Deficits in error monitoring are associated with externalizing but not internalizing behaviors among children with a history of institutionalization

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Background: Children raised in institutions are at increased risk of developing internalizing and externalizing problems. However, not all children raised in institutions develop psychopathology. Deficits in error monitoring may be an important moderating role between a history of institutional rearing and subsequent psychopathology. Methods: We investigated the impact of psychosocial deprivation on behavioral and neural responses (event-related potentials: ERPs) to a Flanker task assessing error monitoring and the relations between these measures and psychopathology for 12-year-old children in the Bucharest Early Intervention Project (BEIP). The BEIP involves two groups of institutionalized children randomly assigned in infancy to receive either a foster care intervention (FCG) or care as usual (CAUG). Results: Children who experienced institutional care, particularly those in the CAUG, showed perturbed behavioral performance and ERPs on the Flanker task. Additionally, an ERP measure of error monitoring [error-related negativity (ERN)] moderated the relations between time spent in institutions and externalizing and ADHD behaviors. When the amplitude of the ERN was smaller, time spent in institutional care was positively related to ADHD and externalizing behaviors, whereas time spent in institutions was unrelated to externalizing problems when children evidenced a larger ERN. Neural correlates of error monitoring did not moderate the relations between time spent in institutionalized care and internalizing behaviors. Conclusions: Exposure to institutional care early in life may affect brain circuitry associated with error monitoring. Perturbations in this neural circuitry in combination with psychosocial deprivation are possibly a risk pathway associated with the development of externalizing and ADHD problems. Keywords: Institutions; externalizing disorder; internalizing disorder; cognition; event-related potentials.

Introduction

Children raised in institutions experience severe psychosocial deprivation and display a myriad of negative developmental outcomes in cognition and psychopathology (Nelson, Fox, & Zeanah, 2014). Children who experience institutional care in early childhood are at increased risk of developing both internalizing and externalizing disorders later in life (Colvert et al., 2008; Humphreys et al., 2015; Tizard & Rees, 1975). Nevertheless, only some children with a history of institutional rearing subsequently develop psychopathology. Identifying factors that differentiate between those children who do and do not develop disorders are of great interest.

A good deal of evidence has linked early deprivation to deficits in executive skills throughout childhood (Colvert et al., 2008; Loman et al., 2013; Merz & McCall, 2011). One cognitive domain that is of particular interest is error monitoring because deficits have been reported among multiple samples of previously institutionalized children (Loman et al., 2013; McDermott et al., 2013). Furthermore, error monitoring has a well-defined neural correlate known as the error-related negativity (ERN; Falkenstein, Hohnsbein, Hoorn, & Blanke, 1991), which is an event-related potential (ERP) believed to be generated in the anterior cingulate cortex (Dehaene, Posner, & Tucker, 1994; Van Veen & Carter, 2002). The ERN is a negative deflection maximal at frontalcentral sites and peaks within the first 100 ms following an error response. The ERN is thought to index error and conflict detection associated with incorrect response selection (Hajcak, 2012). Data suggest that the ability to monitor errors and the ERN component emerge in childhood (Grammer, Carrasco, Gehring, & Morrison, 2014) and develop throughout adolescence, with the ERN becoming more negative and stable in late adolescence and early adulthood (Davies, Segalowitz, & Gavin, 2004).

To our knowledge, only one study from another sample (Loman et al., 2013) and two studies from the present sample (McDermott, Westerlund, Zeanah, Nelson, & Fox, 2012; McDermott et al., 2013) have examined error monitoring and the ERN in...
previously institutionalized children. All three studies examined the ERN in late childhood (ages 8–11) and results are inconsistent. One study showed evidence that children who experienced institutional care exhibited smaller (less negative) ERNs (McDermott et al., 2012), while the other two studies found no or mixed evidence for reduced ERN amplitudes in previously institutionalized children (Loman et al., 2013; McDermott et al., 2013). One reason for these mixed findings may be that the ERN goes through rapid changes, possibly associated with puberty, in late childhood, and adolescence (Davies et al., 2004), which results in individual variability eclipsing group differences. Another reason for inconsistent findings may be that the duration of institutional care experienced by a child may influence ERN development, with longer amounts of time in institutional care associated with larger deficits in error monitoring. Indeed, other cognitive skills have been shown to be negatively impacted by prolonged exposure to institutional care (Troller-Renfree & Fox, 2016; Troller-Renfree, McDermott, Nelson, Zeanah, & Fox, 2015; Troller-Renfree et al., 2016). In addition to its association with early adversity, the ERN has been associated with both internalizing and externalizing disorders (Olvet & Hajcak, 2008). Within the internalizing domain, heightened amplitude of the ERN is associated with greater symptoms of anxiety. The ERN also has been related to externalizing behaviors, such as aggression (Dikman & Allen, 2000; Nelson, Patrick, &Bernat, 2011) and ADHD behaviors (Shiels & Hawk, 2010), especially, impulsivity (Martin & Potts, 2009; Nelson et al., 2011). Reduced amplitude of the ERN is associated with more ADHD and externalizing behaviors. To our knowledge, only one study using the present sample examined whether error monitoring moderates associations between deprived caregiving experience and the expression of socioemotional behavior problems in childhood. In this study, McDermott et al. (2013) demonstrated that among a subset of children – those who had been removed from institutional care and placed into foster care – a larger ERN predicted lower levels of socioemotional behavior problems. While these findings suggest that there may be a relation between the ERN, institutionalized care, and psychopathology, this link has yet to be established in a sample of previously institutionalized children who did not receive intervention. One reason for the lack of previous findings may be that the association between the ERN and psychopathology does not appear until later adolescence (Meyer, Weinberg, Klein, & Hajcak, 2012). Additionally, given that the prevalence of many internalizing and externalizing disorders increase throughout adolescence (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Merikangas, Nakamura, & Kessler, 2009), there may not have been enough variability in internalizing and externalizing problems to detect associations. Another reason for the heterogeneity of past findings may be that prior studies have not examined whether the duration of institutional care effects the development of the ERN and whether reduced ERN amplitudes compound with institutional care to predict the emergence of psychopathology. The identification of the ERN as a possible factor for the development of psychopathology in previously institutionalized children is thus of great interest given that it may identify one of the pathways by which psychopathology emerges as well as suggest a system to target for evidence-based interventions aimed at improving mental health.

This study examined the development of error monitoring in a sample of children who experienced early institutionalization and were enrolled in the Bucharest Early Intervention Project (BEIP) – a randomized controlled trial of foster care as an alternative to institutional care (Nelson et al., 2014; Zeanah et al., 2003). Children in the study were randomized to one of two conditions: removed from an institution and placed into foster care (Foster Care Group; FCG) or remained in institutional care (Care as Usual Group; CAUG). Following randomization, children placed into the FCG received high-quality care that was supported by project staff through age 54 months. While many FCG children remained in their assigned foster care placement, others left their foster care home for variety of reasons including reunion with their biological families. Children randomized to the CAUG received care provided by the local governmental authorities, including continued institutional care for some, but others were reunited with their biological family, placed into government-run foster care (not associated with the BEIP foster care), or adopted domestically (see Figure 1 for CONSORT diagram). In addition, a typically developing sample of children (Never-Institutionalized Group; NIG) was recruited from the community. The primary reason for recruiting children from the Bucharest community was to ensure that measures collected at each age point provided normative data. Behavioral responses, reaction times, and continuous electroencephalography (EEG) were collected during a modified Flanker task when children were 12 years of age.

Consistent with past studies examining the effect of institutionalized care on error monitoring, we predicted that previously institutionalized children would show perturbed behavioral performance and lower ERN amplitude (a neural correlate of error monitoring). Additionally, consistent with findings at 8 years of age (McDermott et al., 2013), we hypothesized that the FCG would show better performance and enhanced neural correlates related to error monitoring. Building on past findings, we also predicted that prolonged institutional care would lead to reduced ERN. Finally, given that past studies using previously institutionalized

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samples have suggested that neural correlates of error monitoring may moderate the relations between early adversity and externalizing behaviors, we predicted that amplitude of the ERN would moderate the relation between prolonged institutionalized care and the presentation of externalizing disorders at age 12. The specificity of this moderating effect across externalizing subscales was not hypothesized, given that no study has previously investigated this question.

Methods

Participants

The sample comprised 136 children, abandoned in infancy ($M = 2.57$ months) and placed into institutional care in Bucharest, Romania. Hallmarks of institutional care include social deprivation (high caregiver-child ratios) and noncontingent caregiving (for more information, see Nelson et al., 2014 and Zeannah et al., 2003). Following assessment, children were randomized into two groups – Care as Usual Group (CAUG) and Foster Care Group (FCG). A group of never-institutionalized community children (NIG) were also assessed. At age 12, 50 CAUG (23 girls), 50 FCG (24 girls), and 48 NIG (26 girls) completed the Flanker task and 38 CAUG, 36 FCG, and 47 NIG provided usable behavioral data. The mean age of assessment was 11.39 for CAUG, 11.92 for FCG, and 12.14 for NIG.

The University Institutional Review Boards of the principal investigators (Fox, Nelson & Zeannah) and of the University of Bucharest, Romania, approved the study protocol. The appropriate legal guardian provided consent for participation of previously and currently institutionalized children.

Flanker task

At age 12, participants completed an adapted version of the Flanker task (Eriksen & Eriksen, 1974). Stimuli were presented electronically using E-Prime 2.0 software (Psychology Software Tools, Pittsburgh, PA) and consisted of five angle brackets presented horizontally. Half of the trials were congruent with all of the arrows facing the same direction (<<<<<< or >>>>>>) and half of the trials were incongruent with the middle arrow facing the opposite direction of the four flanking arrows (<<<<<< or >>>>>>). Participants completed 20 practice trials followed by 160 test trials. Consistent with past tasks used with the present institutional sample (McDermott et al., 2013), the task dynamically adjusted the stimulus presentation time based on each participant’s performance. If a participant had a cumulative accuracy greater than 60%, the subsequent trial presentation time was decreased by 50 ms. Conversely, if cumulative accuracy was less than 60%, the subsequent trial presentation time was increased by 50 ms. Presentation times were between 100 and 1,450 ms with a mean presentation time of 553.34 ms ($SD = 414.46$). Furthermore, to prevent against expectancy effects, stimulus presentation times were jittered by one of three values: $-60$ ms, $-10$ ms, and $+40$ ms. The intertrial interval was randomly assigned to be one of four durations: 773 ms, 790 ms, 807 ms, or...
assess children’s mental health and well-being. The version
HBQ provides dimensional scales to enable researchers to
2002), the HBQ does not yield clinical diagnoses. Rather, the
established clinically meaningful cut-points for some of the
hbq.wordpress.com/). Although several publications have
and Development (for more information, see https://macarthur
MacArthur Foundation Research Network on Psychopathology
The Health and Behavior Questionnaire (HBQ) was developed by

ERP processing
Continuous EEG was recorded using a 64-channel Geodesic
Sensor Net (Electrical Geodesic, Inc., Eugene, OR). Prior to
data collection, electrode impedances were reduced to below
50 kΩ. Data were sampled at 250 Hz throughout collection. All
electrodes were referenced online to electrode Cz and re-
ferenced offline to an average reference. The data were
filtered using a digital band-pass FIR filter from 0.3 to 30 Hz.
Response-locked trials were segmented separately for error
and correct trials. Trials began 400 ms prior to response and
extended to 800 ms after the response (1,200 ms total). Eye
blinks were removed using independent component analysis
(ICA) performed by the ERP PCAToolkit (Delorme & Makeig,
2004; Dien, 2010). Individual blinks were identified for each
participant to create an average blink topography. The ICA
components that correlated at .9 or above with the averaged
blink topography and/or with the ERP PCAToolkit-supplied
blink topography were removed. Next, an automated procedure
was used for artifact rejection. Channels were marked bad if
the fast average amplitude exceeded 130 μV or if the difference
between a channel and the neighboring channels was greater
than 30 μV for an individual segment. Channels were marked
globally bad if the correlation between neighboring channels
was less than .40 or if the channel was bad on greater than
20% of trials. Trials were marked bad if more than 10% of the
channels were determined to be bad. Bad channels on the
remaining good trials were replaced using spherical spline
interpolation (Perrin, Pernier, Bertrand, & Echallier, 1989).
Correct and error trials were separately averaged for each participant and were baseline corrected to the average activity
from 400 ms to 200 ms before the response. The ERN and
correct response negativity (CRN) were evaluated as the
average activity at the frontocentral electrode 4 (FCz) where
the ERN was maximally negative. The ERN and CRN ampli-
tudes were extracted as the mean activity of ~50 to 150 ms.
Participants were included in ERP analyses if they had at least
10 artifact-free error trials in each condition. To examine brain
activity specific to errors, a difference wave was created by
subtracting the brain activity on correct trials from the brain
activity on error trials for the ERN (i.e. ERN – CRN; AERN).
Trials with reaction times faster than 200 ms and slower
than 1,200 ms were removed from the analysis. On average, less than 5% of trials were removed due to extreme reaction
times (M = 7.26 trials, SD = 8.26). The numbers of trials
excluded due to extreme reaction times were not different
between groups, F(2, 118) = 0.622, p = .539. Accuracy was
considered as the number of correct trials divided by the total
number of trials without an extreme response. Reaction times
were averaged separately for correct trials and error trials for
each participant in each condition and did not include nonre-
sponse trials.

MacArthur Health and Behavior Questionnaire
The Health and Behavior Questionnaire (HBQ) was developed by
members and affiliates of the John D. and Catherine T.
MacArthur Foundation Research Network on Psychopathology
and Development (more information, see https://macarthur
hbq.wordpress.com/). Although several publications have
established clinically meaningful cut-points for some of the
mental health scales (Lemery-Chalfant et al., 2007; Luby et al.,
2002), the HBQ does not yield clinical diagnoses. Rather, the
HBQ provides dimensional scales to enable researchers to
assess children’s mental health and well-being. The version
administered to each participant’s primary teacher at age 12
comprises 105 items. For the present paper, two composites and
their subscales from the HBQ were examined: internalizing
behaviors and externalizing/ADHD behaviors. The internalizing
composite comprises items related to depression and overanx-
ious behaviors. The externalizing and ADHD composite com-
prises measures of oppositional defiance, conduct problems,
overhostility, relational aggression, inattention, and impulsiv-
ity. The HBQ was completed by each participant’s primary
teacher.

Analytic plan
Behavioral differences in performance accuracy were exam-
ined using repeated-measures ANOVAs using IBM SPSS,
Version 22.0 (IBM Corp, Armonk, NY). Participant group
(CAUG, FCG, and NIG) served as a between-subjects factor
and Flanker trial type (congruent, incongruent) served as a
within-subjects factor. Next, differences in reaction time were
examined using repeated-measures ANOVAs. Participant
group (CAUG, FCG, and NIG) served as a between-subjects
factor and accuracy (correct and incorrect) served as a
within-subjects factor. For analyses examining group differences in
the AERN, a one-way ANOVA was conducted with group
(CAUG, FCG, and NIG) as the between-subjects factor. Addi-
tionally, a linear regression was conducted to examine whether
percentage of time spent in institutional care predicts AERN
amplitude. Greenhouse-Geisser corrections were applied as
necessary.
For analyses examining the moderating role of the AERN,
two separate path models were conducted in M-PLUS using
FIML estimation for missing data (Muthén & Muthén, 1998–
2010). First, separate moderation analyses were conducted with
Internalizing and Externalizing/ADHD behaviors com-
poses as outcomes. Exploratory follow-up moderation anal-
yses were conducted to examine the moderating role of
individual subscales that compose the externalizing/ADHD
composite.

Results

Behavioral analyses

Accuracy. Table 1 displays the accuracy and reac-
tion times across group. Intent-to-treat results revealed a
main effect of trial type with higher accuracy on congruent
(M = 79.85, SD = 9.69) compared with incongruent trials
(M = 51.28, SD = 14.36), F(1, 72) = 236.54, p < .001, η² = .767.
The main effect of group (F(1, 72) = 2.776, p = .100,
η² = .037) and Group × Trial type interaction
(F(1, 72) = 1.824, p = .181, η² = .025) failed to reach
significance. However, when compared with the
community sample, an additional main effect of
group emerged with the NIG (M = 74.6, SD = 11.32)
outperforming the CAUG (M = 63.8, SD = 7.49) and
FCG (M = 67.4, SD = 10.64), F(2, 118) = 12.658, p < .001, η² = .177.

Reaction time. Intent-to-treat results revealed a
main effect of trial type with quicker responses on
incorrect trials (M = 423.49, SD = 112.52) com-
pared with correct trials (M = 484.25, SD = 93.91),
F(1, 72) = 115.45, p < .001, η² = .616 and a main
effect of group with the FCG (M = 424.71, SD = 91.08) showing significantly faster respon-
ses than the CAUG (M = 481.509, SD = 91.08),
F(1, 72) = 6.35, p = .014, η² = .081. These main effects were qualified by a Group × Accuracy interaction, F(1, 72) = 7.280, p = .009, η² = .092. Post hoc follow-ups revealed that both groups showed faster reaction times on error trials when compared with correct trials. Furthermore, groups did not differ in reaction times on incorrect trials (CAUG: M = 458.60, SD = 128.72; FCG: M = 462.98, SD = 79.87). However, on correct trials, the CAUG was significantly slower to respond than the FCG (CAUG: M = 504.42, SD = 102.5; FCG: M = 462.98, SD = 79.87). Follow-up analyses comparing ever-institutionalized children to community controls were almost identical to the intent-to-treat analyses, with a main effect of trial type (error trials faster than correct trials; Table 1 Reaction Time, accuracy, and demographics (standard deviations in parentheses)

<table>
<thead>
<tr>
<th>Subjects with behavioral data (N)</th>
<th>CAUG</th>
<th>FCG</th>
<th>NIG</th>
<th>Group Differences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall accuracy</td>
<td>63.90% (7.49)</td>
<td>67.37% (10.64)</td>
<td>74.67% (11.32)</td>
<td>-</td>
</tr>
<tr>
<td>Congruent accuracy</td>
<td>79.34% (9.18)</td>
<td>80.39% (10.30)</td>
<td>87.26% (8.97)</td>
<td>-</td>
</tr>
<tr>
<td>Incongruent accuracy</td>
<td>48.34% (14.17)</td>
<td>54.39% (14.08)</td>
<td>61.90% (16.14)</td>
<td>-</td>
</tr>
<tr>
<td>Error reaction time</td>
<td>458.60 (128.72)</td>
<td>386.43 (78.36)</td>
<td>386.75 (81.81)</td>
<td>-</td>
</tr>
<tr>
<td>Correct reaction time</td>
<td>504.42 (102.50)</td>
<td>462.98 (79.87)</td>
<td>459.28 (85.20)</td>
<td>-</td>
</tr>
<tr>
<td>Age of entry into institution (months)</td>
<td>2.48 (3.76)</td>
<td>2.52 (3.46)</td>
<td>-</td>
<td>Not significant</td>
</tr>
<tr>
<td>% Time in institution</td>
<td>44.96 (27.25)</td>
<td>15.76 (10.33)</td>
<td>-</td>
<td>CAUG&gt;FCG</td>
</tr>
<tr>
<td>WISC IQ</td>
<td>67.57 (13.20)</td>
<td>78.75 (14.90)</td>
<td>99.70 (13.97)</td>
<td>NIG&gt;FCG&gt;CAUG</td>
</tr>
<tr>
<td>Gender (% female)</td>
<td>61%</td>
<td>53%</td>
<td>47%</td>
<td>Not significant</td>
</tr>
<tr>
<td>Internalizing (HBQ)</td>
<td>.50 (.34)</td>
<td>.41 (.32)</td>
<td>.24 (.27)</td>
<td>NIG&gt;CAUG</td>
</tr>
<tr>
<td>Externalizing/ADHD (HBQ)</td>
<td>.48 (.46)</td>
<td>.46 (.47)</td>
<td>.11 (.20)</td>
<td>NIG&gt;CAUG &amp; FCG</td>
</tr>
<tr>
<td>Oppositional defiant (HBQ)</td>
<td>.70 (.57)</td>
<td>.65 (.65)</td>
<td>.15 (.27)</td>
<td>NIG&gt;CAUG &amp; FCG</td>
</tr>
<tr>
<td>Conduct problems (HBQ)</td>
<td>.28 (.34)</td>
<td>.24 (.33)</td>
<td>.03 (.07)</td>
<td>NIG&gt;CAUG &amp; FCG</td>
</tr>
<tr>
<td>Overt aggression (HBQ)</td>
<td>.57 (.63)</td>
<td>.53 (.57)</td>
<td>.12 (.26)</td>
<td>NIG&gt;CAUG &amp; FCG</td>
</tr>
<tr>
<td>Relational aggression (HBQ)</td>
<td>.37 (.46)</td>
<td>.41 (.44)</td>
<td>.13 (.24)</td>
<td>NIG&lt;FCG</td>
</tr>
<tr>
<td>Inattention (HBQ)</td>
<td>.90 (.57)</td>
<td>.77 (.65)</td>
<td>.25 (.34)</td>
<td>NIG&lt;CAUG &amp; FCG</td>
</tr>
<tr>
<td>Impulsivity (HBQ)</td>
<td>.72 (.58)</td>
<td>.67 (.60)</td>
<td>.28 (.29)</td>
<td>NIG&lt;CAUG &amp; FCG</td>
</tr>
</tbody>
</table>

aMean of depression and overanxious.

bMean of oppositional defiant, conduct problems, relational aggression, overt hostility, inattention, and impulsivity.

Figure 2 (A) Response-locked event-related potential waveforms for ΔERN (ERN-CRN) at electrode FCz (where activity was maximal). (B) Scalp topographies of the ΔERN for the CAUG (left), FCG (middle), and NIG (right) at 76 ms after response.
\( F(1, 118) = 239.87, p < .001, \eta^2 = .670 \), a main effect of group (CAUG slower than the FCG and NIG; \( F(2, 118) = 5.245, p = .007, \eta^2 = .082 \), and a significant Group \times Accuracy interaction, \( F(2, 118) = 5.100, p = .008, \eta^2 = .080 \). Post hoc follow-ups revealed that all groups showed faster reaction times on error trials when compared with correct trials, and all three groups did not differ in reaction times on incorrect trials. However, on correct trials, the CAUG was significantly slower to respond than the FCG and NIG (CAUG: \( M = 504.42, SD = 102.5 \); FCG: \( M = 462.98, SD = 79.87 \); NIG: \( M = 459.28, SD = 85.20 \)).

**ERP analyses**

Figure 2 shows the ERP waveform for each group. Intent-to-treat analyses examining differences in the ANRN revealed a marginal main effect of group, \( F(1, 68) = 3.57, p = .063 \), with the CAUG (\( M = -.56, SD = 2.40 \)) exhibiting a marginally smaller ANRN than the FCG (\( M = -1.67, SD = 2.50 \)). Given this pattern of results, a linear regression was conducted to see if amount of time spent in institutional care predicted ANRN amplitude. Results showed that amount of time spent in institutional care significantly predicted ANRN amplitude, with smaller (less negative) amplitudes among children who experienced prolonged institutional care, \( \beta = .272, t(68) = 2.332, p = .023 \).

Follow-up analyses of ever-institutionalized children and community controls examining ANRN differences revealed a main effect of group, \( F(2, 111) = 3.69, p = .028 \). Follow-up independent-samples \( t \)-tests revealed that the CAUG (\( M = -.56, SD = 2.40 \)) showed a significantly smaller ANRN than the NIG (\( M = -.21, SD = 3.167 \); \( t(79) = 2.60, p = .011 \)), but no significant difference between the FCG and NIG, \( t(75) = .811, p = .420 \).

**Mental health**

All moderation models are presented in Table 2. To determine whether the ANRN exhibits moderating effects on the relations between amount of time spent in institutional care and internalizing and externalizing/ADHD symptomology, two separate moderation analyses were conducted. Results revealed that the ANRN did not moderate the relation between percent of life spent in institutional care and internalizing behaviors (\( \beta = .002, p = .155 \)); however, the ANRN did moderate the relationship between percent of time spent in institutional care and externalizing/ADHD behaviors (\( \beta = .003, p = .018 \)). Follow-up analyses revealed when the ANRN was smaller, time spent in institutional care was positively related to externalizing problems (\( \beta = .010, p = .021 \)), whereas time spent in institutionalized care was unrelated to externalizing problems when children evidenced a larger ANRN (\( \beta = -.007, p = .110 \)).

<table>
<thead>
<tr>
<th>Table 2 Moderation analyses</th>
<th>B</th>
<th>SE B</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internalizing(^b)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Institutional care</td>
<td>.002</td>
<td>.002</td>
<td>.162</td>
</tr>
<tr>
<td>ΔERN</td>
<td>-.030</td>
<td>.031</td>
<td></td>
</tr>
<tr>
<td>% Inst*ΔERN</td>
<td>.002</td>
<td>.001</td>
<td></td>
</tr>
<tr>
<td>Externalizing/ADHD(^a)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Institutional care</td>
<td>.001</td>
<td>.002</td>
<td>.349</td>
</tr>
<tr>
<td>ΔERN</td>
<td>-.073</td>
<td>.043</td>
<td></td>
</tr>
<tr>
<td>% Inst*ΔERN</td>
<td>.003*</td>
<td>.001</td>
<td></td>
</tr>
<tr>
<td>Oppositional defiance</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Institutional care</td>
<td>.003</td>
<td>.003</td>
<td>.383</td>
</tr>
<tr>
<td>ΔERN</td>
<td>-.102</td>
<td>.053</td>
<td></td>
</tr>
<tr>
<td>% Inst*ΔERN</td>
<td>.005*</td>
<td>.002</td>
<td></td>
</tr>
<tr>
<td>Conduct problems</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Institutional care</td>
<td>.000</td>
<td>.002</td>
<td>.386</td>
</tr>
<tr>
<td>ΔERN</td>
<td>-.054</td>
<td>.030</td>
<td></td>
</tr>
<tr>
<td>% Inst*ΔERN</td>
<td>.003*</td>
<td>.001</td>
<td></td>
</tr>
<tr>
<td>Overt hostility</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Institutional care</td>
<td>.001</td>
<td>.003</td>
<td>.328</td>
</tr>
<tr>
<td>ΔERN</td>
<td>-.106</td>
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</tr>
<tr>
<td>% Inst*ΔERN</td>
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<td>.002</td>
<td></td>
</tr>
<tr>
<td>Relational aggression</td>
<td></td>
<td></td>
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<tr>
<td>% Institutional care</td>
<td>.000</td>
<td>.002</td>
<td>.566</td>
</tr>
<tr>
<td>ΔERN</td>
<td>-.103**</td>
<td>.039</td>
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<tr>
<td>% Inst*ΔERN</td>
<td>.005**</td>
<td>.001</td>
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<tr>
<td>Inattention</td>
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<tr>
<td>% Institutional care</td>
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<td>.003</td>
<td>.037</td>
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<tr>
<td>ΔERN</td>
<td>-.004</td>
<td>.057</td>
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<tr>
<td>% Inst*ΔERN</td>
<td>.001</td>
<td>.002</td>
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<tr>
<td>Impulsivity</td>
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<td>.003</td>
<td>.257</td>
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<tr>
<td>ΔERN</td>
<td>-.068</td>
<td>.052</td>
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<tr>
<td>% Inst*ΔERN</td>
<td>.003*</td>
<td>.002</td>
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\(^aMean of oppositional defiant, conduct problems, relational aggression, overt hostility, inattention, and impulsivity.\)

\(^bMean of depression and overanxious.\)

To further investigate the moderating effects of the ANRN on the relation between amount of time spent in institutional care and externalizing problems, separate moderation analyses were conducted for all subscales of the externalizing/ADHD composite. Results revealed a significant moderation effect for the oppositional defiant subscale (\( \beta = .005, p = .012 \)), conduct problems subscale (\( \beta = .003, p = .010 \), overt hostility subscale (\( \beta = .004, p = .038 \), relational aggression subscale (\( \beta = .005, p < .001 \)), and marginally with the impulsivity subscale (\( \beta = .003, p = .053 \)); however, the inattention subscale (\( \beta = .001, p = .499 \)) did not appear to be moderated by the ANRN. All moderation analyses showed a pattern similar to the externalizing/ADHD composite, with a positive link between time in institutional care and externalizing subscale when the ANRN is smaller.

**Discussion**

We observed that 12-year-old children who had experienced institutionalized care showed decreased accuracy on a modified Flanker task when compared to their never-institutionalized peers. Furthermore,
only children who were randomized to remain in care as usual showed deficits in processing speed. This effect was specific to trials with correct responses and converges with recent work suggesting that early psychosocial deprivation alters white matter structure, which is thought to underlie processing speed (Hanson et al., 2013; Sheridan, Fox, Zeanah, McLaughlin, & Nelson, 2012). Additionally, we found that prolonged institutional care predicted reductions in the neural correlates associated with error monitoring. To our knowledge, this study is the first to link continued exposure to institutional care to decreases in ERN magnitude and to identify the ERN as a moderator of the relation between amount of time spent in institutional care and the development of externalizing behaviors. Interestingly, this moderation effect appears to be driven by multiple behavior domains, including oppositional and defiant behaviors, conduct problems, overt hostility, relational aggression, and impulsivity. Together, these results suggest that perturbed error monitoring may represent a risk for externalizing problems in previously institutionalized children.

The relations between the neural correlates of error monitoring and externalizing behaviors are well established. This study, like a previous one from the same sample using data when children were 8 years of age, establishes the neural correlates of error monitoring as a potential risk pathway for the development of externalizing behaviors in a previously institutionalized sample (McDermott et al., 2013). Extending previous findings, this study shows that the moderating effect of error monitoring is present across a wide range of externalizing and ADHD behaviors, though not with inattention. This relation is most likely driven by the high intercorrelation of externalizing behaviors in this sample (rs between .5 and .8). It is of particular clinical interest given the high prevalence of ADHD and externalizing disorders in children placed into institutional care (see Humphreys et al., 2015 for more information on clinical diagnoses).

Consistent with previous findings from this sample, the neural correlates of error monitoring did not moderate the risk for internalizing behaviors (McDermott et al., 2013), despite a strong relation between the ERN and anxiety in never-institutionalized samples (Olvet & Hajcak, 2008). The lack of this relation may be attributable to the low incidence of clinical anxiety disorders in the present sample at 12 years of age (Humphreys et al., 2015).

There are a number of limitations associated with this study. First, all analyses were conducted within an intent-to-treat framework regardless of each child’s current placement. However, given that many participants no longer reside in their randomized caregiving placement (Figure 1), our findings likely underrepresent the potential benefits of early enhancement of caregiving quality on error monitoring and psychopathology. Second, a rather substantial portion of the sample could not complete the task with sufficient accuracy (n = 27), leading to reduced sample sizes and power. The large number of participants excluded for poor accuracy is not unexpected given the substantial number of children who exhibited low IQs at 12 years.

In sum, this study provides evidence that one possible pathway to the development of externalizing and ADHD behaviors in children who have experienced institutionalized care is through deficits in error monitoring.

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**Key points**

- Institutional care is associated with increased psychopathology in adolescence; however, the risk factors associated with the emergence of these disorders in this sample are unknown.
- Error monitoring has been implicated in the development of both internalizing and externalizing disorders.
- Children randomized to continued institutional care show perturbed behavioral performance and altered neural markers of error monitoring.
- Neural markers of error monitoring moderated the relationship between amount of time spent in institutional care and externalizing and ADHD behaviors, with children with reduced error monitoring exhibiting increased externalizing behaviors when compared with children with better error monitoring.
• Future work should aim to investigate whether interventions targeting error monitoring may protect against the development of externalizing disorders in previously institutionalized populations.

References


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