Early adversity and neural correlates of executive function: Implications for academic adjustment

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A B S T R A C T

Early adversity can negatively impact the development of cognitive functions, although little is known about whether such effects can be remediated later in life. The current study examined one facet of executive functioning – inhibitory control – among children who experienced institutional care and explored the impact of a foster care intervention within the context of the Bucharest Early Intervention Project (BEIP). Specifically, a go/nogo task was administered when children were eight years old and behavioral and event-related potential (ERP) measures were collected. Results revealed that children assigned to care as usual (i.e. institutional care) were less accurate and exhibited slower neural responses compared to children assigned to the foster care intervention and children who had never been institutionalized. However, children in both the care as usual and foster care groups exhibited diminished attention processing of nogo cues as assessed via P300 amplitude. Foster care children also showed differential reactivity between correct and error responses via the error-related negativity (ERN) as compared to children in the care as usual group. Combined, the results highlight perturbations in neural sources of behavioral and attention problems among children experiencing early adversity. Potential implications for academic adjustment in at risk children are discussed.

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1. Introduction

Executive functioning encompasses a range of cognitive factors essential for achieving goal directed behavior. Primary components of executive function include inhibitory control, retention and manipulation of information in working memory and the ability to set-shift between changing rules (Zelazo et al., 2003). These skills have been deemed core components of educational outcomes (Biederman et al., 2004; Morrison et al., 2010; Razza et al., 2010) as they contribute to sustained and flexible attention patterns essential to academic success (Merz and McCall, 2011). Among the range of executive function skills, inhibitory control has received significant focus in relation to children’s academic adjustment with links to specific subject mastery (Bull and Scerif, 2001; Clark et al., 2010), school enjoyment (Valiente et al., 2007) and readiness for school entry (Duncan et al., 2007; Howse et al., 2003; NICHD Early Child Care Research Network, 2003; Pears et al., 2010).
However, problems with executive function are prevalent among children experiencing early adversity, especially among children who have experienced institutionalized care (Bos et al., 2009; Bruce et al., 2009; Colvert et al., 2008; Merz and McCall, 2011; Pollak et al., 2010). Studies suggest that children who spend longer in institutionalized care perform worse on these tasks compared to children who are in institutions for a shorter period of time (Colvert et al., 2008; Pollak et al., 2010). Even after adoption, post-institutionalized children continue to struggle with attention problems (i.e. Fisher et al., 1997; Kreppner et al., 2001; Roy et al., 2000, 2004), have a greater risk of lagging behind non-adopted peers in school, are more likely to need academic learning services (Loman et al., 2009) and have lower rates of school achievement, particularly if they experienced longer periods of early deprivation (Beckett et al., 2007).

Work from the English and Romanian Adoptees (ERA) study suggests that particular patterns of inattention/overactivity (I/O; Kreppner et al., 2001) may be specific to post-institutionalized children as these symptoms emerge across a number of studies that span the age range from childhood into early adolescence (see Stevens et al., 2008 for a review). Similar regulatory control problems are evident in the BEIP sample at 54-months of age with early deprivation increasing the prevalence of externalizing disorders (e.g. ADHD) among children experiencing institutional care regardless of later placement into a high quality foster care intervention (Zeanah et al., 2009). Moreover, patterns of neural activation in infancy and toddlerhood have also been found to correspond to symptoms of impulsivity and hyperactivity during the preschool years (McLaughlin et al., 2010).

Combined, these data accentuate the far-reaching impact early caregiving has on the development of neural systems involved in executive function and associated outcomes. Research in animal models (Sanchez et al., 1998; Spinelli et al., 2009; Uchida et al., 2010), as well as preliminary work with human populations (Chugani et al., 2001; Eivuathingal et al., 2006; Pollak et al., 2010; Tarullo et al., 2011; Tottenham et al., 2011), demonstrate that deprived caregiving environments negatively influence the function of prefrontal circuitry, which is crucial for the development of executive functioning. Further exploration of the neural measures of regulatory control in young populations of at-risk children warrants the use of non-invasive measures such as event-related potentials (ERPs). One major benefit of this approach is the excellent temporal resolution of ERPs, which may reveal subtle differences in neural reactivity that can have substantial impact on functional outcomes. Of particular interest in this study are three ERP components linked to executive function – the N2, the P300 and the error-related negativity (ERN).

The frontal N2 is generally associated with focusing of attention and aspects of inhibitory control (Falkenstein et al., 1999; Jodo & Kayama, 1992; Jonkman et al., 2003; Luck and Hillyard, 1994) as well as conflict detection (Nieuwenhuis et al., 2003). In typically developing children, the latency of the N2 is faster to go versus nogo trials (Davis et al., 2003) and source localization studies identify prefrontal and anterior cingulate (ACC) generators (Bekker et al., 2004; Lamm et al., 2006; Nieuwenhuis et al., 2003). The P300 component is linked to stimuli salience with amplitude differences indexing inhibitory control or sustained attention (Burden et al., 2009; Davis et al., 2003; Donchin et al., 1986; Eimer, 1993; Mantini et al., 2009). Generators of the P300 have been localized to the temporal parietal junction and lateral prefrontal cortex (see Nieuwenhuis et al., 2005 for a review). The ERN is a response-locked component associated with response monitoring and evaluation of actions (Falkenstein et al., 1991; Gehring et al., 1993) and is theorized to signal the need for behavior adjustment (Coles et al., 2001; van Veen and Carter, 2002). This component has been localized to the ACC in the rostral cingulate zone (Hermann et al., 2004; Roger et al., 2010).

Overall, the precise behavioral and neural sources of executive function problems among children experiencing early adversity are incompletely understood. In the current study, the impact of early institutionalization on childhood executive function is examined as well as the potential amelioration of negative consequences via a foster care intervention. Participants were part of a sample in the Bucharest Early Intervention Project (BEIP). This project is the first randomized control trial of a foster care intervention for institutionalized children (see Zeanah et al., 2003, for details). Three groups of children are included in the study: institutionalized children who were either randomized to be taken out of the institution and placed into foster/family care (Foster Care Group; FCG) or randomized to remain in the institution (Care As Usual Group; CAUG), and typically developing children from the community (Never Institutionalized Group; NIG). A go/nogo task was administered when the children were eight years old and behavioral and event-related potential (ERP) measures were collected. Two sets of predictions were made. First, the CAUG would generally perform worse than the NIG. Second, if our foster care intervention was successful, performance among the FCG would (a) be superior to children in the CAUG and (b) comparable to performance among children in the NIG.

2. Methods

2.1. Participants

The study sample comprised an initial group of 136 children who had been abandoned or placed in institutions in Bucharest, Romania. These children were free of major genetic or neurological disease/disorders and did not show signs of fetal alcohol syndrome. Half of these children were randomly assigned to remain in institutional care (Care As Usual Group; CAUG) and the other half were placed in a high-quality foster care program (Foster Care Group; FCG) (see Smyke et al., 2010 for details of the foster care program). Typically developing children (n = 72), who had never spent time in an institution (Never Institutionalized Group; NIG), were also recruited from pediatric clinics in Bucharest (see Fox et al., 2011, Fig. 1 for a Consort Diagram providing information on the sample at age 8).

Children were initially assessed at enrollment into the study (baseline) and then followed up at 30-, 42- and
54-months. The current assessment was conducted at 8 years of age (Mean = 8.53 years, SD = .40). To confirm that children had the capacity to complete a go/nogo task participants who scored less than 70 on their full-scale IQ, as measured on the Wechsler Test of Intelligence, were excluded from analysis (15 CAUG, 10 FCG, 1 NIG). The final sample for analysis of behavioral data included 33 (13 female) CAUG children, 43 (21 female) FCG children and 41 (17 female) NIG children. Children were excluded from electrophysiological analysis due to excessive eye or body movement artifact. The final sample for analysis of ERP data included 29 (11 female) CAUG, 37 (16 female) FCG, and 29 (15 female) NIG children.

The study protocol was approved by the University Institutional Review Boards of the principal investigators (Fox, Nelson & Zeanah) and the University of Bucharest. As dictated by Romanian law, consent was given by the local Commission on Child Protection for each child participant who lived in their sector of Bucharest. Assent for the electrophysiology was also obtained from each caregiver who accompanied a child to the visit.

2.2. Go/nogo task

Behavioral responding and ERPs were recorded during a letters version of the go/nogo task (i.e. Casey et al., 1997; Davis et al., 2003). Children were instructed to press a button for every letter (go trials) except for the letter ‘X’ (nogo trials). The letter stimuli were presented for 500 ms and were followed by a 1100 ms blank screen with an average random inter-trial interval (ITI) of 200 ms (range: 100–300 ms). A total of 220 trials were split between two blocks. The first block consisted of 40 go trials to prime responding. The second block of 180 trials was composed of both go and nogo trials that were presented in a randomly mixed order at a 70:30 ratio (126 go trials and 54 nogo trials).

2.3. Procedures and experimental design

Children were fitted with an electrode cap (Electro-Cap International, Inc., Eaton, OH) while seated in front of a computer screen. Data were recorded from 13 sites (Fz, F3, F4, Cz, C3, C4, Pz, P3, P4, T7, T8, O1, O2) and the left and right mastoids in accordance with the International 10/20 system (Jasper, 1958). Channels were referenced to Cz, and Afz served as ground. Impedances were kept at or below 10 kΩ. Electro-oculogram (EOG) was recorded from two mini-electrodes, placed directly above and below the left eye. EEG and EOG signals were amplified by SA Instrumentation Company (San Diego, CA) by factors of 5000 and 2500, respectively. Filter settings were 1–100 Hz. Data were digitized online with customized acquisition software and sampled at a rate of 512 Hz using a 12-bit analog-to-digital converter (±2.5 V input range) and Snap-Master acquisition software (HEM Data Corporation, Southfield, MI).

A 30-Hz digital lowpass filter was applied using the ERP Analysis Systems software from James Long Company (Caroga Lake, NY). Epochs containing signals that exceeded ±200 mV were excluded from analysis, and eye movement artifact was regressed. Data were re-referenced to an average mastoids configuration, and individual averages were created using the 100 ms prior to stimulus onset and 200–100 ms prior to response for baseline correction. Participants with fewer than 10 usable trials per condition for the stimulus-locked ERPs, and fewer than 8 for the response-locked ERPs (see Olvet and Hajcak, 2009), were excluded from further analysis.

Components of interest were identified by inspection of the grand mean waveforms. Two stimulus-locked components, the fronto-central N2 component and the midline parietal P300 component, and one response-locked component, the error-related negativity (ERN), were examined. Only correct responses to go trials were included in the analysis for the N2 and P300. For the ERN, response-locked averages were computed for correct go trials and incorrect nogo trials. Peak amplitude (µV) and latency to peak amplitude (ms) were extracted for the N2 (250–500 ms) at frontal electrodes (F3, Fz, F4). Mean amplitude was examined for the ERN (−50 to 100 ms) at the midline frontal electrode (Fz) and for the P300 (400–700 ms) at the midline parietal electrode (Pz).

3. Results

3.1. Statistical procedures

Accuracy and reaction time measures of behavioral performance were analyzed with a series of repeated-measures analyses of variance (ANOVAs). Greenhouse–Geisser corrections were applied when necessary to control for violations of sphericity. Group (CAUG, FCG, NIG) was a between-subjects factor and for the stimulus-locked components (N2 and P300) condition (go versus nogo) served as a within-subjects factor. Three levels of electrode (F3, Fz, F4) were included for the N2 analysis. As the P300 is maximal at Pz, the mean amplitude was analyzed at this electrode only. For the response-locked ERN, a difference score was calculated comparing mean amplitude on correct versus incorrect trials for Fz. When significant (p ≤ .05) main or interaction effects emerged, post hoc comparisons were conducted and corrections for multiple comparisons were applied.
3.2. Behavioral data

A main effect of condition emerged for behavioral accuracy, $F(1,114)=246.67$, $p<.001$, such that all children responded more accurately to go ($M=93.9\%$, $SD=6.5$) compared to nogo trials ($M=75.2\%$, $SD=12.3$). This effect was qualified by a condition $\times$ group interaction, $F(2,114)=3.02$, $p<.05$, and follow-up tests reveal significant group differences for the go condition only, $F(2,114)=7.12$, $p<.001$. Specifically, the NIG ($M=95.8\%$, $SD=4.9$) and FCG ($M=94.7\%$, $SD=5.8$) showed the highest accuracy rates for go trials, and both groups performed significantly better than children in the CAUG ($M=90.5\%$, $SD=7.9$; $p<.05$). For reaction time, both main effects of condition, $F(1,114)=64.90$, $p<.001$, and group emerged, $F(2,114)=5.50$, $p<.01$, whereby all children responded faster for incorrect ($M=478$ ms, $SD=12.6$) as compared to correct trials ($M=552$ ms, $SD=7.9$) but children in the CAUG ($M=558$ ms, $SD=11.1$) responded the slowest overall regardless of condition, as compared to the FCG ($M=502$ ms; $SD=7.7$) and NIG children ($M=494$ ms, $SD=8.2$; $p<.05$) (Table 1).

3.3. Stimulus-locked ERP data

3.3.1. N2 findings

For peak amplitude, there was a main effect of electrode, $F(2,184)=19.81$, $p<.001$. A comparison of the electrode means indicated that data from Fz ($M=-6.74\mu V$, $SD=2.7$) showed significantly larger peak amplitudes than F3 ($M=-5.91\mu V$, $SD=2.7$) and F4 ($M=-5.94\mu V$, $SD=2.7$). Latency analyses revealed a main effect of condition, which was qualified by a condition $\times$ electrode $\times$ group interaction, $F(4,184)=3.61$, $p=.009$. Follow-up analyses reveal that latency to peak N2 was faster for go compared to nogo trials across all three frontal electrodes for children in the FCG, $F(1,36)=23.37$, $p<.001$ and NIG, $F(1,28)=4.48$, $p<.05$. Children in the CAUG, however, only showed discrimination between go and nogo trials at electrode F3, $F(2,56)=5.87$, $p=.01$ (see Fig. 1).

3.3.2. P300 findings

There were main effects of condition and group for mean amplitude of the P300, and these effects were qualified by a condition $\times$ group interaction, $F(2,92)=3.04$, $p<.05$. Post hoc comparisons revealed that all children showed equivalent P300 amplitudes on go trials. After correcting for multiple comparisons, analyses suggest that children in the NIG ($M=4.34\mu V$, $SD=2.0$) showed larger amplitudes to nogo trials compared to the FCG ($M=2.84\mu V$, $SD=2.7$, $p<.05$) and CAUG ($M=2.90\mu V$, $SD=2.6$, $p<.07$, see Fig. 2).

3.4. Response-locked ERP data

3.4.1. Patterns of ERN expression

Due to the relatively strong behavioral performance on this task, only a subset of children had a sufficient number of usable error trials to explore the ERN response (16 CAUG, 20 FCG, 23 NIG). The magnitude of the ERN difference score varied significantly between the groups, $F(2,56)=4.62$, $p=.014$, at Electrode Fz with the largest response in the FCG ($M=-11.52\mu V$, $SD=6.0$) followed by the NIG ($M=-9.53\mu V$, $SD=6.9$) and then the CAUG ($M=-5.19\mu V$, $SD=5.7$). Post hoc analyses revealed that the FCG had a significantly larger ERN compared to the CAUG ($p=.01$). The FCG and NIG did not differ from each other (see Fig. 3).

3.5. Timing of intervention effects and between measure associations

We examined possible associations between age at placement in foster care and behavioral and electrophysiological data. No significant correlations between age at
placement and accuracy or reaction time emerged in these analyses. There were also no associations between age at placement and physiological measures. However, significant correlations did emerge between the stimulus-locked ERPs and behavioral measures. For the CAUG a larger P300 response for correct go trials was linked to higher accuracy rates on go trials ($r = .57, p = .001$). A similar pattern emerged for the FCG with larger P300 amplitudes for go trials linked to greater go trial accuracy ($r = .32, p = .05$). Likewise, among both the FCG and NIG greater reaction times to go trials were associated with increased N2 latencies for go trials ($r = .38$ and $r = .49$, respectively, $p$'s < .05).

4. Discussion

Three intriguing patterns emerged from these data. First, children who experienced early institutionalization and then were randomized to the care as usual group (CAUG) were overall less accurate on the go/nogo task compared to children randomized to the foster care intervention (FCG) and the never institutionalized children (NIG). Second, the neural distribution of peak latency for the N2 differed across the hemispheres between go and nogo conditions at frontal sites for both the NIG and FCG, whereas the CAUG only distinguished trial conditions at the left frontal site. Third, magnitude differences in neural reactivity emerged between the groups on ERP components assessing attention allocation and response monitoring. Specifically, the P300 was largest among the NIG compared to the other two groups, and the CAUG displayed the smallest difference in reactivity between correct and error trials assessed via the ERN difference score. Collectively, these patterns suggest a complex constellation of neural processing underlying impaired performance on a go/nogo task for children experiencing early adversity.

This work also highlights areas of potential amelioration in certain attentional and monitoring aspects of executive functioning that may contribute to children’s ability to succeed in the academic setting. Specifically, a positive impact of the caregiving intervention on task performance emerged for accuracy patterns such that the foster care children performed as well as the never institutionalized children and marginally better than institutionalized children who did not receive the intervention. When exploring accuracy rates by condition, an unexpected pattern emerged such that all groups performed similarly for the nogo trials, however, the children in the CAUG made significantly more errors of omission on go trials. Contrary to our initial hypothesis that institutionalization would impair children’s ability to inhibit their actions, the data indicate that by eight years of age children who have experienced early deprivation can complete the nogo portion of a basic inhibitory control task. However, in this type of task, failure to respond to go trials results in a high level of inhibition accuracy that does not necessarily indicate mastery of the task. In fact, the pattern of heightened errors for go trials indicates that problems engaging regulatory control might result from impairments in either the ability to sustain or orient attention toward salient task stimuli. Alternatively, the pattern of increased errors of omission among the CAUG may reflect trouble determining or enacting the appropriate action in response to task cues. Any one of these issues could cause significant difficulty in a school setting where sustained and flexible attention abilities are essential (Merz and McCall, 2010).

To further explore the possibility that institutionalized children may have difficulty interpreting task cues we also examined the neural patterns associated with processing of task stimuli and action evaluation. Prior work suggests that differences in the distribution of neural processing can signal developmental delays or aberrations in cognitive processes. In the current study, group differences in the cortical distributions of processing speed for the go versus nogo stimuli emerged for the N2 component. Specifically, the FCG and NIG groups exhibited faster latencies for go trials across the three frontal sites of F3, Fz, ad F4 whereas the CAUG only distinguished between trial conditions at the frontal site F3. Given that processing speed differences may influence children’s ability to rapidly process and respond to instructions in the classroom setting, further investigation is warranted to determine whether and how hemispheric patterns in speed of processing could contribute to specific types of regulatory problems and learning difficulties.

According to Falkenstein et al. (1999), N2 latency measures of processing speed are positively linked to performance accuracy in adults. Although no connections between N2 latency and accuracy were observed in the current sample of children, positive associations did emerge between N2 latency and reaction times for go trials among both the FCG and NIG. The nature of these correlations may reflect more efficient attention allocation as the FCG and NIG had greater accuracy for go trials then the CAUG. Although additional study is needed to determine precisely how distribution and processing speed of neural activity combine to impact cognitive function, these findings are in line with the current literature on distribution and peak latency patterns of ERP components in children. Namely, altered ERP distribution and latency patterns have been linked to variations in cognitive processing within typically developing populations that differ in age (Molfese et al., 2008) or learning skills (Maatta et al., 2005), as well as among children with specific disorders such as dyslexia (Penolazzi et al., 2006) or autism (McPartland et al., 2004).
Despite group differences for the N2 component, the FCG did not differ from the CAUG for the P300. A general trend for increased P300 amplitude to nogo trials emerged for all children, supporting the notion that this component reflects neural mechanisms involved with the inhibitory control aspect of executive function, however, the magnitude of this response was greatest among the NIC as compared to both the FCG and CAUG. Similar patterns of diminished P300 responding for nogo trials have been interpreted as a developmental lag in younger children as compared to older children and adults (van der Stelt et al., 1998; Johnstone et al., 2001), and in children with ADHD as compared to controls (van Leeuwen et al., 1998; Doehnert et al., 2010). It is possible that a longer period of intervention is required to elicit changes in this component among children who have experienced institutionalized care. Nonetheless, the current findings do indicate a strong role for early experience in shaping the development of neural systems that can contribute to inhibitory control via attention regulation processes. Hopefully, additional longitudinal work will help ascertain the potential for plasticity in this component with altered rearing experiences or cognitive training programs.

In contrast to the P300 results, the foster care intervention did have a significant positive impact on children's reactivity to their behavioral responses on the inhibitory control task, such that the FCG displayed a larger ERN difference score between correct and error trials compared to children in the CAUG and a comparable response to the NIC. These patterns illuminate a strong disparity between the FCG and CAUG in the monitoring of one's own actions. Developmental theories highlight the importance of monitoring in the transition from automatic to voluntary control of behavior (Bandura, 1986; Karoly, 1993) and in establishing a balance between an individual's own performance goals and external demands on their behaviors (Kopp, 1982). Thus, the diminished neural reactivity to behavioral responding in the CAUG may index future risk for externalizing problems and academic trouble due to lack of flexibility in behavioral adjustment (Hall et al., 2007; Olvet and Hajcak, 2008). In fact, recent work by Hirsh and Inzlicht (2010) found a link between ERN response and educational success in a study of college age adults, such that students with smaller ERN responses had worse academic performance. Moreover, patterns of impoverished ERN responses have also been noted in children with ADHD (Groen et al., 2008; van Meel et al., 2007). Further work is needed to ascertain whether increases in monitoring skills can compensate for other impairments in executive function.

4.1. Implications for educational outcomes

The current data highlight both behavioral and neural variations in processes supporting executive functioning among post-institutionalized children. Differences emerged for children experiencing institutionalization as well as children who experienced a subsequent foster care intervention. Although this study tested children in the context of a well-controlled laboratory experiment, it is feasible that the added complexity of a school classroom may serve as a more challenging environment. Given that children who have difficulty regulating themselves in kindergarten are less likely to complete high school (Vitaro et al., 2005), delineating specific sources of executive function problems at an early age has significant long-term educational implications.

Due to the extremely poor academic trajectories of children with impaired executive function, and inhibitory control in particular, a greater understanding of the causes and composition of such impairments may be highly beneficial to the design of more effective intervention programs. Early caregiving conditions may be one such major contributor to the development of executive function as the parent–child relationship is among the first contexts in which a child learns to self-regulate. At the moment, the precise paths through which early caregiving shapes the neural systems underlying executive function have not yet been determined. Although the neglect experienced in the context of institutional rearing represents a very specific context, this study does underscore that high quality caregiving can ameliorate specific deficits in neural reactivity linked to executive functioning.

Overall, these results emphasize that a deprived caregiving environment may have a substantial impact on children's mastery of executive function skills that are linked to academic adjustment. The United Nations Children's Fund estimates that 8 million children currently live in institutionalized care worldwide (Committee on the Right of the Child, 2004) thus deprived early caregiving places an enormous amount of children at risk for educational problems. Data from training programs conducted with typically developing children (e.g. Diamond et al., 2007; Rueda et al., 2005) indicate that interventions aimed at enhancing inhibitory control skills could train brain circuitry to more efficiently learn complex constructs and engage in higher order problem solving skills that are required ever more frequently as a child progresses through school. Therefore a dual approach design that simultaneously focuses on improving caregiving conditions and children's executive function skills prior to the start of formal schooling might have the most profound (Pears et al., 2010), and potentially long-lasting effects on scholastic achievement among at risk children.

5. Conclusions

In sum, the current study revealed that early deprivation impairs specific behavioral and neural aspects of executive functioning. Amelioration of these effects via a high quality foster care intervention varied across both behavioral and neural measures. These patterns suggest a complex interplay between early caregiving environments and the developmental plasticity of neural systems underlying regulatory control. Perturbations in these systems are likely to have serious consequences for learning outcomes among children experiencing early adversity, therefore these data call attention to the relevance of intervention efforts aimed at enhancing the development of executive function skills of children at risk. Given the influence of these skills on early school adjustment, future work should aim to further unpack the neural and behavioral consequences of
deprived caregiving environments on children's academic and psychosocial outcomes.

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