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Undergraduate

# Behavioral and EEG Correlates of Reduced Executive Functioning in Adolescents

By Joshua Espano

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## **Abstract**

Exposure to orphanage care or other deprived conditions represent a contributing risk factor in the development of ADHD behaviors. Upon leaving these contexts, the resting EEG patterns found in post-institutionalized (PI) children resemble the EEG profile of children with behavior problems, such as inattention, hyperactivity, and impulsivity. Specifically, this atypical pattern consists of increased theta power relative to other power spectra and decreased alpha power. This study examined if this pattern persists years after adoption and/or whether they were found only in PI youth who developed ADHD. PI and nonadopted (NA) children, between 11-15 year old, comprised the two groups in the study. Approximately half of the participants in each group were diagnosed with ADHD. Parent-reported executive function and attention problems differed by ADHD, but not by adoption classification (PI/NA). The PI youth and NA youth with ADHD

exhibited the atypical EEG pattern related to early deprivation and ADHD-related deficits. In the PI youth only, concentrations in beta power and alpha power were positively and negatively associated with fewer executive functions.

## **Introduction**

Early adverse rearing, such as those experienced by orphans raised in institutions, constitutes a severe risk factor in development. These environments entail deficits in nutrition and health care, cognitive and physical stimulation, and stable interpersonal relationships (Gunnar, Bruce, & Grotevant, 2000). As a result, children who experience these conditions are more likely to be malnourished, as well as cognitively and socio-emotionally delayed relative to non-adopted comparison groups (Johnson, 2002; Maclean, 2003; Smyke et al., 2007). Inadequate caregiving quality is usually highlighted as the central aspect of institutionalization.

Individualized and responsive care is uncommon, even in the high-quality institutions, due to high child:caregiver ratios and constant schedule rotations (Tizard & Rees, 1974). Children in these contexts are unable to form stable attachments and are not responded to when distressed. Several studies demonstrate the effects of this deficit in caregiving. Tizard (1977) studied children in model orphanages used to train caregivers and found that behavioral issues continued in children despite improvements in nutrition and cognitive stimulation relative to regular orphanages. Although better than regular institutional caregiving, these model institutions still lacked continuity in care as the children's primary caregivers changed regularly as new caregivers came into training. In addition, the Bucharest Early Intervention Project (BEIP) group found that caregiving quality within the institution affected competence, cognitive development, and problematic behavior (Smyke et al., 2007). This same research group found an improvement in secure attachment behaviors following an intervention in which they reduced the

child:caregiver ratio (Smyke et al., 2002). Thus, experiencing unresponsive and inconsistent caregiving for prolonged periods produces negative outcomes for institutionalized children.

Researchers have assessed the degree to which consequences, related to institutionalization, continue after a drastic change in context (Maclean, 2003). For example, researchers have studied children adopted out of institutions and into stable, middle-class families. Due to the positive change in the caregiving quality, most post-institutionalized (PI) children recover from health problems and show improvements in growth and cognitive delays (Johnson, 2002; Maclean, 2003; Rutter, 1998). Despite remarkable recovery, behavioral problems persist relating to deficits in executive function that consist of capacities to self-regulate, inhibit responses, make decisions, shift attention, and manipulate information (Wilcutt et al., 2005). Researchers consistently identify attention and inhibitory control, as diminished in PI children and adolescents implicating deficits in their brain development responsible for executive functioning (Bos et al., 2009; Gunnar et al., 2007; Kreppner et al., 2001; Loman et al., 2013; McDermott et al., 2012). We can conclude from these findings that while behavioral and cognitive issues improve following an improvement in care, recovery is not complete and problems remain for many children.

It is important to determine how early adverse experiences shape the developing brain. Researchers have identified abnormalities in PI children relative to comparison groups, (Marshall et al., 2004; Tarullo, Garvin, & Gunnar, 2011) indicating the effects of institutional rearing on neural mechanisms. For instance, children who experienced or are experiencing institutional care tend to show cortical hypoactivation in their baseline electroencephalogram (EEG) waveform (Marshall et al., 2004; Tarullo et al., 2011). The EEG signal, a measure of electrical activity along the scalp, consists of fluctuating frequencies ranging from low to high. The frequencies, from low to high, commonly associated with brain activity are theta, alpha, and beta (Tarullo et

al., 2011). Cortical hypoactivation, or a lack of activation, reflects predominance in the lower frequencies, such as theta. Having an EEG distribution in the higher frequencies indicates faster processing and maturation of neural mechanisms. In contrast to PI individuals, the baseline EEG of non-adopted comparison groups consists of relatively more concentrations in the higher frequencies, specifically alpha and beta (Clarke et al., 2001; Gasser et al., 1988).

The sensitive period model and maturational lag model have both been used to explain the EEG abnormalities in PI children. The former proposes permanent damage following physical and/or psychological deprivation at a critical developmental period; the latter proposes that neural mechanisms are delayed and potentially recover. Since, evidence suggests that the effects of institutionalization persist at least 6 months after institutionalization (Marshall et al., 2004; Tarullo et al., 2011) more research is needed to determine if EEG abnormalities eventually normalize after a longer period in an improved environment.

The goal of the current study was to determine if the EEG band concentrations of PI adolescents resemble the atypical pattern previously found in younger PI and currently-institutionalized children (Marshall et al., 2004; Marshall et al., 2008; Tarullo et al., 2011), and children who have an Attention Deficit/Hyperactivity Disorder (ADHD) diagnosis (Bresnahan et al., 1999; Mann et al., 1992). The current study addresses the question of whether the effects of institutionalization on neural mechanisms are permanent. In contrast, finding typical EEG band concentrations in the PI adolescents would lend support to the plasticity and recovery capacity of neural mechanisms, especially when caretaking improves. A non-adopted group, half of which met the diagnostic criteria for ADHD, was included to possibly distinguish between ADHD related to institutionalization and ADHD in a non-adopted population. Loman's (2012) results regarding the same sample being studied highlight the similarity of PI and NA adolescents with ADHD on both behavioral and event-related potential (ERP) measures. The youth with ADHD

were similar on parent reported behavior commonly associated with ADHD. Also, they showed similar neural responses (ERP components such as N2 and P300) to specific time-locked stimuli. It was expected that the adolescents with ADHD would have more documented ADHD-related behavior problems and more pronounced atypical EEG profiles than the adolescents without ADHD. It was also predicted that having an atypical EEG profile would correlate with more executive function deficits.

## **Methods**

### *I. Participants*

The participants were 11 to 15 year old adolescents, divided into post-institutionalized (PI) and nonadopted (NA) groups. Half of the participants in each group met the diagnostic criteria for ADHD. The following are the four subgroups:

- post-institutionalized with ADHD (*PI-A*,  $n = 26$ ),
- post-institutionalized without ADHD (*PI-N*,  $n = 20$ ),
- non-adopted with ADHD (*NA-A*,  $n = 19$ ), and
- non-adopted without ADHD (*NA-N*,  $n = 20$ ).

The post-institutionalized groups were institutionalized for at least 6 months prior to their adoption. The ADHD groups met the criteria for an ADHD diagnosis, which is defined by exhibiting 4 or more inattentive behaviors and 4 or more hyperactive/impulsive behaviors. Internationally adopted participants were recruited from the Minnesota International Adoption Project Registry and non-adopted participants were recruited locally.

## *II. Procedures*

The data used for the current study were collected as a part of a larger study (See Loman, 2012 for detailed procedures). The current study will only examine the baseline EEG data and one of the behavioral measures collected. The data for the fourth group was not collected at the same time as the others since they were not included in Loman's dissertation (2012).

## *III. Measures*

*EEG.* Baseline EEG was recorded with a 128-electrode Hydrocel Geodesic Sensor Net (Electrical Geodesic, Inc.) during eyes-open and eyes-closed trials for a total of 8 trials, for one minute each. The participants were told to stare straight into the middle of a blank computer screen, to minimize movement artifacts that would otherwise obscure the EEG signal, during eyes-open trials. The participants were instructed to close their eyes for the eyes-closed trials and told to open their eyes after 1 minute. Impedances were checked prior to collection to ensure impedances at all sites were below 100K $\Omega$  prior to beginning collection; they were fixed by adding drops of water to the sponges of the electrodes. In a few instances impedences for sites were above the 100K $\Omega$  threshold due to broken electrodes.

The EGI software Netstation (Electrical Geodesic, Inc., Eugene, OR) was used for data collection and data processing. All channels were referenced to Cz (midline of scalp) during collection. The difference in electrical activity between a reference site and a recording site functions as the voltage of a specific channel or electrode (Light et al., 2010). In Netstation software, data were filtered to .3 and 40Hz, segmented into individual epochs, and baseline corrected across the entire epochs before individuals scorers manually artifacted for bad channels. Channels that were marked bad were imputed using bad channel replacement in Netstation. Data were then re-referenced to an average reference and eye-channels were

excluded from the averaged reference because of consistent noise in some of the eye-channel data. Data sets were then exported into EEGLab software where artifacts due to eye-blinks and movement were manually removed from the data in order to only pick up brain activity. Inter-rater reliability was calculated using inter-class correlations which were all above 0.9 in order to make sure the different coders were consistent with each other.

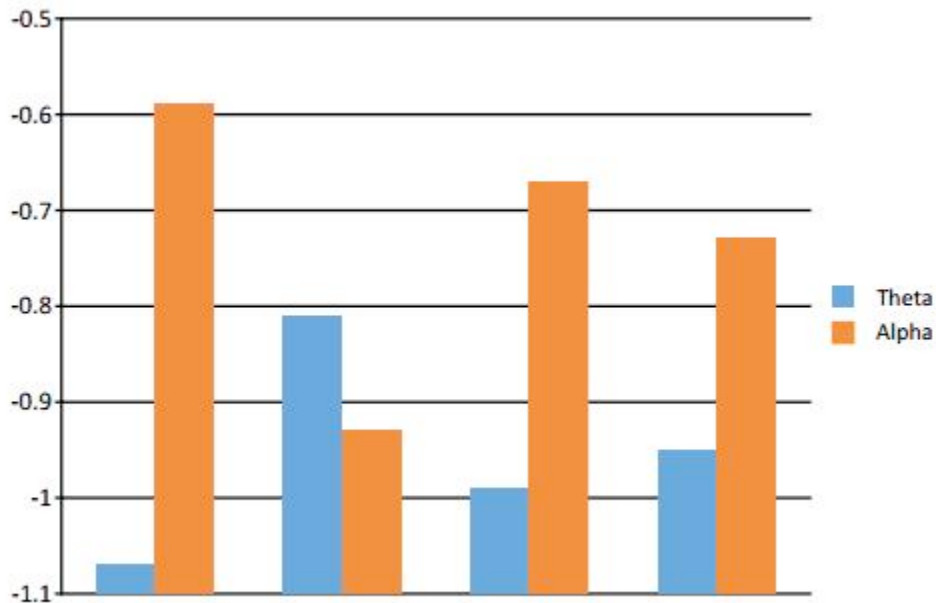
Data were analyzed using a Fast Fourier Transform using a window size of 1000 and a 50% overlap. Data from electrodes in regions of interest were exported for analysis and mean power across each region and band of interest were calculated. Consistent with other studies, EEG data were log (ln) transformed (Marshall et al., 2004; Tarullo et al., 2011). Theta was defined as 4-7.5Hz, alpha was defined as 8-12Hz, and beta was defined as 13-25Hz. Relative power was calculated by taking the mean power in each region of interest in the specific band, relative to the total power in that region in the alpha, theta and beta bands combined and then creating a mean for relative alpha across all regions of interest. The EEG processing has been completed for the majority of the participants (*PI-A*,  $n = 16$ ; *PI-N*,  $n = 15$ ; *NA-A*,  $n = 15$ ; *NA-N*,  $n = 13$ ). Relative power was used in order to account for individual differences such as skull thickness, which affects the absolute band power or the total output of a band. Using the relative power measure allows us to determine how much of a specific band power is in a waveform. Executive Function (EF). The parent-report version of Conners 3<sup>rd</sup> edition (Conners, 2008) Short Form was used to assess ADHD-related cognitive, emotional, and behavioral problems. We only used the executive functioning problems, hyperactivity/impulsivity, and inattention subscales for the purposes of this study. This measure is a validated questionnaire, which used a large, representative sample that also considered gender norms.



## Results

### *I. Relative power*

We conducted a 2 (ADHD/No-ADHD) by 2 (PI/NA) Multivariate Analysis of Variance (MANOVA) with relative theta, alpha, and beta as dependent variables. There was a significant multivariate effect of ADHD/No-ADHD, Wilk's  $\lambda = 0.84$ ,  $F(3, 54) = 3.37$ ,  $p < 0.05$ . Univariate tests showed that there were no significant effects for relative theta,  $F(1,55) = 3.38$ ,  $p > 0.05$ . For relative alpha, there was a main effect of ADHD/No-ADHD,  $F(1,55) = 7.26$ ,  $p < 0.05$ , with higher relative alpha in adolescents with ADHD. For relative beta, there was again a main effect of ADHD/No-ADHD,  $F(1,55) = 7.01$ ,  $p < 0.05$ , with higher relative beta only in the PI adolescents without ADHD compared to all other groups. There was no multivariate effect of PI/NA, Wilk's  $\lambda = 0.94$ ,  $F(3,54) = 1.11$ ,  $p > 0.05$ . Also, there was no significant effect of ADHD/No-ADHD x PI/NA interaction, Wilk's  $\lambda = 0.87$ ,  $F(3,54) = 2.62$ ,  $p = 0.60$ .



*Fig. 1. Relative alpha and theta power in the PI and NA youth.*

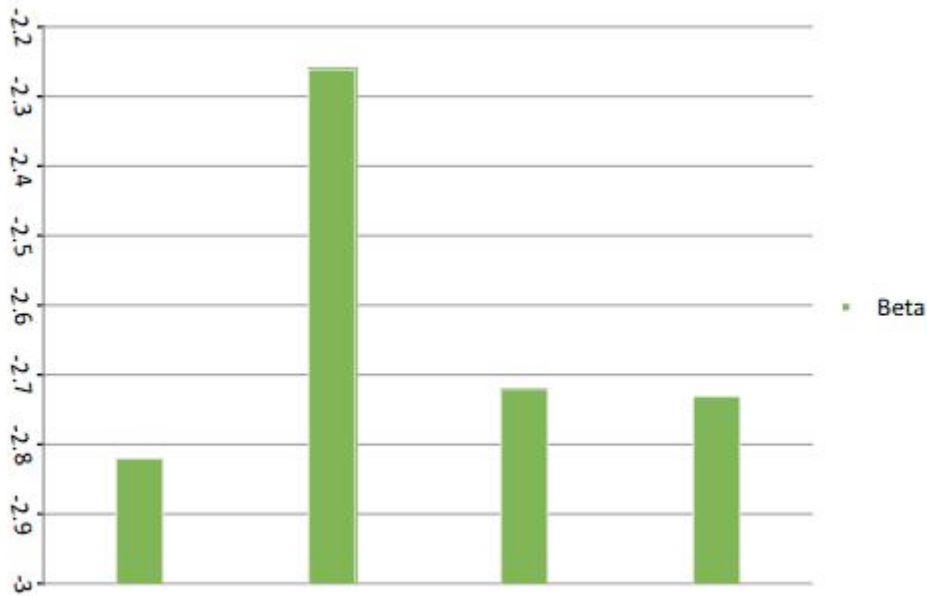


Fig. 2. Relative Beta power in the PI and NA youth.

## II. Executive Functioning

A 2 (ADHD/No-ADHD) by 2 (PI/NA) MANOVA, with parent reported EF problems, inattention, and hyperactivity/impulsivity as dependent variables, revealed a significant multivariate effect of ADHD/No-ADHD, Wilk's  $\lambda = 0.26$ ,  $F(3,69) = 64.88$ ,  $p < 0.001$ . Univariate tests showed that for EF problems, there was a main effect of ADHD,  $F(1,70) = 62.80$ ,  $p < 0.001$ . For inattention, there was another main effect of ADHD,  $F(1,70) = 159.99$ ,  $p < 0.001$ . For hyperactivity/impulsivity, there was again a main effect of ADHD,  $F(1,70) = 104.74$ ,  $p < 0.001$ . There was no significant multivariate effect of PI/NA Wilk's  $\lambda = 0.97$ ,  $F(3,69) = 0.63$ ,  $p > 0.05$ . Again, there was no significant multivariate effect of ADHD x PI interaction Wilk's  $\lambda = 0.99$ ,  $F(3,69) = 0.33$ ,  $p > 0.05$ .

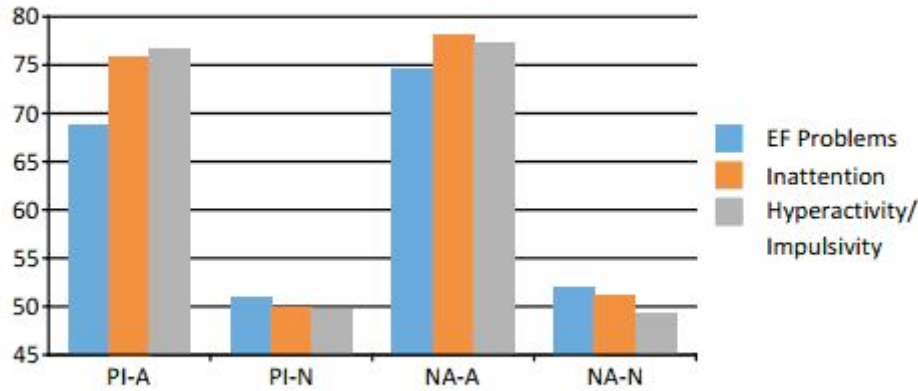


Fig. 3. Parent reported behavioral issues related to Executive Functioning across all groups.

### III. EEG correlates of Executive Function

Relative theta power was not significantly correlated with any of our parent reported EF behaviors. In the PI youth, relative alpha was positively correlated with EF problems,  $r(27) = 0.44$ ,  $p < 0.05$ , and inattention,  $r(27) = 0.50$ ,  $p < 0.05$ , but not hyperactivity/impulsivity,  $r(27) = 0.31$ ,  $p > 0.05$ . For the NA youth, relative alpha was not significantly correlated with EF problems,  $r(26) = 0.09$ ,  $p > 0.05$ , inattention,  $r(26) = 0.00$ ,  $p > 0.05$ , and hyperactivity/impulsivity,  $r(26) = -0.03$ ,  $p > 0.05$ . Tests of the significance of the difference between these correlations were computed. None of the differences were significant.

Relative beta power was negatively correlated with EF-problems, inattention and hyperactivity/impulsivity for the PI, but not the NA youth [PI, EF problems  $r(27) = -0.61$ ,  $p < 0.05$ , inattention  $r(27) = -0.62$ ,  $p < 0.05$ , and hyperactivity/impulsivity  $r(27) = -0.51$ ,  $p < 0.05$ ; NA, EF problems,  $r(26) = -0.02$ ,  $p > 0.05$ , inattention,  $r(26) = -0.07$ ,  $p > 0.05$ , and

hyperactivity/impulsivity,  $r(26) = -0.01, p > 0.05$ ]. Tests of differences between these correlations revealed significant difference for two and trend level difference for one of three measures: EF problems,  $Z = -2.46, p < 0.05$ , inattention  $Z = -2.34, p < 0.05$ , and hyperactivity/impulsivity,  $Z = -1.97, p < 0.06$ .

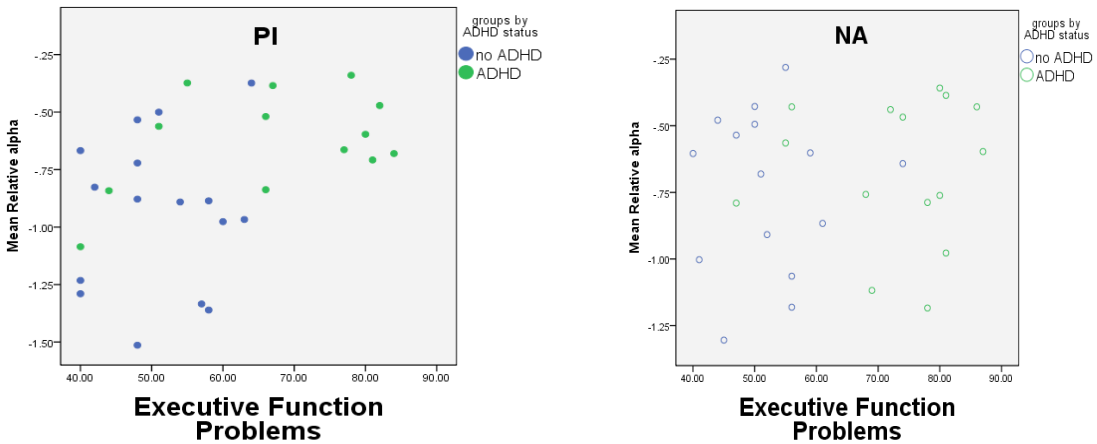


Fig. 4. Parent reported executive function problems and relative Alpha power in PI and NA youth.

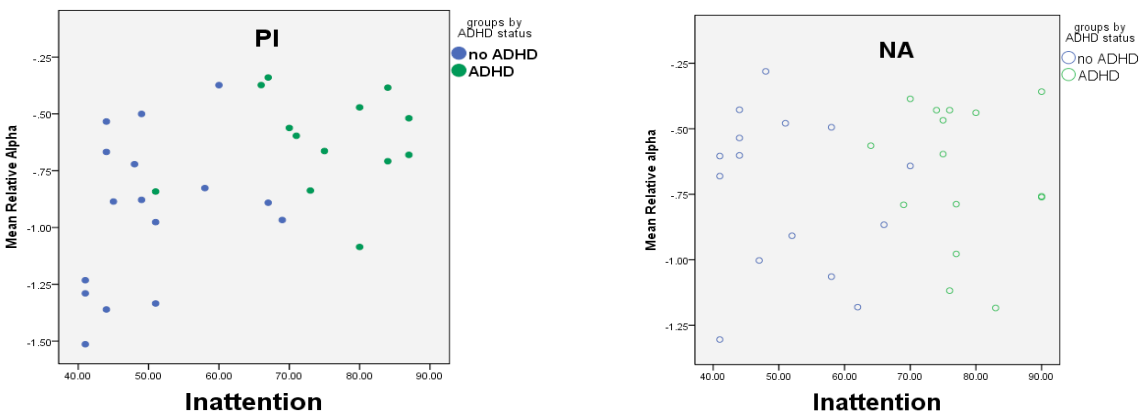


Fig. 5. Parent reported inattention and relative Alpha power in the PI and NA youth.

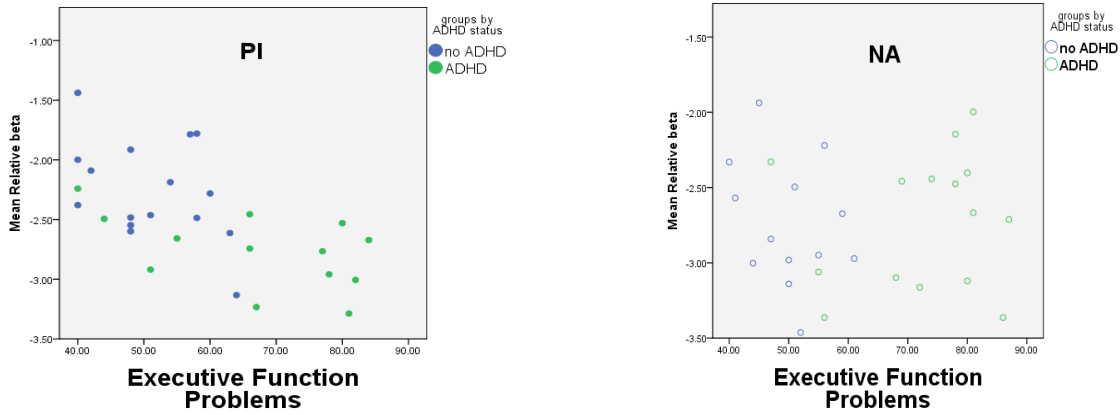


Fig. 6. Parent reported executive function problems and relative Beta power for PI and NA youth.

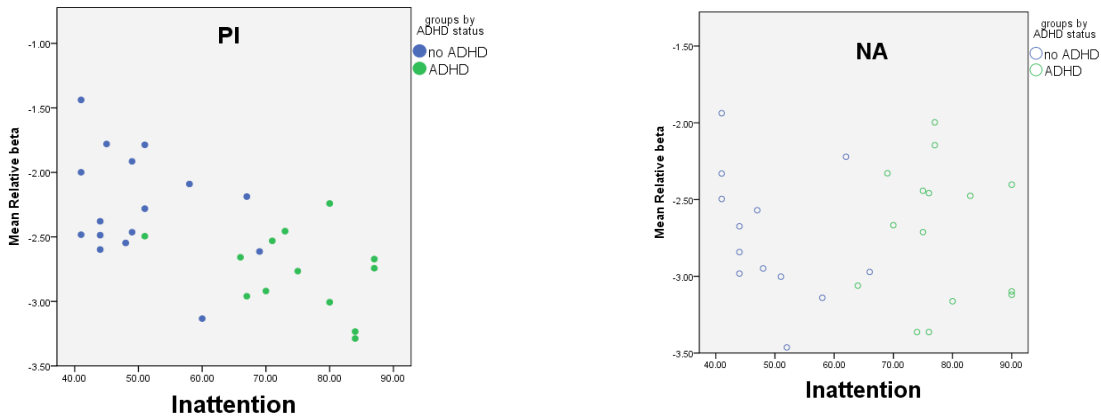


Fig. 7. Parent reported inattention and relative Beta power in PI and NA youth.

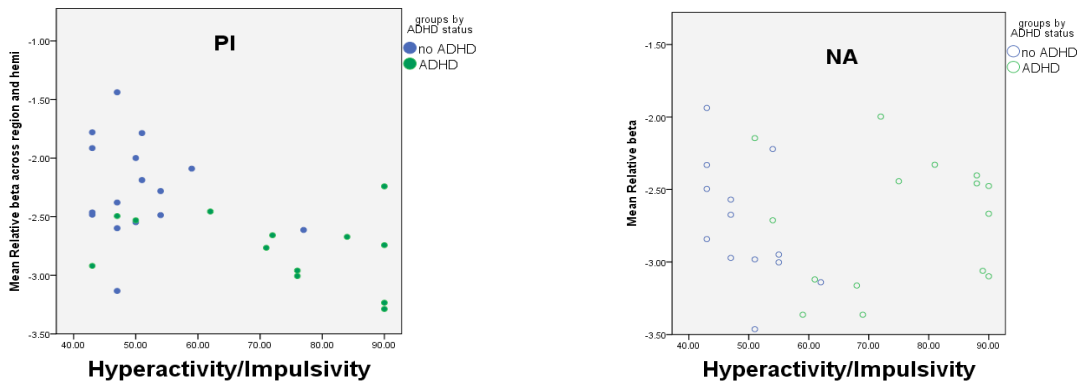


Fig. 8. Parent reported hyperactivity/impulsivity and relative Beta power in PI and NA youth.

## **Discussion**

Patterns of baseline EEG differed between youth with and without ADHD, furthermore, they differed in the direction of basal activity reflecting higher frequency power spectra in those without ADHD compared to those with ADHD. The difference was seen in the higher frequencies: more alpha than beta power, instead of a greater percentage in the theta band in youth with ADHD. Importantly, we found no differences in EEG power spectra distributions between the PI and NA children regardless of ADHD status. We did find that relative alpha and beta power were more closely correlated with ADHD symptoms and executive functioning among the PI than NA youth.

Although the current study found an atypical pattern in the youth with ADHD relative to comparison groups, the findings are not consistent with previous literature which points to theta power as being elevated (Bresnahan et al., 1999; Lazzaro et al., 1999). There are several reasons that explain why theta power was not elevated in the youth with ADHD. First, the ages of the participants within the sample and their stage of brain development should be taken into consideration. The EEG waveform of adolescents differs from that of children due to neuronal maturation processes, such as myelination (Brady et al., 1999; Marshall et al., 2004). Further, EEG power distributions shift into the higher frequencies as slow wave activity decreases with age (Bresnahan et al., 1999; Clarke et al., 2001). Therefore, we should expect to see the differences, if any, in the higher frequencies between adolescents with and without ADHD.

The EEG waveform of children with ADHD shows an atypical predominance in the lower frequencies, specifically in the theta band, compared to children without ADHD (Mann et al., 1999). Therefore, this pattern will most likely continue with age in the higher frequencies, specifically in alpha (the band range following theta), because of the frequency shift due to brain maturation. The shift in frequencies partially explains the lack of differences in the theta band

between youth with and without ADHD in the current study. Findings of increased alpha power in adolescents and adults with ADHD suggest the shifting of atypical activity into higher frequencies with age (Bresnahan & Barry, 2002; Koehler et al., 2009; Lazzaro et al., 1999). The EEG profile of the youth with ADHD in our current sample may reflect the neurophysiological progression of individuals with ADHD. However, the frequency shifting explanation still fails to fully account for the lack of differences in theta. The studies that implicated elevated alpha power also found elevated theta power in adolescents and adults with ADHD (Bresnahan & Barry, 2002; Bresnahan et al., 1999; Koehler et al., 2009; Lazzaro et al., 1999). The continued presence of theta power in individuals with ADHD implies a difference between samples of previous studies and the sample of the current study.

In contrast with the current study, previous researchers tested participants who never took or currently were not taking medication (Bresnahan & Barry, 2002; Lazzaro et al., 1999). These same participants showed the elevations in theta power. The medication taken by the youth with ADHD in this study may have influenced their EEG waveform. Atypical EEG distributions of individuals with ADHD seem to be more pronounced in the absence of medication use. The medication use of the current sample may have reduced activity in the theta band. Some researchers suggest that ADHD medication normalizes the brain activity of individuals with ADHD, evident in the reduction of theta power in their EEG waveform (Clarke et al., 2002). It is unclear whether the discrepancy between the data found in this study and previous research is in fact due to the medication as we do not have adequate information regarding the medication use of our participants. However, medication use should be considered when evaluating the EEG profiles of individuals with ADHD based on the normalizing effects found by Clarke and colleagues (2002) and the findings of the current study. Future studies need to take into account

and further examine the effects of medication on brain activity. The similar brain activity of the PI and NA youth with ADHD corresponds to similarities in their behavior.

PI and NA youth with ADHD did not differ in terms of executive function deficits measured with the parent report of their behavior. The youth with ADHD were similar, contrary to Sonuga-Barke and Rubia's (2008) finding that PI adolescents with ADHD were more impaired than a standard ADHD sample. The discrepancy may take root in the difference of measurement. Sonuga-Barke and Rubia's study used investigator-based interviews to determine problem behaviors, while the current study used a parent-report questionnaire. In examining inhibitory control and planning with the Go-No-Go and Stockings of Cambridge tasks, respectively, Loman (2012) noted that PI adolescents with ADHD did differ relative to NA adolescents with ADHD. This suggests that these two subgroups do differ in certain respects, which the measures of the present study failed to detect. Further research is needed in order to determine if PI adolescents with ADHD and NA adolescents with ADHD are actually significantly different in terms of their executive function deficits and behavior. It is also noteworthy that PI and NA youth without ADHD did not differ. This indicates more resilience, and/or less deprivation regarding the experiences of the PI youth without ADHD relative to the PI youth with ADHD. It cannot be stated with certainty that different institutional experiences produced the differences within the PI youth; but, it is important to note that the PI youth with ADHD and PI youth without ADHD were adopted from different regions (Loman, 2012). The majority of the PI youth without ADHD were adopted from regions with better institutional care relative to those regions where the PI youth with ADHD were adopted (Gunnar et al., 2007; Loman, 2012). Future research needs to acquire more accurate information on the different institutionalization experiences in order to elucidate the factors that contribute to the heterogeneous outcomes of PI adoptees.



The correlations between executive functioning deficits and relative beta power in the PI youth were consistent with previous literature, implicating increased high-frequency band power in self-regulation (Tarullo et al., 2011); however, this was not the case for relative theta power. Relative theta power did not correlate with the measures of executive functioning deficits in this study, contrary to the findings of Tarullo and colleagues (2011). The positive correlation between alpha power and executive functioning issues fits with the frequency shift explanation. Since hypoactivation shifts into the higher frequencies with age, alpha and not theta power should correlate with executive functioning deficits. However, the lack of differences found in the relative alpha correlations between the PI and NA suggests that the correlation might not be significant. It may just be the case that the effects were more detectable in the PI youth. It is difficult to interpret the lack of significant correlations in the NA youth and this is due to the low statistical power of the current study. The correlations between ADHD-related behavior and certain types of brain wave patterns are consistent with previous research (Tarullo et al., 2011). Namely, relative beta correlated with reduced ADHD-related behavior suggesting the role of high frequency distributions in self-regulation, attention, and inhibitory control.

The results should be interpreted with caution because of several limitations. Certain aspects of the current study's methodology need to be addressed in future research. Some of the results in the current study may reflect an amplified sex difference due to the unbalanced gender distribution in the PI-N subgroup ( $n = 20$ ,  $males = 2$ ). Clarke et al.'s (2001) comparison of EEG power distributions between genders revealed that pre-adolescent females showed a significant maturational lag evident in their increased low-frequency and decreased high-frequency band power. Therefore, a subgroup comprised of mostly females may have contributed to the observed elevation of the alpha band in youth with ADHD relative to the NA-N subgroup; it is important to note that the youth with ADHD still had the highest relative alpha elevation. The gender

imbalance is especially difficult to address in future studies due to the prevalence of ADHD-related problems of PI males (Loman, 2012). Other considerations for future research include increasing the sample size, separating eyes-open and eyes-closed trials, controlling for medication use, investigating any gender differences in the ADHD subgroups, and examining activity in specific regions as opposed to using a whole-head mean. Lastly, the cross-sectional design of the current study constrains our interpretations regarding the persistence of EEG abnormalities in PI adolescents. A longitudinal design will potentially provide more information as to whether the brains of children who experienced deprivation recover and normalize with age.

The aim of this study was to expand on previous findings of atypical EEG patterns in PI children and children with ADHD. In the current study, the hypoactivation pattern persisted in the form of the alpha band. Thus, researchers need to evaluate the utility of power in the alpha band when diagnosing adolescents, because this pattern is found in adults with ADHD. The EEG patterns found in the youth with ADHD in the current study indicate the persistence of abnormal brain activity. Additionally, the lack of theta power elevations may reflect the effects of medication, and not the amelioration of neural mechanisms. The findings of the current study can potentially improve ADHD diagnoses of adolescents. The similarities in executive function behavior within the ADHD and non-ADHD groups (Fig. 3) indicate the heterogeneity in the outcomes of PI children, which may be due to the differences in their respective institutional experiences. The persistence of neural abnormalities and behavioral problems in the PI youth with ADHD emphasizes the need for improving pre-adoption and post-adoption services to help families that adopt children internationally, as well as domestically. Researchers and practitioners need to improve interventions and information available to the families who adopt in order to reduce and prevent negative outcomes associated with adopted children (Gunnar et

al., 2000). The adversity PI children faced in the institutions and are facing in their current contexts must be addressed in future research and policy.

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