Early Adverse Experiences and the Neurobiology of Facial Emotion Processing

Margaret C. Moulson
Massachusetts Institute of Technology

Charles H. Zeana
Tulane University Health Science Center

To examine the neurobiological consequences of early institutionalization, the authors recorded event-related potentials (ERPs) from 3 groups of Romanian children—currently institutionalized, previously institutionalized but randomly assigned to foster care, and family-reared children—in response to pictures of happy, angry, fearful, and sad facial expressions of emotion. At 3 assessments (baseline, 30 months, and 42 months), institutionalized children showed markedly smaller amplitudes and longer latencies for the occipital components P1, N170, and P400 compared to family-reared children. By 42 months, ERP amplitudes and latencies of children placed in foster care were intermediate between the institutionalized and family-reared children, suggesting that foster care may be partially effective in ameliorating adverse neural changes caused by institutionalization. The age at which children were placed into foster care was unrelated to their ERP outcomes at 42 months. Facial emotion processing was similar in all 3 groups of children; specifically, fearful faces elicted larger amplitude and longer latency responses than happy faces for the frontocentral components P250 and Nc. These results have important implications for understanding of the role that experience plays in shaping the developing brain.

Keywords: early experiences, face processing, event-related potentials, facial emotion, institutionalization

The tragic consequences of early institutionalization are well documented across multiple domains of development. Children raised in institutions often suffer from growth restriction and other medical sequelae (Johnson, 2000), and their cognitive and language development is delayed (Beckett et al., 2006; Carlson & Earls, 1997; Nelson et al., 2007; O’Connor, Rutter, Beckett, Keavney, & Kreppner, 2000). They also display a number of behavioral problems (e.g., attention-deficit/hyperactivity disorder and autistic-like behaviors; Beckett et al., 2002; Fisher, Ames, Chisholm, & Savoie, 1997; Kreppner, O’Connor, & Rutter, 2001; Rutter et al., 1999; Vorria, Rutter, Pickles, Wolkind, & Hobshaum, 1998) and socioemotional difficulties, such as deficits in joint attention (Tarullo, 2007), attachment problems (O’Connor, Bredenkamp, & Rutter, 1999; O’Connor & Rutter, 2000; Zeanah, Smyke, Koga, & Carlson, 2005), and indiscriminate friendliness (Chisholm, 1998; O’Connor et al., 1999; O’Connor & Rutter, 2000; Rutter et al., 2007). However, few studies have explored the origins of these deficits by examining the effects of early institutionalization on brain development.

The small number of studies that have examined brain development in institutionalized children find consistent evidence of alterations in neural structure and functioning. For example, Chugani et al. (2001) found that postinstitutionalized children have decreased glucose metabolism in limbic and paralimbic regions of the brain, as well as structural changes in brain connectivity from the amygdala to areas of the prefrontal cortex (Eluvathingal et al., 2006). Marshall and colleagues found that currently institutionalized children display a pattern of increased low-frequency (theta) power and decreased high-frequency (alpha and beta) power in their electroencephalogram (EEG) compared to family-reared children (Marshall & Fox, Nelson, & Zeanah, 2004), as well as increased short-distance coherence compared to children placed in foster care (Marshall, Reeb, Fox, 2008). Additionally, Parker and Nelson (2005a, 2005b) found that institutionalized children display...
decreased-amplitude event-related potentials (ERPs) in response to pictures of faces compared to family-reared children.

To explore further the neural consequences of early institutionalization, the present investigation used ERPs to examine brain activity in response to facial expressions of emotion in children with histories of institutionalization. ERPs are a noninvasive measure of the brain’s electrical activity that reflect the response of large populations of neurons firing synchronously in response to discrete stimulus events (Handy, 2005). Examining differences between children with and without histories of institutionalization in terms of the amplitudes, latencies, and overall morphology of their ERP waveforms across multiple brain regions may provide insight into the neural changes that accompany a history of institutionalization. The neural responses to facial expressions of emotion were of particular interest because there is reason to suspect that the neural systems that subserved face perception may be particularly disrupted by early institutionalization, due to the experience-dependent nature of this ability. Additionally, it is possible that the social difficulties observed in institutionalized children (e.g., deficits in joint attention, indiscriminate friendliness) have their origins in atypical processing of social stimuli such as emotional faces; therefore, examining the neural responses to facial expressions of emotion may offer insight into higher level social deficits in institutionalized children.

Although there are no studies that have quantified the perceptual exposure to faces experienced by children in different rearing environments, the characteristics of institutional care make it likely that institutionalized children have atypical experiences with faces compared to children reared in typical families. The documented high child-to-caregiver ratios, high caregiver turnover, and limited adult–child interactions within institutions (as reviewed in Zeanan et al., 2003) may lead to less exposure to adult faces in institutionalized compared to family-reared children. It is also possible that institutionalized children have access to a more limited range of facial expressions of emotion or have disproportionate experience with particular facial expressions (e.g., negative or neutral expressions) compared to children reared in typical homes. Certainly, the social interactions in which institutionalized children experience face stimuli differ from those of family-reared children, although it is unknown to what extent the broader social context in which faces are experienced affects the low-level perceptual discrimination of faces and facial emotions.

If institutionalization does lead to atypical experiences with faces, previous research suggests that those experiences might have a powerful effect on the developing face-processing system. Nelson (2001) argues that the development of face processing reflects an experience-dependent process, in which experiences with faces over the first years of life drive cortical specialization. Numerous studies with both typically and atypically developing children have now demonstrated that early experiences shape the development of face perception (Bar-Haim, Ziv, Lamy, & Hodes, 2006; Geldart, Mondloch, Maurer, de Schonen, & Brent, 2002; Kelly et al., 2007; Le Grand, Mondloch, Maurer, & Brent, 2001, 2003; Pascalis, de Haan, & Nelson, 2002; Sangrigoli & de Schonen, 2004).

Particularly relevant to the current investigation are behavioral studies that have examined facial emotion processing in children reared in adverse early environments. Pollak and colleagues (Pollak, Cicchetti, Hornung, & Reed, 2000; Pollak & Kistler, 2002) found that children raised in abusive households show atypical processing of angry faces but not other emotional faces. These children show a response bias for angry faces (they are more likely to match any emotional situation to a picture of an angry face; Pollak et al., 2000). They also overidentify the emotion of anger but do not differ from controls in identifying happiness, sadness, and fear (Pollak & Kistler, 2002), and they correctly identify facial expressions of anger on the basis of less perceptual information than controls (Pollak & Sinha, 2002). In addition, they attend to angry faces more than controls do and have trouble disengaging from angry faces (Pollak & Tolley-Schell, 2003). This pattern of results is likely due to increased exposure to negative emotions in abusive households (Kavanagh, Youngblade, Reid, & Fagot, 1988; Lyons-Ruth, Connell, Zoll, & Stahl, 1987). There is also some evidence that a history of institutionalization negatively impacts facial emotion processing. Wismer Fries and Pollak (2004) demonstrated that 4.5-year-old previously institutionalized children were less accurate than controls at identifying the emotions happiness, sadness, fear, and anger, and they had difficulty matching emotional faces to appropriate emotional scenarios. Similarly, Parker and Nelson (2005a) found some evidence for altered processing of facial emotions in currently institutionalized children, in that they showed larger amplitude ERP components in response to fearful versus sad faces, whereas family-reared children showed larger amplitude ERP components in response to sad versus fearful faces.

Despite this evidence demonstrating the effects of early experience on facial emotion processing, it is still unknown exactly how much and what kinds of experiences are required to drive the typical development of the system. Therefore, it is entirely plausible that even the adverse environment of institutionalization provides sufficient exposure to faces to set up the neural architecture that subserves face perception. Indeed, a recent study demonstrated that monkeys who had no exposure to faces during the first months of life could still perform basic face-discrimination tasks (Sugita, 2008). Potentially, children raised in institutions are simply exposed to enough faces to ensure the typical development of face processing.

Thus, the first two goals of the current investigation were to (a) determine whether early institutionalization results in widespread neural changes as assessed by ERPs and (b) examine whether early institutionalization leads to altered perceptual processing of facial expressions of emotion. The third goal of the study was to determine whether placement in high-quality foster care could ameliorate any adverse neural changes that resulted from early institutionalization. This study is the first randomized controlled trial of foster care as an intervention for institutionalization, and it provides a unique opportunity to examine questions about the possibility for recovery of neural functioning among previously institutionalized children. We were particularly interested in whether the timing and duration of the intervention (i.e., foster care) were related to the extent of any improvement observed in previously institutionalized children. If so, it would give us insight into whether or not there is a sensitive period during which placement in foster care allows for optimal recovery and beyond which placement in foster care is less effective in ameliorating any neural dysfunction that results from institutionalization. Previous research with internationally adopted children has shown that children adopted before approximately 6 months of age are indistinguish-
able from family-reared children, whereas children adopted after 6 months of age are more likely to have persistent problems spanning physical, cognitive, and socioemotional domains. This is true even when children are assessed several years after adoption, and the risk increases the longer a child resides in an institution (Beckett et al., 2006; Morison & Ellwood, 2000; O’Connor et al., 2000; O’Connor & Rutter, 2000; Rutter et al., 2007).

To explore these questions, the current study focused on the neurological functioning of three groups of children enrolled in the Bucharest Early Intervention Project (BEIP), a longitudinal study of the effects of institutionalization on brain and behavioral development (Zeanah et al., 2003). The BEIP assessed children residing in institutions across Bucharest, Romania, on a number of physical, cognitive, socioemotional, and neurological measures. Following a baseline assessment, institutionalized children were randomly assigned to either continued institutional care or placement in high-quality foster care. As state-run foster care was virtually nonexistent when the project began, foster families were recruited, screened, trained, and supported by BEIP itself (Zeanah et al., 2003). Institutionalized and foster-care children were compared to a group of family-reared children in Bucharest during follow-up assessments at 9, 18, 30, 42, and 54 months of age. The ethical considerations of this project have been discussed in detail elsewhere (Zeanah et al., 2006a, 2006b), but it is important to highlight two key points. First, children who were placed in BEIP foster care were never returned to institutional care. Second, institutionalized and foster-care children were not prevented from moving into alternate environments (e.g., reunion with their biological family, placement into state-run foster care, or placement with an adoptive family) with the permission of the appropriate authorities.

At the baseline, 30-, and 42-month assessments, ERPs were recorded while children passively viewed pictures of faces expressing different emotions, thereby allowing a comparison of the neurological functioning of institutionalized, previously institutionalized, and never-institutionalized children in the context of a socioemotional processing task. This investigation, an extension of the work of Parker and Nelson (2005a), consists of additional analyses of the baseline data, and new analyses of the follow-up assessments.1 On the basis of previous research, it was predicted that institutionalized children would show decreased amplitude brain activity compared to never-institutionalized children at all assessments. It was also predicted that foster care would be at least somewhat effective in remedying any differences in the amplitude and pattern of brain activity associated with institutionalization and that children who were placed in foster care earlier in life and had correspondingly longer durations of intervention would show the greatest improvement.

Regarding the effects of institutional care specifically on facial emotion processing, we offer two competing hypotheses. First, it is possible that both the perceptual exposure to faces and overall social environment in institutions are sufficiently atypical to adversely affect the development of the face processing system, which would be manifested in differing neural responses to particular facial emotions in institutionalized versus never-institutionalized children. The alternative is that institutions do provide adequate input to set up the neural architecture responsible for the perceptual processing of facial emotion, which would result in minimal differences between institutionalized and never-institutionalized children in their neural processing of different facial emotions.

Method

Participants

Participants included 208 children recruited in Bucharest, Romania, to take part in the BEIP. At the baseline assessment, which occurred between 5 and 31 months of age, the institutionalized group (IG) consisted of 136 children recruited from six institutions within Bucharest who had spent at least half of their lives institutionalized. Over 50% of these children had spent all of their lives in institutions. The never-institutionalized group (NIG) consisted of 72 children who were recruited through birth records from the maternity hospitals where the institutionalized children were born and were matched to the IG on age and sex. To the best of our knowledge, based on health information collected from both the IG and NIG, none of these children had a history of seizures. Exclusion criteria were genetic syndromes, overt signs of fetal alcohol syndrome, and microcephaly. Eleven of the IG children initially recruited for this study retrospectively met exclusion criteria and were subsequently excluded from all analyses, reducing the total number of IG children to 125. Following the baseline assessment, children in the IG were randomly assigned to continued institutional care (IG, n = 62) or foster care (FCG, n = 63). An additional 24 participants (IG, n = 11; FCG, n = 1; NIG, n = 12) were not included in the longitudinal sequence of assessments, but were recruited for cross-sectional data collection.

ERP data from 85, 81, and 75 participants at the baseline, 30-, and 42-month assessments, respectively, are reported [baseline: IG n = 62 (31 females), NIG n = 23 (9 females); 30-month: IG n = 26 (11 females), FCG n = 33 (16 females), NIG n = 22 (12 females); 40-month: IG n = 29 (13 females), FCG n = 33 (15 females), NIG n = 13 (8 females)]]. An additional 100 (54%), 72 (47%), and 48 (39%) participants at the baseline, 30-, and 42-month assessments, respectively, were excluded from data analysis because of technical error, having fewer than 10 artifact-free trials per condition, blinking while the picture was on the screen on 25% or more trials, or excessive eye or body movement artifact. Due to the wide range of ages at the baseline assessment (5 to 31 months), age (younger, older) based on median split was included as a factor in the analyses for the baseline assessment. The median age of the IG at baseline was 23.6 months (31 younger, 31 older), whereas the median age of the NIG at baseline was 21.1 months (12 younger, 11 older). Although many children assigned to the IG and FCG no longer resided in those environments at 42 months due to the noninterference policy of the BEIP (Figure 1), original group assignments were preserved during data analysis. This intent-to-treat approach is conservative, as it likely underestimates any differences between the groups.

1 Our primary motivation for performing additional analyses at the baseline assessment was to report data from occipital electrodes, which were not analyzed in Parker and Nelson (2005a) due to a technical error that was described in an erratum to the original article.
Stimuli and Procedure

At each assessment, ERPs were recorded from children while they viewed color pictures of faces expressing the emotions anger, happiness, fear, and sadness (Figure 2). The stimuli were taken from the MacBrain Face Stimulus set (the “NimStim”) (Tottenham et al., in press). Each participant saw one of three Caucasian females posing all four emotions. Each of the four emotions was presented on 25 trials, in random order, for a maximum of 100 trials.

Children sat on the parent’s or caregiver’s lap in front of a computer screen that was surrounded by black panels that blocked the children’s view of the room. The presentation of each stimulus was preceded by a 100-ms baseline period, followed by the presentation of the stimulus for 500 ms, followed by a 1,200-ms poststimulus recording period during which a blank blue screen was presented. The intertrial interval varied randomly between 500 and 1,000 ms. An experimenter observed the child from behind the black screen and eliminated trials during which the child looked away from the computer screen. The experimenter also tapped on the screen or shook a rattle to direct the child’s attention back to the screen when necessary. The study session continued until the child had seen the maximum number of trials (100) or had become too fussy or distracted to continue.

Electrophysiological Recording and Processing

ERPs were recorded from 13 scalp leads (Fz, F3, F4, Cz, C3, C4, Pz, P3, P4, T7, T8, O1, O2) and left and right mastoids using a Lycra Electro-Cap (Electro-Cap International, Inc., Eaton, OH) with sewn-in tin electrodes and referenced online to Cz. Additionally, vertical electro-oculogram was recorded from electrodes placed above and below the left eye, bisecting the midline, to record blinks and other eye movements. Following cap placement, abrasive gel was inserted into each of the electrode sites, after which the scalp under each electrode site was gently abraded. A small amount of electrolytic gel was then inserted into each electrode site. Impedances were at or below 10 kΩ for each electrode. EEG and electro-oculogram signals were amplified by factors of 5,000 and 2,500 respectively, with a 0.1 to 100 Hz band-pass filter, using custom bioelectric amplifiers from SA Instrumentation Company (San Diego, CA). All channels were digitized at 512 Hz.

Figure 1. Status of participants in the institutionalized group, foster care group (FC), and never-institutionalized group of the Bucharest Early Intervention Project at 54 months of age.
onto the hard drive of a PC using a 12-bit analog-to-digital converter (± 2.5 V input range) and Snap-Master acquisition software (HEM Data Corporation, Southfield, MI). ERP equipment and testing parameters remained the same for all three assessments.

Data processing was carried out using the ERP32 data analysis software package (Version 3.82; New Boundary Technologies, Minneapolis, MN). Trials 1,400 ms long (100 ms baseline + 500 ms stimulus presentation + 800 ms poststimulus recording) were scored for artifact. Channels that exceeded ± 200 μV were marked as bad in a particular trial. After the data were re-referenced to an average mastoids configuration, individual averages were created for each of the four conditions (anger, happiness, fear, sadness). During the averaging process, a trial was rejected if there were more than two channels marked bad due to artifact. Additionally, a blink correction algorithm was applied based on methods described in the literature (Gratton, Coles, & Donchin, 1983). Participants with fewer than 10 good trials per condition were excluded from further analysis (see Table 1 for average numbers of good trials by group and assessment). Grand means were created by averaging the individual averages together.

Table 1
Average Numbers of Good Trials by Group and Assessment

<table>
<thead>
<tr>
<th>Assessment</th>
<th>IG M (SD)</th>
<th>FCG M (SD)</th>
<th>NIG M (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>17.1 (2.88)</td>
<td>19.6 (3.68)</td>
<td>19.6 (3.68)</td>
</tr>
<tr>
<td>30-month</td>
<td>18.2 (3.19)</td>
<td>19.8 (2.86)</td>
<td>21.6 (1.18)</td>
</tr>
<tr>
<td>42-month</td>
<td>20.7 (1.84)</td>
<td>20.6 (1.94)</td>
<td>21.3 (2.29)</td>
</tr>
</tbody>
</table>

Note. IG = institutionalized group; FCG = foster care group; NIG = never-institutionalized group.

Grand means (Figures 3, 8, and 11) were inspected to identify components of interest. At all three assessments, three occipital components (P1, N170, P400) and two frontocentral components (P250, Nc) were analyzed. Time windows that captured the components of interest were identified separately for each assessment, as previous research has shown that there are maturational changes in the latency of various components across age (Batty & Taylor, 2006; Taylor, Batty, & Itier, 2004; Webb, Long, & Nelson, 2005). Thus, the time windows for some components differed in duration and/or starting latency across assessments.

Results

For each component of interest at each assessment, peak amplitude, peak latency, and area under the curve (if appropriate) were analyzed. For occipital components, 4 (condition: angry, happy, fearful, sad) × 2 (hemisphere: left, right) × 3 (group: IG, FCG, NIG) repeated-measures analyses of variance (ANOVA)s were conducted. For frontocentral components, data were collapsed across frontal and central sites to create composite frontocentral
variables and 4 (condition: anger, happiness, fear, sadness) \( \times 3 \) (laterality: left, central, right) \( \times 3 \) (group: IG, FCG, NIG) repeated-measures ANOVAs were conducted. For the baseline assessment, there were only two groups (IG, NIG), as random assignment had not yet occurred, and an additional factor (age: younger, older) was included in all ANOVAs. Where the omnibus ANOVAs revealed significant main effects, pair-wise comparisons were carried out using \( t \) tests with a Bonferroni correction for multiple comparisons. Where the omnibus ANOVAs revealed significant interactions, independent samples \( t \) tests or one-way ANOVAs were carried out. An alpha level of .05 was used for all statistical tests. On the basis of a priori hypotheses, follow-up analyses were conducted for marginally significant (.05 < \( p < .07 \)) main effects of group or condition.

**Baseline Assessment**

**Occipital Components**

**P1** (90–190 ms). There was a main effect of group for peak amplitude, \( F(1, 74) = 13.90, p < .001 \), indicating that the NIG showed a larger amplitude P1 than the IG (Figure 3). There was a significant Group \( \times \) Age interaction for latency, \( F(1, 74) = 5.20, p = .025 \). Follow-up \( t \) tests revealed that younger IG and NIG children showed similar latency to the P1, \( t(36) = -0.59, ns \), whereas older NIG children showed a significantly faster latency to the P1 than did older IG children, \( t(38) = 2.90, p = .006 \) (Figure 4).

**N170** (150–325 ms). There was a significant Hemisphere \( \times \) Group interaction for peak amplitude, \( F(1, 74) = 4.24, p = .043 \). Follow-up \( t \) tests revealed that children in the NIG showed larger N170 amplitudes than children in the IG, but only over the right hemisphere, \( t(76) = -2.48, p = .015 \). These effects were qualified by a significant Hemisphere \( \times \) Group interaction, \( F(1, 74) = 5.05, p = .028 \). Follow-up analyses revealed that the NIG showed larger P400 amplitudes than the IG over the right hemisphere only, \( t(76) = -2.48, p = .015 \). Additionally, the NIG showed larger amplitudes over the right than left hemisphere, \( F(1, 21) = 7.58, p = .012 \), whereas the IG did not (Figure 6A). This was due primarily to the older NIG children, as further follow-up analyses revealed that older NIG children showed this hemispheric asymmetry, \( F(1, 9) = 37.90, p < .001 \), whereas younger NIG children did not. A Condition \( \times \) Hemisphere interaction was also found, \( F(3, 222) = 3.67, p = .023 \). Follow-up analyses revealed that there were larger P400 amplitudes over the right than the left hemisphere for happy faces, \( F(1, 77) = 3.36, p = .071 \) and fearful faces, \( F(1, 77) = 5.54, p = .021 \), but not angry and sad faces (Figure 6A).

Latency analyses revealed main effects of hemisphere, \( F(1, 74) = 7.12, p = .009 \), group, \( F(1, 74) = 6.20, p = .015 \), and age, \( F(1, 74) = 5.08, p = .027 \). These effects were qualified by a significant Hemisphere \( \times \) Group \( \times \) Age interaction, \( F(1, 74) = 5.00, p = .028 \). Follow-up analyses revealed that P400 latency did not differ for children in the IG as a function of hemisphere or age. However, in the NIG, older children showed faster P400 latencies over the right than the left hemisphere, \( F(1, 9) = 10.26, p = .011 \), whereas younger children did not show this asymmetry. Also, over the right hemisphere older NIG children showed faster latencies than younger NIG children, \( t(20) = 2.21, p = .039 \) (Figure 6B).

---

\( ^2 \) The subset of BEIP participants used for the baseline analyses in this study differed somewhat from the subset of participants used for the baseline analyses in Parker and Nelson (2005a). This was due in part to slightly more stringent criteria for artifact rejection in this study (\( \geq 500 \mu V \) vs. \( \geq 250 \mu V \) in Parker and Nelson, 2005a) and the number of good trials required for inclusion in the analyses (10 trials vs. 9 trials in Parker and Nelson, 2005a). It is also likely that the inclusion of occipital electrodes in our analyses led to the exclusion of additional participants. Our findings from the baseline assessment were generally consistent with the results of Parker and Nelson (2005a). The only difference of note was that Parker and Nelson (2005a) found some evidence for group differences in the neural processing of facial emotion, whereas we did not. This discrepancy is explored in the Discussion.
Frontocentral Components

**P250 (185–385 ms).** For peak amplitude, there was a main effect of laterality, $F(2, 142) = 5.60, p = .006$. Post hoc comparisons indicated that the P250 was larger over right and midline leads than left leads. There were no significant main effects or interactions for P250 latency.

**Nc (350–600 ms).** There was a main effect of laterality for the amplitude of the Nc, $F(2, 142) = 8.68, p < .001$. Post hoc comparisons demonstrated that the Nc was larger over midline than left or right leads. There was also a main effect of condition, $F(3, 213) = 2.97, p = .035$. Post hoc comparisons revealed that fearful faces elicited significantly larger Nc amplitudes than did angry faces (Figure 7). Inspection of the means indicated that Nc amplitudes to fearful faces were also larger than to happy and sad faces, although these differences were not significant. Latency analyses revealed a main effect of group, $F(1, 71) = 6.24, p = .015$, indicating that IG children had faster latencies to the Nc than NIG children. There was also a main effect of laterality, $F(2, 142) = 3.43, p = .035$, where latency to the Nc was faster over left leads than midline leads.

30-Month Assessment

**Occipital Components**

**P1 (80–190 ms).** There was a main effect of group for peak amplitude, $F(2, 71) = 3.12, p = .05$. Post hoc comparisons indicated that both NIG and FCG children showed larger P1 amplitudes than IG children (Figure 8), although these differences were not significant. There were no significant main effects or interactions for P1 latency.

**N170 (175–325 ms).** For the peak amplitude, there was a main effect of hemisphere, $F(1, 70) = 5.69, p = .02$, indicating that N170 amplitude was larger over the left hemisphere than the right. There were no significant main effects or interactions for N170 latency.

---

3 Our analysis approach for the frontocentral components at the baseline assessment differed from Parker and Nelson (2005a) because it was based on what analysis we felt was most appropriate at the 30- and 42-month assessments.
P400 (250–500 ms). Analyses of peak amplitude revealed a main effect of hemisphere, $F(1, 69) = 6.33, p = .014$, indicating that the P400 was larger over the right than the left hemisphere. There were no significant main effects or interactions for P400 latency.

Frontocentral Components

P250 (185–350 ms). Peak amplitude analyses revealed main effects of condition, $F(3, 186) = 2.80, p = .044$, and laterality, $F(2, 124) = 10.47, p < .001$. Post hoc comparisons indicated that fearful faces elicited larger P250 amplitudes than happy faces (Figure 9A), and that P250 amplitudes were larger over midline than left or right leads. Latency analyses also revealed main effects of laterality, $F(2, 124) = 3.20, p = .046$, and condition, $F(3, 186) = 3.09, p = .03$. The main effect of condition was qualified by a significant Condition $\times$ Group interaction, $F(6, 186) = 3.13, p = .007$. However, follow-up analyses revealed no significant group differences for any of the conditions. Post hoc comparisons collapsing across group indicated that latency to the P250 was faster for happy faces than angry faces (Figure 9B) and faster over midline than left or right leads.

Nc (350–550 ms). For peak amplitude, there was a main effect of laterality, $F(2, 124) = 5.97, p = .003$, indicating that Nc amplitudes were larger over midline than left or right leads. This effect was qualified by a significant Condition $\times$ Laterality interaction, $F(6, 372) = 2.30, p = .041$. Follow-up analyses revealed that the main effect of laterality was largely driven by the angry condition, as it was the only condition that showed larger Nc amplitudes over midline than left and right leads, $F(2, 128) = 9.57, p < .001$. Latency analyses revealed a main effect of condition, $F(3, 186) = 6.08, p = .001$. Post hoc comparisons indicated that happy and sad faces generated faster latencies to the Nc than fearful faces (Figure 10).

Occipital Components

P1 (80–210 ms). Peak amplitude analyses revealed main effects of group, $F(2, 69) = 3.65, p = .031$, and hemisphere, $F(1, 69) = 4.77, p = .032$. The amplitude of the P1 was larger over the right than the left hemisphere, and post hoc comparisons indicated that the NIG showed significantly larger amplitudes than the IG, whereas the FCG was not different from either group (Figure 11). Latency analyses revealed a main effect of group that approached significance, $F(2, 69) = 2.88, p = .063$. Post hoc comparisons indicated that P1 latency was faster in the NIG than the IG, with the FCG falling in between; however, the differences between the three groups were not significant on follow-up.

N170 (160–300 ms). There were no significant main effects or interactions for N170 peak amplitude. There was a significant Condition $\times$ Hemisphere $\times$ Group interaction for N170 latency, $F(6, 136) = 2.70, p = .019$. Follow-up analyses revealed no condition or hemisphere effects for either the IG or FCG, but a significant Condition $\times$ Hemisphere interaction in the NIG, $F(3, 36) = 4.96, p = .025$. Further follow-ups indicated a hemispheric asymmetry for the sad condition, in that the N170 latency for the sad condition was faster over the left than the right hemisphere, $F(1, 12) = 5.08, p = .044$. This hemispheric asymmetry was not apparent for the other conditions.

P400 (250–500 ms). Inspection of the grand means indicated that the P400 was more broadly distributed at this assessment
compared to earlier assessments. As such, peak amplitude and latency were not analyzed; instead, area under the curve was. For P400 area under the curve, there was a main effect of group, $F(2, 69) = 3.13, p = .05$. Post hoc comparisons revealed that NIG children showed significantly larger area under the curve than IG children, while FCG children were not different from either group (Figure 11).

Frontocentral Components

**P250 (185–350 ms).** For peak amplitude, there was a main effect of laterality, $F(2, 142) = 14.21, p < .001$, and a main effect of condition that approached significance, $F(3, 213) = 2.47, p = .07$. Post hoc comparisons indicated that P250 amplitude was significantly larger over midline than left or right leads, and that

![Figure 9](image-url)

*Figure 9.* Condition differences for (A) P250 amplitude and (B) P250 latency at the 30-month assessment. Error bars represent standard errors of the means. * $p < .05.$
P250 amplitude was larger in response to fearful faces than angry, happy, or sad faces, although these differences were not significant on follow-up. Latency analyses revealed a main effect of laterality, $F(2, 142) = 6.82, p = .002$, that was qualified by significant Group $\times$ Laterality, $F(4, 142) = 2.83, p = .03$, and Condition $\times$ Laterality, $F(6, 426) = 3.20, p = .006$, interactions. Follow-up analyses revealed that both the IG and FCG showed larger amplitudes over midline than left or right leads, IG: $F(2, 54) = 12.69, p < .001$; FCG: $F(2, 64) = 3.94, p = .027$, although the NIG did not. Additionally, over the left hemisphere happy faces elicited faster latencies than angry or fearful faces, whereas over midline leads both happy and sad faces elicited faster latencies than fearful faces (Figure 12). Over the right hemisphere there were no differences between the conditions.

$N_c$ (350–500 ms). For peak amplitude, there was a main effect of condition, $F(3, 213) = 5.74, p = .001$, that was qualified by a significant Condition $\times$ Laterality $\times$ Group interaction, $F(12, 134) = 3.03, p = .001$. Follow-up analyses revealed that all three groups showed larger amplitudes for fearful faces than happy, angry, or sad faces (Figure 13A), but only the FCG and NIG showed significant laterality effects. In the FCG, condition differences were only present over right leads, and in the NIG, condition differences were present over left and right, though not midline leads. For $N_c$ latency, there was a main effect of condition, $F(3, 213) = 3.79, p = .013$. Post hoc comparisons indicated that happy faces elicited faster $N_c$ responses than angry, fearful, or sad faces (Figure 13B).

**Analysis of Intervention Effects**

To evaluate the effect of the foster care intervention, the IG and FCG were compared at the 42-month assessment. The NIG was used for comparison purposes only and is not included in the analyses. Because placement in foster care occurred at variable ages following the baseline assessment, the FCG was divided into two groups (early-placed, late-placed) based on a median split at 24.5 months. Because age at placement is directly related to the duration of intervention experienced by the FCG—that is, the early-placed group had correspondingly longer durations in foster care, whereas the late-placed group had correspondingly shorter durations in foster care—we used only one of these variables to analyze the effect of the foster care intervention. To evaluate the overall effect of the intervention, independent samples $t$ tests were performed on the amplitudes and latencies of the occipital components comparing the IG and FCG. To evaluate whether the timing and/or the duration of foster care affected outcomes at 42 months of age, two analysis techniques were used. First, separate one-way ANOVAs were performed comparing the IG ($n = 27$), the early-placed FCG ($n = 16$), and the late-placed FCG ($n = 16$). Second, correlations between age at placement into foster care and the amplitudes and latencies of the occipital components were performed for the FCG only.

Independent samples $t$ tests by group revealed few significant differences between the IG and FCG. P1 latency was marginally faster in the FCG compared to the IG, $t(57) = 1.89, p = .064$. No other comparisons were significant. However, it is possible that the timing of placement in foster care and/or duration of institutionalization affected ERP outcomes at 42 months of age, such that children who were placed earlier and had correspondingly longer...
durations in foster care (the early-placed group) show more improvement than children placed in foster care later (the late-placed group). If this were the case, we would expect the early-placed group to show significantly larger amplitudes and shorter latencies than both the IG and the late-placed FCG for the occipital components.

One-way ANOVAs revealed no significant differences between the IG, early-placed FCG, and late-placed FCG for any of the occipital components. In addition, from an examination of the means for all three groups (Table 2), there does not seem to be any discernible pattern whereby children in the early-placed FCG had generally larger amplitudes and shorter latencies for the occipital components than the late-placed FCG. However, it is possible that a correlational analysis might reveal more clearly any differences in outcome based on age at placement into foster care. Thus, Pearson correlations were computed between age at placement into foster care and the occipital variables. Only one significant correlation was found, for the peak amplitude of the N170, \( r = -0.374, p = 0.035 \) (Figure 14). As the N170 is a negative-going component, the negative correlation here between age at placement and N170 amplitude indicates that children who were older when placed in foster care tended to show larger (i.e., more negative) amplitudes for the N170.

Discussion

The purpose of the current study was to examine the effects of early institutionalization on the neural circuitry that subserves facial emotion processing using event-related potentials (ERPs) and to evaluate the efficacy of foster care in ameliorating any adverse changes caused by institutionalization. The first important finding that emerged from this investigation was that in the context of a facial emotion processing task, institutionalized children demonstrated dramatically smaller amplitudes and longer latencies for the occipital components compared to never-institutionalized children, which persisted until at least 42 months of age. This is consistent with previous ERP findings in this sample of children. Specifically, Parker and Nelson (2005a, 2005b) reported that institutionalized children in the BEIP showed smaller amplitudes than never-institutionalized children for four ERP components located over frontal, central, parietal, and temporal regions at the baseline assessment. Additionally, we have reported that institutionalized children also display decreased amplitudes for occipital components across the three assessments compared to never-institutionalized children in the context of a caregiver–stranger face recognition task (Moulson, Westerlund, & Nelson, in press).

These findings are also broadly consistent with other findings documenting the neural consequences of institutionalization. Marshall and colleagues found altered patterns of EEG power and coherence in institutionalized compared to never-institutionalized children in the BEIP (Marshall & Fox, 2004; Marshall et al., 2008), and Chugani et al. (2001) found reduced metabolic activity in limbic and paralimbic regions of the brain in postinstitutionalized children. Taken together, these results suggest that institutionalized children display widespread cortical hypoarousal. The term cortical hypoarousal was initially used to describe the pattern of EEG activity observed in children with disorders of learning and attention (for review, see Barry, Clarke, & Johnstone, 2003)—specifically, increased low frequency (theta) power and decreased high frequency (alpha and beta) power—which is mirrored by the pattern of EEG activity observed in institutionalized and previously institutionalized children (Marshall & Fox, 2004; Marshall et al., 2008; Tarullo, 2007), as well as children reared in other adverse environments (e.g., families of low socioeconomic status; see Otero, 1997; Otero, Pliego-Rivero, Fernandez, & Ricardo, 2003). Although it seems likely that the findings reviewed above...
regarding the neural consequences of early institutionalization are all manifestations of cortical hypoarousal, the underlying neuro-physiological sources of this phenomenon are unknown.

A key feature of the current study was the randomized controlled trial of foster care as an intervention for early institutionalization. Following placement in foster care, previously institutionalized children showed some evidence that the cortical hypoarousal resulting from early institutionalization was diminished, in that their ERP amplitudes and latencies for occipital components were intermediate between the institutionalized and never-institutionalized children at the 30- and 42-month assessments. In conjunction with other findings from the BEIP (Marshall et al., 2008; Nelson et al., 2007), this suggests that placement in high-quality foster care may be somewhat effective in remediating adverse neural changes caused by early institutionalization. Whether or not children placed in foster care will eventually show full recovery remains an open question. Notably, the extent of improvement of cortical hypoarousal was unrelated to the age at which children had been placed in foster care. This suggests that the timing of placement in foster care, and correspondingly its duration, were not significant factors in predicting children’s response to intervention following institutionalization, at least in terms of their ERP outcomes.

That the timing and duration of intervention were unrelated to the extent of improvement in children’s ERP outcomes suggests either that the sensitive period for general brain development is sufficiently long to allow remediation following even extended periods of deprivation, and children will eventually show full recovery; or that the sensitive period for full recovery closed before these children were placed in foster care. Because children were not assessed beyond 42 months of age in the current study, it is impossible to determine whether they will eventually show full remediation of cortical hypoarousal; thus, at present we cannot distinguish between these alternative interpretations of the current results.

The lack of timing effects on ERP outcomes is surprising, as numerous previous studies have demonstrated that earlier intervention leads to better outcomes across multiple domains of development (for review, see Maclean, 2003). Indeed, even other studies with children in the BEIP have shown that earlier placement in foster care is associated with better outcomes—specifically, increased cognitive recovery (Nelson et al., 2007) and improvements in EEG power and coherence (Marshall et al., 2008). This is strongly suggestive of domain specificity in the presence, onset, and offset of sensitive periods underlying these different domains of development.

Another key finding in the current study was that all three groups of children showed differential neural processing of the four emotions. This differential emotion processing was present across all three assessments and was found primarily for the frontocentral components P250 and Nc. That fearful faces elicited generally larger amplitudes and longer latencies than the other three emotions, whereas happy faces elicited smaller amplitudes and shorter latencies, is consistent with previous literature. For example, 7-month-old infants show a larger amplitude Nc in response to fearful versus happy faces (Nelson & de Haan, 1996). In older children, other negative emotions in addition to fear (e.g., anger, sadness, disgust) also elicit larger amplitudes for the Nc compared to happy faces (Batty & Taylor, 2006; Nelson & Nagent, 1990).

It is surprising that children who experienced early deprivation as a result of institutional care did not show atypical neural processing of facial emotions at any assessment. Converging ev-
idence from the BEIP that early institutionalization does not disrupt the ability to discriminate facial emotions is found in the performance of the BEIP children on a visual paired comparison task at both the baseline and 42-month assessments. Children were familiarized to the same emotional faces used in the ERP task and were tested on familiar versus novel emotions. Children in all groups showed similar discrimination of facial emotions (Jeon, Nelson, & Mousson; Nelson, Parker, & Guthrie, 2006). These results indicate that the ability to discriminate facial emotions is left largely intact following a period of early deprivation, which suggests that the likely atypical experience that institutionalized children have with faces is sufficient to correctly shape the neural architecture that subserves the perceptual discrimination of facial emotions. However, this finding is in contrast to previous ERP findings with this sample of children. Parker and Nelson (2005a) found that institutionalized and never-institutionalized children showed different neural responses specifically for the fearful and sad expressions at the baseline assessment. It is difficult to determine the cause of this discrepancy, although certainly the differences in analysis strategy may have played a role. However, the fact that our findings were replicated at the 30- and 42-month assessments and were consistent with behavioral findings at baseline and 42 months lends some confidence to our interpretation that institutionalized children do not differ radically from family-reared children in their perceptual processing of facial emotions.

Despite the evidence for intact processing of facial emotion found in this study, it is currently unknown whether these children will show deficits further downstream in processing facial emotion (e.g., in recognizing and understanding facial emotions). Neither ERP nor visual paired comparison tasks are particularly sensitive measures of sophisticated social–cognitive understanding of facial emotion. Indeed, studies that have used more complex behavioral tasks have found subtle behavioral deficits in facial emotion processing in postinstitutionalized children (Wismer Fries & Pollak, 2004). It is also possible that a longer period of deprivation (i.e., beyond 42 months of age) would lead to corruption of the neural systems that subserve even the perceptual discrimination of facial emotions.

In sum, the findings from this study provide evidence that children raised in institutions display cortical hypoarousal compared to children raised in their biological families. Despite this general hypoarousal, these findings also indicate that institutionalized children show similar neural processing of facial emotions compared to never-institutionalized children, at least to the extent that this task was a sensitive measure of facial emotion processing. Results from children who were randomly assigned to the foster care intervention suggest that the cortical hypoarousal that results from early institutionalization is amenable to improvement following this change in rearing environment.

The current study is a valuable addition to the existing literature documenting the neural consequences of early institutionalization. Not only do these findings have important implications for our scientific understanding of the effects of early psychosocial deprivation on the brain and the existence of sensitive periods during development, but they also have far-reaching implications from a policy standpoint. The possibility that foster care is somewhat effective in ameliorating the neural dysfunction associated with early institutionalization holds promise for the many children still living in institutional care throughout the world.

References


face processing requires visual input to the right hemisphere during infancy. *Nature Neuroscience*, 6, 1108–1112.


Received September 30, 2007
Revision received June 11, 2008
Accepted August 19, 2008