggesting PIs may struggle with sustaining attention to the tasks. PIs' smaller ERN amplitudes similarly mapped onto the behavioral results, indicating decreased error monitoring, and suggesting that perhaps they are not attending well enough to realize an error was made. Despite **not** demonstrating more false alarms behaviorally on the Go/No-go task, PIs did demonstrate differential brain activity (smaller amplitude) associated with response inhibition-related N2. Similar results have also been reported in studies involving children with ADHD (e.g., Karayanidis et al., 2000) and with other early adversity samples (e.g., Bruce, McDermott, Fisher, & Fox, 2009; McDermott et al., 2012). This pattern may represent processing deficits that behavioral measures are not sensitive enough to detect thus suggesting PIs may have an

underlying inhibitory control deficit. It could also be that PIs struggled with stimulus discrimination, perhaps due to poor sustained attention, explaining the decreased N2 during Go/No-go (e.g., Nieuwenhuis et al., 2003).

Together with reports that PI children demonstrate difficulties with inattention, impulsivity, and hyperactivity (Kumsta et al., 2010; Maclean, 2003), these results suggest that PI children display attention regulation similar to non-institutionalized children with ADHD. Children with ADHD demonstrate increased omission errors on continuous performance tasks (e.g., Sartory et al., 2002), but also generally make more commission errors. Similar to our results, there is evidence supporting decreased N2 in ADHD samples (Kenemans et al., 2005) al., 2005); however, there is also evidence for decreased P300 among children with ADHD (e.g., Spronk et al., 2008). Further, children with ADHD have increased error rates and decreased ERN amplitude on Flanker tasks (van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2007). While these results cannot address specifically the similarity of, and differences between, the attention regulatory difficulties in PIs to those experienced by non-PI children with ADHD, they do indicate a need to directly compare executive attention task performance and ERPs across the groups.

Objectives and Hypotheses

The primary objective of the current study was to fill an apparent gap in the literature and to address the question of whether deprivation-related ADHD is different from ADHD in children without histories of deprivation. It was hypothesized that the overall profile of inattention and hyperactivity demonstrated by PI adolescents would

have overlapping features with that of NA adolescents with ADHD; however, it would not map on precisely and would instead have a unique symptom profile.

The first goal of the study was to directly compare the specific ADHD-related symptoms across adolescents internationally-adopted from institutions (PI), with and without ADHD diagnoses, and never insitutionalized, non-adopted (NA) children with ADHD. This comparison included assessment of differences in clinical symptom patterns and severity across the groups. Although few studies involving ADHD-related symptoms in PI adolescents explicity examined the specific types of ADHD symptoms, Roy and Rutter (2006) suggested that inattentive behaviors may make up the majority of the inattentive/overactive profile of PI adolescents. Similarly, Loman et al. (in press) found behavioral deficits more specific to sustained attention rather than impulsive behavior. Therefore, it was expected that PI adolescents would demonstrate more inattentive behaviors that NA adolescents with ADHD. Based on the findings of Sonuga-Barke and Rubia (2008) of gender differences in symptom profiles among their sample of PIs with ADHD, it was anticipated that severity would differ by gender such that PI males with ADHD would differ from the other PI males without ADHD whereas PI females with ADHD would differ from the NA females with ADHD. This suggests that PI males with ADHD would be more similar to NA males with ADHD whereas PI females with and without ADHD would be similar to each other. Given the commonly described clinical profile of PI children noting presence of both externalizing and internalizing problems (e.g., Juffer & van Ijzendoorn, 2005; Zeanah et al., 2009) and the common comorbidities of ADHD including oppositional defiant disorder, conduct disorder, and mood and anxiety disorders (e.g., Biederman, 2005; Fischer, Barkley,

Smallish, & Fletcher, 2002; Jensen, Martin, & Cantwell, 1997), it was also anticipated that PI adolescents with ADHD would have more co-occurring emotional and behavioral difficulties than NA adolescents with ADHD and PI adolescents without ADHD.

The evidence that inattentive/hyperactive and disinhibited social behaviors are correlated among PI children contributed to a second goal of exploring whether disinhibited social behaviors may be a unique feature of deprivation-related ADHD and better understand the relation between ADHD symptoms and disinhibited social behavior in PI adolescents. We expected PI adolescents, both with and without ADHD, to demonstrate higher levels of disinhibited social behavior than the NA adolescent group and that when considered in association with displayed ADHD symptoms, disinhibited social behavior would be strongly associated with ADHD symptomatology for the PI adolescents, but not for the NA adolescents.

As described, ADHD has been conceptualized as being related to deficits in both executive function and reward/delay aversion. In order to gain a better understanding of the deficits associated with deprivation-related ADHD and investigate whether insititutional rearing has a differential impact on the two different, yet related, systems, a third goal of the study was to compare group performance on behavioral tasks related to both executive function and delay aversion. Overall, it was thought that executive functioning as a whole would not differ universally and instead there would be a specific pattern of deficits. Following from the neuropsychological impairment findings of Sonuga-Barke and Rubia (2008), it was expected that PI adolescents with ADHD diagnoses would demonstrate the poorest performance on the executive function tasks. Then, based on previous executive function findings (Colvert et al., 2008; Loman et al.,

in press; Pollak et al., 2010), it was anticipated that PI adolescents without ADHD would perform better than NA adolescents with ADHD on vigilance and spatial working memory tasks. However, these two groups would not differ for inhibitory control and planning. Examination of PI adolescents' performance on the delay aversion tasks was exploratory and no firm predictions were made.

Finally, a fourth goal of the study was to compare patterns of brain activity during a sustained attention/inhibitory control task across groups and extend the findings of our previous ERP study (Loman et al., in press). It was hypothesized that PI adolescents with ADHD would demonstrate decreased N2 and P300 amplitudes and later N2 latency relative to PI adolescents without ADHD and NA adolescents with ADHD.

Methods

Participants

Eleven- to 15-year-old participants were recruited from three separate groups: 1) *Post-institutionalized with ADHD (PI-A, n* = 26*)* were internationally adopted at 10 months of age or older, having spent at least 6 months of their pre-adoptive care in an institution and met criteria for an ADHD diagnosis; 2) *Post-institutionalized without ADHD (PI-N, n* = 20*)* met the same pre-adoptive criteria as group 1 and demonstrated 3 or fewer of the 6 inattentive behaviors and 3 or fewer of the 6 hyperactive/impulsive behaviors required for an ADHD diagnosis; 3) *Non-adopted with ADHD (NA-A, n* =19*)* were Minnesota-born children raised in their families of origin that currently met criteria for an ADHD diagnosis. See Table 2 for participant demographics including region of origin for the adopted groups. The current age range for the participants was chosen in order to best capture the full range of possible ADHD symptoms and behaviors. It has

been suggested that symptoms of the inattentive subtype of ADHD emerge in middle childhood (ages 8 to 12 years) whereas the hyperactive/impulsive symptoms emerge earlier (Barkley, 2003). The age criteria of 10 months or older at adoption was chosen for the PI groups because much of the literature involving internationally-adopted children suggests that older age at adoption is associated with higher risk for demonstrating inattentive and hyperactive behaviors (e.g., Gunnar et al., 2007; Kreppner et al., 2001; Stevens et al., 2008).

Internationally-adopted participants were recruited from the Minnesota International Adoption Project Registry, a list of community families interested in participating in research, and from advertisements shared with support groups for families created through adoption. Non-adopted particpants were recruited from fliers placed in local pediatric and behavioral health clinics and advertisements shared with local schools. In the final sample of participants, the three groups did not differ in the family-level demographics of parental education, (parent completing survey: $\chi^2(8) = 8.97$, *ns*; parent's spouse/partner: $\chi^2(12) = 13.83$, *ns*) or reported household income range, $\chi^2(18) = 16.24$, *ns*. Across groups, the majority of parents completed 4-year college degrees or higher (75% of parent completing survey; 57% of parent's spouse/partner) and reported family incomes greater than $75,000 (71%). There was, however, a disparity in gender distribution, $\chi^2(2) = 12.9$, $p < .01$ in that females were over-represented in the PI-N group. Importantly, the two groups with ADHD did not differ from each other in gender distribution, $\chi^2(1) = 0.0$, *ns*.

In order to reduce the likelihood of group differences being due to prenatal and congenital conditions, adolescents were excluded from the laboratory sessions if they

reported having a known genetic condition (n = 5), a pervasive developmental disorder (n = 6), or medically diagnosed Fetal Alcohol Spectrum Disorder (n = 12). Photographic screening using the FAS Facial Photographic Analysis Software (Astley, 2003) was also used to analyze the CDC guidelines regarding FASD associated facial dysmorphia (i.e., smooth philtrum, thin vermillion border, and small palpebral fissures) based on digital pictures taken during the laboratory session. One participant was excluded based on this screening procedure. Participants' near vision, wearing corrective lenses as prescribed, was screened through the use of a symbol based eye chart to ensure proper completion of the computer tasks. Based on the criteria of correctly identifying at least half of the symbols at a Snellen fraction of 20/40 or better with each eye, all participants were included. Approval for this study was granted by the University of Minnesota Institutional Review Board.

Procedure

There were two phases to this project. In Phase 1, parents of participants completed a web-based survey answering questions regarding their child and family-level demographcis. Responses were provided for 211 adolescent participants. Parents were mailed a small gift card upon completion of the survey. A subset of children were invited for Phase 2 based on ADHD diagnosis and pre-adoptive history provided during Phase 1. Demographic variables including age, gender, and reported family income were also considered when possible to allow for matching across groups. A total of 65 of 102 invited adolescents participated in Phase 2. Primary reasons for declining Phase 2 participation were: being too busy, adolescents' lack of interest, and not returning calls. Phase 2 involved a single laboratory session lasting approximately 3 hours. Participants

taking stimulant medications were asked to participate on days when they had not taken their medication or during a time of day when the stimulant was less effective (e.g., the afternoon for a medication taken in the morning). Families received a gift card for volunteering to participate in Phase 2.

Measures

Phase 1.

Parent questionnaires. Parents completed questions from the Minnesota International Adoption Project Survey (Hellerstedt et al., 2008) regarding their child's medical history including behavioral and emotional diagnoses and current medications, the child's educational history including any services received, and family-level demographics such as household income and parent education. Parents of adopted children also completed questions regarding their child's life experiences prior to adoption. All parents also provided a rating of "not all," "just a little," "pretty much," and "very much" for each of the behaviors off medication if applicable associated with ADHD diagnosis based on the DSM-IV diagnostic criteria (e.g., talks too much, has difficulty organizing tasks and activities). Behaviors were considered clinically significant if there were rated as "pretty much" or "very much." Clinically significant behaviors were then counted to determine if an individual met criteria for a diagnosis of ADHD.

Phase 2.

General cognitive abilities. Participants' general cognitive ability was assessed with the Wechsler Abbreviated Scale of Intelligence (WASI). Nonverbal reasoning abilities were estimated through administration of the Matrix Reasoning subtest and

verbal reasoning abilities were estimated through administration of the Vocabulary subtest. Performance on these two subtests contributed to a Full Scale IQ score (FSIQ-2).

Puberty. Participants completed a questionnaire evaluating their pubertal status using the Pubertal Development Scale (PDS). Questions were answered on a 1 ('not yet started') to 4 ('seems completed') scale and were averaged into a puberty status score. Items reflected the main axes of puberty: Adrenal changes included pubic/body hair and skin changes; Gonadal changes included growth spurt, breast development, and menarche for females and growth spurt, deepening of voice, and facial hair growth for males.

ADHD and psychopathology symptoms. Participants and their parents each completed the self-report and parent-report versions of the Conners 3rd Edition (Conners 3S) Short Form to assess the cognitive, emotional, and behavioral problems associated with ADHD. The Conners 3S is a well-validated measure that provides age-stratified norms across different ADHD-related domains (Inattention, Hyperactivity/Impulsivity, Learning Problems, Executive Functioning, Defiance/Aggression, and Peer Relations) based on a large, nationally representative sample (Conners, 2008). Combined gender norms were used in this study. This measure is often used in clinical settings to assist with diagnosis of ADHD. Parents also completed the Swanson, Nolan, and Pelham version IV questionnaire (SNAP-IV; Swanson et al., 2001). This assessment was used in the primary analyses of the large NIMH Collaborative Multisite Multimodal Treatment Study of Children with Attention-Deficit/Hyperactivity Disorder (MTA). Items are rated on a 0 ("Not at all") to 3 ("Very Much") scale. As in Swanson et al. (2001), scores across the three domains of Inattention, Hyperactivity/Impulsivity, and Opposition/Defiance were averaged into an overall symptom severity scale. To explore co-occurring

psychopathology, participants and their parents also each completed the clinical scales of Behavioral Assessment System for Children-2 (BASC-2) to assess the emotional and behavioral functioning across externalizing, internalizing, and behavioral domains.

Disinhibited social behavior. Participants and their parents each completed ratings assessing how the child participant typically responded in different social situations to assess disinhibited social behavior (e.g., I have wandered off or gone somewhere with someone I just met, even someone my parent(s) didn't know very well) Items were based on questions utilized by the English and Romanian Adoptees Study (e.g., Rutter et al., 2007; Stevens et al., 2008). Questions were included about current behavior as well as behavior at younger ages. Participants and their parents responded to items on 0 ('not at all true') to 2 ('always true') scale. A summary score was calculated for the self-report (total possible = 20) and parent-report separately (total possible = 32), with higher scores indicative of more disinhibited behavior.

Executive function tasks. Participants completed a battery of computer-based tasks found to demonstrate the largest effect sizes when comparing groups with and without ADHD (Willcutt et al., 2005). 1) Go/ No-go task: The same task parameters were used as in our previous study (i.e., Loman et al., in press). This computerized task required participants to press a button as quickly and accurately as possible for each letter presented (Go trials) except the letter X (No-go trials). Stimuli were presented for 600 ms with 1600 ms allowed for a response. Intertrial intervals were varied (200-400 ms). Following a short practice period (8 Go trials, 6 No-go trials), two blocks of 164 trials were completed. Within each block, 20 Go trials were presented, followed by 144 trials consisting of 75% Go intermixed with 25% No-go trials. Accuracy and response time were variables of

analysis. 2) Cambridge Neuropsychological Test Automated Battery (CANTAB) Stop/signal task: This task measures the ability to inhibit a response when signaled. During the initial practice portion of the task, participants were asked to press the appropriate button corresponding to the direction of an arrow on the screen. In the second part, the participant was told to continue pressing the buttons for the arrow direction, but, if they heard a beep, they should withhold their response and not press the button. Response speed, accuracy, and time to inhibit the response to the stop stimulus (SSRT) were variables of analysis. Staircase functions generated an estimate of SSRT. 3) CANTAB Spatial Working Memory Task: This is a self-ordered task that assesses strategy use and the ability to retain spatial information and manipulate remembered items. The participant was instructed to look for and collect tokens hidden inside colored boxes; once a token was found within a box, another token would be in that particular box. The number of errors was the variable of analysis. 4) CANTAB Stockings of Cambridge Task: This task is a measure of spatial planning wherein participants were asked to copy the pattern of stacked colored balls shown in an upper display by moving colored circles between three locations in a specific number of moves. Problems increased in difficulty as the number of required moves increases. The mean time to start a problem (i.e., initial thinking time) and problems solved in minimum number of moves were dependent variables of interest.

Delay aversion tasks. Participants completed a battery of computer-based delay aversion tasks. The Choice Delay Task (e.g., Maudsley's Index of Childhood Delay Aversion [MIDA]; Kuntsi, Stevenson, Oosterlaan, & Sonuga-Barke, 2001) assessed preferences for immediate, smaller rewards versus for later, larger rewards. Using

spaceships as stimuli, the participant was told to destroy enemy spacecraft using their own spaceship, controlled via the computer mouse. The participant was told to earn as many points as possible by choosing between two options: waiting for 2 seconds to destroy one spaceship and receive 1 point as a reward, or wait for 30 seconds in order to destroy two spaceships and get 2 points as a reward. The percentage of choices for the 2 points delayed reward was the variable of analysis. Due to programming errors, 9 participants did not complete this task (PI-A = 2, PI-N = 6, NA-A = 1). Another 6 declined to participate in or finish the task (PI-A = 2; NA-A = 4). The Delay Reaction Time (Sonuga-Barke et al., 2010) assessed reaction time when a delay is imposed. A stimulus (either a left or a right arrow) appeared on the center of the computer screen for either 3 or 20 seconds. When the screen turned blank, the participants were to respond quickly and accurately to the disappearance of the stimulus by pressing the left or right mouse button. During another portion of the task, participants responded to the direction of stimulus, but there was no delay prior to the required response. The variable of analysis was delay sensitivity calculated from mean reaction time for the delay levels minus the reaction time from the no delay task portion.

Event-related potentials (ERPs). ERPs were recorded using a 128 channel HydroCel Geodesic Sensor Net (Electrical Geodesics, Inc.) sampled at 500 Hz during the Go/No-go task. Data collection and data processing were completed using EGI software (Net Station; Electrical Geodesic, Inc., Eugene, OR). All channels were first referenced to Cz during recording and then rereferenced against an average reference corrected for the polar average reference effect (PARE correction). Eye blink artifacts (160 μV threshold), signals exceeding 120 μV, and fast transits exceeding 60 μV were removed

during the averaging process. Data were filtered using a FIR bandpass filter with a lowpass frequency of 30 Hz and a highpass frequency of .3 Hz. Data were segmented into epochs from 400 ms before to 700 ms after stimulus onset, were baseline corrected (based on 200 ms prior to stimulus onset), and averaged within each condition for each participant. ERP data from approximately 12% of the sample were visually inspected for excessive mastoid, vertical EOG, and movement artifact that appeared to bias the waveform. Because analyses examining the correlations between the visual and automatic artifact yielded an average Pearson's *r* correlation of $r(8)=.99$, $p < .001$, all artifact detection was completed automatically. A minimum of 9 usable ERP trials were required for analyses; however, the majority of the sample had a minimum of 20 usable trials. ERPs were associated with correct trials. Automatic detection identified peak amplitude (μV) and latency to peak (ms) for the stimulus-locked component of N2 (negative deflection between 205-460 ms) maximal at medial-frontocentral scalp electrodes (Fz, F1, F2, FCz). Average amplitude (μV within specified window) was used for P300 (positive deflection between 220-650 ms) maximal at parietal electrodes (P1, Pz, P2). Due to software errors, ERP data were not collected from three participants (PI-A = 2, NA-A = 1).

Data Analysis

Background Characteristics

Descriptive statistics were computed for each of the following variables for the participants that completed the Phase 2 laboratory session: age, pubertal status, IQ, and for the PI groups only, age-at-adoption, duration of institutionalization, and region-of-

origin. Group differences across these variables were assessed using Pearson chi-square for categorical variables and ANOVA for continuous variables.

Primary Analyses

For all of the analyses that follow, General Linear Models (GLM) were used to compute multivariate analysis of variance (MANOVA) and analysis of variance (ANOVA).

Clinical symptom pattern. Differences in ADHD symptom counts for Inattentive Symptoms and Hyperactive/Impulsive behaviors were examined with repeated measures ANOVA with group (PI-A, PI-N, and NA-A) and gender as between-subject variables with ADHD symptoms as the within-subject variable. Group differences in ADHD features (Conners 3S scales T-scores) of Inattention, Hyperactivity/Impulsivity, Learning Problems, Executive Functioning, Defiance/Aggression, and Peer Relation reports were examined using MANOVA with group and gender as independent variables. Univariate ANOVAs for each significant dependent variable were conducted as follow-up tests to the MANOVA. Group differences in ADHD symptom severity as assessed by the SNAP-IV were examined using ANOVA with group and gender as the independent variables and the SNAP-IV severity scale as the dependent variable. Differences in co-occurring emotional and behavioral symptoms (i.e., parent and self-report BASC-2) across groups were explored with MANOVAs for each of the overall symptom domains (e.g., Externalizing: hyperactivity, aggression, conduct problems; Internalizing: anxiety, depressive symptoms, somatic complaints; Behavioral: atypical behavior, withdrawal, attention

problems). Univariate ANOVAs for each significant dependent variable were conducted as follow-up tests to the MANOVA

Relation between ADHD symptoms and disinhibited social behavior. Group differences in levels of disinhibited social behavior were examined using MANOVA with group and gender as the independent variables and self-report ratings of behavior and parent-report ratings of behavior as the dependent variables. Univariate ANOVAs for each significant dependent variable were conducted as follow-up tests to the MANOVA. Bivariate correlations of ADHD symptoms and symptom severity with disinhibited social behavior were conducted for the overall sample, as well as by group. The Bonferroni method for controlling Type I error rates for multiple comparisons was applied when considering correlations within each group. Group differences in the magnitude of correlation were tested using Fisher's z-test.

Performance on behavioral tasks related to executive function and delay aversion. Because the previous literature suggests that group differences may vary for the different types of executive function tasks, group differences on each the executive function tasks were examined individually. Analysis of the Go/No-go task included repeated measures ANOVA comparing group differences and the mean rate of commission errors (false alarms) to the mean rate of omission errors and an ANOVA comparing mean reaction time to target (go) trials across groups. Greenhouse-Geisser correction was applied when the sphericity assumption was not met. Significant interactions were explored using simple effects analyses. Stop-signal analyses included three univariate ANOVAs comparing median reaction time, mean accuracy, and the mean time required to inhibit the response to the stop stimulus across groups. ANOVAs

exploring group differences on Spatial Working Memory total errors, and Stockings of Cambridge initial thinking time and problems solved in the minimum number of moves were also conducted.

Group differences on the delay aversion tasks were examined using MANOVA with group and gender as the independent variables and the percentage of choices for the delayed reward and delay sensitivity score as the dependent variables. ANOVAs for each significant dependent variable were conducted as follow-up tests to the MANOVA.

Associations between symptom and behavioral profiles. Bivariate correlations of the Conners 3S Inattentive and Hyperactive/Impulsive subscales and ADHD symptom severity with executive function task and delay aversion task behavioral performances were computed. Correlations were computed across the entire sample of adolescents with an ADHD diagnosis, as well as by group with ADHD. Group differences in the magnitude of correlations were tested using Fisher's z-test.

Brain activity. Group differences in the amplitude of N2 and P300 and latency for N2 were evaluated with repeated measures ANOVAs with group and gender as the between subjects factors with condition (go vs. no-go trials) and electrode as the within subjects factors. Greenhouse-Geisser correction was applied when the sphericity assumption was not met. Significant interactions were explored using simple effects analyses.

Results

Background Characteristics Differentiating the Groups

See Table 2 for a summary of participant demographics by group. Groups differed in mean age at assessment, $F(2,62) = 4.74$, $p < .05$, such that the PI-N as a group

were older than the PI-A. NA-A did not differ from either group. Notably, age at assessment was not correlated with the primary variables of interest and was therefore not included as a covariate in the primary analyses. Pubertal status did not differ across groups (PI-A: $M = 2.44$, $SD = .93$; PI-N: $M = 3.01$, $SD = .46$; NA-A: $M = 2.77$, $SD = .72$) when this age difference was accounted for, $F(2,57) = .811$, *ns*. There was a trend toward group differences in IQ, $F(2,54) = 3.09$, $p = .054$, suggesting that the PI-N had a higher average IQ than the PI-A. Evaluation of the individual subtests revealed a similar pattern and trend toward significance for Vocabulary, $F(2,54) = 3.01$, $p = .058$; however, no group differences were found for Matrix Reasoning, $F(2,56) = 2.36$, *ns*. Examination of pre-adoptive experiences for the PI-A and PI-N groups revealed that the PI-A group was older at the time of adoption, $F(1,43) = 7.89$, $p < .01$, and correspondingly spent more time in an institution prior to adoption, $F(1,41) = 5.92$, $p < .05$. The groups also differed in region of origin, $\chi^2(3) = 9.17$, $p < .05$, with more PI-A children adopted out of Eastern Europe and more PI-N adopted out of Asia. Notably, group ($\beta = 1.88$, Walds $\chi^2(1) = 6.42$, $p < .05$), and not duration of institutionalization ($\beta = -.014$, Walds $\chi^2(1) = .24$, *ns*), was a predictor of region of origin.

Primary Analyses

For all of the analyses that follow, group and gender were included as independent variables. Unless otherwise noted, there were no significant gender effects or group by gender interactions.

Clinical symptom pattern. Examination of ADHD symptom counts revealed a main effect for symptom type, $F(1,58) = 27.14$, $p < .001$, and a main effect for group, $F(2,58) = 28.06$, $p < .001$. The entire sample endorsed more inattentive symptoms than

hyperactive/impulsive symptoms. The PI-N group endorsed fewer symptoms than the PI-A and NA-A groups. PI-A and NA-A did not differ from each other. See Table 3 for group means. Multivariate analysis considering parent report of ADHD features on the Conners 3S revealed main effects of group, Wilks' λ =.38, $F(12, 96) = 4.97$, $p < .01$, and of gender, Wilks' λ =.75, $F(6, 48) = 2.73$, $p < .05$. The groups had different scores based on parent report for the ADHD features of Inattention, $F(2, 53) = 22.53$, $p < .001$, Hyperactivity/Impulsivity, $F(2, 53) = 9.73$, $p < .001$, and Executive Functioning, $F(2, 53) = 9.05$, $p < .001$. Tukey's post hoc tests revealed that for all three parent-rated domains, PI-N had lower T-scores than PI-A and NA-A. See Table 3. Males were found to have higher T-scores than females for Inattention, $F(1, 53) = 4.03$, $p = .05$ (males: $M = 76.72$, $SD = 9.8$, females: $M = 61.63$, $SD = 15.9$), and Hyperactivity/Impulsivity, $F(1,53) = 16.15$, $p < .001$ (males $M = 81.02$ $SD = 11.3$, females $M = 58.37$, $SD = 15.7$). There was no effect of group for self-report of ADHD features on the Conners 3S, Wilks' λ =.77, $F(10, 102) = 1.38$, *ns*. Evaluation of ADHD symptom severity revealed a main effect of group, $F(2,55) = 16.98$, $p < .01$, such that PI-N were rated as less severe than both PI-A and NA-A. A group by gender interaction, $F(2, 55) = 9.4$, $p < .001$, revealed that the NA-A females ($M = 1.97$, SD =.45) had higher severity than PI-A females ($M = 1.19$, SD = .46) followed by PI-N females ($M = .40$, $SD = .33$) whereas males with ADHD did not differ (NA-A: $M = 1.52$, $SD = .44$; PI-A: $M = 1.86$, $SD = .49$).

Multivariate analyses of co-occurring emotional and behavioral problems based on parent report yielded group effects for externalizing symptoms, Wilks' λ =.78, $F(6, 96) = 2.51$, $p < .05$, and behavioral symptoms, Wilks' λ =.63, $F(6, 96) = 4.10$, $p < .01$. No effects were found for internalizing symptoms, Wilks' λ =.82, $F(6, 96) = 1.68$, *ns*.

Within the externalizing domain, groups differed in parent report of hyperactivity only, $F(2,50) = 6.59$, $p < .01$. Tukey's post-hoc analyses revealed that both the PI-A and NA-A had higher hyperactivity than PI-N. See Table 3 for group means. Among behavioral symptoms, groups differed in atypicality, $F(2,50) = 5.31$, $p < .01$, and attention problems, $F(2,50) = 11.92$, $p < .001$. Post-hoc analyses indicated that PI-A and NA-A had both higher atypicality and attention problems than PI-N (see Table 3). Self-report of co-occurring emotional and behavioral problems yielded no effect of group for internalizing symptoms, Wilks' $\lambda = .56$, $F(14, 58) = 1.38$, *ns,* or inattention/hyperactivity, Wilks' $\lambda = .86$, $F(4, 90) = 1.72$, *ns.*

ADHD symptoms and disinhibited social behavior. Group differences were noted at a trend level for the omnibus test of disinhibited social behavior, Wilks' $\lambda = .84$, $F(4, 106) = 2.36$, $p = .058$. While parent-report of disinhibited behavior did not differ by group, $F(2,54) = 1.67$, *ns*, self-report yielded a main effect of group, $F(2,54) = 3.77$, $p < .05$. Tukey's post-hoc analyses revealed that PI-A demonstrated higher disinhibited social behavior than PI-N and NA-A (see Figure 2). Across the entire sample, greater parent-reported disinhibited social behavior was associated with greater number of inattentive symptoms, $r(60) = 0.35$, $p < .01$, greater number of hyperactive symptoms, $r(60) = 0.40$, $p < .01$, and greater symptom severity, $r(60) = 0.42$, $p < .01$. Self-report of disinhibited social behavior was positively correlated with number of hyperactive symptoms, $r(60) = 0.30$, $p < .05$ and symptom severity, $r(60) = 0.27$, $p < .05$. Examination of the correlations within each group following adjustment to the significance threshold by the Bonferroni correction ($\alpha = .003$) found that self-reported disinhibited social behavior was positively correlated with the number of hyperactive

symptoms for NA-A, $r(18) = 0.66$, $p = .003$. There were no differences in the magnitude of correlations across groups.

Behavioral task performance: executive function. See Table 4 for group means across the tasks. As expected, analysis of Go/No-go accuracy revealed that mean accuracy was higher for Go trials ($M = .86$, $SD = .20$) than for No-go trials ($M = .63$, $SD = .18$) across groups, $F(1,56) = 36.71$, $p < .001$. There was no group by trial type (i.e., Go vs. No-go) interaction, $F(2,56) = .67$, *ns*. Indeed, exploratory analyses focusing only on Go trials following from the results of Loman et al. (in press) revealed that PI-A were less accurate than PI-N, $F(1, 42) = 5.03$, $p < .05$, but did not differ from NA-A, $F(1,42) = 1.57$, *ns* (see Figure 3). Analysis of reaction time for correct Go trials on the Go/No-go revealed a trend toward a group effect, $F(2,58) = 3.06$, $p = .055$, in that NA-A were slower to respond than PI-N, whereas PI-A did not differ from either group. On the Stop-Signal task, there was a trend toward significance in group differences of stopping accuracy $F(2,50) = 2.77$, $p = .07$. Follow-up analyses suggested that PI-A had poorer stopping accuracy than PI-N; NA-A did not differ from either group. Evaluation of reaction time for Go trials on the Stop-signal task revealed no group effect, $F(2,50) = 1.44$, *ns*; however, there was a main effect of gender, $F(1,50) = 5.65$, $p < .05$, such that females ($M = 403.50$, $SD = 69.2$) responded more quickly than males ($M = 460.63$, $SD = 169.21$). Analysis of the SSRT (mean time required to inhibit the response to the stop stimulus) yielded a trend toward a group effect, $F(2,50) = 2.65$, $p = .08$, suggesting PI-A and NA-A required more time to inhibit a response than PI-N. In addition, there was a group by gender interaction, $F(2,50) = 3.24$, $p < .05$, indicating PI-A males ($M = 282.8$, $SD = 118.22$) required more time to inhibit their responses than PI-A females ($M =$

196.95, *SD* = 64.4) whereas the other groups did not differ across gender. On the Spatial Working Memory task, there was a group effect, $F(2,60) = 4.88$, $p < .05$, in that PI-A made more errors than PI-N; NA-A did not differ from either group. Assessment of the Stockings of Cambridge task yielded no group effect for the number of problems solved in the minimum number of moves, $F(2,56) = 1.29$, *ns,* and no group effect for the time required before the first move was made, $F(2,56) = 1.03$, *ns*.

Behavioral task performance: delay aversion. Multivariate analysis investigating delay aversion task performance yielded a group by gender interaction, Wilks' $\lambda = .81$, $F(2,42) = 4.93$, $p < .05$, whereas the main effects were not significant (group: Wilks' $\lambda = .91$, $F(4,84) = 1.04$, *ns*; gender: Wilks' $\lambda = .97$, $F(2,42) = .68$, *ns*). The interaction was found for the MIDA percentage of choices for the larger, delayed reward, $F(2,44) = 5.06$, $p < .05$, and the delay sensitivity score, $F(1,56) = 3.5$, $p < .05$. Post-hoc analyses revealed that PI-A males ($M = 51.41$, $SD = 30.3$) chose the larger delayed reward less often than did the NA-A males ($M = 91.3$, $SD = 14.6$) whereas the females did not differ across groups. For delay sensitivity, follow-up analyses revealed that within the females, PI-As had shorter reaction times than the PI-Ns on trials with longer inter-stimulus-intervals (females: PI-A $M = 180.69$, $SD = 95.8$; PI-N $M = 253.61$, $SD = 82.63$; NA-A $M = 253.29$, $SD = 80.69$), whereas the males did not differ across groups.

Associations between symptoms and behavioral profiles. Across the sample of adolescents with an ADHD diagnosis (i.e., PI-A and NA-A), there was a trend toward significance for the correlation between Conners 3S Hyperactivity/Impulsivity subscale and SSRT, $r(34) = -0.33$, $p = .06$. Correlations computed within each group with ADHD

indicated that within the PI-A group, a higher Conners 3S inattentive score was associated with less time to start the Stockings of Cambridge task, $r(20) = -0.46$, $p < .05$. Although this association was not present for the NA-A group, the magnitude of correlation was significantly different across groups, Fishers z-test = 2.112, $p < .05$. Additionally, within the NA-A group, faster Go trial reaction time was associated with lower ADHD symptom severity, $r(19) = -0.46$, $p < .05$; however this did not hold within the PI-A group. The magnitude of the correlations did not differ from each other, Fishers z-test = 1.63, *ns*. When the Bonferroni correction was applied ($\alpha = .002$), these correlations were no longer significant. For the delay aversion tasks, there was no significant correlation across the sample of adolescents with ADHD between percentage of choices for the larger delayed reward and any of the ADHD symptom measures. Within the PI-A group, the Conners 3S Inattentive score was negatively correlated with percentage of choices for the larger delayed reward, $r(18) = -.57$, $p < .05$. Additionally for the PI-A group, the SNAP-IV severity score negatively correlated with percentage of choices for the larger delayed reward, $r(19) = -.52$, $p < .05$. Indeed these correlations were not significant after accounting for Bonferroni correction ($\alpha = .008$). None of the correlations were significant within the NA-A group and the magnitude of the correlations did not differ across groups. There was no significant correlation between delay sensitivity and the ADHD symptom measures either across the entire ADHD sample or within each ADHD group.

Brain activity. Analysis for N2 amplitude associated with correct trials on the Go/No-go task revealed a main effect of condition, $F(1,53) = 6.21$, $p < .05$, such that No-go amplitude ($M = -6.50$ μV, $SD = 4.7$) was more negative than Go amplitude ($M = -4.61$

μV, *SD* = 3.5). This was qualified by a trend toward a condition by gender interaction, $F(1,53) = 3.54$, $p = .065$; No-go amplitude was only more negative than Go amplitude for females (No-go: *M* = -6.93 μV, *SD* = 4.5; Go: - 4.00 μV, *SD* = 3.3) as for males the amplitudes did not differ (No-go: *M* = -5.30 μV, *SD* = 3.6; Go: -6.01 μV, *SD* = 4.8). There was additionally a trend toward a group by gender interaction, $F(2,53) = 3.03$, $p = .057$. Follow-up analyses suggested that females did not differ in N2 amplitude across groups; however, PI-A males showed a smaller N2 (e.g., less negative) than NA-A males. (See Figure 4). For N2 latency, there were no group effects; however, there was a trend toward a main effect of condition, $F(1,53) = 3.72$, $p = .059$ suggesting that average latency for No-go trials (*M* = 355.03 ms, *SD* = 60.0) was later than for Go trials (*M* = 333.26 ms, *SD* = 66.9). There was also a main effect of lead, $F(2.6, 137.98) = 11.10$, $p < .01$, with FCz (*M* = 312.46 ms, *SD* = 52.8) peaking earlier than the other leads (F1: *M* = 356.44 ms, *SD* = 60.1; F2: *M* = 348.60 ms, *SD* = 61.9; Fz: *M* = 343.19 ms, *SD* = 75.3) .

Analysis of P300 average amplitude revealed a main effect for condition, $F(1,53) = 25.80$, $p < .001$, in that the amplitude was larger for No-go trials (*M* = 5.95 μV, *SD* = 4.1) than for Go trials (*M* = 2.31 μV, *SD* = 2.4). While there was no group effect, there was main effect of gender, $F(1,53) = 7.90$, $p < .01$, such that males (*M* = 5.54 μV, *SD* = 2.6) had larger average amplitude than females (*M* = 2.86 μV, *SD* = 2.3).

Discussion

Despite the fact that the presence of ADHD-like behaviors in PI children and adolescents has been well-documented in the literature, there was only a preliminary understanding to date of the associated deficits and neurobehavioral development. Previous research lent some support to understanding ADHD-like behaviors in PI

children and adolescents, or "deprivation-related ADHD" as being related to executive functioning difficulties, particularly spatial working memory, vigilance, and perhaps inhibitory control (Bos et al., 2009; Loman et al., in press; Pollak et al., 2010). Although similar deficits have been reported among children without histories of institutionalization with ADHD, additional difficulties with planning, problem-solving, and impulsivity are also common for children and adolescents with "standard ADHD" (Willcutt et al., 2005). In addition, ADHD has been described as being associated with unusual sensitivity to reinforcement (Luman et al., 2005) or suboptimal reward processing (Sonuga-Barke, 2005). Evidence for differential reward processing is only starting to be explored in samples with a history of deprivation with some suggestion of altered reward-related behavior (e.g., Fries, 2008). Importantly, the majority of studies to date have reached conclusions regarding the presence of ADHD symptoms and ADHD-related deficits in executive functioning and reward processing following early deprivation by comparing PI children and adolescents to non-adopted children, other internationally adopted children, or domestically adopted children. In the current study, we set out to better understand deprivation-related ADHD by directly comparing PI children, both with and without ADHD diagnoses, to non-institutionalized, non-adopted children with ADHD. Across these three groups, this study examined the clinical symptom profile, the presence of disinhibited social behaviors, performance on behavioral tasks related to executive function and delay aversion, and brain activity during a sustained attention/inhibitory control task. This design allowed for investigation into whether deprivation-related ADHD is different from ADHD in children without histories of deprivation and provided a more thorough description of the deprivation-

related ADHD features and symptom profile than has been seen in the literature previously.

Investigation into group characteristics revealed that among the PI groups, the adolescents with ADHD were older at the time of adoption and had spent more time in the institution prior to adoption. These differences occurred despite sampling parameters limiting inclusion of participants to those adopted at relatively older ages (i.e., 10 months) following a substantial amount of time in an institution. Importantly, these results are in fact consistent with the well documented findings of older age at adoption, and correspondingly longer duration of institutionalization, being associated with symptoms of increased inattention and hyperactivity (Audet & Le Mare, 2011; Gunnar et al., 2007; Kreppner et al., 2001). The PI groups also differed in their pre-adoptive region of origin as more PI-A adolescents were adopted out of Eastern Europe and PI-N out of Asia. This difference is consistent with findings from Gunnar et al. (2007) that children adopted from Russia/Eastern Europe, relative to those adopted from other countries, were at higher risk for demonstrating behavior problems. Notably, duration of institutionalization was not found to be a significant predictor of region of origin within the current sample of internationally adopted adolescents. Beyond this, it is unclear from the current study whether there are inherent differences in the pre-adoptive experiences within the institutions that may have contributed to children from one region over the other to be more likely to have ADHD. Prevalence rates of ADHD within Asia and Europe, for example, have been reported to be similar to that of North America (Polanczyk et al., 2007). Notably, the use of alcohol among women of child-bearing age has been reported to be relatively high in Eastern European countries (D. E. Johnson,

2001). It is well-documented that prenatal exposure to alcohol is associated with increased risk for inattentive and hyperactive behaviors (e.g., O'Malley & Nanson, 2002). To account for this, participants were excluded from Phase 2 participation (i.e., the laboratory tasks) if they had a diagnosis of a fetal alcohol spectrum disorder (FASD) or displayed facial features consistent with this diagnosis. Of course, the effects of alcohol use during pregnancy outside of those associated with an FASD diagnosis or facial features were not known or controlled for and may in fact have been present across participants in any of the groups.

In addition to being adopted at older ages after having stayed longer in an institution most likely in Eastern Europe, there was a trend for PI-A adolescents to have lower IQs, perhaps due to lower verbal reasoning abilities, than the PI-N adolescents. Living longer in an institution prior to adoption has been reported to be significantly correlated with IQ; this association appears to be found particularly for measures of verbal reasoning (Beckett et al., 2006; Loman et al., 2009). Therefore, the current result of a trend for IQ differences among the PI groups is not surprising. Additionally, the PI-A adolescents did not differ in IQ from the NA-A adolescents. Studies of ADHD in non-deprivation samples report that children with ADHD consistently have cognitive deficits, including lower IQ, relative to those without ADHD (see Biederman et al., 2009 for review). For example, one study reported a correlation of -0.3 between ADHD symptoms and IQ (Kuntsi et al., 2004). While what is driving the association between low IQ and ADHD is not precisely clear, the results of a longitudinal study implied that the cognitive deficits are independent of the course of ADHD (Biederman et al., 2009). Exactly how attention difficulties play a role in IQ testing in the current sample is

unknown. IQ might affect attention regulation; at the same time, IQ testing is highly susceptible to attention. Importantly, the lower IQ of PI children noted in the literature has not been found to account for group differences in performance on behavioral tasks (Colvert et al., 2008). Additionally, covarying IQ in our previous study (i.e., Loman et al., in press) did not significantly change the findings. Overall, the suggestion of a lowered IQ is another characteristic that separates PI-A adolescents from PI-N adolescents and implies a similarity in this associated feature between deprivation-related and standard ADHD.

Beyond a description of the preadoptive and general factors associated with deprivation-related ADHD, a goal of the study was to compare ADHD-related symptoms, features, and severity across groups. Analyses of reported symptoms of ADHD revealed that the PI-N group endorsed fewer symptoms than both groups with ADHD, who did not differ from each other. It was hypothesized that PI-A adolescents would be more likely to demonstrate inattentive symptoms; however, the reported symptom type did not differ in prevalence across the groups. Instead, the entire sample endorsed more inattentive symptoms than hyperactive/impulsive symptoms. Given the age-range of the sample, the higher prevalence of inattentive symptoms over hyperactive/impulsive symptoms is consistent with the ADHD literature. Barkley (2003) has described, for example, that the hyperactive/impulsive symptoms of ADHD may emerge in the preschool years whereas the inattentive symptoms may emerge in middle childhood, around ages 8- to 12-years of age. Investigation into features of ADHD resulted in a corresponding pattern. Specifically, group differences across the scales on the parent-report of the Conners 3S existed such that both PI-A and NA-A adolescents had elevated T-scores (i.e., were more

problematic) relative to PI-N adolescents. This was true for the scales of Inattention, Hyperactivity/Impulsivity, and Executive Functioning. Congruently, inspection of the T-scores across groups for these scales (see Table 3) revealed that scores for only the ADHD groups reached the clinically significant range (T-score >70). Although self-report on the Conners 3S did not yield group differences across the scales, inspection of group means (see Table 3) suggested a similar pattern as the parent-report; specifically, both ADHD groups had means within the clinically relevant ranges for Inattention and Hyperactivity/Impulsivity. Again, these results are contrary to expectations that specific problems with inattentive behaviors would be present for PI adolescents. Instead, both group of adolescents with ADHD were rated as having more problematic behavior than the PI-N adolescents. Indeed, these findings are suggestive of similarities in the clinical features of ADHD among both PI and NA adolescents.

Corresponding to the results noting that PI-Ns were often rated as having the least problematic behavior, they were also rated as having the lowest symptom severity. Beyond this, severity ratings confirmed hypotheses suggesting differences across gender. Similar to the findings of Sonuga-Barke and Rubia (2008), PI-A males were similar in symptom severity to NA-A males. Different from Sonuga-Barke and Rubia, however, PI-A females were neither similar to NA-A females or PI-N females; instead their severity ratings were in the middle of the two groups, with NA-As rated among the females as having the highest symptom severity. Notably, the groups did not differ by gender in terms of reported symptoms or associated features. Among the whole sample, males were reported to have higher Inattention and Hyperactivity/Impulsivity than females on the parent-report Conners 3S. This finding is consistent with the literature

noting that males are consistently found to outnumber females in diagnoses of ADHD (e.g., Biederman, 2005; Wilens, Biederman, & Spencer, 2002). It additionally supports the findings within the previous PI literature (e.g., Roy et al., 2004; Vorria et al., 1998; Wiik et al., 2011) noting higher prevalence of ADHD behaviors in males.

The final portion of investigation into the clinical symptom pattern across groups involved comparison of co-occurring emotional and behavioral problems. Beyond the similar findings of increased attention and hyperactive behavior for the two groups with ADHD relative to PI-N, PI-A and NA-A were also both found based on parent-report to have higher levels of atypical behavior. This scale corresponds to behaviors described as the child sometimes acting strangely, seeming unaware of others, or repeating actions. Elevations on this scale for the ADHD groups relative to the PI-N group are not surprising as similar elevations have been reported among ADHD samples previously (Harrison, Vannest, & Reynolds, 2011). Notably, visual inspection indicated that none of the group means fell within the clinically significant range. Moreover, these results did not support hypotheses that PI-A would have more co-occurring emotional and behavioral difficulties than the other two groups. Instead, the results followed what may be expected based on the ADHD literature (e.g., Biederman, 2005) that adolescents diagnosed with ADHD are more likely to demonstrate elevations on clinical scales relative to those without an ADHD diagnosis.

Beyond understanding the clinical symptom profile, an additional study goal was to assess if disinhibited social behaviors are in fact a unique feature of deprivation-related ADHD. The current findings supported the previous literature (e.g., Bruce, Tarullo, et al., 2009; Kreppner, et al., 2001; Rutter et al., 2001; Stevens et al., 2008) as PI-A

adolescents reported having higher disinhibited social behavior. Interestingly, PI-A adolescents reported more disinhibited social behavior than both PI-N and NA-A adolescents. Contrary to expectations, PI-Ns did not differ from NA-As. These results suggest that high levels of disinhibited social behavior are not present for PI adolescents in general but instead may be a unique feature of deprivation-related ADHD. Indeed, the findings are also consistent with the conclusions drawn by Rutter and colleagues that disinhibited attachment and ADHD behaviors are part of a deprivation specific syndrome (Kumsta et al., 2010). Following from some of the literature drawing links between disinhibited social behavior and inattentive and impulsive symptoms, higher disinhibited social behavior was also associated with higher levels of inattentive and hyperactive symptoms and greater symptom severity across the entire sample. Bruce et al. (2009) similarly reported that across their sample of internationally-adopted and non-adopted children, those who were poorly regulated were more likely to display disinhibited behaviors. Divergent from hypotheses, however, these associations between disinhibited social behavior and ADHD symptoms within the current study did not exist within the PI-A group when correlations were examined within each of the groups individually. This finding is somewhat surprising given that Stevens et al. (2008) noted correlations with disinhibited attachment behavior and inattention/overactivity in their Romanian PI sample of similarly-aged children. Indeed, the lack of a significant correlation for inattention and/or hyperactivity within the current study may suggest that the higher rates of disinhibited social behavior in the PI-A group are not specifically attributable to these specific ADHD behaviors but, are instead related to a different factor not assessed in the current study. Stunted growth at the time of adoption, for example, has recently been

reported to be associated with greater disinhibited social approach for PI children (A. E. Johnson, Bruce, Tarullo, & Gunnar, 2011). On the other hand, there is no evidence to suggest that disinhibited social behavior does *not* exist for some children with standard ADHD. Although the NA-A adolescents overall reported lower levels of disinhibited social behavior, the current finding of increased hyperactive behavior being related to higher levels of disinhibited social behavior for the NA-As may in fact suggest that this type of behavior does exist for some individuals with standard ADHD. Further investigation into disinhibited social behavior among both groups of adolescents with ADHD is warranted.

Beyond comparison of the clinical symptoms and associated features, ADHD has been linked to performance on behavioral tasks related to both executive functioning and reward/delay aversion. As noted above, analysis of associated features of ADHD via parent-report on the Conners 3S was indicative of both ADHD groups having elevated (i.e., problematic) executive functioning based on behavior associated with daily living. The pattern of executive deficits based on behavioral task performance, however, was slightly different. For example, with Go/No-go, as expected, all groups were more accurate on Go trials than on No-go trials thus indicating increased difficulty on the trials requiring inhibitory control. Analyses following from the results of our previous study (i.e., Loman et al., in press) specifically focusing on Go trials revealed that PI-As made more omission errors than PI-Ns although NA-A performance did not differ from either group. Inspection of group accuracy across the current study and Loman et al. for the Go/No-go task confirms that PI-As have the poorest accuracy across all group (See Figures 1 and 3) and support the conclusions that early deprivation has an impact on

sustained attention. These findings also support the ADHD literature. Despite findings that both omission and commission errors are present within ADHD samples, Willcutt et al. (2005) reported that omission errors on continuous performance tasks (e.g., "Go" errors on Go/No-go) consistently distinguish individuals with ADHD from those without ADHD. The other variable reported by Willcutt et al. to consistently discriminate ADHD was reaction time to the stop signal (SSRT) from the Stop-signal task. The current findings support this previous research as there was a trend for PI-Ns to have faster SSRT than both groups of adolescents with ADHD. Notably, PI-A males were found to be particularly slow in their SSRT and their performance may best account for the PI-A trend of increased time to inhibit an automatic response. Importantly, the slowed SSRT for the PI-As was not likely due to slower general processing as the groups did not differ in reaction time for Go trials on the Stop-signal task. Instead, the PI-A's slowed SSRT performance is likely indicative of impulsive behavior (Logan, Schachar, & Tannock, 1997), particularly when combined with the trend of PI-A adolescents to have poor accuracy in stopping on this task. Moreover, performance of the PI-Ns on the Stop-signal task relative to groups with more clinical impairment suggests consistency with previous research findings (Loman et al., in press; McDermott et al., 2012) that PI children without ADHD diagnoses do not demonstrate errors specific to response inhibition on Go/No-go tasks. Importantly, there has been some discussion in the literature that Go/No-go and Stop-signal tasks may be tapping different types of inhibitory control. Verbruggen and Logan (2008), for example, suggest that inhibitory control within the Go/No-go task may become automatic over the course of the task whereas more control is required throughout the Stop-signal task. A consideration of this distinction may account for

group differences in inhibitory control during the current Stop-signal task and a lack of group differences in commission errors among the Go/No-go studies.

Moving beyond specific attention and cognitive control tasks, group performance on the Spatial Working Memory task followed a similar pattern to the one described thus far: PI-As had the poorest performance compared to PI-Ns, and NA-A performance fell in the middle. For the Stockings of Cambridge planning task, on the other hand, the groups did not differ in performance. Results from these two tasks correspond to previous PI research indicating that the Stockings of Cambridge task did not discriminate samples of PI children from controls whereas the Spatial Working Memory task did (Bos et al., 2009; Pollak et al., 2010). The current findings support previous suggestions that problem solving on a planning task is not an area of specific weakness for PI children. Following from this, it is also worth noting that it is somewhat surprising that NA-As did not differ from the PI adolescents on this planning task given samples with ADHD often perform differently from those without ADHD on planning tasks (Willcutt et al., 2005) and on the CANTAB Stockings of Cambridge task specifically (Gau & Shang, 2010).

Taken together, group performance on the executive function tasks was generally indicative of impairment in functioning for PI-As, thus at least partially supporting study hypotheses and previous research (Sonuga-Barke & Rubia, 2008). Results differed from expectations in that PI-As did not demonstrate the poorest performance overall, but rather tended to perform only more poorly than their post-institutionalized peers. These findings suggest that having ADHD along with a history of institutionalization may be associated with more severe executive dysfunction than does a history of institutionalization alone. Rather than differing from either PI group, the performance of

NA-As tended to fall in between the two groups. Because of this, drawing conclusions regarding whether the PI-A group performed differently from the NA-A group is difficult and speculative at best. Inspection of group means may suggest that PI-As tended to perform most poorly on tasks assessing vigilance and spatial working memory; however, these conclusions are likely limited by low power and thus additional study is warranted. One exception to this pattern is the SSRT result which indicates that PI-A and NA-A adolescents may in fact have similar inhibitory control impairments, thus noting a potential overlapping neuropsychological feature between deprivation-related ADHD and standard ADHD.

Group performance on delay aversion tasks was more explorative in nature and indeed, demonstrated a different pattern than the executive function tasks. While no group effects were identified on either task, results were indicative of group by gender interactions. Among the males, PI-As were less likely than NA-As to wait for the larger, later reward. In fact, PI-A males as a group chose the larger, later reward only about 50% of the time. These results suggest that PI-A males may have even more significant discomfort (e.g., frustration, agitation; Marco et al., 2009) with waiting out longer delays than do NA-A males when given a choice to respond sooner. When not given a choice and instead participants had to respond to an imposed delay, male PI-As did not differ from male NA-As. Instead, the difference was attributable to females. Following from the delay aversion model, these results suggest that both groups of males with ADHD disengaged from the trials with long stimulus presentations in order to speed up the passage of time which then had an effect on their reaction time (Bitsakou et al., 2009). Notably, both groups of males with ADHD demonstrated mean reaction times similar to

that reported in the previous literature for similarly-aged individuals with ADHD (i.e., PI-A $M = 237.03$, $SD = 103.9$; NA-A $M = 242.57$, $SD = 81.7$; ADHD participants in Bitsakou et al., 2009 $M = 246$, $SD = 132$). Interestingly, the pattern of performance among the females was such that PI-As responded more quickly than PI-Ns. In other words, PI-A females responded more similarly to how control (i.e., non-ADHD) participants performed the delay reaction time test (Bitsakou et al., 2009). While slower reaction times on trials with long inter-stimulus-intervals have been described as indicative of delay avoidance, variability in reaction time within individuals has also been seen in ADHD (e.g., Andreou et al., 2007). Although variability in reaction time from the delay reaction time task itself has not been investigated previously, future research could include specific examination of intra-individual response time variability across the different trials of this task to determine if this pattern of results holds. On the other hand, these results may be another indication of gender differences in the deprivation-related ADHD profile. More research examining the sensitivity to delay is clearly needed to better understand if and how deficits in processing and motivation for reward exist within PI adolescents.

Associations between behavioral task performance and ADHD features and severity were examined to assess how different executive functions or delay aversion may be associated with deprivation-related ADHD differently from standard ADHD. As expected, across all individuals with ADHD, hyperactivity and impulsivity on the Conners 3S was associated with SSRT, a measure of impulsivity. Exploratory results examining these associations within each group revealed that more elevated inattention concerns were associated with less initial planning time for the PI-As and that this

relation existed at a higher magnitude than for the NA-As. This finding seems to indicate that poor planning associated with impulsive responding contributes to difficulties in attention regulation for PI-As. Further associations found only for the PI-As suggest a preference for sooner, smaller rewards may also be a marker for increased attention problems and a more severe ADHD symptom presentation within deprivation-related ADHD. These findings further highlight that a more thorough investigation into reward processing may be an important piece in understanding the deprivation-related ADHD profile in addition to the developmental profile of children who have experienced early institutional care.

Finally, a fourth goal of this study was to expand upon the ERP findings of Loman et al. (in press) suggesting PI children have similar neurobiology as children with ADHD. Results for both the N2 and P300 components in the current study demonstrated the expected patterns of demonstrating larger amplitude (i.e., more negative for N2 and more positive for P300) for the No-go trials relative to the Go trials. Similar to our previous findings, there was no group effect in P300 amplitude. In addition, despite demonstrating more sustained attention behavioral errors, in contrast to inhibitory control errors on the Go/No-task, PI-A males demonstrated differential brain activity (i.e., smaller amplitude) relative to NA-A males associated with the response inhibition component N2. As noted in the discussion of our previous study (Loman et al., in press), similar results of noting ERP differences in the absence of behavioral differences have been reported within other early adversity samples (e.g., Bruce, McDermott, et al., 2009; McDermott et al., 2012) and in studies involving children with standard ADHD (e.g., Karayanidis et al., 2000). As before, this may represent processing deficits that the

Go/No-go behavioral measure is not sensitive enough to detect. As described by Verbruggen and Logan (2008), inhibiting a response to the No-go stimuli could be learned and become automatic over time. Thus, within the current study, the Go/No-go behavioral results may be indicative of learning skills to compensate for underlying inhibitory control deficits. Unlike our previous study, within the current study we have the benefit of including the Stop-signal task, which has been reported to be a better task for assessing controlled, rather than automatic, inhibition (Verbruggen & Logan, 2008). Findings from this task were in fact suggestive of the PI-A group having difficulties controlling inhibitory responses and thus correspond to the N2 findings. On the other hand, as was suggested in our previous Go/No-go study (Loman et al., in press), it could also be that the decreased N2 during Go/No-go was associated with difficulties with discriminating a visual Go stimulus from a No-go stimulus due to poor sustained attention (Nieuwenhuis et al., 2003). Future studies will benefit from collecting ERP during the top-signal task in order to replicate the current behavioral results and allow for investigation into these differing interpretations of N2 findings as the Stop-signal task does not include the same stimulus discrimations demands as the Go/No-go task.

Summary and Conclusions

The results from this study suggest that deprivation-related ADHD has a similar clinical and behavioral profile to standard ADHD in addition to a few possible unique markers. Elevated levels of inattentive and hyperactive/impulsive behaviors similar to those associated with ADHD have been well documented within studies of post-institutionalized children and were confirmed within the current study. Akin to the ADHD literature describing higher prevalence of symptoms in males over females

(Biederman, 2005), males in the current study were also reported to have more problematic inattentive and hyperactive/impulsive behaviors. Both ADHD groups were also found to have lower IQ scores, share elevated scores for the same co-occurring emotional and behavioral problems, and demonstrate inhibitory control deficits relative to NA-A adolescents. The few unique features of deprivation-related ADHD included reports from PI-A adolescents of elevated disinhibited social behavior and among males, behavioral performance indicative of more aversion to delay and differential brain activity relative to NA-A adolescents with group differences suggesting PI-As had more problematic functioning. Importantly, the findings noting significant differences between the male PI-As and NA-As are limited by a lack of a comparison with PI-Ns due to the small sample size and warrant replication.

Overall, the combination of the overlapping profiles across the ADHD groups along with the few differing features is supportive of Sonuga-Barke and Rubia (2008)'s suggestion that deprivation-related ADHD may be a phenocopy of ADHD. Based on the PI literature to date, the underlying proposed difference between "standard ADHD" and "deprivation-related ADHD" is that in the case of PI children and adolescents, elevated levels of inattentive and hyperactive/impulsive behaviors are tied closely with the species atypical early caregiving environments associated with institutional care. The altered behavior and neurobiological functioning found within PI-A adolescents is likely secondary to the remarkable stress associated with extended periods of living within an institution. Indeed, the current results highlight that not all children adopted from institutions are equally affected by their experiences. As increased ADHD symptoms have been reported previously for PI children adopted at older ages (e.g., Rutter,

Kreppner, & O'Connor, 2001), in the current study age at adoption differed across the two groups of PI children such that those with ADHD as a group were adopted older after having spent a longer time in the institution. Loman & Gunnar (2010) proposed that low caregiver interaction, such as that experienced within institutions, results in chronic stress for the young child. This stress system activity then biases the developing threat system to organize larger defense responses. The resultant overactivity of both the stress and threat response systems affects the development of neural regulatory systems and hence increases the risk for a behavioral regulation disorder. PI children may be at particular risk for developing regulatory difficulties because the caregiving regulatory system is suggested to have a larger influence earlier in life (Loman & Gunnar, 2010). Correspondingly, evidence from animal models has linked prolonged stress to architectural changes in the prefrontal cortex (see review Arnsten, 2009). For example, rat pups with a history of extensive maternal deprivation were found to have enduring alterations in prefrontal dendritic outgrowth (Pascual & Zamora-Leon, 2007). Indeed, this result in particular supports assertions that it may be more difficult for developing systems to reorganize after exposure to severe or prolonged periods of deprived nurturance (Loman & Gunnar, 2010). Importantly, demonstration of behavior and functioning viewed as impaired within the context of typical development may have been adaptive during an earlier developmental period. As suggested by Gatzke-Kopp (2011), delay aversion preferences for the sooner, smaller reward may be adaptive in unstable environments wherein immediate reward is of higher certainty than a delayed potential reward and the goal is immediate survival. Similarly, disinhibited social behavior may be adaptive in the revolving caregiving environment of an institution when not being

selective in interactions allows for some form of a relationship with whoever is present (Rutter, O'Connor, & the English and Romanian Adoptees (ERA) Study Team, 2004). The current finding of the PI-As having resided in institutions for longer lengths of time supports previous literature noting that "early life experience can have long-term phenotypic consequences" (Crespi & Denver, 2005, p. 14).

Stress early in life similarly has also been reported to have an effect on dopamine neurophysiology, with evidence suggesting that chronic stress can be associated with low dopamine functioning (Gatzke-Kopp, 2011). Hypodopaminergic functioning has been reported to result in less willingness to expend effort for a reward and is consequently associated with a preference for an immediate over a delayed reward, even if the delayed reward is larger (Gatzke-Kopp, 2011; Phillips, Walton, & Jhou, 2007). Notably, the current findings of PI-A males demonstrating this same type of preference appear to be in line with this evidence from animal models. On the other hand, it is similarly possible within the current study that the PI-As were genetically predisposed to demonstrate ADHD behaviors and then the experience of early deprivation further elicited, or perhaps amplified, the behaviors. Stevens et al. (2009), for example, examined genes involved within the dopaminergic system (DRD4 and DAT1) within a sample of Romanian PI children. Elevated levels of ADHD symptoms were noted for children with a history of extended institutional care (i.e., $\geq$ 6months) carrying the DAT1 risk haplotype (Stevens et al., 2009). Future studies investigating gene by developmental history interactions within samples of PI children with ADHD will aid in understanding why it is that this behavioral profile develops.

Beyond the models focusing on the pre-adoptive environment of PI children, it is also possible that current or post-adoptive environmental factors may play a role in which PI children demonstrate deprivation-related ADHD. Importantly, in the current sample, groups did not differ in terms of family-level demographics including parental education and household income. These factors have each been linked to the provision of a supportive and enriching environment for children (Bradley et al., 2000). At the same time, Audet and LeMare (2011) have reported that both interactions between current authoritarian parenting style and duration of institutionalization, and between attachment ratings and duration of institutionalization, predict inattentive and overactive behavior in their sample of Romanian children adopted into Canada. Specifically, authoritarian parenting was associated with more behavior problems for children with mild deprivation (i.e., less than 5 months) and fewer behavior problems for children with severe deprivation (i.e., greater than 48 months). Attachment ratings were negatively associated with inattentive and overactive behaviors for children with less than 19 months of deprivation and were not associated for those with more than 19 months (Audet & Le Mare, 2011). A limitation of the current study is the lack of a measurement of the post-adoptive caregiving environment. Future studies will benefit from including assessment of parenting and attachment within investigations of deprivation-related ADHD, particularly when considering variability in which PI children demonstrate deprivation-related ADHD.

There are several limitations to consider when interpreting the results of this study. The reported findings are most notably limited by the small number of participants that completed the Phase 2 (i.e., laboratory) portion of the study. However,

electrophysiological studies often have smaller sample sizes. This methodology was coupled here with recruitment within unique populations of PI and children with ADHD without co-occurring genetic conditions or medically diagnosed, or facial features associated with, Fetal Alcohol Spectrum Disorder. Nevertheless, even with this sample size, previously reported patterns of behavior were replicated. In addition, a larger sample would particularly allow for more definitive conclusions regarding the level of impairment of PI-As relative to the NA-As. For example, although inspection of the means across the majority of the executive function tasks suggest that PI-As may have the most impairment in the functioning across the groups, this was of course not confirmed statistically.

Conclusions drawn from these current results are also somewhat limited because there was no inclusion of a non-adopted, without ADHD group. Inclusion of this additional comparison group could allow for more robust comparisons to determine similarities in the ADHD samples to each other relative to others and better document impaired functioning relative to "healthy controls." Notably, the majority of the measures utilized within the current study have been included in studies of PI children versus non-adopted, community controls and their performance relative to each other is well-documented. Given that many of the current behavioral tasks were in fact identical to previous studies, comparison with the findings from those studies is also possible. Additionally, the behavioral tasks included in the current study are those that have been well-documented as distinguishing samples of children with ADHD from those without ADHD (e.g, Sonuga-Barke et al., 2008; Willcutt et al., 2005). Moreover, within the current study, the scores from the standardized questionnaires are based on normative

data and thus provide some indication of functioning relative to non-clinical samples. Standardized norms are also available for some measures from the CANTAB; therefore, future analyses could benefit from examination of group performance relative to those norms.

Another potential limitation within the sample is that the PI-N group is primarily female. Detecting and interpreting gender effects or interactions involving males may be limited within the current sample. Despite trying to recruit more males meeting PI-N criteria, there was a smaller pool of male participants available from Phase 1 as females were more represented within those participants as well. Therefore, the higher percentage of PI-N females completing Phase 2 participation may accurately reflect the distribution of males versus females among the PI adolescents.

Finally, the initial design of this study also did not include investigation into differences into the proportion of each of the groups obtaining scores on the standardized questionnaires (i.e., Conners 3S and BASC-2) within the clinical range. Although visual inspections of group means falling within the clinical range were possible, it may actually be more beneficial to know whether groups differed statistically in the number of individuals meeting clinical criteria. Such an investigation would allow for a better understanding regarding what are problem behaviors across each of the groups and if a particular behavior/set of symptoms is associated with more impairment for one group than another. Future studies will benefit from this type of analysis to aid in the understanding of the clinical profile of deprivation-related ADHD and also allow for a more thorough investigation into potential different types of deprivation-related ADHD and consideration of individual differences.

Future Directions

Ultimately, this early description of the deprivation-related ADHD profile has implications for both identification and treatment. Although not reflected in the literature to date, intervention efforts, both for behavior and medication, designed for standard ADHD may not be effective for these children with histories of early deprivation. Stimulant medications, for example, are often used to treat standard ADHD; however, parents of PI children with ADHD symptoms have reported that these medications are not beneficial (M. Kroupina, personal communication, May 15, 2012). A review of treatment of other samples of children with developmental disorders with comorbid ADHD-like behaviors (e.g., genetic disorders, developmental delay, pervasive developmental disorders) indicated that although stimulants are often prescribed, side effects are often more burdensome (Rowles & Findling, 2010). In addition to pharmacological treatment, the use of behavioral intervention has been supported in the standard ADHD literature (e.g., Fabiano et al., 2009; Pelham & Fabiano, 2008). Evidence-based models of behavioral parent training focus on generally teaching parents to work with their children on manipulating the antecedents (e.g., commands) and consequences (e.g., time out, rewards) of child behaviors to increase desired behavior (e.g., compliance, self-monitoring). While portions of this type of individualized behavioral intervention will likely be an effective part of treatment for deprivation-related ADHD, incorporating components from treatments of the indiscriminate form of reactive active disorder will likely be beneficial. This may include focus on the child forming positive interactions with caregivers (Boris & Zeanah, 2005) and on effectively detecting threat and safety (Schuengel, Oosterman, & Sterkenburg, 2009). There is much need for

research focusing on testing interventions designed explicitly for post-institutionalized children and their families.

This study is one of the first to directly compare post-institutionalized children with ADHD to non-adopted children without ADHD. The current findings support previous research (e.g., Carlson et al., 1995) outlining the role of the caregiving environment in the development of regulatory difficulties and suggest that high-quality caregiver-child interactions support the development of attention regulation, and potentially reward processing, systems. They additionally highlight the need for future research to examine which specific aspects of early experiences support the development of attention, regulatory, and reward-processing systems. In addition, the PI literature on reward processing is in its infancy. The current finding of a strong delay aversion for PI-A male adolescents emphasizes the need to expand upon the current knowledge of how early deprivation relates to reward processing and motivation. The replication and expansion of group differences in ERP brain activity combined with the previous results of differential relative alpha and theta powers for both PI children and children with ADHD indicate that further investigation incorporating resting electroencephalogram (EEG) power will assist in further defining the profile of deprivation-related ADHD.

Future studies will also benefit from incorporating magnetic resonance imaging into investigations of deprivation-related ADHD. The few neuroimaging studies to date suggest an impact of early deprivation on prefrontal networks and reward processing neural circuitry (Bauer et al., 2009; Chugani et al., 2001; Eluvathingal et al., 2006; Mehta et al., 2009; Mehta et al., 2010). The current results particularly highlight the need to investigate the functional circuitry associated with reward sensitivity in deprivation-

related ADHD. Incorporation of structural magnetic resonance imaging into the comparisons of children with deprivation-related ADHD to those with standard ADHD will also be useful. A very recent study (i.e., Sheridan, Fox, Zeanah, McLaughlin, & Nelson, 2012) investigating the neural development of institutionalized Romanian children (i.e., BEIP study) found that cortical white matter was smaller for the children who remained in care as usual within the institution; however, it did not differ between the previously institutionalized children placed into foster care and the never institutionalized children. While the authors speculate that these findings may be indicative of "catch-up" following time in a more enriching environment for previously institutionalized children, they do not directly address these findings within the context of increased attention problems for these children. Yet, studies of children with ADHD, on the other hand, have identified that individuals with ADHD have disrupted white matter relative to controls (e.g, Nagel et al., 2011). Therefore, investigation into the cortical white matter of PI-A relative to PI-N and NA-A could be quite informative in understanding of the neurobiological signature of both early institutional care and deprivation-related ADHD. A pre-post design of comparing individuals first at the time of adoption and then again after years within the enriching post-adoption environment could also be beneficial to determine whether, and if so how, there is differential "catch-up" across the groups.

Finally, since it is possible that genes associated with ADHD risk may correlate with risk of being abandoned and placed in an institution, future studies must also incorporate consideration of possible genetic contributions and possible interactions between genes and developmental history. The study by Stevens et al. (2009) offers a promising

glimpse into furthering the understanding of deprivation-related ADHD; however, further study may benefit from inclusion of genes beyond those involved in the dopaminergic system. While gene by developmental history interaction studies will further aid in the understanding of deprivation-related ADHD, findings may be incomplete as it is possible that the environmental effects may mask genetic effects, or that the interactions may be due to other hidden factors (Plomp, van Engeland, & Durston, 2009). Interaction focused studies also fail to provide information about mechanism. Therefore, research designs which include both neuroimaging measures and genetics will be crucial within future studies of PI children. As first described by Durston et al. (2008), such studies will be beneficial in understanding of how different neurobiological and cognitive processes work together in the etiology and course of the behavioral symptom profile of ADHD, both for typically and atypically developing samples.

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Appendix: Tables

Table 1.

Means for Each Symptom Cluster by Group and Effect Sizes (Cohen's d) for the Differences Between Internationally Adopted and Non-adopted Children (From Wiik et al., 2011)

	Parent report					Child report				
	Marginal means			Effect size		Marginal means			Effect size	
	PI	EA/FC	NA	PI vs. NA	EA/FC vs. NA	PI	EA/FC	NA	PI vs. NA	EA/FC vs. NA
ADHD	$.25_a$	$.15_b$	$.13_b$	.46	–	$.44_a$	$.40_a$	$.33_b$	.27	.20
Clinical percentage	22.7	5.6	2.7			19.7	7.0	8.3		
Externalizing	$.12_a$	$.10_a$	$.07_b$	.29	.19	$.26_a$	$.25_a$	$.19_b$	.20	.18
Clinical percentage	9.0	9.6	1.3			13.6	7.0	4.1		
Internalizing	$.13_a$	$.12_a$	$.09_b$	.25	.17	$.36_a$	$.33_{a,b}$	$.29_b$	.19	–
Clinical percentage	11.9	9.7	2.7			15.2	4.2	4.1		

Note: Means with different subscripts are significantly different at $p < .05$. Mean comparisons control for age, sex, family income, average parental education, and enrichment. Effects of child age, sex, parental education, income, and enrichment are controlled before effect sizes are calculated. Effect sizes are shown for significant contrasts.

Table 2.

Descriptive Statistics for Group Background Characteristics

	PI-A *n* = 26 (11 females) M (SD)	PI-N *n* = 20 (18 females) M (SD)	NA-A *n* = 19 (8 females) M (SD)
Age (years) at session	13.0 (1.3)	14.0 (0.9)	13.8 (1.2)
Age (months) at adoption	36.2 (23.1)	19.5 (14.7)	-
Median age (months) at adoption	37.0	37.0	-
Age at adoption range	11-96	10-66	-
Time (months) in institution	24.9 (12.3)	15.8 (11.3)	-
Median time (months) in institution	13.5	12.0	-
Time in institution range	9-54	6-54	-
Region of origin (*n*)			
Eastern Europe	18	6	-
Asia	7	13	-
Central & South America	0	1	-
Other (Ethiopia)	1	0	
WASI			
Vocabulary (T-Score)	45.8 (11.5)	53.5 (12.0)	52.8 (9.6)
Matrix Reasoning (T-Score)	47.5 (10.1)	53.3 (8.2)	51.6 (7.7)
FSIQ-2 (Standard Score)	95.4 (14.1)	106.3 (14.2)	101.4 (13.5)

Table 3.

ADHD Symptoms, Associated Features, Severity, and Co-occurring Emotional and Behavioral Problems across Groups

	PI-A M (SD)	PI-N M (SD)	NA-A M (SD)
Reported ADHD Symptoms***			
# Inattentive	7.08 (2.3)	.95 (1.3)	7.63 (2.0)
# Hyperactive/Impulsive	4.36 (2.70	.40 (0.8)	3.74 (2.8)
Conners 3S Parent-report (T-score)			
Inattention***	75.90 (9.4)	49.90 (8.5)	78.05 (8.6)
Hyperactivity/Impulsivity***	76.67 (15.2)	49.65 (7.9)	77.21 (13.4)
Executive Functioning***	68.71 (14.0)	51.00 (7.8)	74.63 (11.3)
Learning Problems	69.38 (15.5)	54.85 (13.1)	64.37 (13.7)
Aggression/Defiance	59.90 (16.1)	52.15 (12.6)	64.84 (15.8)
Peer Relations	71.57 (18.9)	57.70 (14.4)	71.05 (17.6)
Conners 3S Self-report (T-score)			
Inattention	65.90 (13.1)	53.55 (11.6)	63.90 (10.0)
Hyperactivity/Impulsivity	66.76 (12.7)	60.35 (11.9)	63.00 (10.3)
Learning Problems	60.67 (14.9)	55.65 (12.4)	56.42 (9.3)

Aggression	56.80 (15.9)	53.30 (12.5)	57.53 (9.7)
Family Relations	54.24 (12.7)	50.10 (6.9)	57.16 (12.2)
SNAP-IV severity**	1.56 (0.6)	0.45 (0.4)	1.71 (.5)
BASC-2 Parent report (T-score)			
Hyperactivity**	67.77 (11.3)	50.65 (7.6)	67.71 (14.4)
Aggression	55.55 (11.8)	47.94 (7.8)	58.41 (8.7)
Conduct problems	58.91 (13.2)	49.59 (7.6)	59.71 (12.6)
Anxiety	54.86 (17.2)	51.94 (9.3)	59.18 (12.1)
Depression	56.18 (11.5)	50.71 (8.0)	61.00 (8.8)
Somatization	48.05 (10.9)	45.24 (9.2)	58.47 (16.8)
Atypicality**	59.32 (12.6)	45.00 (4.6)	59.47 (11.2)
Attention Problems***	62.68 (6.7)	45.35 (7.2)	63.82 (8.5)
Withdrawal	53.32 (6.7)	51.12 (11.4)	58.82 (10.3)
BASC-2 Child report (T-score)			
Atypicality	54.47 (14.0)	50.56 (12.9)	48.29 (7.1)
Locus of control	52.35 (9.9)	45.31 (6.0)	55.06 (12.3)
Social stress	52.53 (9.6)	46.94 (8.1)	51.76 (10.8)
Anxiety	50.74 (11.1)	47.94 (9.8)	51.71 (10.2)

Depression	49.29 (12.1)	44.19 (4.7)	48.88 (11.2)
Sense of inadequacy	53.26 (11.7)	42.31 (6.6)	55.00 (13.2)
Somatization	52.62 (16.5)	46.56 (8.7)	57.00 (14.7)
Attention Problems	55.40 (11.5)	45.19 (9.1)	58.35 (12.9)
Hyperactivity	57.16 (14.0)	47.94 (11.8)	57.76 (8.8)

Note. Significant differences in group means were due to the PI-N group differing from both the PI-A and NA-A groups. For the Conners 3S and BASC-2, T-Scores ranging from 60-69 are considered "at-risk" and T-Scores >70 are considered to be "clinically significant."
***$p < .001$ **$p < .01$

Table 4.

Executive Function Task Performance for Each Group

	PI-A M (SD)	PI-N M (SD)	NA-A M (SD)
Go/No-go			
Go trial accuracy*	.78 $(0.3)_a$	.95 $(0.1)_b$	.88 (.2)
No-go trial accuracy	.62 (0.2)	.63 (0.2)	.65 (.2)
Reaction time (Correct go trials)$^{+}$	323.4 $(35.7)_{a,b}$	311.4$(35.4)_a$	340.5 $(38.4)_b$
Stop-Signal Task			
Stop trial accuracy^{++}	.45 $(0.2)_a$	.52 $(0.1)_b$	.49 $(0.1)_{a,b}$
Reaction time (Correct go trials)	444.4 (142.4)	414.0 (116.0)	415.7 (100.7)
SSRT$^{\infty}$	241.74 $(104.0)_a$	191.96 $(66.2)_b$	230.46 $(70.9)_a$
Spatial Working Memory			
Total errors*	39.40 $(17.8)_a$	24.68 $(13.9)_b$	31.68 $(13.9)_{a,b}$
Stockings of Cambridge			
# Problems solved in minimum moves	6.92 (1.5)	8.56 (2.1)	7.58 (1.9)
Initial thinking time	4154.65 (2869.1)	2901.66 (12310.7)	6977.4 (8041.2)

Note. Means with different subscripts are significantly different at $*p < .05$, $^{+}p = .055$, $^{++}p = .07$, $^{\infty}p = .08$

Appendix: Figures

Figure 1.

Accuracy for Each Group on Go and No-go trials of Go/No-go in Loman et al. (in press)

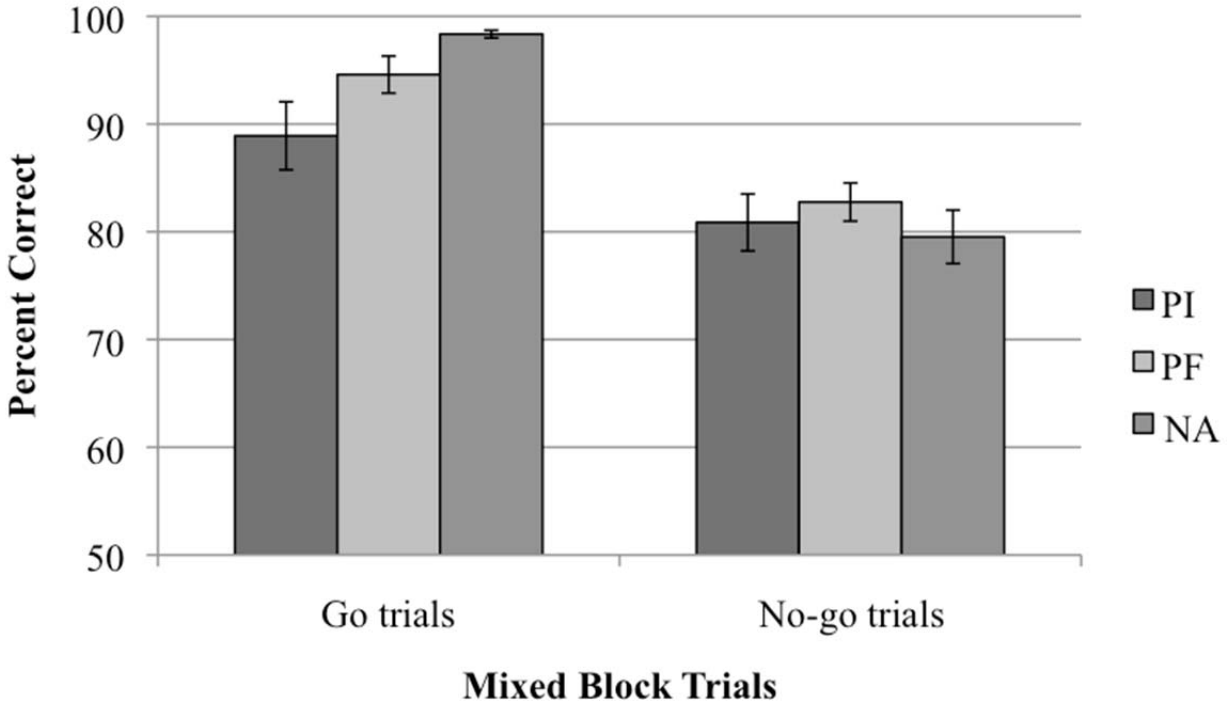

Figure 2:

Disinhibited Social Behavior for Each Group

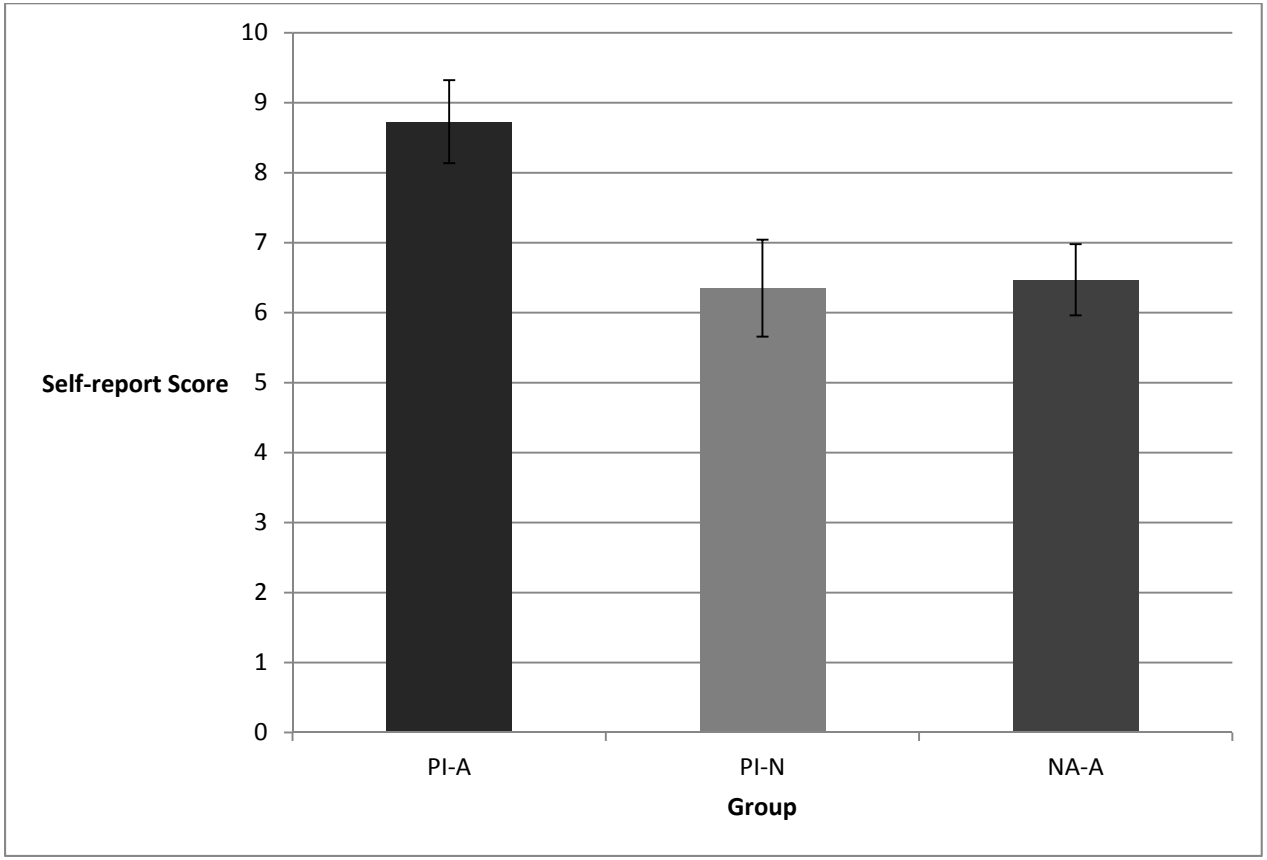

Figure 3.

Accuracy for Each Group on Go trials of Go/No-go

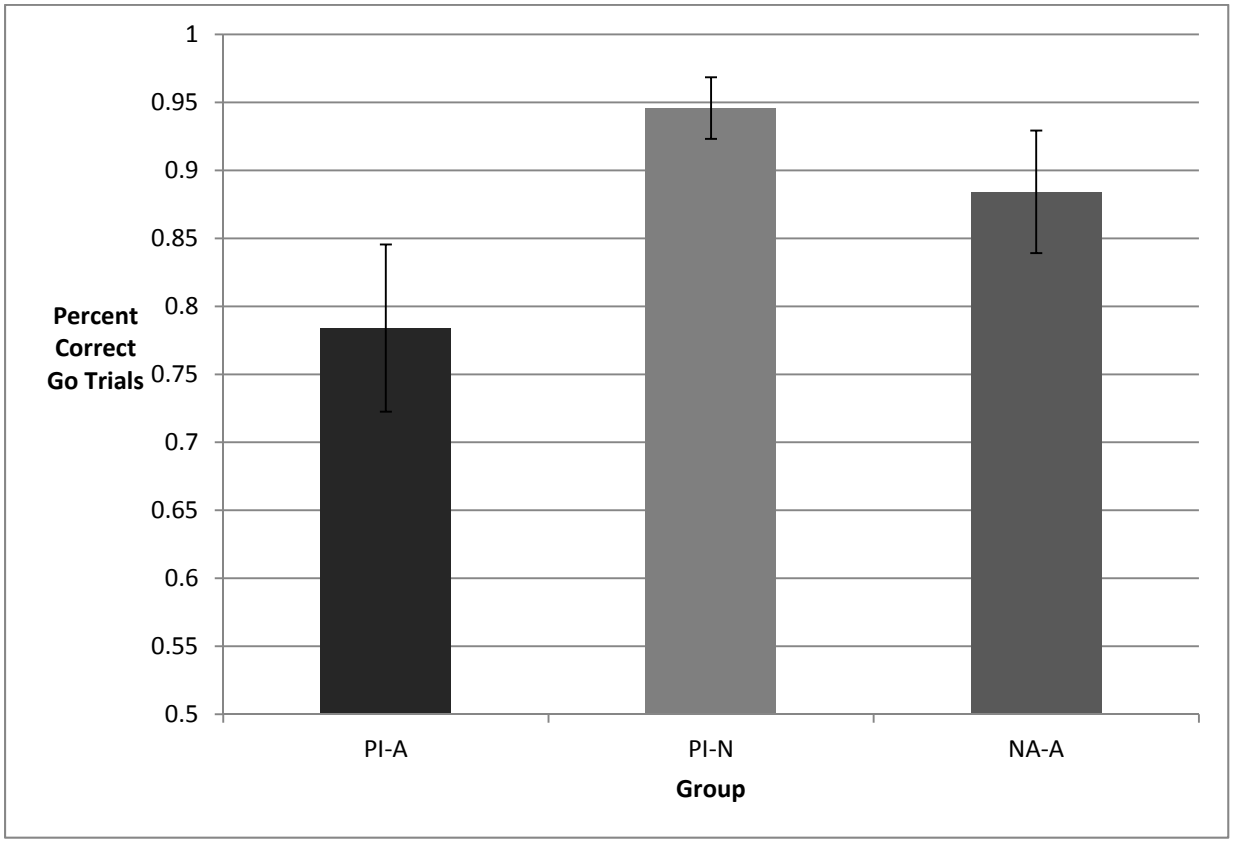

Figure 4.

Grand-averaged Group ERP Waveforms (Averaged No-Go and Go trials) at Electrode Site FCz for Males in Each Group with ADHD