Behavioral Differences in Aggressive Children Linked with Neural Mechanisms of Emotion Regulation

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ABSTRACT: Children with aggressive behavior problems may have difficulties regulating negative emotions, resulting in harmful patterns of interpersonal behavior at home and in the schoolyard. Ventral and dorsal regions of the prefrontal cortex (PFC) have been associated with response inhibition and self-control—key components of emotion regulation. Our research program aims to explore differences among aggressive and normal children in the activation of these cortical regions during emotional episodes, to the extent possible using electrophysiological techniques, to identify diagnostic subtypes, gain insights into their interpersonal difficulties, and help develop effective treatment strategies. This report reviews several recent studies investigating individual and developmental differences in cortical mechanisms of emotion regulation, corresponding with different patterns of interpersonal behavior. Our methods include event-related potentials (ERPs) and cortical source modeling, using dense-array electroencephalography (EEG) technology, as well as videotaped observations of parent–child interactions, with both normal and aggressive children. By relating patterns of brain activation to observed behavioral differences, we find (i) a steady decrease in cortical activation subserving self-regulation across childhood and adolescence, (ii) different cortical activation patterns as well as behavioral constellations distinguishing subtypes of aggressive children, and (iii) robust correlations between the activation of cortical mediators of emotion regulation and flexibility in parent–child emotional communication in children referred for aggressive behavior problems. These findings point toward models of developmental psychopathology based on the interplay among biological, psychological, and social factors.

KEYWORDS: aggression; cortical source modeling; emotion; psychopathology; event-related potentials (ERPs)

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INTRODUCTION

The capacity to regulate emotional impulses effectively is critical for normal development, and children lacking these skills often develop serious behavior problems. Specifically, aggressive children may not adequately regulate the thoughts, feelings, and actions arising from negative emotional states such as anger and anxiety, and this may greatly diminish the quality of their relationships, first with their parents and later with their peers. Yet research on emotion regulation in child clinical populations has not made much progress. This may be due, in part, to the problems inherent in assessing emotion regulation using behavioral measures, which inevitably conflate emotional expression, regulation, and behavior itself. Thanks to the new tools of cognitive neuroscience, however, the biological substrates of emotion regulation can now be explored in the laboratory and related to individual differences in interpersonal behavior. Linking neural indices of emotion regulation with observed behavioral differences has been the goal of our research over the last few years, and we have pursued these objectives through the use of dense-array electroencephalography (EEG) and videotaped observations of parent–child interactions. By examining these measures for clinically referred children, and by comparing them with data from age-matched controls, we are advancing a model of developing emotion regulation capacities—a model that can help explain typical and atypical developmental trajectories and guide prevention and intervention policies.

In this report we outline the theoretical and empirical considerations that guide the search for brain mechanisms of emotion regulation in children, relate them to research on aggressive behavior problems, review the methods we have developed for analyzing brain and behavior patterns, and highlight results from several recent studies. Our most interesting finding to date is that reduced neural activity related to emotion regulation corresponds with an overall decrease in behavioral flexibility in children with aggressive behavior problems.

Scope of the Problem

Approximately half of all referrals to children's mental health agencies are for oppositional or aggressive behaviors.[1] Childhood aggression is associated with a host of serious difficulties. Most notably, early onset of aggression predicts later delinquency and adult criminality[1] and is linked to severe psychosocial maladjustment across several domains including peer relations[2] and academic functioning.[3] Adolescent antisocial behavior is predictive of later occupational instability, unemployment, marital problems, depression, and substance abuse.[1,3] Moreover, it is widely accepted that childhood anxiety and aggression problems co-occur.[4] Several studies suggest that children

with both aggression and anxiety problems are at higher risk for a number of negative outcomes, compared with "pure" aggressive children.[5]

Emotion Regulation and Child Psychopathology

Clinically significant aggression and anxiety problems can be understood as disorders of emotion regulation.[6,7] Children with these problems may have failed to develop the capacity to appropriately modulate their feelings of anger and anxiety and the behaviors that flow from them. Research with young children indicates an association between poor emotion regulation and aggressive outcomes. Young children who are less able to voluntarily shift their attention and inhibit their emotional impulses have higher levels of aggression.[8] In contrast, children with good emotional control are able to shift attention away from anger-inducing cues and use nonhostile verbal methods.[9] In these and related studies, behavior regulation and emotion regulation are sometimes considered extensions of a more fundamental capacity for executive or "effortful" control.[10]

Neurocognitive Mechanisms of Emotion Regulation

Individual differences in emotion regulation become deeply entrenched, they reliably predict psychopathological outcomes, and they become increasingly resistant to intervention as children mature. For these reasons, most investigators assume that different styles of emotion regulation express distinct biological mechanisms.[11] Developmental psychologists are becoming increasingly interested in the neurobiological substrates of these mechanisms. Neural approaches use imaging techniques, lesion studies, and electrophysiological methods to specify cortical regions and activation profiles that mediate them. Research with adults has made progress linking these control mechanisms with normal and abnormal emotional processes. However, *developmental* neuroscience is only beginning to tackle emotion and its regulation, despite wide agreement on the importance of this agenda.

Neuroimaging and lesion studies have focused on prefrontal systems that mediate appraisal, inhibitory control, and self-monitoring, which may all be critical components of emotion regulation. The dorsal anterior cingulate cortex (ACC), on the medial wall of each frontal lobe, is a key structure for selecting among competing choices, making judgments, monitoring one's performance, and learning.[12,13] The ACC can also be involved in processing emotion, and it is specifically implicated when individuals are in control of their emotional responses or judgments.[14] The orbitofrontal cortex (OFC), on the ventral surface of the prefrontal cortex (PFC), is responsible for assigning emotional significance, especially in social situations, and for maintaining a response set such

as avoidance or inhibition in anticipation of emotional consequences.[15] Thus, dorsal and ventral prefrontal systems have unique cognitive styles: dorsal systems (e.g., dorsal ACC) appear to mediate the smooth, deliberate control of behavior in a supervisory or top-down fashion, whereas ventral systems (e.g., ventral ACC and OFC) control impulses rigidly, in anticipation of negative consequences. Importantly, both children and adults show increased activation in both the ACC and OFC during response inhibition.[16] Hence, both structures may play a role in emotion regulation in children as well as adults.[17]

In adults, externalizing and internalizing psychopathologies are linked with emotion dysregulation corresponding to anomalies in both these frontal systems. Aggressive individuals typically show deficits in both ACC and OFC activation,[18] implying under-regulation of behavior. Blair[15] suggests that the OFC is especially important for the regulation of reactive aggression, and Hoptman[19] found aggression to be associated with decreased metabolism in anterior, inferior, and medial–frontal systems. Conversely, anxious and depressed individuals show greater-than-normal activation in ventral systems including the OFC and ventral ACC.[20]

EEG methods are particularly appealing for clinical research because they are noninvasive, versatile, and relatively inexpensive. EEG or electrical brain wave activity is recorded at the scalp from an array of electrodes. Event-related potentials (ERPs) are computed by averaging EEG data over many trials on a given task. Several ERP components recorded over the PFC are thought to tap aspects of cognitive control. The frontal N2 is seen 200–400 msec poststimulus on trials requiring participants to withhold a prepotent response, and it is often assumed to tap inhibitory control mechanisms. Negative emotional evaluations predict higher amplitude N2s[21] and the N2 is enhanced by negative feedback concerning one's performance.[22] Thus, greater N2 amplitudes may reflect the ramping up of inhibitory controls when negative emotions arise. Another ERP component, the error-related negativity (ERN), is recorded approximately 50–100 msec postresponse and is thought to tap action monitoring or response control.[23] The ERN has been linked to anxiety and negative affect. Less impulsive, more controlled individuals show enhanced ERNs[24] as do individuals with obsessive-compulsive styles.[23] Similarly, individuals with negative mood or trait negative affect show higher amplitude ERNs,[25,26] whereas undersocialized individuals show lower amplitude ERNs.[27] Thus, the cognitive controls tapped by the N2 and ERN may be recruited, to different degrees by different individuals, for the regulation of emotion and emotional behavior. Researchers have now begun to examine these ERP components in children,[17] but few studies to date have investigated their role in children's emotional processes.

Dense-array EEG techniques (e.g., recording from 128 channels rather than just a few) allow researchers to model the generators of ERPs using source analysis methods. Source modeling programs place hypothetical generators in a model of the cortex and test for goodness-of-fit against the fine-grained

scalp data provided by multiple electrodes. We are particularly interested in this methodology, because it allows us to test hypotheses about the approximate location of cortical activities that may underpin unique mechanisms of emotion regulation. Source analyses of medial–frontal ERPs (including the N2 and ERN) indicate a key generator in the region of the ACC for adults.[28,13] Similarly, the region of the OFC, particularly in the right hemisphere, has been identified as a generator of the N2 in studies of adults and children.[28,29] Source analysis of scalp EEG cannot provide definitive anatomical information, but we have utilized source modeling to examine the *relative* contributions of global prefrontal systems to ERP variables that may differentiate styles of emotion regulation.

Parent–Child Interactions and the Development of Psychopathology

Poor parent–child interactions are one of the central causal factors implicated in the development of childhood psychopathology.[30] Most notably, decades of direct observational studies conducted in the home and the laboratory have established a clear link between particular patterns of parent–child relations and childhood aggression.[31,32] Our recent work[33] points to the flexibility versus rigidity of parent–child relations as an especially relevant dimension for predicting clinical disorders. Both aggressive and anxious children tend to have inflexible parent–child interactions (they become "stuck" in habitual emotional exchanges) in contrast to nonclinical family interactions that flexibly shift to accommodate contextual demands.

Linking Parent–Child Interactions with Neurocognitive Mechanisms of Emotion Regulation

How might parent–child interaction patterns that contribute to aggressive behavior problems be associated with distinct neurobiological constellations of impaired emotion regulation? According to our model, aggressive children who are "pure" externalizers (EXT) cannot control their angry impulses when confronted with blocked goals because they do not anticipate negative consequences from their overly permissive parents. We hypothesize that these children fail to recruit both dorsal and ventral frontocortical controls, and thus their angry emotions remain unregulated. However, children who are comorbid for internalizing and externalizing problems (MIXED) may have parents who are intermittently permissive and punitive, and they may attempt to regulate their resultant anxiety through excessive reliance on ventral control systems. Thus, compared with age-matched controls, both subpopulations may be unable to control their emotional impulses in a smooth, deliberate manner using the dorsal ACC, and such differences should show up in ERPs associated with specific cortical regions and specific regulatory functions.

In conclusion, our working hypothesis is that unique regulatory dysfunctions depend on particular cortical modes of control that develop in parallel with distinct parent–child interaction styles and predict distinct constellations of behavior problems. These modes may preclude flexible, top-down self-regulation and thus contribute to the behavioral rigidity that characterizes the parent–child interactions of aggressive children.

REVIEW OF METHODS AND SELECTED EMPIRICAL FINDINGS

Development of State Space Grid Methodology

We have recently developed state space grid (SSG) analysis, a graphical and quantitative tool based on dynamic systems (DS) principles. This method allows researchers to examine several coexisting interaction patterns and explore movement from one to the other in real time. DS theorists use the concept of a state space to represent the range of behavioral habits, or attractors, for a given system. Behavior is conceptualized as moving along a real-time trajectory on this hypothetical landscape, being pulled toward certain attractors and freed from others.[34] Based on these abstract formalisms, Lewis *et al*.[35] developed a graphical approach that utilizes observational data and quantifies these data according to two ordinal variables that define the state space for any individual psychobehavioral system. Granic and Lamey extended this methodology to represent dyadic behavior (e.g., parent–child interactions).[36] The dyad's trajectory (i.e., the sequence of emotional states) is plotted on a grid representing all possible combinations (FIG. 1). Much like a scatterplot, one dyad member's (e.g., parent's) coded behavior is plotted on the x-axis and the other member's (e.g., child's) behavior is plotted on the y-axis. Thus, each point on the grid represents a simultaneously coded parent–child event (i.e., a dyadic state).

Differences in Parent–Child Interactions

We began our investigation of the etiology and treatment of clinical subtypes by trying to discover the distinct parent–child interaction patterns that differentiate pure EXT and MIXED children.[36] Parents and clinically referred children were asked to discuss a problem for 4 min and then try to "wrap up" in response to a signal (or perturbation). The perturbation was intended to increase the emotional pressure on the dyad, triggering a reorganization of their behavioral system. It was hypothesized that, as a function of differences in the underlying structure of their relationships, EXT and MIXED dyads would be differentially sensitive to the perturbation and would reorganize to different regions of the state space. Subtyping was determined by scores on parent- and

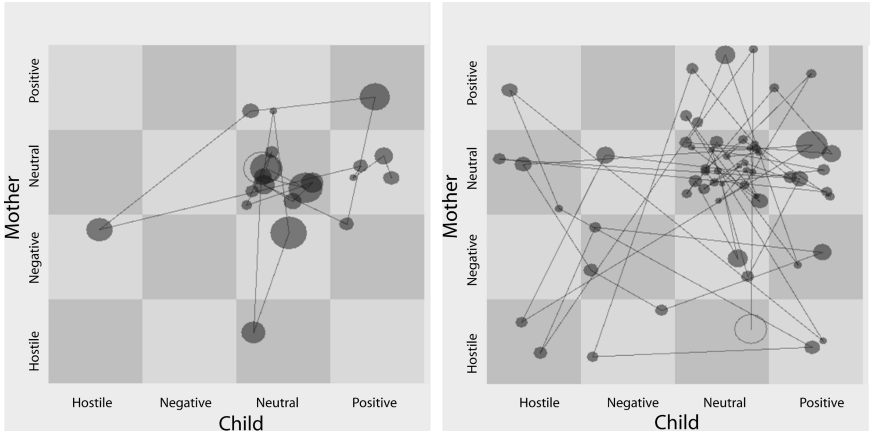

FIGURE 1. Examples of dyadic SSGs depicting low behavioral flexibility (left panel) and high behavioral flexibility (right panel).

teacher-rated behavior scales: EXT children were those with externalizing scores in the clinical range, whereas MIXED children scored in the clinical range on the internalizing *and* externalizing scales. Separate grids were constructed for the pre- and postperturbation interaction sessions. Both EXT and MIXED dyads tended toward the permissive region of the SSG (child hostile–parent neutral/positive), as well as other regions (i.e., mutual neutrality and negativity), *before* the perturbation. After the perturbation, EXT dyads tended to stabilize in the permissive region. MIXED dyads, however, tended toward the mutual hostility or mutual negativity region of the grid. These graphical observations were subsequently confirmed by case-sensitive, multivariate analyses including log-linear modeling.

Emotion-Induction ERP Methodology

We then developed a novel ERP paradigm that integrates an emotion induction process with a go/no-go procedure. On standard go/no-go tasks, participants respond as quickly as possible with a button-click on most trials but withhold clicking given particular cues. ERPs recorded during successful no-go trials (e.g., the N2) are seen as tapping cognitive processes involved in response inhibition, whereas ERPs following errors (e.g., the ERN) are thought to tap cognitive processes recruited for performance monitoring. In our task, the children are shown desirable toys or gift certificates prior to the procedure, and they are reminded several times that they need to earn a high number of points to receive one of these rewards. They are then instructed to click each time a letter appears but to avoid clicking when the same letter appears twice.

The stimulus presentation speed is adjusted dynamically to maintain an error rate (on no-go trials) of approximately 50%.[37] This innovation ensures that task difficulty is consistent across children, regardless of age and concentration skills. The task is divided into three blocks, and points (displayed on-screen every 20 trials) rise steadily for all children during block A. However, due to an adjustment in the algorithm, points begin to drop sharply in block B and end up back at zero. This block induces negative emotions such as anxiety, anger, and distress, as confirmed by emotion rating scales administered following the task. Finally, points rise again during block C so that a prize can be awarded, but negative emotions are presumed to remain active.

Developmental Differences in Cortical Mechanisms of Emotion Regulation

In the first complete study using the new task, we examined developmental differences in two inhibitory ERPs, the N2 and frontal P3, before and after the negative emotion induction (block A vs. blocks B and C).[17] Fifty-eight normal children, 5–16 years of age, were tested. We hypothesized that ERP amplitudes would diminish with age, consistent with fMRI and ERP studies suggesting that cortical efficiency improves with development, but that amplitudes would increase with the emotion induction in blocks B and C, indicating greater efforts at inhibitory control. Indeed, both the frontal N2 and frontal P3 components decreased in amplitude as well as latency across five age points in a fairly linear profile, $F(4, 48) = 2.66, P = 0.04$ and $F(4, 49) = 4.75, P = 0.003$ for the main effect of age on amplitudes. Amplitudes were also greater following the emotion induction phase of the task, suggesting increased inhibitory control in the service of emotion regulation. Source modeling indicated more central-posterior activation in younger children, giving way to medial–dorsal activation, suggestive of the dorsal ACC, as children matured. The finding of developmental "frontalization" is consistent with other recent work,[38] and it provides a useful backdrop for studying clinical groups.

Neurocognitive Differences among Subtypes of Aggressive Children

In our first study comparing clinically referred and normal children, we examined differences in the emotion regulatory mechanisms of subtypes of aggressive children.[39] Children (aged 8–12 years) were recruited from outpatient group treatment programs for aggressive children along with gender- and age-matched controls. Subtyping was determined by scores on parent- and teacher-rated behavior scales, as before. Only the MIXED children's N2s increased in response to the emotion induction, $F(2, 31) = 3.56, P < 0.05$, resulting in greater amplitudes than EXT children in block C, mean difference $= 4.08\ \mu V, P < 0.05$. As shown in FIGURE 2, ERN amplitudes were greatest for control children and smallest for EXT children, with MIXED children

in between, but these differences were significant only prior to the emotion induction, $F(2, 33) = 3.68$, $P < 0.05$. These results suggest that anticipatory self-regulation recruited unusually high cortical activation for MIXED children in the presence of negative emotion, but that EXT children actually required negative emotion to recruit near-normal levels of activation following their errors. Also shown in FIGURE 2 are striking differences in the source models of peak ERN activity across the three groups. The black squares superimposed on the Brain Electrical Source Analysis (BESA) head models depict the two regions of interest: that of the dorsal ACC (above) and the ventral PFC (OFC and ventral ACC) (below). Across all blocks, normal children showed

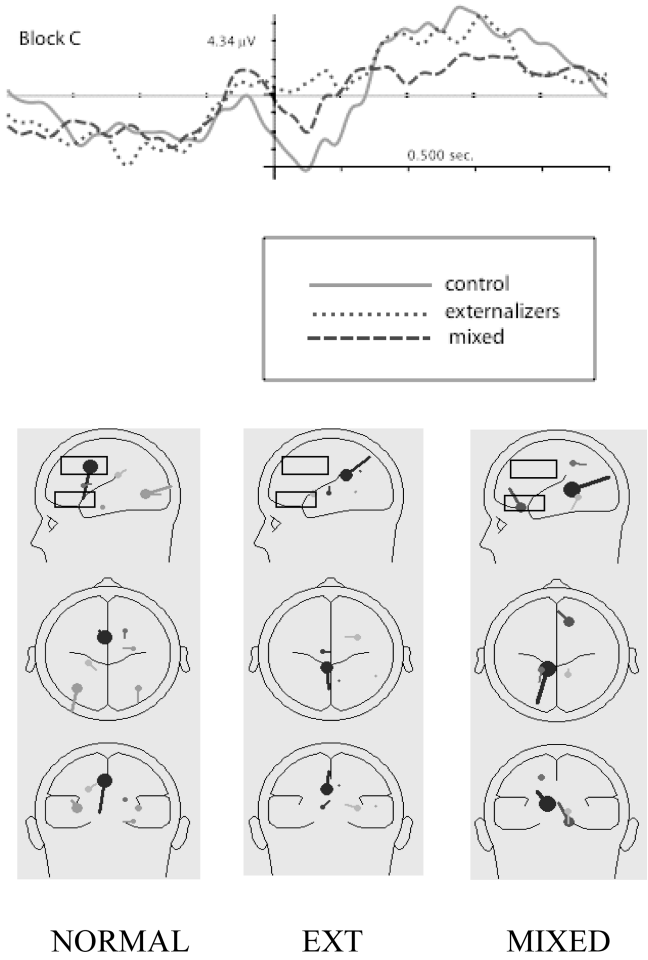

FIGURE 2. Grand-averaged waveforms and source models shown at peak ERN amplitudes, for normal, pure externalizing, and comorbid (externalizing/internalizing) children.

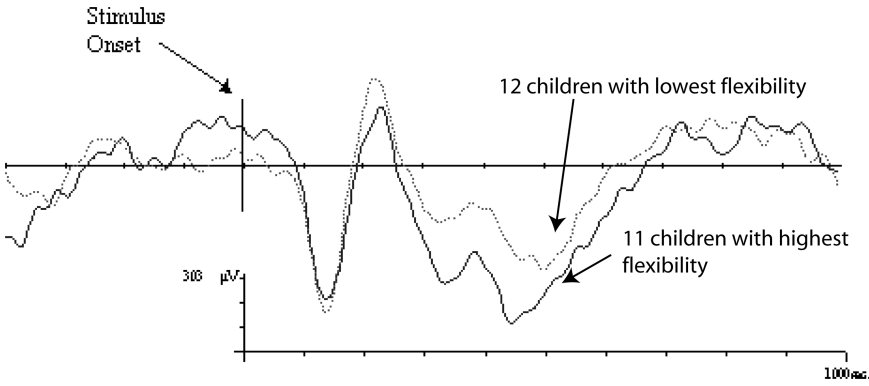

FIGURE 3. Grand-averaged waveforms for the most flexible and least flexible subgroups of children ($n = 11$ or 12). The N2, indicated by the arrow, shows greater mean amplitude for the flexible subgroup.

a strong source in the region of the dorsal ACC, consistent with models of adult brain activity during performance monitoring. EXT children showed no frontal activity whatever, but displayed a posterior source in the region of the posterior cingulate cortex. MIXED children demonstrated a similar posterior source and, importantly, a source in the region of the right OFC or right ventral ACC. These findings provide initial support for our model of subtype-specific differences in neurocognitive mechanisms of emotion regulation.

Neurophysiological Substrates of Behavioral Flexibility: Combining SSG and ERP Measures

Using the same sample of referred children, parent–child interactions were videotaped in the home before treatment. At each home visit, parents and children discussed consecutively: a positive topic, a mutually unresolved, anger-provoking problem, and another positive topic. Dyadic SSGs were constructed based on second-by-second codes derived from a modified version of the Specific Affect Coding System (SPAFF).[40] The flexibility of parent–child interactions was then assessed for the problem-solving discussion (the second topic) using three SSG measures: transitions—the number of movements from cell to cell, dispersion—the overall spread of behavior durations across the cells of the grid, and mean cell duration—the tendency for behavior to remain "stuck" within cells (a measure of rigidity, the converse of flexibility). We then examined associations between behavioral flexibility, tapped by these measures, and N2 amplitudes thought to tap neural mechanisms of emotion regulation derived from our task.

Values for children with complete observational and ERP data ($n = 33$) were entered into a three-step regression model, with N2 amplitudes as

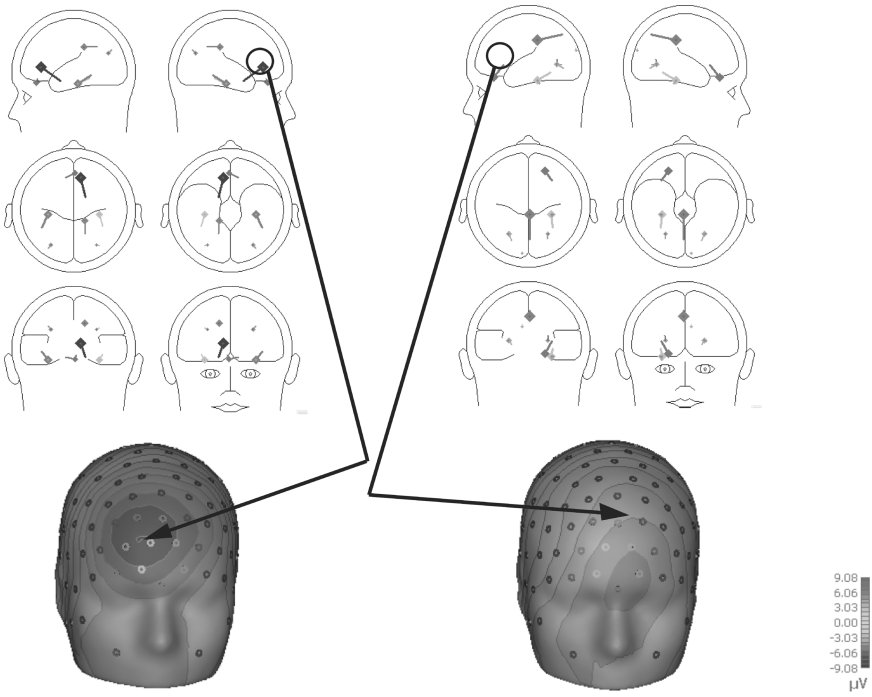

FIGURE 4. BESA head models for extreme groups ($n = 7$) of flexible (left panel) versus nonflexible (right panel) subgroups of aggressive children. A source in the region of the rostral ACC, at the peak of the N2, is evident for the flexible dyads only.

the dependent variable. The first step controlled for age, gender, ERP segment count, mean stimulus time, and medication (stimulant vs. nonstimulant). The second step controlled for the proportion of time spent in negative states, as negative emotion itself might be assumed to increase N2 amplitudes. In the third step, each of the three flexibility measures was entered in turn.

Flexibility measures did not predict N2 amplitudes in block A. However, these same measures predicted N2 amplitudes in block C (following the negative emotion induction; see FIG. 3): for transitions, $\Delta R^2 = 0.24$, $F(1, 25) = 10.26$, $P = 0.004$; for dispersion, $\Delta R^2 = 0.17$, $F(1, 25) = 6.52$, $P = 0.02$; for mean cell duration, $\Delta R^2 = 0.19$, $F(1, 25) = 7.57$, $P = 0.01$. In all analyses greater flexibility predicted greater amplitude N2s, suggesting greater recruitment of frontocortical control systems. A source analysis was performed on the most flexible and least flexible children ($n = 7$ in each group). As shown in FIGURE 4, a generator in the midline region of the PFC, corresponding to the rostral ACC, was observed for the flexible group but not for the nonflexible group. These results indicate higher amplitude cortical activations, probably

mediated by anterior cingulate areas, following the induction of negative emotions, for children showing more flexible interpersonal behavior during emotionally challenging interactions with their parents. Although all children in this sample were referred for aggressive behavior problems, these differences in behavioral flexibility corresponding with biological mediators of emotion regulation may help determine which families are most likely to benefit from treatment. Current research activities are aimed at testing this prediction.

CONCLUSION

Developmentalists are increasingly interested in refining and testing models of brain-behavior relations that can help explain individual differences in socioemotional development in general and childhood psychopathology in particular. Our research represents one approach for linking neural mechanisms of emotion regulation with behavioral habits that differ both in emotional content and overall flexibility. The work reviewed in this article represents an early phase of a relatively untested research paradigm, based on the integration of DS thinking and methods of cognitive neuroscience. However, the results so far have been encouraging, and they suggest future research directions that may ultimately benefit troubled children, their families, and their communities.

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