

ADHD Symptoms, Executive Attention, and  
Electrophysiological Activity in Post-Institutionalized Children

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## Abstract

Prior research indicates children who experience early deprivation in institutional care have increased rates of attentional problems (e.g., ADHD) years after adoption. Limited and conflicting evidence suggests post-institutionalized (PI) children may demonstrate behavioral deficits on measures of attentional control, response inhibition, and conflict monitoring and resolution, capacities broadly referred to as executive attention. Previous research has documented increased concentration of electroencephalogram (EEG) relative power in low frequency (e.g., theta) bands in PI children, compared to never institutionalized children. Similar patterns of low frequency EEG power have been observed in individuals with ADHD, suggesting a potentially shared neurobiological correlate. The current study examined both behavioral and electrophysiological correlates of attention in PI children. Parent reported ADHD symptoms, behavioral performance on two computerized executive attention measures, and resting EEG power were collected in a sample of 5 ½-year-old internationally adopted PI children ( $n = 25$ ) and compared to same age non-adopted (NA) children ( $n = 33$ ). Resting EEG collected when the children were 18 months of age was examined in longitudinal analyses. Consistent with prior research, PI children had increased levels of ADHD symptoms, although most children's symptoms were below a clinical threshold. No group differences were found in behavioral performance on executive attention measures. Analysis of longitudinal EEG data indicated PI children demonstrated greater concentration of low frequency EEG power (e.g., theta) than NA children at 18 months of age, and this group difference remained at age 5 ½, despite the PI group having spent an

average of 4 ½ years in adoptive care. Contrary to hypotheses, concentration of frontal relative EEG power in the theta band at age 5 ½ was associated for PI children with more accurate performance on an executive attention task. In addition, in PI children increased concentration of low frequency frontal relative EEG power at age 18 months was associated with lower ADHD symptoms at age 5 ½ years. Interpretation of these results is limited by small sample size, particularly for the EEG analyses. However, these findings suggest the importance of considering brain-behavior relationships in respect to developmental context (e.g., resource poor vs. resource rich environments).

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## Introduction

### *Developmental Effects of Institutional Care: An Overview*

Scientific inquiry into the developmental effects of social and environmental deprivation provides the opportunity to explore fundamental questions regarding human development, plasticity, and resilience. The study of children who have spent time in institutional care settings offers an unfortunate, but unique “natural experiment” pertaining to the developmental impact of early adversity. Institutional care continues to be relatively common in many areas of the world. Often children are abandoned or placed into these settings due to the inability of their parents and families to provide care as the result of social, economic, or political pressures, illness, or death. In addition, many of these children experience numerous genetic, prenatal, and postnatal risk factors that may increase the likelihood of developmental difficulties. While in institutional care, children experience varying degrees of physical and social/emotional deprivation including a lack of necessary nutrition, medical care, and stimulation and interaction with the environment necessary to support cognitive, motor, and social development (Gunnar, Bruce, & Grotevant, 2000). In addition, a lack of consistent and long-term caregiving relationships is a fundamental characteristic of institutions, which are typically staffed by caregivers on a rotating schedule (Zeanah, Smyke, & Settles, 2006). The adoption of children from institutional care and their placement into adoptive families provides a distinct and measurable end to this deprivation. Within the United States, children internationally adopted from institutional care are a significant and growing population. More than 215,000 children were internationally adopted into the U.S. between 1998 and 2008 (U.S.

Department of State, 2009). Although not all of these children experienced institutional care, previous estimates suggest as many as 85% of internationally adopted children spend at least some portion of their pre-adoptive life in an institutional setting (Johnson, 2000).

Descriptive research on institutionalized children has documented significant adverse developmental effects of institutional care including delays in physical growth and cognitive functioning (Ames, 1997; Goldfarb, 1945; Johnson, 2000; O'Connor et al., 2000; Provence & Lipton, 1962; for review see Rutter, 1981; Rutter & the ERA Team, 1998; Smyke et al., 2007), as well as language development (Goldfarb, 1945; Provence & Lipton, 1962; Windsor, Glaze, Koga, & the BEIP Core Group, 2007). Social, emotional and behavioral difficulties such as disturbances in attachment behaviors (Zeanah, Smyke, Koga, Carlson, & the BEIP Core Group, 2005) and peer interactions (Vorria, Rutter, Pickles, Wolkind, & Hobsbaum, 1998) have also been observed.

Although there is considerable evidence for the harmful effects of institutional care, remarkable developmental catch-up typically occurs after children are removed from institutions and placed into family based care. Catch-up in physical growth and general cognitive abilities (i.e., IQ) proceeds rapidly, with many children demonstrating IQ within the broad normal range several years after adoption (Rutter & the ERA Team, 1998). However, despite this catch-up, post-institutionalized (PI) children as a group still lag behind their never institutionalized peers in numerous domains including physical growth (Cohen, Lojkasek, Zadeh, Pugliese, & Keifer, 2008; van IJzendoorn, Bakermans-Kranenburg, & Juffer, 2007), cognitive abilities (Beckett et al., 2006; Loman et al.,

2009), academic functioning (Beckett et al., 2007; Loman et al., 2009), and social functioning (Hodges & Tizard, 1989). Increased rates of psychiatric disorders have also been found in institutionalized and PI children compared to community controls (Zeanah et al., 2009). Particularly strong evidence is present for a link between institutional care experience and an increased risk of Attention-Deficit/Hyperactivity Disorder (ADHD).

#### *Inattentive and Hyperactive-Impulsive Symptoms Following Institutional Care*

Difficulties with inattention and hyperactivity-impulsivity have been repeatedly observed in studies of institutionalized children (Provence & Lipton, 1962; Roy, Rutter, & Pickles, 2000; Vorria et al., 1998), including children who experienced high quality institutional care (adequate nutrition, stimulation, low caregiver to child ratio) (Tizard & Rees, 1974). Problems with inattention and hyperactivity-impulsivity appear to remain years after adoption and have been documented across multiple samples of PI children (Ames, 1997; Chugani et al., 2001; Gunnar et al., 2007; Stevens et al., 2008; Wiik et al., in press; Zeanah et al., 2009). Consistent with these findings, caregiver reported rates of ADHD diagnosis are significantly elevated in PI children (Beverly et al., 2008: 42%; Le Mare & Audet, 2002 as reviewed by Maclean, 2003: 29%, Zeanah et al., 2009: 19%) compared to never institutionalized children. The prevalence and persistence of these symptoms has led researchers working with a well known longitudinal sample of Romanian PI children [the English and Romanian Adoptees (ERA) study] to argue that inattention and overactivity are part of an “institutional deprivation syndrome” (Kreppner et al., 2001), which also includes problems with attachment, quasi-autistic symptoms, and cognitive impairment (Rutter, Kreppner, O’Connor, & the ERA Study Team, 2001).

However, recent follow-up of the ERA sample suggests that ADHD is prevalent (> 20% of the sample) in PI adolescents (> 6 months of institutional care) regardless of whether they demonstrated other deprivation specific patterns (i.e., disinhibited attachment, quasi-autistic symptoms) earlier in childhood (Sonuga-Barke et al., 2009).

Research indicates that inattentive and hyperactive-impulsive symptoms in children with institutional experience are not solely accounted for by IQ (Roy, Rutter, & Pickles, 2000), and may instead represent institutional effects distinct from general cognitive impairment. Risk for inattentive and hyperactive-impulsive symptoms has been positively associated with time in institutional care in the ERA study (Kreppner et al., 2001). However, recent research in this sample suggests a possible threshold effect for duration of institutionalization. Although the precise timing of this threshold has yet to be identified, it appears that institutional care during the first 6 to 24 months of life significantly increases risk for inattention and hyperactivity-impulsivity even after prolonged time in the adoptive home (Stevens et al., 2008). Additional support for a threshold effect is observed in research indicating no difference in ADHD rates at 4 ½ years of age between still institutionalized children and PI children who had been placed into foster care at a mean age of 23 months (Zeanah et al., 2009). It should be noted that the findings reported by Zeanah et al. (2009) reflect a carefully designed large scale research project based in Bucharest, Romania [the Bucharest Early Intervention Project (BEIP)] which randomly assigned children into foster care placement. When examined together, these findings strongly suggest that experience in institutional care during the first two years of life is associated with increased risk for difficulties with attention and

self-regulatory abilities, although the precise mechanism of these effects remains to be identified. In addition, the persistence of these difficulties years after adoption indicates that developmental and neurobiological deficits underlying these symptoms may not be fully ameliorated by the general intervention of placement into family based care.

#### *Executive Attention: Development, Neural Circuitry, and Stress Vulnerability*

Infancy and early childhood are crucial periods for the development of basic self-regulation skills including abilities for attentional control, inhibition, and monitoring and resolution of conflict; all of which are capacities associated with executive attention (Posner & Rothbart, 2000; Rueda, Posner, & Rothbart, 2005). Physiologic regulation of arousal (e.g., sleep, wake) begins prenatally and has important influence on attentional functioning throughout life. In early infancy, attentional control skills begin to emerge as infants learn to regulate distress, a process that may be fostered by soothing methods typically utilized by caregivers (e.g., holding, rocking, engaging visual attention). Young infants' ability to orient to and disengage from a stimulus is one of the early achievements in attentional regulation, with significant increases in these abilities evident by 3 to 4 months of age (Posner & Rothbart, 1994; 1998 for review). More complex executive attention abilities such as inhibition and conflict monitoring and resolution develop into early childhood (Gerstadt et al., 1994; Posner & Rothbart, 2000; Rueda et al., 2004).

Arousal and attention functions are supported by multiple neural circuits involving structures in the brainstem, limbic system, and cerebral cortex (Geva & Feldman, 2008; Jones, 2003). Numerous neurotransmitters including glutamate,

acetylcholine, norepinephrine, dopamine, and serotonin contribute to the functioning of these brain systems (Jones, 2003). Recent neuroimaging research has attempted to pinpoint specific structures associated with executive attention. The results of this work indicate that the anterior cingulate cortex (ACC) and lateral prefrontal cortex are active during completion of tasks requiring executive attention (Fan, Flombaum, McCandliss, Thomas, & Posner, 2003; Posner & Rothbart, 2007). The ACC is believed to play a greater role in the monitoring and detection of conflict, while activation in the lateral prefrontal cortex may be more closely related to the resolution of conflict (Rothbart & Rueda, 2005). The content of the stimuli has also been found to be important in regard to the neural activation associated with executive attention. For example, Bush, Luu, and Posner (2000) review neuroimaging evidence for differential activation in portions of the ACC based on whether a task requires cognitive or emotional regulation, with the dorsal portion of the ACC being associated with cognitive demands and regulation and the ventral portion of the ACC being associated with emotional processing and regulation. In regard to developmental changes, research suggests that brain activity associated with executive attention becomes more focal with maturation, as children display greater cortical activation as measured by fMRI and broader activation as indexed by ERP when performing tasks requiring executive attention than do adults (Rothbart & Rueda, 2005; Rueda et al., 2005).

Although the impact of early adversity on brain regions associated with executive attention (e.g., ACC, prefrontal cortex) has not been fully explicated, their extended period of postnatal development suggests they may be particularly vulnerable to stress

and adversity in early life (Teicher et al., 2003). Consistent with this hypothesis, evidence of neuronal loss in the ACC has been found in maltreated children with PTSD (De Bellis, Keshavan, Spencer, & Hall, 2000). Negative effects of stress on the development and functioning of the prefrontal cortex have also been well documented (Arnsten, 2009; Sanchez, Ladd, & Plotsky, 2001). In addition, alteration in neurotransmitter systems, including dopaminergic functioning has been associated with stress (Pani, Porcella, & Gessa, 2000) and observed in non-human primates following maternal deprivation (Sanchez et al., 2001). Although further understanding of the impact of these stress related changes in neurotransmitter functioning is needed, it is notable that dysregulation of catecholamines (dopamine, norepinephrine) has been clearly implicated in ADHD (Pliszka, 2005; Prince, 2008). Therefore, it appears plausible that stress induced changes in brain structures (ACC, prefrontal cortex) and neurotransmitter systems (DA, NE) may provide one pathway toward problems with executive attention and inattentive and hyperactive-impulsive symptoms in PI children.

#### *Behavioral Measures of Executive Attention in PI Children*

The majority of literature to date regarding inattentive and hyperactive-impulsive symptoms in institutionalized and PI children has relied on parent/caregiver reports, teacher reports, and behavioral observations of behavior. However, recently researchers have become increasingly interested in utilizing behavioral tasks (e.g., go/no-go tasks, flanker tasks) designed specifically to assess executive attention capacities including inhibition, attentional control, and conflict monitoring and resolution. Go/no-go tasks require that participants respond to the majority of stimuli (go trials) while inhibiting

their prepotent response to specific non-targets (no-go trials), thus offering a measure of response inhibition. Flanker tasks require response to a centrally located target stimulus, which is flanked on either side by congruent or incongruent stimuli (e.g., Fan, McCandliss, Fossella, Flombaum, & Posner, 2005). Thus, successful completion of a flanker task requires attentional control, monitoring and resolution of conflict.

Limited research completed with these executive attention tasks suggests that children with institutional care experience display poorer performance relative to comparison children, although there is conflicting evidence as to the precise pattern of these deficits. Research with 10 and 11-year-old children indicated that compared to never institutionalized controls, PI children demonstrated poorer sustained attention on a letter go/no-go task as measured by accuracy to target (go) trials. However, PI children did not differ from controls in their accuracy to no-go trials, a measure of response inhibition (Loman, 2009). In contrast, an additional sample of PI children in this 10 to 11-year-old age range was found to demonstrate greater difficulty with response inhibition on letter and emotion go/no-go tasks compared to controls (E. Zobel, personal communication, July 18, 2008). It is notable that this group effect was present on the emotion go/no-go task only for commission errors to non-target (no-go) neutral faces, but not for non-target sad or angry faces, suggesting that emotional information may facilitate PI children's response inhibition. However, this hypothesis is not supported by the findings of Tottenham and colleagues (2010) who reported that PI children performed more poorly than controls on an emotion go/no-go task with angry, fearful, sad faces, with no group difference for trials with happy faces (Tottenham et al., 2010). These

conflicting results suggest that further study is needed to determine whether or not PI children as a group display deficits in sustained attention and response inhibition, as well as the pattern of these deficits in regard to non-emotional (e.g., letter) and emotional information.

Results of research utilizing flanker tasks suggest that institutional care is associated with poorer conflict monitoring and resolution. For example, when compared to never institutionalized controls, institutionalized children and PI children living in foster care as part of the BEIP study were found to demonstrate poorer accuracy and slower reaction time on a flanker task at 8 years of age (Fox, McDermott, Vanderwert, Nelson, & Zeanah, 2008). Ten and 11-year-old PI children were also found to demonstrate poorer accuracy to both congruent and incongruent flanker trials (Loman, 2009). However, reaction time on a flanker task was not found to differ between 6 to 11-year-old PI children and community controls (Chapman et al., 2009). Analyses of group differences in accuracy to congruent and incongruent trials were not reported in this sample. Although research to date is suggestive of impairments in PI children's performance on measures of conflict monitoring and resolution, further research is needed to more clearly elucidate the impact of early adversity on these skills.

#### *Neurobiological Correlates of Early Adversity and Executive Attention in PI Children*

In addition to the increasing use of laboratory-based measures of executive attention, there is growing interest in identifying neurobiological correlates of these abilities in PI children. However, much of the neuroimaging research to date has focused on documenting associations between a history of institutional care and alterations in

brain structure and circuitry more broadly. One of the earliest neuroimaging studies of PI children was completed with positron emission tomography (PET). This study compared a group of 10 PI children with adults and epileptic children and found alterations in glucose metabolism in the brain stem, lateral temporal cortex, medial temporal structures (hippocampus and amygdala), infralimbic prefrontal cortex, and the orbital frontal cortex of the PI children (Chugani et al., 2001). The PI children also demonstrated difficulties with impulsivity and attention, as well as other mild cognitive deficits. More recent research using diffusion tensor imaging (DTI) by the same research group (Eluvathingal et al., 2006) compared a small sample of PI ( $n = 7$ ) and control ( $n = 7$ ) children and found evidence in the PI group of an underdeveloped left uncinate fasciculus, a white matter tract connecting the anterior temporal lobe and frontal lobe. Eluvathingal et al. (2006) hypothesized that this underdevelopment may be due to inadequate stimulation of this pathway during a sensitive period of brain development. The PI children in this sample were also found to demonstrate increased impulsivity, although the relationship between these structural and behavioral findings requires further investigation. Additional evidence of structural brain differences in PI children was provided by Tottenham et al. (2010) who found increased adjusted amygdala volume in PI children adopted from orphanage care at 15 months of age or older, compared to either PI children adopted before 15 months of age or community controls. A significant positive association was present between age at adoption from institutional care and amygdala volume, and this association was not explained by IQ or anxiety disorder diagnosis. Larger amygdala

volume was also found to be related to poorer performance on an emotional go/no-go task utilizing negative emotional faces (Tottenham et al., 2010).

Turning to neurobiological correlates of executive attention more specifically, a recent functional MRI study reported that PI girls adopted from Eastern Europe displayed greater neural activation in the ACC, left cerebellum, medial frontal gyrus, and superior frontal gyrus during completion of a flanker task than did community controls. Notably, no such group difference was found for PI girls adopted from China (Chapman et al., 2009). On average, the Eastern European adoptees were adopted at a later age (mean = 24.9 months) than were the Chinese adoptees (mean = 18.5 months), suggesting that duration of institutionalization may have contributed to these group differences. However, other variables (e.g., genetic factors, reason for placement in institutional care) may also co-vary with region of origin. Therefore, additional research is needed before such region of origin differences can be fully understood. Recordings of event-related potentials (ERPs) have also been used to study neurobiological correlates of executive attention in PI children. Returning to results of Fox et al. (2008) and the BEIP study discussed earlier, 8-year-old children with institutional care experience displayed lower amplitude error-related negativity (ERN) during completion of a flanker task than did controls. The ERN is believed to reflect error detection after an incorrect response and relate to correction on approaching trials (Rothbart & Rueda, 2005). Although neuroimaging research with PI children is currently limited to a small number of studies, it provides emerging evidence for associations between early institutional care and alterations in brain regions implicated in executive attention and emotion processing.

Recordings of resting electroencephalogram (EEG) provide further evidence for associations between early adversity and alterations in neurobiological correlates of brain function. However, appropriate interpretation of this research requires an understanding of typical developmental changes in EEG activity. EEG power is believed to reflect different functional correlates based on the frequency band, with increased EEG frequency being associated with greater alertness and cognitive activity. In typically developing populations, developmental change in EEG power has been found to occur from infancy through childhood, with a decrease in relative low frequency slow wave activity (delta and theta) and a corresponding increase in higher frequency (alpha and beta) activity (Clarke, Barry, McCarthy, & Selikowitz, 2001; Marshall, Bar-Haim, & Fox, 2002). Sex differences in EEG power have also been found in childhood, with males demonstrating less theta and more alpha power than females, suggesting a maturational lag in EEG power for females when compared to males (Clarke et al. 2001; Diaz De Leon, Harmony, & Marosi, & Becker, 1988; Harmony et al., 1988).

Developmental studies must also consider the metric (absolute power vs. relative power) in which EEG is reported and compared. Absolute power is believed to be more susceptible to changes in skull thickness and impedance and for these reasons the use of absolute power in developmental EEG research has been argued against (e.g., Benninger et al., 1984). Relative EEG power is the proportion of power occurring in a specific frequency band at a particular scalp site or region relative to the total EEG power across all frequency bands recorded at that scalp site or region. Because relative power is a ratio, changes in absolute power in any band will impact relative power across bands. Several

strengths of utilizing relative power rather than absolute power in developmental EEG research have been put forth in the literature. First, relative power has been found to have better test-retest reliability than absolute power (John et al., 1980). In addition, relative power is believed to be more sensitive than absolute power to developmental changes in the frequency composition of EEG over time (Clarke et al., 2001).

Limited prior research has demonstrated associations between psychosocial adversity and EEG power. Specifically, children 6 to 12 years of age with low socioeconomic status in Mexico and Venezuela were found to display a less mature pattern of EEG power (relative power concentrated at lower frequencies) compared to children from middle or high socioeconomic class (Harmony et al., 1988; Harmony et al., 1990). The authors hypothesized that low socioeconomic status may interfere with the provision of an adequate caregiving environment, particularly in regard to nutrition. Prospective longitudinal study of children in Mexico with high psychosocial risk (i.e., low parental education, many children in the family, multiple families sharing one home), but normal neurologic and medical history, indicated that at 18 to 30 months of age these high risk children displayed less mature patterns of EEG power (concentration of power at lower frequencies) than children with low psychosocial risk. Although these group differences in EEG power decreased over time, the pattern remained significant in frontal and occipital regions at 6 years of age (Otero, Pliego-Rivero, & Ricardo, 2003). In addition, a large scale study of adults demonstrated associations between early life stress and a significant decrease in overall power across the EEG spectrum in adulthood (McFarlane et al., 2005). Although replication of these findings is needed, this research

suggests that early adversity is associated with reductions in EEG power which remain over time.

Evidence for associations between early adversity and alterations in EEG power has also been observed in children with institutional care history. In the BEIP sample, Marshall, Fox, and the BEIP Core Group (2004) found increased low frequency (theta) absolute power at occipital leads and decreased high frequency (beta and alpha) absolute power in frontal and temporal sites in 5 to 31-month-old institutionalized children compared to never institutionalized community controls. Additionally, the institutionalized group demonstrated increased relative theta power in frontal, parietal, and occipital regions, with lower relative alpha power overall compared to community controls. The institutionalized group also displayed lower relative power in the beta band at occipital leads compared to controls (Marshall et al., 2004). Similar EEG findings of increased relative theta power and decreased absolute and relative alpha power compared to controls were observed in a sample of 18-month-old PI children who had spent an average of 6 months in their adoptive homes (Tarullo, 2008). These findings suggest that reduction in EEG power remains months after children are removed from institutional care.

Given the evidence for a developmental shift in EEG power toward concentrations at higher frequencies (e.g., alpha) (Marshall et al., 2002), the patterns of EEG power observed in PI children [concentration of power at lower frequencies (theta)] may reflect a maturational delay in brain development associated with institutional care. Consistent with this hypothesis, these patterns of EEG power have been found to shift

toward more typical patterns after prolonged time in family based care. Follow-up of the Marshall et al. (2004) BEIP sample revealed greater relative alpha power at 3 ½ years of age for children who spent 24 to 36 months in foster care, compared to children who remained in institutional care (Marshall et al., 2008).

Findings of reduced EEG power in institutionalized and PI children are particularly notable because highly similar patterns of EEG power (increased relative theta power and decreased relative alpha power) have been observed in individuals with ADHD (Barry, Clarke, & Johnstone, 2003), with maximal theta power often being found in frontal regions. The consistency of these EEG patterns in ADHD has encouraged some researchers to argue that patterns of EEG power, particularly in the frontal region may serve as a valuable diagnostic tool for ADHD when paired with a standard psychiatric evaluation (Quintana, Snyder, Purnell, Aponte, & Sita, 2007). However, other scientists caution against this usage of EEG power due to a lack of specificity (Bush, 2008). It remains unclear whether or not the concentration of EEG power in lower frequency bands observed in ADHD indicates general cortical immaturity consistent with a maturational delay model of ADHD (Rubia, 2007), or if it is indicative of developmental deviation or hypoarousal models of brain functioning in ADHD (Barry et al., 2003). Support for the maturational delay model of ADHD has emerged from recent neuroimaging evidence documenting a delay in cortical maturation (achievement of peak cortical thickness) in children with ADHD (Shaw et al., 2007).

The similarity in EEG findings across PI and ADHD samples and the high rates of ADHD symptoms in children with institutional care history suggests these populations

may share both electrophysiological correlates and behavioral outcomes. However, before this conclusion can be made, careful research is needed to specifically examine whether associations exist among ADHD symptoms, executive attention, and patterns of EEG power in PI children. Longitudinal research may be particularly useful in further elucidating the relationships among these variables.

### Objectives and Hypotheses

The current study aimed to address this gap in the literature by examining parent reported ADHD symptoms, behavioral performance on executive attention measures, and EEG power in a sample of 5 ½-year-old internationally adopted PI children and comparing the results to those found in same age non-adopted children. In addition, this study served as a longitudinal follow-up to collection of resting EEG completed at 18 months age in this sample (Tarullo, 2008). Both cross-sectional and longitudinal analyses were incorporated into the current study objectives.

The first goal of this study was to examine whether 5 ½-year-old PI children differed from same age comparison children in regard to 3 domains: 1) parent reported ADHD symptoms, 2) behavioral performance on two executive attention tasks, a go/no-go task and flanker task, designed to assess response inhibition, attentional control, and conflict monitoring and resolution abilities, and 3) EEG power, specifically in regard to relative theta and relative alpha power within the frontal region. Relative theta and relative alpha power were identified as EEG variables of interest based on prior research (Marshall, 2004; Tarullo, 2008) demonstrating associations between early adversity and increased relative theta power and decreased relative alpha power, as well as evidence for

similar patterns of relative power in ADHD samples (Barry et al., 2003). The frontal region was selected as the scalp region of interest due to its proximity to brain structures (lateral prefrontal cortex, ACC) believed to be associated with executive attention (Rothbart & Rueda, 2005) and prior documentation of altered relative EEG power patterns in this region for children with institutional care experience (Marshall et al., 2004) and children with ADHD (Barry et al., 2003).

Given the prior literature documenting increased ADHD symptoms in children with institutional care experience (Ames, 1997; Gunnar et al., 2007; Kreppner et al., 2001; Stevens et al., 2008; Zeanah et al., 2009), it was expected that the PI group would have greater parent reported levels of ADHD symptoms than the non-adopted children. Based on the limited research (Fox et al., 2008; Loman, 2009; Tottenham et al., 2010; E. Zobel, personal communication, July 18, 2008) regarding PI children's performance on executive attention tasks, it was anticipated that the PI group would demonstrate poorer performance on the go/no-go and flanker tasks compared to the non-adopted group. Considering the evidence for change toward more typical patterns of EEG power in PI children after 2 to 3 years in family based care (Marshall et al., 2008), it was expected that relative theta and relative alpha EEG power in the frontal region would be similar between the PI and non-adopted groups at 5 ½ years of age.

The second goal of the study was to investigate whether relative theta and relative alpha power in the frontal region at 5 ½ years of age were associated with parent reported ADHD symptoms and performance on behavioral attention tasks across and within the PI and non-adopted groups. Given prior research linking EEG power and ADHD (Barry et

al., 2003), it was hypothesized that concentration of frontal EEG power in lower frequency bands (e.g., theta) would be positively associated with parent reported ADHD symptoms, as well as poorer performance on executive attention tasks (go/no-go and flanker).

A third study goal was to examine longitudinal relationships between EEG power in infancy (at 18 months) and behavioral outcomes in early childhood (at 5 ½ years). If the concentration of EEG power in lower frequency bands observed at 18 months in the PI group (Tarullo, 2008) represented a simple *maturational lag* in brain development which disappears over time in the adoptive home, associations with behavior (e.g., ADHD symptoms, performance on executive attention tasks) later in childhood may not be expected. However, if these alterations in EEG power at 18 months of age represented a *developmental deviation* in PI children's brain development, they may indeed be associated with poorer behavioral outcomes in later life, even if more typical patterns of EEG power emerged over time in the adoptive home. To explore this question, analyses were completed to test for associations between frontal relative theta and frontal relative alpha power at 18 months of age and 1) parent reported ADHD symptoms at 5 ½ years of age, and 2) behavioral performance on executive attention tasks (go/no-go and flanker) at 5 ½ years of age, across and within participant groups.

### Method

#### *Participants*

Participants were 5 ½-year-old children recruited from 2 groups: 1) the *post-institutionalized (PI) group* ( $n = 25$ , 24 girls) who were internationally adopted into the

United States between 10 and 16 months of age and had spent 75% or more of their pre-adoptive life in institutional care, with no more than 2 months in family based care (e.g., foster home, relative care) and 2) the *non-adopted (NA) group* ( $n = 33$ , 27 girls) who were born in the United States and raised in their biological family. The PI and NA groups did not differ in age at assessment,  $t(56) = .52, ns$ , (PI:  $M = 5.60$  years,  $SD = .11$ ; NA:  $M = 5.58$  years,  $SD = .08$ ), family income,  $t(55) = .62, ns$ , parent education level,  $t(55) = .62, ns$ , or number of stressful life events while in their family environment,  $t(54.31) = 1.78, ns$ , as measured by the Child Life Events Scale (Coddington, 1972). In addition, groups did not differ on estimated verbal cognitive ability,  $t(54) = 1.50, ns$ , or estimated nonverbal cognitive ability,  $t(55) = .01, ns$ , as measured respectively by the Vocabulary and Matrix Reasoning subtests of the Wechsler Preschool and Primary Scale of Intelligence, Third Edition (WPPSI-III) (Wechsler, 2002). Both subtests have been found to correlate highly with Full Scale IQ (Sattler & Dumont, 2004).

Children in the PI group were primarily adopted from China (76%,  $n = 19$ ) with a limited number being adopted from Russia (16%,  $n = 4$ ), Ukraine (4%,  $n = 1$ ), and Guatemala (4%,  $n = 1$ ). PI children were adopted at a mean age of 11.84 ( $SD = 1.84$ ) months and spent an average of 94% ( $M = 11.44$  months,  $SD = 1.75$ ) of their pre-adoptive lives in institutional care. Overall, 20% of PI parents endorsed the belief that their child experienced a neglect of basic physical needs prior to adoption, while 64% endorsed the belief that their child experienced a neglect of basic social needs prior to adoption. No parent endorsed the belief that their child experienced physical or sexual abuse prior to adoption.

All participants were previously recruited for a laboratory assessment at 18 months (Tarullo, 2008). Sex distribution of the PI group reflects the children available for recruitment at the time of the 18 month assessment. The NA group was recruited to match the sex distribution of the PI group at the 18 month assessment. Attrition rates of 27% ( $n = 10$ ) and 28% ( $n = 13$ ) were present in the PI and NA groups respectively from the 18 month assessment to the 5 ½ year assessment. Major reasons for attrition were inability to locate and/or contact the family and an unwillingness to participate due to time demands, prior participation in research, or concerns regarding the child's ability or willingness to complete the assessment tasks.

#### *Procedures*

Post-institutionalized participants were recruited from the Minnesota International Adoption Project Registry, a list of families created through international adoption who expressed interest in research participation. Non-adopted children were recruited from the Institute of Child Development Infant Participant Pool, a list of community families interested in research participation. The initial collection of resting EEG was completed when children were approximately 18 months of age during part of a broader laboratory research assessment (Tarullo, 2008). All other measures described below were collected when children were 5 ½ years of age during a laboratory research session that lasted approximately 2 ½ to 3 hours. Additional measures (e.g., language, social cognition) were collected at the session, but are not included in the current analysis. In order to reduce the likelihood of group differences due to prenatal and congenital conditions rather than post-natal care experiences, children were excluded from the sample if they

were reported to have a known congenital condition or medically diagnosed Fetal Alcohol Spectrum Disorder. One non-adopted child was excluded from the sample due to congenital skull abnormalities (bioronial craniosynostosis) and prior occipital skull fractures. Photographic screening using the FAS Facial Photographic Analysis Software (Astley, 2003) was completed during the lab assessment. Digital pictures of participants were analyzed using the 2004 CDC guidelines regarding FASD associated facial dysmorphia (i.e., smooth philtrum, thin vermillion border, and small palpebral fissures). One PI child was excluded from the sample due to facial features associated with FASD.

### *Measures*

*Parent report of ADHD symptoms.* The parent report version of the MacArthur Health and Behavior Questionnaire (HBQ) (Armstrong, Goldstein, & the MacArthur Working Group on Outcome Assessment, 2003) was administered. The HBQ provides measures of social, emotional, and academic functioning. The ADHD Symptoms scale and its component subscales, the Inattention subscale and the Impulsivity subscale were analyzed. A clinical threshold for the ADHD Symptoms scale has been identified in previous literature (Luby et al., 2002) and was utilized to identify participants with parent reported ADHD symptoms in the clinical vs. subclinical range.

*Go/no-go task.* Participants completed an animal themed go/no-go task designed to index sustained attention and response inhibition. Participants were directed to press a button to all animal pictures except for one non-target animal (monkey). This task has been used previously with young children (J.M. McDermott, personal communication). Each stimulus was presented for 700 msec followed by a black screen for 2300 msec.

Participants' responses were recorded at any time during the 3000 msec following onset of the stimulus. The trial ended when the child pressed the button or upon completion of the 3000 msec response window. Intertrial intervals were 500 msec, during which time a black screen was presented. Targets (go stimuli) were presented with a 75% probability. No-go stimuli were presented following either 1, 3, or 5 target (go) stimuli. Participants completed a practice session consisting of 12 trials. A total of 120 test trials were then administered in two blocks of 60 trials (45 targets/go, 15 no-go) (see Figure 1). Order of block administration was balanced between groups. One PI child completed only one block of the task and was excluded from analysis. Consistent with prior research with this task (J.M. McDermott, personal communication), reaction times of less than 200 msec were removed from analysis. Variables of analysis were accuracy to target (go) trials, accuracy to non-target (no-go) trials, and mean reaction time to target (go) trials. Accuracies to no-go trials following 1, 3, and 5 go trials were examined to test for possible differential accuracy as interference from proceeding go trials increased.

*Flanker task.* Child participants completed a color flanker task requiring them to respond to red and green circles on a computer screen. This task has been previously used in research with young children (McDermott, Perez-Edgar & Fox, 2007). Participants were presented with five colored circles on the screen. They were directed to attend to the middle circle and indicate the color of the middle circle by pressing a green or red button. Congruent trials were those in which the target (middle) circle was flanked on both sides by circles of identical color to the target. Incongruent trials were those in which the target (middle) circle was flanked on both sides by circles of opposite color to the target. A

white fixation point remained at the center of the screen throughout the task. At the beginning of each trial a warning cue was provided for 300 msec, followed by a fixation point for 500 msec. The target stimulus was then presented for 700 msec. Participants were given 1300 msec from the onset of the stimulus to respond and indicate the color of the middle circle. Visual feedback regarding the accuracy of the child's response was provided for 800 msec, with correct responses being followed by a smiling face and incorrect responses being followed by a frowning face. The intertrial interval ranged randomly between 3,900 and 4,400 msec (see Figure 2). Participants completed 8 practice trials. A total of 180 test trials (3 blocks of 60 trials) were presented in random order. Participants were required to complete at least 90 test trials with greater than 50% accuracy to be included in the analysis. Stimulus presentation and recording of behavioral responses was completed using EPrime. Reaction times of less than 200 msec were removed from analysis. To ensure that participants' performance was not impacted by difficulty identifying colors or red-green color blindness, all children completed a screening requiring them to recognize and name various colors including red and green. All participants successfully completed this screening. Flanker data from 15 participants were excluded from analysis due to computer programming error ( $n = 5$ ; 1 PI, 4 NA), completion of less than 90 test trials ( $n = 5$ ; 3 PI, 2 NA), and overall task accuracy of 50% or poorer ( $n = 5$ ; 2 PI, 3 NA). Variables of analysis were accuracy and mean reaction time to congruent and incongruent trials.

*Resting electroencephalogram (EEG) at 5 ½ years of age.* Electroencephalogram activity (EEG) was recorded using a 128 channel HydroCel Geodesic Sensor Net

(Electrical Geodesics, Inc.). A total of 6 minutes of EEG was collected over three 2 minute segments. During each 2 minute segment, data were collected with the lights on and lights off in the assessment room in alternating periods of 30 seconds. This design was utilized to simulate the traditional eyes open and eyes closed collection procedures for resting EEG in a sample of children too young to reliably comply with these condition demands. Due to evidence of differences in EEG power and topography between eyes open and eyes closed conditions (Barry, Clarke, Johnstone, Magee, & Rushby, 2007), resting EEG collection was split evenly between conditions (3 minutes in each condition). For both conditions, participants were directed to keep their body still while looking quietly at decorated pages containing glow-in-the-dark stars. Based on this experimental design both the lights on and light off conditions involved cortical processing of visual input, although the level of ambient light was greater in the lights on condition.

Electrodes were prepared by briefly soaking in a distilled water solution with potassium chloride and a small amount of baby shampoo. Impedances were measured at each electrode and accepted if they were at or below 50 kΩ. EEG data collection was completed with Net Station software. Sampling rate was set to 250 Hz and all data were referenced to the vertex (Cz) during collection. EEG data were processed in Net Station and filtered using a basic finite impulse response (FIR) filter with settings at .3Hz (high pass) and 30 Hz (low pass). Data were segmented into 2 second epochs within lights on/lights off conditions. Segments containing gross artifact (change in EEG signal amplitude of 350 µv or more) were rejected and bad channels replaced. Eye blinks were

modeled and removed through use of the Net Station Ocular Artifact Removal tool. To mark for remaining gross artifact, eye blinks, and eye movements, automated artifact detection was completed and segments containing EEG signal with a change of more than 150  $\mu$ v were rejected. Segmented data were visually inspected after automated processing to ensure the quality of remaining EEG data. Eye channels were excluded from analysis and data were re-referenced to an average reference using PARE correction and exported to EEGLab supported by Matlab for analysis with a fast Fourier Transformation (FFT). A Hanning window of 2 seconds with an overlap of 50% was used during the FFT. To maintain consistency with prior research (Marshall et al., 2004) and methods utilized during the 18 month EEG data collection in this sample (Tarullo, 2008), absolute power was calculated for 3-5 Hz (theta), 6-9 Hz (alpha), and 10-18 Hz (beta) bands. EEG data were exported into SPSS. Natural log (ln) transformation was used to normalize the distribution of absolute power variables. Relative theta and relative alpha power were calculated at each electrode site by determining the ratio of absolute power at a band level (e.g., theta) and electrode site to the total absolute power (sum of power in theta, alpha, and beta bands) at that electrode site. Mean relative theta power and mean relative alpha power across the frontal region was then computed. A difference score between frontal relative theta and frontal relative alpha was also calculated. The frontal region of interest was identified by using the 10/20 electrode placement locations (Fz, F3, F4, F7, F8) as anchors with adjacent electrodes included in the region average. A total of 27 electrodes (as labeled on the EGI GSN 128 Hydrocel net: 2, 3, 4, 5, 9, 10, 11,

15, 16, 12, 18, 19, 20, 22, 23, 24, 26, 27, 28, 33, 34, 116, 117, 118, 122, 123, 124) were included in the frontal region of interest.

Five PI children did not provide any EEG data due to refusal of the sensor net. Data from an additional 15 children (PI:  $n = 4$ , NA:  $n = 11$ ) were lost due to excess artifact. No difference was found between conditions (lights on vs. lights off) for relative theta or relative alpha power in the frontal region. Therefore, EEG data were combined between conditions to maximize statistical power. Following data processing, participants had a mean duration of 130.74 seconds ( $SD = 74.10$ ) of EEG data for analysis. No group difference was present in the duration of EEG recording available for analysis,  $t(36) = .929, ns$ . See Table 1 for available data across measures by group.

*Resting electroencephalogram (EEG) at 18 months of age.* Resting EEG was collected at 18 months of age as described previously by Tarullo (2008). EEG was recorded from 16 electrode sites based on the 10/20 electrode placement system (Jasper, 1958). Electrodes were located in frontal, central, temporal, parietal, and occipital scalp regions (Fp1, Fp2, Fz, F3, F4, F7, F8, Cz, C3, C4, T3, T4, P3, P4, O1, and O2). All sites were referenced to the vertex (Cz) during data collection. Abrasive and conducting gels were inserted into the electrode sites of interest and the scalp was gently abraded with the blunt end of a q-tip. Impedances were measured and accepted if below 5K ohms. Amplifier output was set so the signal was 50 uV peak to peak, with a gain of 10,000. The signal was digitized at 512 samples per second to prevent aliasing from affecting the data. Baseline EEG was recorded for 6 minutes while the child was seated in a high chair next to the parent. To keep the child quiet and focused with reduced motor activity, the

experimenter blew bubbles, showed the child a rotating ferris wheel toy, and performed silent puppet shows. Data were collected using the continuous recording mode of ERP-W, re-referenced to an average reference, and exported for analysis in software by James Long Company. EEG data were then scored for artifact due to eye movement or motor activity. Epochs containing amplitudes larger than 175 uV were considered to reflect motor artifact and eliminated from analysis. Eye blinks were identified in the FP1 channel, which is sensitive to eye movement, and these epochs were also eliminated from analysis. The data were analyzed with a Discrete Fourier Transform (DFT) using a Hanning window of 1 second with a 50% overlap. Absolute power was computed for the 3-5 hz (theta), 6-9 hz (alpha), and 10-18 hz (beta) bands, and expressed in microvolts squared. EEG data were then exported into SPSS for statistical analysis. To normalize the distribution of EEG variables, natural log (ln) transformation was used. Relative theta and relative alpha power were calculated at each electrode site by determining the ratio of absolute power at a band level (e.g., theta) and electrode site to the total absolute power (sum of power in theta, alpha, and beta bands) at that electrode site. Mean relative theta power and mean relative alpha power across the scalp were then calculated. These power variables were analyzed to test whether participants with longitudinal EEG (EEG recordings at both 18 months and 5 ½ years) demonstrated group differences in relative theta and relative alpha power previously found in the original sample at 18 months (Tarullo, 2008). Mean relative theta power and mean relative alpha power across the frontal region (Fp1, Fp2, Fz, F3, F4, F7, F8) was calculated at 18 months in order to create frontal power variables analogous to those computed at 5 ½ years. A difference

score between frontal relative theta power and frontal relative alpha power at 18 months was also calculated.

### Analysis Plan

#### *Evaluating Possible Covariates*

Gender and age of participants at assessment were examined as possible covariates to be used in analysis.

#### *Parent Report of ADHD Symptoms*

An independent samples t-test was used to test for a group difference between PI and non-adopted children in parent reported overall ADHD symptoms. Follow-up independent samples t-tests were completed separately for the Inattention subscale and Impulsivity subscale to determine if symptoms in either domain differed by group (PI vs. NA). A chi-square test was used to test for an effect of group on the number of children with parent reported ADHD symptoms in the clinical range, based on a clinical threshold identified in prior literature (Luby et al., 2002).

#### *Go/No-Go Task Performance*

A repeated measures ANOVA with trial type (go vs. no-go) as the within participants factor and group as the between participants factor was utilized to assess for group differences in accuracy on the animal themed go/no-go task. A repeated measures ANOVA with the number of proceeding go trials (1, 3, and 5 go trials prior to no-go trial) as the within participants factor and group as the between participants factor was used to test for group differences in accuracy as interference from proceeding go trials increased. An independent samples t-test was utilized to test for a group difference between PI and

NA children in mean reaction time to correct go trials. Associations between parent reported ADHD symptom variables (overall ADHD symptoms, Inattention, Impulsivity) and performance measures (go and no-go accuracy and correct go RT) on the go/no-go task were examined with Pearson correlations across and within groups.

#### *Flanker Task Performance*

A repeated measures ANOVA with trial type (congruent vs. incongruent) as the within participants factor and group as the between participants factor was used to examine group differences in accuracy on the flanker task. A repeated measures ANOVA with trial type (congruent vs. incongruent) as the within participants factor and group as the between participants factor was utilized to assess for group differences in mean reaction time to correct trials. Associations between parent reported ADHD symptom variables (overall ADHD symptoms, Inattention, Impulsivity) and performance measures (congruent and incongruent accuracy and congruent and incongruent reaction time) on the flanker task were examined with Pearson correlations across and within groups.

#### *Relative EEG Power at 18 Months in Longitudinal EEG Participants*

Two x two (group, retention status) way ANOVAs were used to test for differences in EEG at 18 months of age between participants with and without longitudinal EEG data (EEG at both 18 months and 5 ½ years of age). Separate 2 x 2 way ANOVAs were completed with relative theta power across the scalp, relative alpha power across the scalp, and relative theta-alpha difference score across the scalp at 18 months as dependent variables. Group (PI vs. NA) and retention status (retained with longitudinal EEG data or lost at 5 ½ years) were the independent variables. Independent

samples t-tests were completed to test whether the longitudinal EEG subsample demonstrated previously documented (Tarullo, 2008) group differences (PI vs. NA) in mean relative theta power and mean relative alpha power across the scalp at 18 months of age. Independent samples t-tests were also completed for frontal relative theta power and frontal relative alpha power at 18 months of age.

#### *Relative EEG Power at 5 ½ Years of Age & EEG Associations Over Time*

Group differences in EEG variables (frontal relative theta power, frontal relative alpha power and frontal relative theta-alpha difference score) at 5 ½ years of age were examined through independent samples t-tests. Associations over time (18 months to 5 ½ years) in frontal relative EEG power were examined through calculation of separate Pearson correlations between frontal relative theta power at 18 months and 5 ½ years, frontal relative alpha power at 18 months and 5 ½ years, and frontal relative theta-alpha difference scores at 18 months and 5 ½ years. Pearson correlations were computed across and within groups.

#### *Concurrent Associations Between EEG Power and ADHD Symptoms at 5 ½ Years of Age*

Associations at 5 ½ years of age between EEG variables (frontal relative theta power, frontal relative alpha power and frontal relative theta-alpha difference score) and parent reported ADHD symptom variables (overall ADHD symptoms, Inattention, Impulsivity) were examined using Pearson correlations across and within groups.

#### *Concurrent Associations Between EEG Power and Go/No-Go Task Performance at 5 ½ Years of Age*

Associations at 5 ½ years of age between EEG variables (frontal relative theta

power, frontal relative alpha power, frontal relative theta-alpha difference score) and behavioral performance variables (accuracy to go trials, accuracy to no-go trials, reaction time to correct go trials) on the go/no-go task were examined using Pearson correlations across and within groups.

*Concurrent Associations Between EEG Power and Flanker Task Performance at 5 ½ Years of Age*

Associations at 5 ½ years of age between EEG variables (frontal relative theta power, frontal relative alpha power, frontal relative theta-alpha difference score) and behavioral performance variables (accuracy and reaction time to congruent trials, accuracy and reaction time to incongruent trials) on the flanker task were examined using Pearson correlations across and within groups.

*Longitudinal Associations Between EEG Power at 18 Months and ADHD Symptoms at 5 ½ Years of Age*

Longitudinal associations between EEG variables (frontal relative theta power, frontal relative alpha power, frontal relative theta-alpha difference score) at 18 months of age and parent reported ADHD symptom variables (overall ADHD symptoms, Inattention, Impulsivity) at 5 ½ years of age were tested using Pearson correlations across and within groups.

*Longitudinal Associations Between EEG power at 18 Months and Go/No-Go Task Performance at 5 ½ Years of Age*

Longitudinal associations between EEG variables (frontal relative theta power, frontal relative alpha power, frontal relative theta-alpha difference score) at 18 months of

age and behavioral performance variables (accuracy to go trials, accuracy to no-go trials, reaction time to correct go trials) on the go/no-go task at 5 ½ years of age were tested using Pearson correlations across and within groups.

*Longitudinal Associations Between EEG Power at 18 Months and Flanker Task Performance at 5 ½ Years of Age*

Longitudinal associations between EEG variables (frontal relative theta power, frontal relative alpha power, frontal relative theta-alpha difference score) at 18 months of age and behavioral performance variables (accuracy and reaction time to congruent trials, accuracy and reaction time to incongruent trials) on the flanker task at 5 ½ years of age were tested using Pearson correlations across and within groups.

## Results

*Evaluating Possible Covariates*

*Gender.* A chi-square indicated no group difference in the gender distribution of the samples,  $\chi^2(1) = 2.70, ns$ . Because there were insufficient numbers of males (PI:  $n = 1$ , NA:  $n = 6$ ) to include sex as a factor or covariate, analyses were completed with and without males. The pattern of results for parent reported ADHD symptoms, go/no-go and flanker behavioral performance, and frontal relative theta and alpha EEG power did not change when males were excluded. Therefore, both males and females were included in all between and within group analyses.

*Age at assessment.* The groups did not differ in age at assessment,  $t(56) = .52, ns$ . Pearson correlations indicated that child age at the 5 ½ year assessment was not associated with any 5 ½ year variables of interest (parent reported ADHD symptoms,

accuracy or reaction time on the go/no-go task, accuracy or reaction time on the flanker task, frontal relative theta or alpha power). Based on these results, age at assessment was not included as a covariate in analyses.

#### *Parent Report of ADHD Symptoms*

An independent samples t-test indicated that PI children demonstrated greater parent reported overall ADHD symptoms,  $t(55) = 3.01, p < .01$ , than did NA children. When the HBQ subscales of Inattention and Impulsivity were examined in separate independent samples t-tests, group differences were found for both scales, Inattention:  $t(56) = 2.65, p = .01$ ; Impulsivity:  $t(55) = 2.99, p < .01$ , with PI children reported to demonstrate greater symptoms (see Table 2). A chi-square test indicated no effect of group,  $\chi^2(1) = 1.69, ns$ , for the percentage of children with ADHD symptoms within the clinical range by parent report (PI: 12%; NA: 3%). No associations were present in the PI group between duration of institutional care and parent reported overall ADHD symptoms, Inattention, or Impulsivity.

#### *Go/No-Go Task Performance*

*Response accuracy.* Results of a repeated measures ANOVA revealed a main effect of trial type,  $F(1,55) = 70.88, p < .001$ , with greater accuracy to go trials (93%) compared to no-go trials (66%) across participants. No main effect of group,  $F(1,55) = .22, ns$ , or trial x group interaction,  $F(1,55) = .30, ns$ , was found (see Table 3). A repeated measures ANOVA revealed a main effect of the number of proceeding go trials,  $F(1.75, 95.98) = 11.46, p < .001$ , on the accuracy to no-go trials. No main effect of group,  $F(1,55) = .26, ns$ , or number of proceeding go trials x group interaction,  $F(1.75, 95.98) =$

.30, *ns*, was found. Follow up paired samples t-tests indicated that participants demonstrated poorer accuracy on no-go trials following 1 go trial (58% accuracy) compared to those following 3 go trials (68% accuracy),  $t(56) = 2.67, p < .05$ , or 5 go trials (73% accuracy),  $t(56) = 4.73, p < .001$ .

A negative association between parent reported overall ADHD symptoms and accuracy to no-go trials was present across groups,  $r(57) = -.29, p < .05$ . This finding was carried by a correlation between Inattention and accuracy to no-go trials,  $r(57) = -.35, p < .01$ . When examined separately for PI and NA participants, correlations with no-go accuracy were present only in the PI group for overall ADHD symptoms:  $r(24) = -.45, p < .05$  and for Inattention:  $r(24) = -.49, p < .05$ . No other correlations were present between parent reported ADHD symptom variables and accuracy variables on the go/no-go task.

*Reaction time.* An independent samples t-test revealed no group difference in mean reaction time to correct go trials,  $t(55) = .72, ns$  (see Table 3). No correlations between parent reported ADHD symptom variables (overall ADHD symptoms, Inattention, Impulsivity) and mean reaction time to go trials were found across or within groups.

#### *Flanker Task Performance*

*Response accuracy.* Results of a repeated measures ANOVA revealed a main effect of trial type,  $F(1,40) = 16.99, p < .001$ , with participants demonstrating greater accuracy to congruent trials (78%) compared to incongruent trials (73%). No main effect of group,  $F(1,40) = 2.64, ns$ , or trial x group interaction,  $F(1,40) = .31, ns$ , was found (see

Table 4). A negative association between Impulsivity and accuracy to incongruent trials was found only in the PI group,  $r(19) = -.47, p < .05$ . No other correlations were present between parent reported ADHD symptom variables and accuracy variables on the flanker task.

*Reaction time.* Results of a repeated measures ANOVA revealed a main effect of trial type,  $F(1,40) = 21.93, p < .001$ , with slower mean reaction time to incongruent compared to congruent trials. No main effect of group,  $F(1,40) = 1.05, ns$ , or trial x group interaction,  $F(1,40) = .50, ns$  was found (see Table 4). No correlations between parent reported ADHD symptom variables (overall ADHD symptoms, Inattention, Impulsivity) and mean reaction time to congruent or incongruent trials were found across or within groups.

#### *Relative EEG Power at 18 Months of Age in Longitudinal EEG Participants*

A two-way ANOVA revealed a main effect of retention status (retained in longitudinal EEG sample vs. non-retained) for relative theta power across the scalp at 18 months,  $F(1,44) = 4.77, p < .05$ , with retained participants having greater relative theta power across the scalp at 18 months than non-retained participants. A marginally significant retention status by group interaction was found for relative theta power across the scalp at 18 months,  $F(1,44) = 3.84, p = .056$ , with inspection of the means suggesting that PI children retained in the longitudinal sample had greater relative theta power across the scalp than those who lost at follow up. No main effect of retention status or retention status by group interaction was found for relative alpha power across the scalp at 18 months of age. A main effect of retention status was found for the relative theta-alpha

difference score across the scalp at 18 months,  $F(1,44) = 4.49, p < .05$ , with retained participants demonstrating greater concentration of relative theta power (e.g., more relative theta power compared to alpha power) than non-retained participants.

Independent samples t-tests completed with the longitudinal EEG subsample revealed group differences in EEG power at 18 months that were generally consistent with prior analyses (Tarullo, 2008). When analyzed across the scalp, a marginally significant group effect was found for relative theta power,  $t(9.41) = 2.17, p = .057$ , with PI children having marginally greater theta power across the scalp than NA children. PI children were also found to have a greater concentration of relative theta power compared to relative alpha power across the scalp (relative theta-alpha difference score),  $t(26) = 2.51, p < .05$ , at 18 months of age than NA children. However, no group difference was found for relative alpha power across the scalp. Thus, the previously reported group difference in alpha power (NA > PI) (Tarullo, 2008) did not hold in the subsample retained longitudinally. A similar pattern of results was found when examining 18 month EEG power specifically in the frontal region. The PI group demonstrated greater relative theta power in the frontal region,  $t(26) = 2.88, p < .01$ , and a greater concentration of relative theta power compared to relative alpha power in the frontal region (relative theta-alpha difference score),  $t(26) = 2.66, p < .05$ , at 18 months of age than NA children. No group difference was found for relative alpha power in the frontal region at 18 months of age (see Table 5).

#### *Relative EEG Power at 5 ½ Years of Age & EEG Associations Over Time*

Independent samples t-tests revealed a group difference in frontal relative theta

power at the 5 ½ year assessment,  $t(36) = 2.16, p < .05$ , with PI children having greater frontal relative theta power than NA children. A marginally significant group difference was present for relative theta-alpha difference score in the frontal region,  $t(36) = 1.84, p = .073$ , with PI children having a marginally greater concentration of frontal relative theta power compared to frontal relative alpha power at 5 ½ years of age. No group effect was found for frontal relative alpha power at 5 ½ years of age,  $t(36) = .17, p = ns$  (see Table 6).

Pearson correlations across groups indicated marginally significant associations between frontal relative theta power at 18 months and 5 ½ years,  $r(32) = .33, p = .066$ , and frontal relative theta-alpha difference scores at 18 months and 5 ½ years,  $r(32) = .33, p = .065$ . No significant correlation between frontal relative alpha power at 18 months and 5 ½ years was found across groups,  $r(32) = .24, p = ns$ . When correlations were examined separately by group, longitudinal associations between 18 month and 5 ½ year EEG variables were found only for NA children. In the NA group, a significant longitudinal correlation was present between frontal relative theta-alpha difference scores at 18 months and 5 ½ years,  $r(20) = .62, p < .01$ , while marginally significant longitudinal correlations were found for frontal relative theta power,  $r(20) = .42, p = .065$ , and frontal relative alpha power,  $r(20) = .44, p = .055$ . Correlation coefficients in the PI group were not significant and ranged from -.06 to .08,  $n = 12$ , with all  $ps > .05$  (see Table 7).

#### *Concurrent Associations Between EEG Power and ADHD Symptoms at 5 ½ Years of Age*

Pearson correlations across groups revealed no concurrent associations between 5

$\frac{1}{2}$  year EEG variables (frontal relative theta power, frontal relative alpha power, frontal relative theta-alpha difference score) and parent reported ADHD symptom variables (overall ADHD symptoms, Inattention, Impulsivity), with correlation coefficients ranging from -.28 to .27,  $n = 37$  ( $n = 38$  for Inattention), with all  $ps \geq .10$ . When correlations were examined separately by group, a marginally significant association was present in the NA group between the frontal relative theta-alpha difference score and Impulsivity,  $r(21) = .43, p = .054$ . No other EEG – ADHD symptom associations were present in the NA group, with correlation coefficients ranging from -.38 to .38,  $n = 21$  ( $n = 22$  for Inattention), with all  $ps > .09$ . No associations between EEG and ADHD variables were found in the PI group at  $5 \frac{1}{2}$  years of age, with correlation coefficients ranging from -.36 to .15,  $n = 16$ , with all  $ps > .10$  (see Table 8).

*Concurrent Associations Between EEG Power and Go/No-Go Task Performance at  $5 \frac{1}{2}$  Years of Age*

Pearson correlations across groups revealed no associations between  $5 \frac{1}{2}$  year EEG variables (frontal relative theta power, frontal relative alpha power, frontal relative theta-alpha difference score) and go/no-go task performance variables (go accuracy, no-go accuracy, correct go RT), with correlation coefficients ranging from -.25 to .07,  $n = 38$ , with all  $ps > .10$ . When correlations were examined separately by group, no significant associations were found in either group (NA: correlation coefficients ranged from -.16 to .11,  $n = 22$ , with all  $ps > .10$ ; PI: correlation coefficients ranged from -.31 to .30,  $n = 16$ , with all  $ps > .10$ ) (see Table 9).

*Concurrent Associations Between EEG Power and Flanker Task Performance at  $5 \frac{1}{2}$*

*Years of Age*

Pearson correlations across groups revealed no associations between 5 ½ year EEG variables (frontal relative theta power, frontal relative alpha power, frontal relative theta-alpha difference score) and flanker task performance variables (accuracy and reaction time to congruent trials, accuracy and reaction time to incongruent trials), with correlation coefficients ranging from -.18 to .23,  $n = 29$ , with all  $p > .10$ . When correlations were examined separately by group, no significant associations between EEG and flanker performance variables were found in the NA group, with correlation coefficients ranging from -.36 to .26,  $n = 16$ , with all  $p > .10$ . A marginally significant association was found in the PI group between frontal relative theta power and mean reaction time to congruent trials,  $r(13) = -.51, p = .077$ . However, this finding was driven by one participant and disappeared when this outlier was removed. Marginally significant associations were also found in the PI group between frontal relative theta-alpha difference score and accuracy to congruent trials,  $r(13) = .55, p = .052$  (see Figure 3), and frontal relative theta-alpha difference score and accuracy to incongruent trials,  $r(13) = .51, p = .077$  (see Figure 4). No other associations were found between EEG and flanker task variables in the PI group (see Table 10).

*Longitudinal Associations Between EEG Power at 18 Months and ADHD Symptoms at 5 ½ Years of Age*

Pearson correlations across groups revealed no longitudinal associations between EEG variables (frontal relative theta power, frontal relative alpha power, frontal relative theta-alpha difference score) at 18 months and parent reported ADHD symptom variables

(overall ADHD symptoms, Inattention, Impulsivity) at 5 ½ years of age, with correlation coefficients ranging from -.05 to .12,  $n = 49$  ( $n = 50$  for Inattention), with all  $ps > .10$ . When correlations were examined separately by group, no associations were present in the NA group between 18 month EEG variables and 5 ½ year parent reported ADHD symptoms, with correlation coefficients ranging from -.33 to .27,  $n = 28$  ( $n = 29$  for Inattention), with all  $ps \geq .09$ . Within the PI group, significant correlations were present between frontal relative alpha power at 18 months and all three parent report ADHD symptoms variables at 5 ½ years of age (overall ADHD symptoms:  $r(21) = .53, p < .05$ ; Inattention:  $r(21) = .48, p < .05$ ; Impulsivity:  $r(21) = .52, p < .05$ ) (see Figures 5, 6, and 7, respectively). Marginally significant associations were present in the PI group between frontal relative theta-alpha difference score at 18 months and two ADHD symptom variables at 5 ½ years, overall ADHD symptoms:  $r(21) = -.40, p = .07$ , and Impulsivity:  $r(21) = -.43, p = .053$  (see Figures 8 and 9). No other associations were found between 18 month EEG and 5 ½ year ADHD symptom variables in the PI group (see Table 11).

#### *Longitudinal Associations Between EEG power at 18 Months and Go/No-Go Task*

##### *Performance at 5 ½ Years of Age*

Pearson correlations across groups revealed no longitudinal associations between 18 month EEG variables (frontal relative theta power, frontal relative alpha power, frontal relative theta-alpha difference score) and go/no-go task performance variables (go accuracy, no-go accuracy, correct go RT), with correlation coefficients ranging from -.22 to .01,  $n = 49$ , with all  $ps > .10$ . When correlations were examined separately by group, no significant associations were found in either group (NA: correlation coefficients

ranged from  $-.23$  to  $.27$ ,  $n = 29$ , with all  $ps > .10$ ; PI: correlation coefficients ranged from  $-.36$  to  $.23$ ,  $n = 20$ , with all  $ps > .10$ ) (see Table 12).

#### *Longitudinal Associations Between EEG Power at 18 Months and Flanker Task*

##### *Performance at 5 ½ Years of Age*

Pearson correlations across groups revealed no associations between 18 month EEG variables (frontal relative theta power, frontal relative alpha power, frontal relative theta-alpha difference score) and flanker task performance variables (accuracy and reaction time to congruent trials, accuracy and reaction time to incongruent trials), with correlation coefficients ranging from  $-.14$  to  $.14$ ,  $n = 34$ , with all  $ps > .10$ . When correlations were examined separately by group, no significant associations were found in either group (NA: correlation coefficients ranged from  $-.26$  to  $.25$ ,  $n = 19$ , with all  $ps > .10$ ; PI: correlation coefficients ranged from  $-.22$  to  $.16$ ,  $n = 15$ , with all  $ps > .10$ ) (see Table 13).

#### Discussion

This study examined parent reported ADHD symptoms, performance on two laboratory-based tasks of executive attention, and longitudinal recordings of resting EEG power in 5 ½-year-old PI children and never institutionalized comparison children. The goal of this study was to contribute to current research regarding the neurobehavioral development of PI children, particularly in regard to behavioral and electrophysiological correlates of attention. The design of this study allowed for comparison between groups (post-institutionalized vs. non-adopted) as well as investigation of associations among

measures at 5 ½ years of age, and in the case of resting EEG power, over time (18 months of age to 5 ½ years of age).

Analysis of parent reported ADHD symptoms revealed that PI children had higher levels of symptoms, in regard to both inattention and impulsivity, than did NA comparison children. These results are consistent with well documented findings of increased inattentive and hyperactive-impulsive symptoms and ADHD diagnosis rates in institutionalized and PI children (e.g., Ames, 1997; Gunnar et al., 2007; Provence & Lipton, 1962; Roy et al., 2000; Stevens et al., 2008; Tizard & Rees, 1974; Wiik et al., in press; Zeanah et al., 2009). Although the PI group had higher mean levels of ADHD symptoms, most PI children's symptoms were reported to be below the severity associated with a diagnosis of ADHD. That is, only 12% ( $n = 3$ ) of the PI sample had parent reported ADHD symptoms above an empirically derived clinical threshold associated with ADHD diagnosis (Luby et al., 2002). This rate is lower than those previously reported in PI samples (Beverly et al., 2008: 42%; Wiik et al., in press: 23%; Zeanah et al., 2009: 19%) and only moderately above the rate expected in the general population of school age children (3 - 7%) (American Psychiatric Association, 2000).

Based on the finding of increased parent reported ADHD symptoms in the PI group, it seems likely that these children would also demonstrate poor performance on computerized tasks designed to assess executive attention abilities. However, analyses of the executive attention (go/no-go and flanker) tasks found no differences in PI and NA children's sustained attention, response inhibition, or monitoring and resolution of conflict as measured by the go/no-go and flanker tasks. This is somewhat surprising

given the limited prior evidence (e.g., Fox et al., 2008; Loman, 2009; Tottenham et al., 2010) suggesting that PI children have difficulty on executive attention tasks. However, the pattern of these difficulties has been inconsistent in prior literature, with the presence of group differences varying based on task measures and design (e.g., emotional stimuli vs. non-emotional stimuli). Given the current inconsistencies in research findings, additional investigation into the presence and pattern of possible executive attention deficits in PI children is needed.

Future research must carefully consider the design of tasks used to assess executive attention abilities in PI children. For example, the go/no-go task utilized in the current study was designed to be child friendly, with an animal theme and structured reinforcement (a break halfway through the task during which the child received a prize). Although this design likely made the task more enjoyable for child participants, it also may have elicited a high level of task engagement and motivation. This is in contrast to measures utilized in clinical neuropsychological assessment (e.g., Test of Variables of Attention, TOVA) which place a high demand on sustained attention and response inhibition without explicit reinforcement or breaks in the task. Therefore, it is possible that deficits in executive attention may be observed in PI children if task demands are sufficiently challenging. Additional testing of this hypothesis is needed.

Task demands should also be considered when interpreting the flanker task results in this study. The flanker task appeared to be somewhat challenging for both groups of children, with 20% ( $n = 5$ ) of PI and 15% ( $n = 5$ ) of NA children being excluded from analysis due to poor performance or early termination of the task. This task has been

previously used in research with 5-year-old children (McDermott et al., 2007) and produced expected interference effects (greater accuracy to congruent compared to incongruent stimuli) in the current study, suggesting that the task functioned as designed. However, differences in task demands (e.g., the type of stimuli used) have previously been found to impact children's performance (McDermott et al., 2007). Thus, it remains unclear whether differences between PI and NA children's performance would be present on alternative flanker tasks.

The PI children included in this study differed from prior research samples in regard to their age at removal from institutional care. Children in the current PI sample were removed from institutional care at an average of 12 months of age, with no child spending more than 16 months in institutional care. Numerous prior studies have included children removed from institutional care well beyond this age range (e.g., Marshall et al., 2004; Stevens et al., 2008). Past research has also consistently documented differences between PI and NA children in regard to general intellectual abilities (IQ) (Beckett et al., 2006; Loman et al., 2009); however, no such group difference was found in the current sample. Although the relationship between general cognitive abilities and attention problems is complex, the PI and NA children in this study appear to be functioning at a similar cognitive level, which is consistent with the lack of group differences on the executive attention tasks.

Analysis of correlations between parent reported ADHD symptoms and children's behavioral performance on attention tasks suggested that PI parents' perceptions of their children's behavior corresponded more consistently with their actual performance on

attention based tasks than did NA parents' perceptions. With the PI group, elevated parent reported inattention was associated with decreased accuracy on no-go trials (response inhibition), while elevated impulsivity was associated with decreased accuracy on incongruent flanker trials (requiring conflict monitoring and resolution). No such associations were present in the NA group. This may be partially due to low variability in the level of ADHD symptoms reported by NA parents. However, these findings are also consistent with previous research suggesting that adoptive parents are particularly sensitive to their children's emotional and behavioral functioning (Miller et al., 2000).

Discussion and interpretation of group differences in relative EEG power must take into consideration the small sample size available for between group and longitudinal analyses. Collection of resting EEG proved difficult at both 18 months and 5 ½ years of age, with a significant amount of data being lost to electrode cap refusal and excess artifact. Given this data loss, it was necessary to test whether the subjects available for longitudinal analyses demonstrated the previously reported (Tarullo, 2008) pattern of increased relative theta power compared to NA children at 18 months of age. This was indeed the case, with group differences present in frontal relative theta power, as well as relative theta power across the entire scalp (although this was marginally significant). Compared to NA children, PI children with longitudinal EEG data also demonstrated a greater concentration of relative theta power compared to relative alpha power both across the scalp and in the frontal region at 18 months of age. These findings are consistent with prior research (Marshall et al., 2004) documenting increased relative theta power in frontal, parietal, and occipital regions for PI children compared to NA children.

When relative alpha power at 18 months of age was examined in the subsample of participants with longitudinal EEG, previous findings of reduced relative alpha power in PI compared to NA children (Marshall et al., 2008, Tarullo, 2008) were not replicated. Inspection of the means indicated that PI children with longitudinal EEG had lower mean relative alpha power both in the frontal region and across the scalp than NA children. However, the small sample sizes (PI:  $n = 9$ , NA:  $n = 19$ ) and large variability in relative alpha power in the PI group likely contributed to the lack of significant group differences.

Prior research has demonstrated a shift toward more age typical patterns of EEG power (i.e., increased relative alpha power) in PI children after 2 to 3 years in family based care (Marshall et al., 2008). Based on this finding, it was hypothesized that an average of more than 4 ½ years in adoptive care would be sufficient to support developmental “catch-up” in EEG power for the PI children in this study. Therefore, patterns of relative EEG power were expected to be similar between PI and NA groups at 5 ½ years of age. Contrary to these expectations, PI children continued to display increased relative theta power in the frontal region compared to NA children. These results may be interpreted in several ways. First, these findings may be seen as evidence of persistent cortical hypoarousal in PI children, which appears to be resistant to remediation by the general intervention of family based care. An alternative hypothesis is that the PI group is demonstrating developmental catch-up consistent with a maturational delay model, but additional time in family care is needed for the distribution of EEG power to reach age typical patterns. It is notable that Marshall and colleagues (2008) found intervention effects after 2 to 3 years of foster care for relative alpha power, but not

relative theta power. Therefore, the continued presence of group differences in relative theta power at 5 ½ years of age is not entirely inconsistent with prior research.

When EEG power measures were examined over time, longitudinal correlations between 18 month and 5 ½ year frontal relative EEG power measures were present in the NA group. However, EEG measures were less stable in the PI group, with no longitudinal associations found between 18 month and 5 ½ year EEG power. This finding is consistent with evidence of rapid developmental change in the years following adoption (e.g., Van IJzendoorn et al., 2007) and variability in long-term outcomes for PI children.

Given literature linking EEG power and ADHD symptoms (Barry et al., 2003; Quintana et al., 2007), it was expected that concentration of frontal EEG power in lower frequency bands (e.g., theta) at 5 ½ years of age would be positively associated with parent reported ADHD symptoms, as well as poorer performance on the executive attention tasks (go/no-go and flanker). In regard to ADHD symptoms, a marginally significant association with EEG power at 5 ½ years of age was present in the NA group with increased concentration of frontal relative theta compared to alpha power being associated with increased parent reported impulsive symptoms. However, frontal relative EEG power was not found to be a significant electrophysiological correlate of parent reported ADHD symptoms for the PI children. The absence of clinical level ADHD symptoms in the majority of PI and NA participants may provide one explanation for the lack of associations between EEG and ADHD symptoms. Although this EEG-ADHD link has been found in clinical samples of individuals with ADHD, this brain-behavior association may not be present in individuals without severe inattentive and hyperactive-

impulsive symptoms. Additional research examining relationships between resting EEG power and ADHD symptoms is necessary to further clarify the presence and pattern of these associations, particularly in non-clinical populations.

When concurrent associations between relative frontal EEG power and behavioral performance on the two executive attention tasks at 5 ½ years of age were examined, findings varied by task and group. No associations were present between frontal relative EEG power and performance measures on the go/no-go task, either across or within participant groups. Marginally significant associations were found between frontal relative EEG power and flanker performance measures; however these associations were present only within the PI group. The direction of these findings is puzzling, as a theoretically less mature pattern of EEG power at 5 ½ years of age (characterized by a greater disparity between the proportion of frontal relative theta power to frontal relative alpha power) was associated with more accurate responding on the flanker task for PI children. These results are contrary to what was expected based on the limited research linking ADHD and lower frequency EEG power (Barry et al., 2003). Further investigation is needed regarding the relationship between resting EEG power and behavioral performance on tasks indexing aspects of executive attention.

Analyses of longitudinal associations between resting EEG power at 18 months of age and behavioral outcomes at 5 ½ years of age provided additional interesting results. No associations were present between frontal relative EEG measures at 18 months of age and go/no-go or flanker performance measures at 5 ½ years of age, either across or within groups. In addition, no associations were present between frontal relative EEG measures

at 18 months of age and parent reported ADHD symptoms at 5 ½ years, when analyzed across all participants or separately in the NA group. However, these longitudinal correlations were significant in the PI group and again indicated that a presumably less mature pattern of frontal relative EEG power (lower frontal relative alpha power) in PI children at 18 months of age was associated with lower levels of parent reported ADHD symptoms at 5 ½ years. This pattern of associations held when examined separately for the Inattention and Impulsivity subscales of the ADHD symptoms index. In addition, a greater proportion of frontal relative theta power compared to frontal relative alpha power was marginally associated with lower levels of parent reported Impulsivity symptoms and overall parent reported ADHD symptoms.

These results are unexpected and somewhat puzzling. Although less mature patterns of brain activity (e.g., decreased relative alpha power at 18 months, increased concentration of relative EEG power in the theta band compared to the alpha band at 18 months and 5 ½ years of age) were expected to be risk factors for ADHD symptoms and poor behavioral performance on executive attention tasks, it appears the opposite was true in this sample of PI children. These less mature patterns of brain activity not only remain years after adoption, but appear to be associated with better behavioral outcomes in regard to parent reported ADHD symptoms and behavioral performance on the flanker task at 5 ½ years of age. Given the small sample size and unexpected pattern of findings, replication of these results is needed before any firm conclusions can be drawn regarding associations between EEG power and attention measures in children who have experienced institutional care in early life.

However, these findings do suggest the importance of considering developmental context when interpreting deviations in presumed normative processes. Lessons from developmental psychopathology suggest that developmental changes (e.g., developmental shift in EEG power toward higher frequency bands) in one context (e.g., family based care since birth), may not be adaptive within another context (e.g., early experience of institutional care). Based on this perspective, concentration of EEG power in lower frequency bands (theta) may be adaptive in resource poor environments such as institutional care, possibly by preserving resources and later function. In contrast, this same pattern of low frequency EEG power may reflect a deviation or delay in typical brain development and function when in the context of a well resourced environment.

The EEG data collection procedures at 18 months of age should also be considered when interpreting these unexpected findings. The initial EEG collection at 18 months of age was completed with tasks (e.g., showing the child a rotating ferris wheel toy, performing silent puppet shows) designed to elicit quiet engagement of attention. Therefore, it is possible that PI children who demonstrated a low level of calm engagement (with activity in the theta band) went on to have better attentional and behavioral regulation at 5 ½ years of age, while those who displayed a high level of arousal (increased alpha band activity) during these quiet tasks were more likely to have difficulty with attentional and behavioral regulation later in childhood.

There are several limitations that require consideration when interpreting the results of this study. The first major challenge is the small sample size across measures, particularly in regard to longitudinal resting EEG power. It should be acknowledged that

this sample was not originally recruited with the intention of a longitudinal study. This distinction is important because longitudinal study designs typically recruit a relatively high number of participants for early data collection waves in order to account for expected attrition over time. In addition, longitudinal participants are aware at the beginning of the study that they will be asked to participate again at a later date, thus increasing their awareness of, and possibly commitment to, the longitudinal aspect of the study. Although the majority of families in this study generously participated in follow-up data collection when their child was 5 ½ years old, a significant portion of the sample (27% for PIs, 28% for NA) was lost to attrition.

An additional limitation of the current study is that the PI sample was primarily comprised of children adopted from China (76%). Although this may limit the generalizability of the findings to children adopted from other world regions, this study represented an important broadening of the current literature regarding EEG power in PI children, which to date is limited to Romanian children (e.g., Marshall et al., 2008). In addition, both participant groups were almost exclusively comprised of girls. Thus, it is possible that gender effects may have impacted the findings, although this hypothesis cannot be directly tested with the current samples. As discussed previously, the PI children included in this study were adopted relatively early, with all children joining their adoptive families by 16 months of age. Given this, these study results do not address the possible developmental impact of more prolonged time in institutional care. The addition of a later adopted post-institutionalized sample would assist in clarifying potential duration effects of institutional care. It is also possible that the PI findings are

more generally related to factors associated with being an internationally adopted child (e.g., prenatal stress, disruption in caregiving relationships). The addition of an internationally adopted group without significant institutional care experience would provide an opportunity to partially control for these shared risk factors. Based on these considerations, two additional samples of participants are currently being collected to compare to the PI and NA children reported on in this study. These additional participant groups include: 1) a later adopted sample of PI children (adopted between 18 and 36 months of age) and 2) a comparison sample of internationally adopted children who spent the majority of their time in foster care prior to adoption.

An additional challenge to research with PI children, and internationally adopted children more generally, is the lack of information regarding their pre-adoptive life. Adoptive parents often have limited information regarding genetic risk factors (e.g., family history of medical and/or psychological problems), prenatal care, stress, and exposure to substances, experience of birth complications (e.g., prematurity), and quality of pre-adoptive care (e.g., nutrition, caregiving practices). This dearth of information also makes it difficult for researchers to directly link experiences of institutional care with any particular developmental outcome, as multiple unknown risk factors for development may be present. For example, previous literature has documented increased risk for attention problems in children born preterm (van de Weijer-Bergsma, Wijnroks, & Jongmans, 2008), as well as children with prenatal exposure to alcohol (O'Malley & Nanson, 2002). Although these risk factors are likely present in only a portion of internationally adopted children, the lack of early life information makes it difficult to

eliminate such potential confounding factors from analyses of PI children's development.

Future research regarding behavioral and electrophysiological correlates of attention in PI children must also consider the likely impact of genetic factors and gene-environment interactions. Research with monozygotic and dizygotic twins suggests that genetic factors make important contributions to performance on laboratory-based executive attention measures such as a flanker task (Fan, Wu, Fossella, & Posner, 2001). Research with adult and child participants has revealed that polymorphisms in genes associated with dopamine (e.g., COMT, DRD4, MAOA, and DAT-1 genes) are associated with variations in behavioral performance on executive attention tasks (e.g., Diamond et al., 2004), as well as greater activation in the anterior cingulate during completion of a flanker task (Fan, Fossella, Summer, Wu, & Posner, 2003). In addition, recently published research exploring gene-environment interactions in PI children revealed that the risk for ADHD symptoms is moderated by the presence of a specific polymorphism in the dopamine transporter gene (DAT1) (Stevens et al., 2009). These research findings clearly suggest the need for additional investigation into the complex gene-environment interactions contributing to ADHD symptoms and executive attention functioning in PI children.

This study sought to contribute to the current literature on behavioral and electrophysiological correlates of attention problems in PI children. This work represents an early step toward the larger goal of elucidating developmental pathways associated with attentional and behavioral regulation difficulties in children who have experienced early adversity. Consistent with prior research, PI children in this study were reported by

their parents to have increased levels of ADHD symptoms, although most children's symptoms fell below the level associated with ADHD diagnosis. Surprisingly, no differences were found between PI and NA children in their behavioral performance on computerized measures of executive attention. Analysis of relative EEG power collected at 18 months of age indicated that PI children demonstrated greater concentration of low frequency EEG power (e.g., theta) than did NA children, suggesting the presence of a maturational delay or developmental deviation in brain activity patterns. Contrary to expectations, when relative EEG power was examined again when participants were 5 ½ years of age, PI children continued to demonstrate greater concentration of low frequency (e.g., theta) frontal relative EEG power, despite having spent an average of 4 ½ years in their adoptive homes. PI children's patterns of EEG power were less stable over time than those of NA children, which is consistent with evidence of significant change and variation in developmental outcomes once PI children are placed in family based care.

Analyses of concurrent and longitudinal associations between EEG and behavioral measures of attention regulation (e.g., parent reported ADHD symptoms, go/no-go and flanker task performance) revealed unexpected findings. Presumably less mature patterns of brain activity at 18 months of age in PI children (e.g., increased concentration of relative power in the theta band vs. the alpha band, decreased relative alpha power) were associated with better behavioral outcomes in regard to parent reported ADHD symptoms at 5 ½ years of age. These findings suggest the importance of considering developmental context (e.g., resource poor vs. resource rich environments). Concentration of brain activity at low frequencies may be adaptive in poorly resourced

environments by preserving resources and later functioning, while this same pattern of brain activity may represent a developmental deviation or delay in resource rich environments.

The results of this study should be interpreted with caution, due to the small sample size and unequal sex distribution. In addition, PI children were primarily adopted from China and at an earlier age than many prior samples, thus potentially limiting the generalizability of these findings. These results require replication in a larger and more diverse sample. Despite these limitations, the findings suggest the need for further investigation regarding developmental pathways and neurophysiological correlates of attention problems and ADHD symptoms in PI children, as well as the ways these pathways and correlates may differ from those of typically developing children and never institutionalized children with ADHD. Limited research has been completed regarding this issue (e.g., Sonuga-Barke & Rubia, 2008). However, additional work utilizing larger samples and multiple levels of analysis (e.g., measures of the post-adoption environment, neuropsychological assessment, neuroimaging, gene-environment interactions) will be necessary to elucidate the complex interplay between experiences of early adversity and the development of capacities for attentional and behavioral regulation.

Such research is particularly important given promising early evidence for the effectiveness of specific interventions focused on improving attentional and behavioral control in young children (e.g., Diamond, Barnett, Thomas, & Munro, 2007). It is currently unclear whether these interventions will be effective in specific populations such as PI children. A more complete understanding of neurobehavioral factors

associated with the development and persistence of attentional and behavioral regulation problems following early adversity will be needed to inform future evidence based intervention programs.

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## Appendix

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Table 1

*Data Available for Analysis by Group at 5 ½ Year Assessment*

Measure	Parent HBQ	Go/no-go Task	Flanker Task	Resting EEG at 5 ½ years	Resting EEG at 18 months	Longitudinal Resting EEG (EEG at both 18 Months & 5 ½ Year Sessions)
	n (% of group)	n (% of group)	n (% of group)			
PI (n = 25)	25 (100%)	24 (96%)	19 (76%)	16 (64%)	21 (84%)	9 (36%)
NA (n = 33)	33 (100%)	33 (100%)	23 (70%)	22 (67%)	29 (88%)	19 (58%)

Table 2

*Group Differences in Parent Reported ADHD Symptoms*

Parent Reported HBQ Index/Scale	Group		Significance Test
	PI	NA	
	<i>M (SD)</i>	<i>M (SD)</i>	
ADHD Symptoms	.67 (.39)	.39 (.32)	$t(55) = 3.01, p < .01$
Inattention	.57 (.41)	.32 (.31)	$t(56) = 2.65, p = .01$
Impulsivity	.77 (.40)	.47 (.36)	$t(55) = 2.99, p < .01$

*Note.* Parent reported HBQ symptoms were rated on a 3 point scale (0 = Never or not true, 1 = Sometimes or somewhat true, 2 = Often or very true).

PI:  $n = 25$ ; NA:  $n = 33$ .

Table 3

*Group Differences in Go/No-Go Task Performance*

Performance Measure	Group		Significance Test
	PI	NA	
	<i>M (SD)</i>	<i>M (SD)</i>	
Go Accuracy	.93 (.05)	.94 (.07)	Group: $F(1,55) = .22, p = ns$
No-Go Accuracy	.68 (.22)	.65 (.21)	Group x Trial Type: $F(1,55) = .30, p = ns$
Correct Go RT (msec)	625. 58 (87.17)	635. 17 (92.70)	$t(55) = .72, p = ns$

PI:  $n = 24$ ; NA:  $n = 33$ .

Table 4

*Group Differences in Flanker Task Performance*

Performance Measure	Group		Significance Test
	PI	NA	
	<i>M</i> ( <i>SD</i> )	<i>M</i> ( <i>SD</i> )	
Congruent Accuracy	.81 (.11)	.76 (.10)	Group: $F(1,40) = 2.64, p = ns$
Incongruent Accuracy	.77 (.13)	.70 (.13)	Group x Trial Type: $F(1,40) = .31, p = ns$
Congruent RT (msec)	837.91 (125.65)	806.89 (106.81)	Group: $F(1,40) = 1.05, p = ns$
Incongruent RT (msec)	876.05 (118.00)	835.07 (113.63)	Group x Trial Type: $F(1,40) = 0.50, p = ns$

PI: *n* = 19; NA: *n* = 23.

Table 5

*Relative EEG Power at 18 Months of Age in Longitudinal EEG Participants*

EEG Power Measure	Group	Significance Test	
		PI	NA
		<i>M (SD)</i>	<i>M (SD)</i>
<b>Whole Scalp</b>			
Relative Theta		.46 (.06)	.42 (.02)
Relative Alpha		.33 (.03)	.35 (.02)
Relative Theta-Alpha		.13 (.09)	.07 (.04)
<b>Difference</b>			
<b>Frontal Region</b>			
Relative Theta		.46 (.04)	.42 (.03)
Relative Alpha		.33 (.03)	.34 (.02)
Relative Theta-Alpha		.13 (.06)	.09 (.03)
<b>Difference</b>			

PI:  $n = 9$ ; NA:  $n = 19$ .

Table 6

*Frontal Relative EEG Power at 5 ½ Years of Age*

Frontal Relative EEG Power Measure	Group		Significance Test
	PI	NA	
	<i>M (SD)</i>	<i>M (SD)</i>	
Relative Theta	.37 (.01)	.36 (.01)	$t(36) = 2.16, p < .05$
Relative Alpha	.35 (.01)	.35 (.01)	$t(36) = .17, p = ns$
Relative Theta-Alpha	.02 (.01)	.01 (.01)	$t(36) = 1.84, p = .07$
Difference			

PI:  $n = 16$ ; NA:  $n = 22$ .

Table 7

*Longitudinal Correlations in Frontal Relative EEG Power Between 18 Months and 5 ½ Years of Age*

Frontal Relative	All Participants	PI Group	NA Group
<b>EEG Power Measure</b>			
Relative Theta	.33 <sup>a</sup>	.08	.42 <sup>a</sup>
Relative Alpha	.24	.03	.44 <sup>a</sup>
Relative Theta - Alpha	.33 <sup>a</sup>	-.06	.62**
<b>Difference</b>			

All Participants:  $n = 32$ ; PI:  $n = 12$ ; NA:  $n = 20$ .

<sup>a</sup>  $p > .05$  and  $< .08$ , \*\*  $p < .01$ .

Table 8

*Concurrent Associations Between EEG Power and ADHD Symptoms at 5 ½ Years of Age*

Frontal Relative EEG Power Measure	ADHD Symptoms			Inattention			Impulsivity		
	All Participants	PI	NA	All Participants	PI	NA	All Participants	PI	NA
Relative Theta	.14	-.14	.27	.17	-.08	.29	.09	-.18	.22
Relative Alpha	-.26	-.26	-.26	-.21	-.36	-.08	-.28	-.14	-.38
Relative Theta - Alpha	.27	.04	.38	.26	.15	.26	.25	-.08	.43 <sup>a</sup>
Difference									

ADHD Symptoms & Impulsivity: All Participants:  $n = 37$ , PI:  $n = 16$ , NA:  $n = 21$ .Inattention: All Participants:  $n = 38$ ,  $n = 16$ ,  $n = 22$ .<sup>a</sup>  $p > .05$  and  $< .08$ .

Table 9

*Concurrent Associations Between EEG Power and Go/No-Go Task Performance at 5 ½ Years of Age*

Frontal Relative EEG Power Measure	Go Accuracy			No-Go Accuracy			Correct Go RT		
	All	PI	NA	All	PI	NA	All	PI	NA
	Participants			Participants			Participants		
Relative Theta	-.25	-.26	-.06	.07	.10	-.04	.06	.01	-.13
Relative Alpha	-.10	-.31	.11	.02	.21	-.12	.04	.30	-.16
Relative Theta - Alpha	-.14	-.04	-.12	.04	-.05	.05	.02	-.17	.02
Difference									

All Participants:  $n = 38$ , PI:  $n = 16$ , NA:  $n = 22$ .All  $p_s = ns$ .

Table 10

*Concurrent Associations Between EEG Power and Flanker Task Performance at 5 ½ Years of Age*

Frontal Relative EEG Power Measure	Congruent Accuracy			Incongruent Accuracy			Congruent RT			Incongruent RT		
	All	PI	NA	All	PI	NA	All	PI	NA	All	PI	NA
	Participants			Participants			Participants			Participants		
Relative Theta	.23	.40	-.12	.16	.30	-.20	-.18	-.51 <sup>a</sup>	.01	-.09	-.30	-.25
Relative Alpha	-.04	-.25	.18	-.09	-.32	.10	-.15	.01	-.35	-.16	.02	-.36
Relative Theta - Alpha	.23	.55 <sup>a</sup>	-.22	.20	.51 <sup>a</sup>	-.22	-.06	-.48	.26	.02	-.30	.05
Difference												

All Participants:  $n = 29$ , PI:  $n = 13$ , NA:  $n = 16$ .<sup>a</sup>  $p > .05$  and  $< .08$ .

Table 11

*Longitudinal Associations Between EEG Power at 18 Months of Age and ADHD Symptoms at 5 ½ Years of Age*

Frontal Relative EEG Power Measure	ADHD Symptoms			Inattention			Impulsivity		
	All Participants	PI	NA	All Participants	PI	NA	All Participants	PI	NA
Relative Theta	.08	-.21	.13	.12	-.15	.24	.04	-.25	.03
Relative Alpha	.01	.53*	-.27	.07	.48*	-.17	-.05	.52*	-.33
Relative Theta - Alpha	.05	-.40 <sup>a</sup>	.26	.05	-.34	.27	.05	-.43 <sup>a</sup>	.23
Difference									

ADHD Symptoms & Impulsivity: All Participants:  $n = 49$ , PI:  $n = 21$ , NA:  $n = 28$ .Inattention: All Participants:  $n = 50$ ,  $n = 21$ ,  $n = 29$ .<sup>a</sup>  $p > .05$  and  $< .08$ , \*  $p < .05$ .

Table 12

*Longitudinal Associations Between EEG power at 18 Months of Age and Go/No-Go Task Performance at 5 ½ Years of Age*

Frontal Relative EEG Power Measure	Go Accuracy			No-Go Accuracy			Correct Go RT		
	All	PI	NA	All	PI	NA	All	PI	NA
	Participants			Participants			Participants		
Relative Theta	-.19	-.36	-.10	-.15	-.18	-.19	.01	.03	-.08
Relative Alpha	.16	-.05	.27	-.18	-.17	-.16	.01	.23	-.11
Relative Theta - Alpha	-.22	-.23	-.23	-.02	-.05	-.04	.00	-.09	.01
Difference									

All Participants:  $n = 49$ , PI:  $n = 20$ , NA:  $n = 29$ .All  $p$ s = ns.

Table 13

*Longitudinal Associations Between EEG Power at 18 Months of Age and Flanker Task Performance at 5 ½ Years of Age*

Frontal Relative EEG Power Measure	Congruent Accuracy			Incongruent Accuracy			Congruent RT			Incongruent RT		
	All	PI	NA	All	PI	NA	All	PI	NA	All	PI	NA
	Participants			Participants			Participants			Participants		
Relative Theta	.14	.15	.05	.12	.09	.07	-.10	-.11	-.20	-.14	-.22	-.16
Relative Alpha	.01	-.11	.25	-.05	-.12	.13	.10	.14	.20	.08	.09	.22
Relative Theta - Alpha	.10	.16	-.10	.11	.12	-.02	-.12	-.13	-.26	-.14	-.20	-.24
Difference												

All Participants:  $n = 34$ , PI:  $n = 15$ , NA:  $n = 19$ .All  $p$ s = ns.

### Figure Captions

*Figure 1.* Visual presentation of the go/no-go task design.

*Figure 2.* Visual presentation of the animal flanker task design.

*Figure 3.* Within group associations of frontal relative theta-alpha power difference and accuracy to congruent flanker trials at 5 ½ years of age. The x axes refer to percent accuracy to congruent trials. The y axes are the concentration of frontal relative theta power minus frontal relative alpha power at 5 ½ years of age.

*Figure 4.* Within group associations of frontal relative theta-alpha power difference and accuracy to incongruent flanker trials at 5 ½ years of age. The x axes refer to percent accuracy to incongruent trials. The y axes are the concentration of frontal relative theta power minus frontal relative alpha power at 5 ½ years of age.

*Figure 5.* Within group longitudinal associations between frontal relative alpha power at 18 months of age and parent reported ADHD symptoms at 5 ½ years of age.

The x axes are the mean total parent report ADHD symptoms score at 5 ½ years of age. The y axes are the concentration of frontal relative power in the alpha band at 18 months of age.

*Figure 6.* Within group longitudinal associations between frontal relative alpha power at 18 months of age and parent reported inattention symptoms at 5 ½ years of age.

The x axes are the mean parent report inattention symptoms score at 5 ½ years of age. The y axes are the concentration of frontal relative power in the alpha band at 18 months of age.

*Figure 7.* Within group longitudinal associations between frontal relative alpha power at 18 months of age and parent reported impulsivity symptoms at 5 ½ years of age.

The x axes are the mean parent report impulsivity symptoms score at 5 ½ years of age.

The y axes are the concentration of frontal relative power in the alpha band at 18 months of age.

*Figure 8.* Within group longitudinal associations between frontal relative theta-alpha power difference at 18 months of age and parent reported ADHD symptoms at 5 ½ years of age. The x axes are the mean total parent report ADHD symptoms score at 5 ½ years of age. The y axes are the concentration of frontal relative theta power minus frontal relative alpha power at 18 months of age.

*Figure 9.* Within group longitudinal associations between frontal relative theta-alpha power difference at 18 months of age and parent reported impulsivity symptoms at 5 ½ years of age. The x axes are the mean parent report impulsivity symptoms score at 5 ½ years of age. The y axes are the concentration of frontal relative theta power minus frontal relative alpha power at 18 months of age.

Animal Theme Go/No-Go Task  
Frequency of targets (go trials) = 75%  
2 blocks of 60 trials (120 trials)  
Per block: 45 go trials, 15 no-go trials  
No-go trials follow 1, 3, or 5 go trials



Non-Target  
(No-Go)  
Stimuli



Target (Go) Stimuli

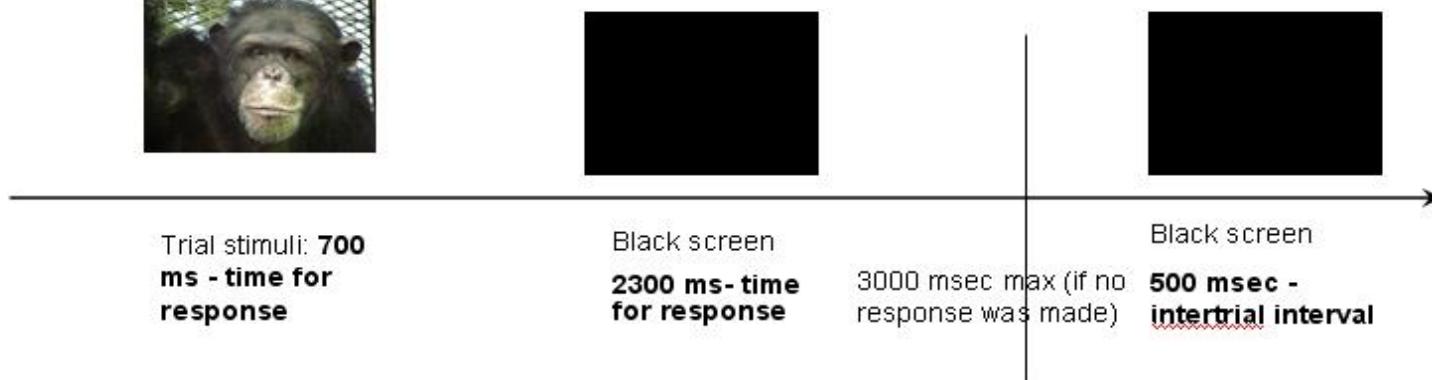


Figure 1. Go/No-Go task design.

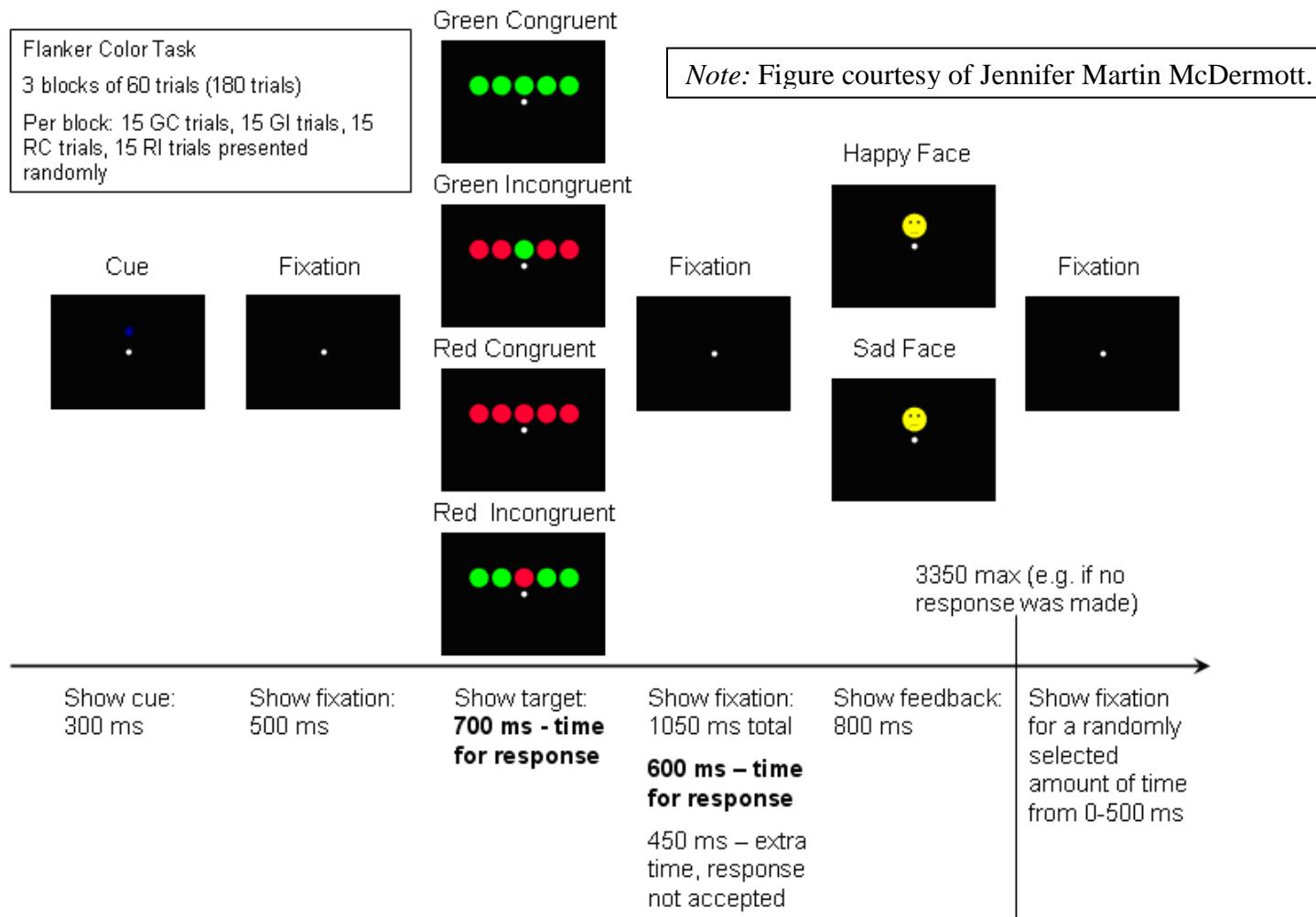


Figure 2. Flanker task design.

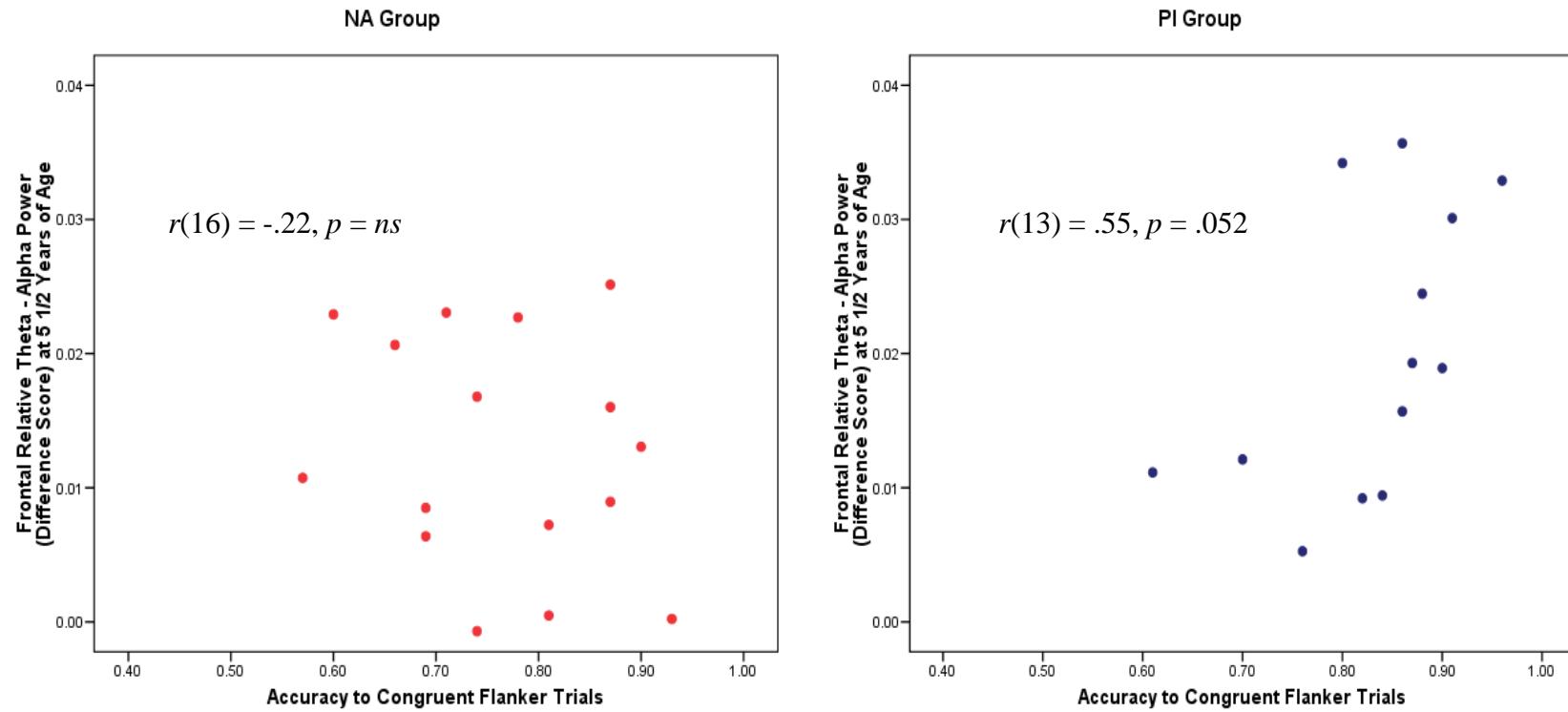


Figure 3. Within group associations of frontal relative theta-alpha power difference and accuracy to congruent flanker trials at 5 ½ years of age.

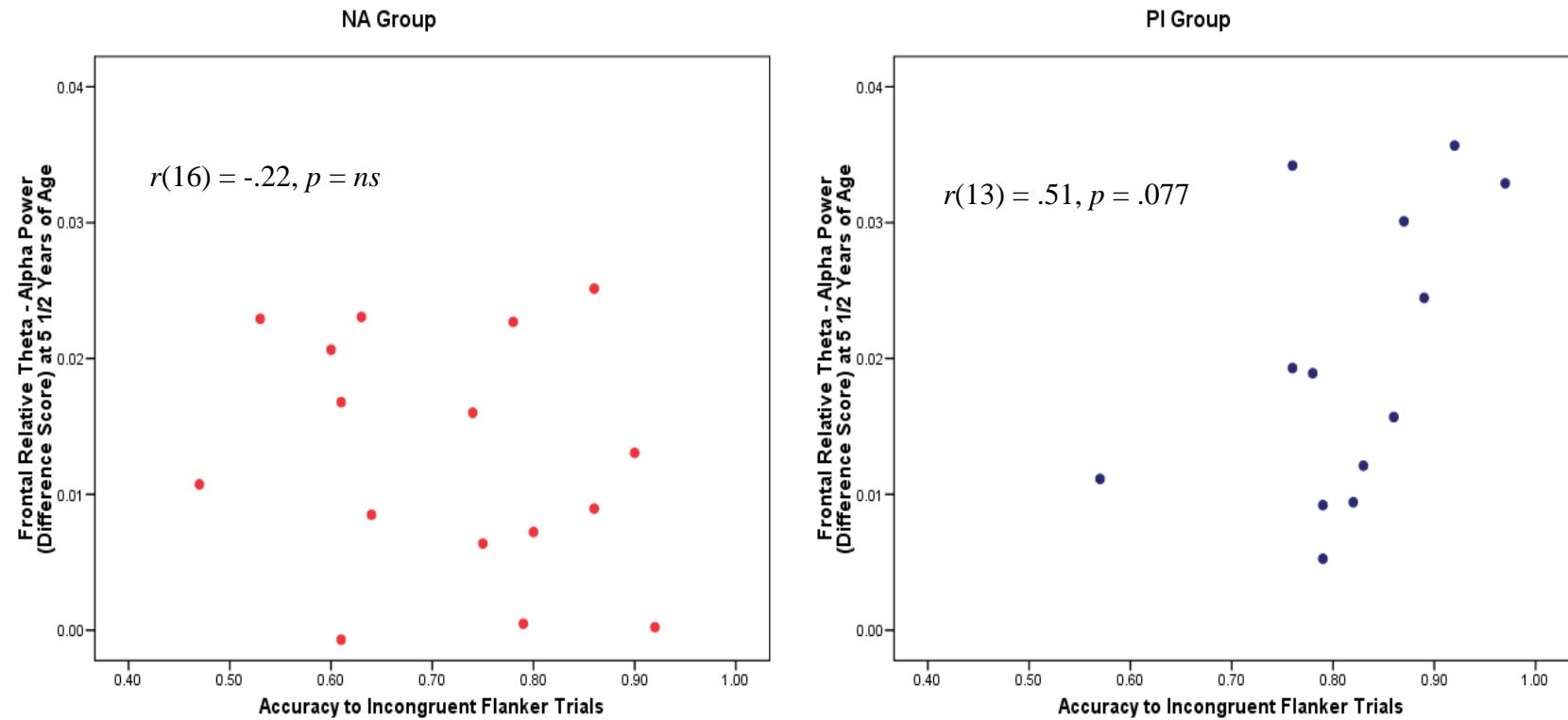


Figure 4. Within group associations of frontal relative theta-alpha power difference and accuracy to incongruent flanker trials at 5 ½ years of age.

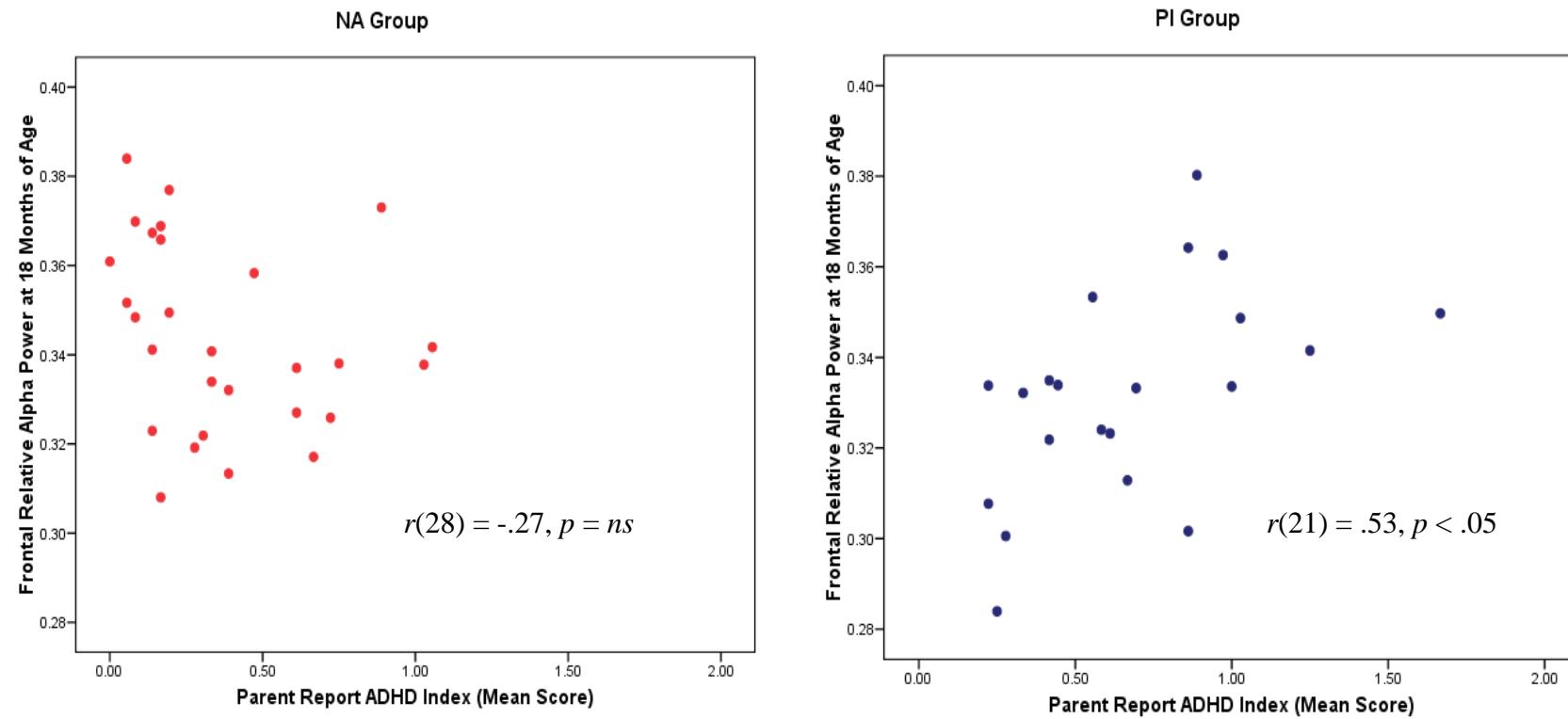


Figure 5. Within group longitudinal associations between frontal relative alpha power at 18 months of age and parent reported ADHD symptoms at 5 ½ years of age.

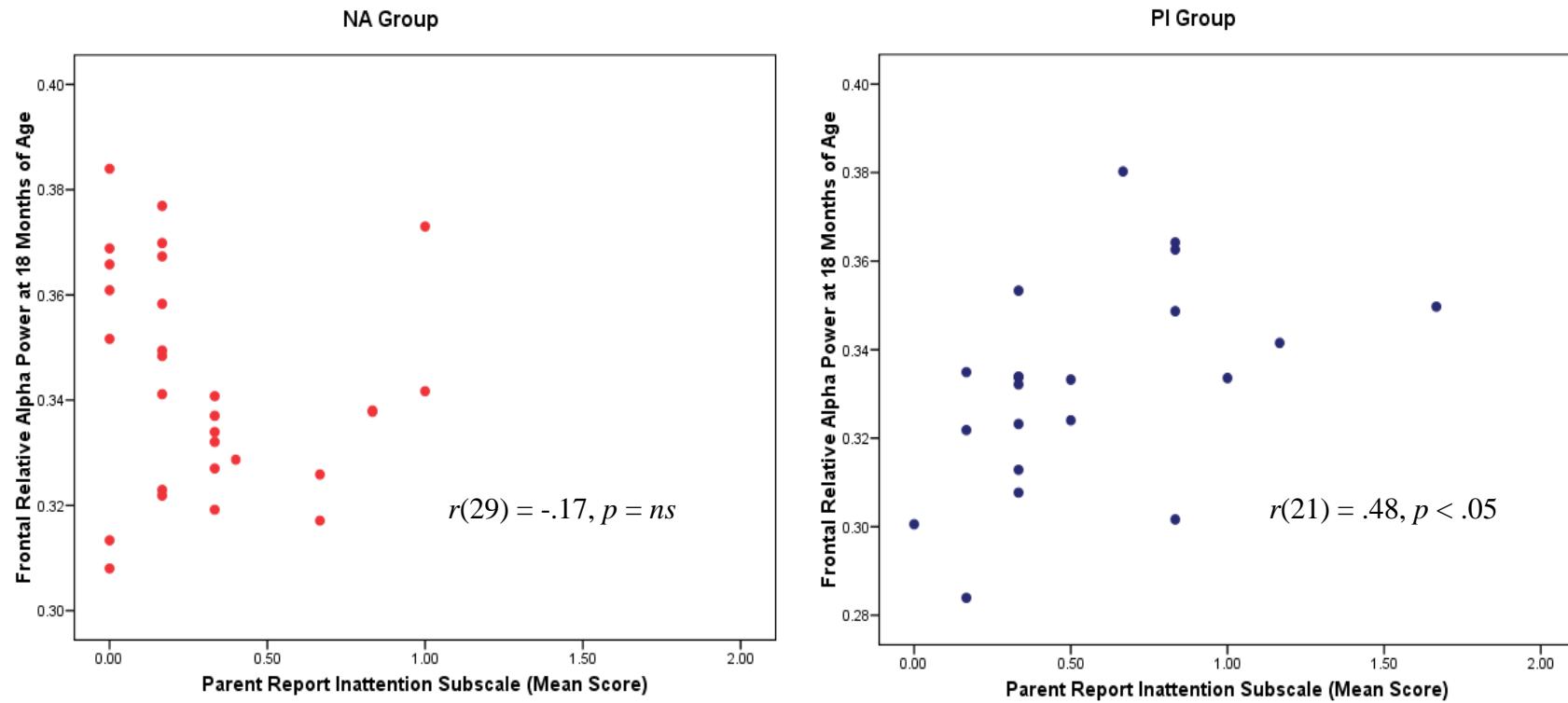


Figure 6. Within group longitudinal associations between frontal relative alpha power at 18 months of age and parent reported inattention symptoms at 5 ½ years of age.

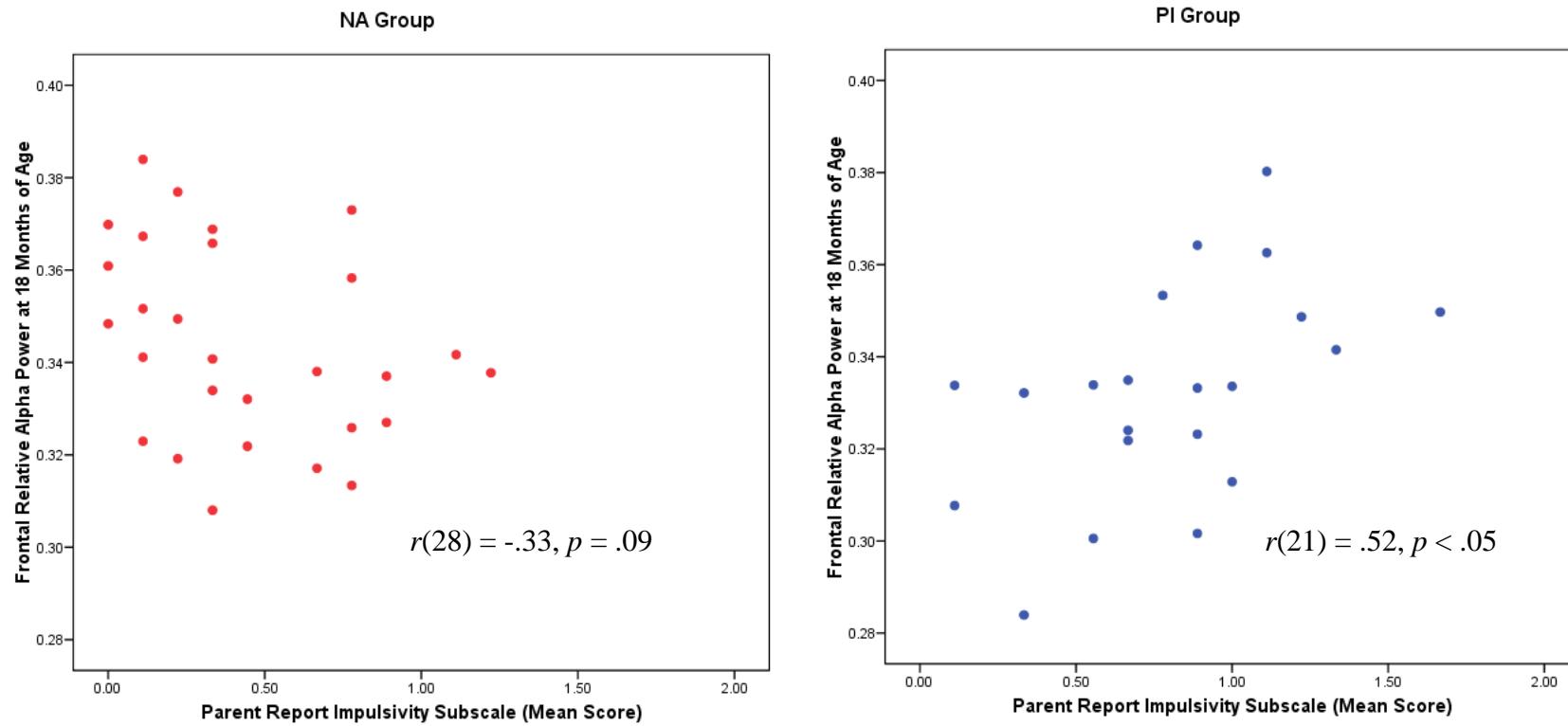


Figure 7. Within group longitudinal associations between frontal relative alpha power at 18 months of age and parent reported impulsivity symptoms at 5 ½ years of age.

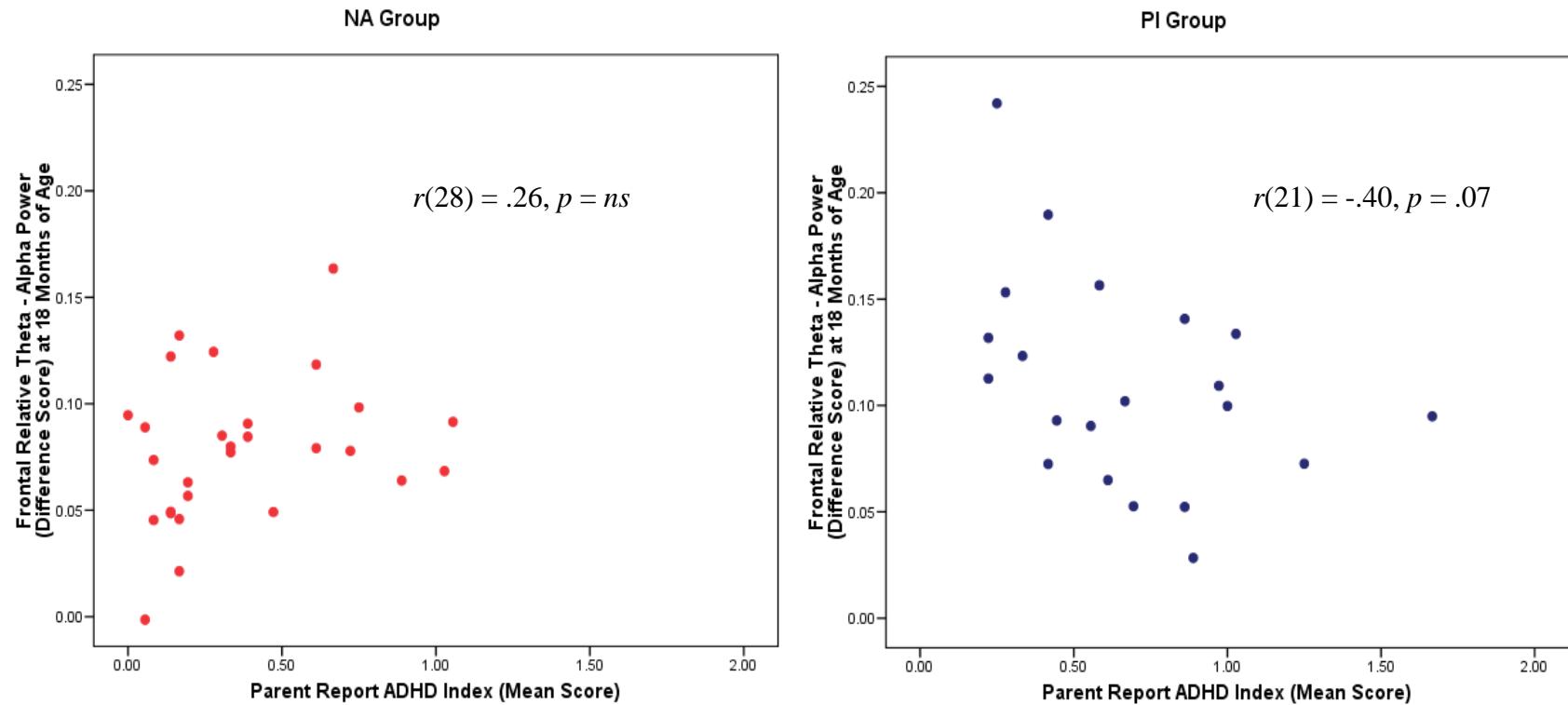


Figure 8. Within group longitudinal associations between frontal relative theta-alpha power difference at 18 months of age and parent reported ADHD symptoms at 5 ½ years of age.

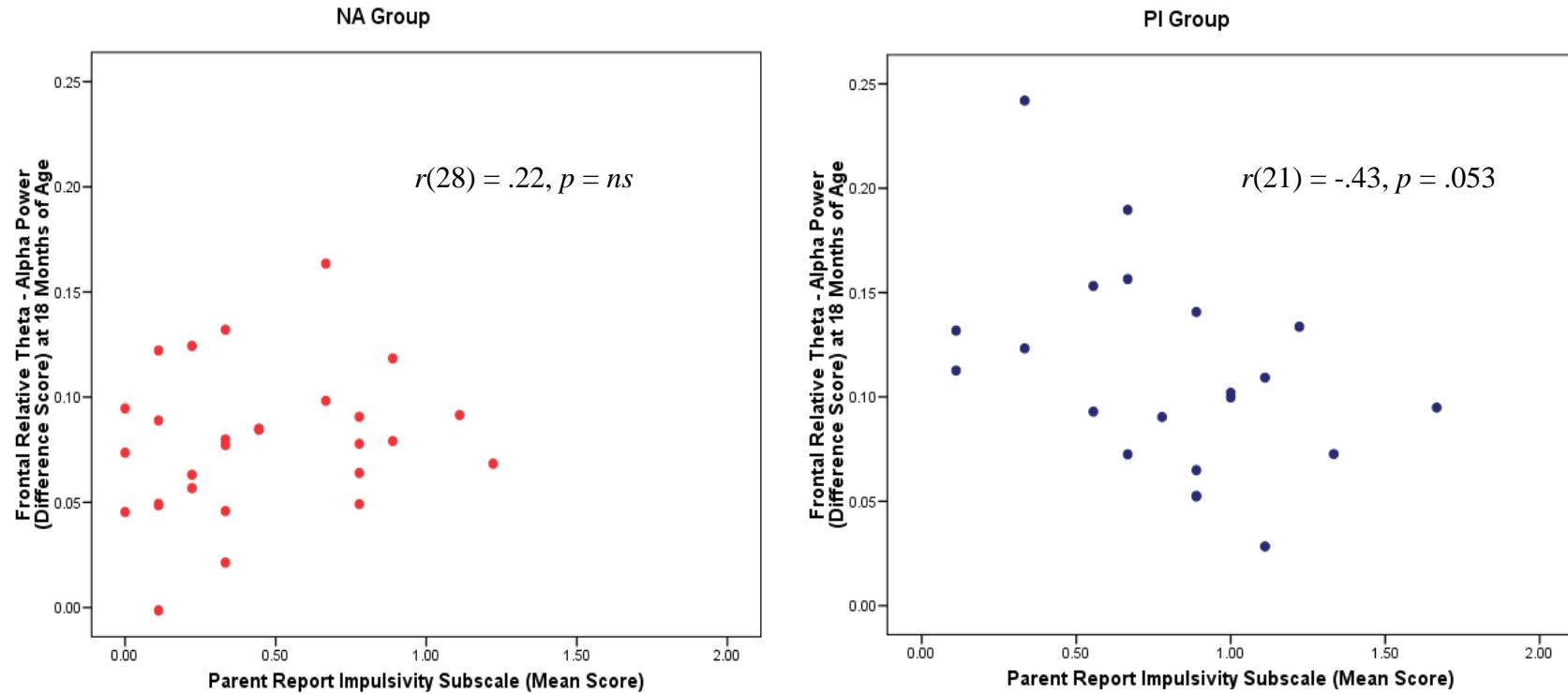


Figure 9. Within group longitudinal associations between frontal relative theta-alpha power difference at 18 months of age and parent reported impulsivity symptoms at 5 ½ years of age.

*Parent Report Health and Behavior Questionnaire (HBQ) Items for Inattention and Impulsivity Subscales*

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Scale	Item
Inattention (mean of 6 items)	1) Distractible, has trouble sticking to any activity 2) Has difficulty following directions or instructions 3) Can't concentrate, can't pay attention for long 4) Jumps from one activity to another 5) Does not seem to listen 6) Loses things
Impulsivity (mean of 9 items)	1) Fidgets 2) Can't stay seated when required to do so 3) Impulsive or acts without thinking 4) Has difficulty awaiting turn in games or groups 5) Interrupts, blurts out answers to questions too soon 6) Has difficulty playing quietly 7) Talks excessively 8) Interrupts or butts in on others 9) Does dangerous things without thinking

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*Note.* Items were rated on a 3 point scale: 0 = Never or not true, 1 = Sometimes or somewhat true, 2 = Often or very true. The ADHD Symptoms Index is the mean of the Inattention and Impulsivity subscales.