Changing the Neural Mechanism of Emotion Regulation in Children with Behavior Problems

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Introduction

Externalizing behavior problems are characterized by high levels of aggression and delinquency. These problems entail serious costs for the person, the immediate social environment, as well as social institutions and society as a whole, and they are thought to be persistent across the lifespan if left untreated (Caspi, Moffit, Newman, & Silva, 1996). Attempts to change the trajectory of externalizing problems through intervention programs that start in childhood seem promising but, unfortunately, they show a lot of variability in their outcomes. Studies generally reveal that about 40 per cent of aggressive children do not show any significant improvement when treatment has ended.

In order to improve treatment efficacy, many researchers call for a better understanding of what makes some children, and not others, respond to treatment. Cognitive mechanisms that children use to regulate emotions may play an important role in determining treatment outcomes. Emotion regulation allows individuals to control their emotional impulses, appraisals, and expressions. An effective strategy of emotion regulation, therefore, helps individuals display emotional responses with appropriate intensity and form, at the correct time and place, and modulate or inhibit inappropriate responses. Externalizing problem behaviors may be due to a lack of effective emotion regulation with respect to anger, and that is why they are characterized by excessive and disproportionate aggressive responses. Not surprisingly, the development of effective emotion regulation is thought to be crucial for adaptive socialization. Ineffective emotion regulation, on the other hand, can lead to the development of externalizing pathologies when aggressive responses become habitual and uncontrolled. These behavioral patterns are often highly resilient and may interfere with successful treatment outcomes once these pathologies have emerged.

Recently, with the discovery of neural mechanisms and neural markers of psychological processes, clinical-developmental psychology has increasingly been informed by neuroscientific research. The current chapter will discuss what we have learned in our lab about changes in the neural mechanisms of emotion regulation with treatment aimed at reducing children’s aggressive behavior. We
will first ask what networks in the brain are associated with emotion regulation. Second, we will ask if successful treatment aimed at improving emotion regulation yields changes in those hypothesized neural systems. Finally, we will speculate on how these results may improve clinical-development theory and stimulate future research, by postulating a hypothesis that underlying internalizing problems play a key role in the development of externalizing behavior problems.

**Emotion Regulation and its Neural Correlates**

Emotion regulation is an umbrella term for a suite of cognitive strategies, such as inhibitory control and error-monitoring, which are used to modulate emotions. Generally, two types of emotion regulation can be distinguished: reactive and deliberate regulation. Reactive regulation is fast and stimulus-driven, and it includes implicit evaluations of objects or events than can be aversive or rewarding. Possibly as a result of such appraisals, reactive regulation includes the rapid execution of automatic behavioral tendencies, such as avoidance or, in the case of externalizing behavior, approach. An effective reactive regulation, for example, could inhibit physical or verbal aggression toward others when it gets crowded upon entering the school bus. By contrast, a child who impulsively insults others shows ineffective, socially non-adaptive, reactive control. In the latter example, this behavior is likely to be at odds with long-term goals, such as establishing reliable friendships or avoiding punishment for harming other people. Reactive regulation can rely on attentional biases of threat perception which appraise the situation in a negative light and generate a rapid fight-or-flight response. With reactive regulation, the line between what is regulation and what is emotion blurs somewhat, because processes of emotional arousal and emotional regulation can be seen as overlapping or adjacent (Kappas, 2011). Deliberate emotion regulation, on the other hand, can be seen as less automatic than reactive regulation. It is characterized as slower, reflective, and more sensitive to strategy. Deliberate strategies, such as reappraisal and planning, may reduce the state of frustration and provide a sense of control. For example, a child who feels anxious at school because of a disappointing grade may reduce this feeling by realizing that other smart children also had large difficulties. This way, the child will not feel as a failure, and may maintain his commitment to academic achievement.

Both reactive and deliberate types of regulation can occur within the same person and at the same moment, and the effectiveness of these coping strategies can differ according to the situation and emotional state. It is possible that the coordination between reactive and deliberate regulation is crucial for effective emotion regulation. For example, an initially weak reactive regulatory response can be countered by strong deliberate regulation. However, little is known about the coordination of these processes, and future research could better ground these speculations.

Each type of regulation has been associated with a particular neural network (Phillips, Ladouceur, & Drevets, 2008; Ray & Zald, 2011). The anterior cingulate
cortex (ACC) is a structure at the midline of the brain, which is associated with reactive as well as deliberate emotion regulation. The ACC has typically been divided into a more “affective” ventral and a more “cognitive” dorsal component. The ventral ACC has strong connections with other ventral prefrontal regions, such as the orbitofrontal cortex, and subcortical regions involved in rapid affective processing such as the amygdala, hypothalamus, and brain stem. Because these networks mediate rapid, impulsive, and visceral regulation, they are thought to be associated with reactive emotion regulation. The dorsal ACC has strong connections to lateral prefrontal regions implicated in working memory, decision making, error monitoring, and response control, such as the dorsolateral prefrontal cortex (PFC). These networks support “higher order” reasoning and are therefore thought to mediate deliberate emotion regulation. Because of its dorsal and ventral divisions, the ACC has been dubbed a “hub” of emotion regulation, involved in the coordination of deliberate and reactive types of regulation.

Changes in Emotion Regulation with Treatment of Externalizing Behavioral Problems

Intervention studies investigating brain regions associated with emotion regulation have been rare, especially with child populations, because such studies are time-consuming and challenging to conduct. However, intervention studies are particularly valuable for understanding mechanisms of change. Instead of studying components of emotion regulation through experimental manipulation, treatment supposedly changes social behaviors that rely on emotion regulation capacities. Thus, changes in observed social behaviors can be inferred to tap changes in emotion regulation, and brain activity in regions hypothesized to mediate emotion regulation can then be examined in relation to those changes. We will next review a recent intervention study from our lab that investigated the neural correlates of emotion regulation.

The study (Woltering, Granic, Lamm & Lewis, 2011) tested seventy-one 8-to 12-year-old children and their families before and after they had completed a 14-week treatment program. The treatment program was aimed to increase children’s self-regulatory capacity. It consisted of Cognitive Behavioral Therapy (CBT) and Parent Management Training (PMT) and was conducted by community agencies. CBT targets effective regulation of emotion and impulses through strategies such as cognitive restructuring, problem solving, role-playing, social and token reinforcements, and generalization activities. PMT promotes positive parenting practices such as skill encouragement, problem solving, and monitoring, as well as the replacement of coercive or lax discipline strategies with mild sanctions targeting misbehavior. Treatment success was assessed by various parent and clinician reports, such as the Child and Adolescent Functional Assessment Scale and the Child Behavior Checklist (CBCL). Children were included in the study when their scores revealed clinical levels of externalizing behavior problems on the CBCL. We note that, although the criteria were aimed at externalizing behavior problems, it was remarkable that the sample appeared largely comorbid for internalizing problems.
Since it is difficult to measure broad psychological constructs such as emotion regulation directly, the cognitive process of inhibitory control, which is thought to be a component of emotion regulation, was measured by means of a go/nogo task. Children were fitted with an electroencephalography (EEG) net and instructed to press a button as fast as possible when a letter appeared on the screen, but to inhibit their response when a letter was repeated. To ensure engagement with the task, children were told that they needed to obtain a lot of points to receive a desirable prize. Points were given for correct responses and subtracted when mistakes were made. A dynamically adjusted algorithm ensured that the difficulty-level of the task remained challenging for each child. Neural correlates of inhibitory control were then investigated by examining event related potentials (ERPs—averaged fluctuations in electrophysiological activity) and source models estimating where in the brain this activity takes place.

ERP components called the N2, occurring 200–400 ms after the nogo stimulus, and the frontal P3, occurring between 300 and 900 ms after the nogo stimulus, have been related to inhibition and attentional control specifically (Falkenstein, Hoormann, & Hohnsbein, 1999) and self-regulation in general (Lewis et al., 2008; Cappadicia, Desrocher, Peppler, & Schroeder, 2009). Converging evidence points to a source or generator of N2 and frontal P3 activity in medial prefrontal regions such as the dorsal and ventral ACC (Bekker, Kenemans, & Verbaten, 2005). Activity in these regions, and other regions, such as the lateral PFC (indicative of reappraisal, a specific regulatory strategy viewed as deliberate) as well as para-amygdalar regions in the temporal lobe (suggestive of reactive regulation) were investigated. We note that although it is unlikely that amygdala activity can be detected directly through EEG technology, para-amygdalar activity is likely to reflect it. We predicted that these components, and their underlying sources, would change for those children who improved with treatment compared to those who did not. Larger N2 magnitudes, specifically, had been associated with comorbid anxious/aggressive behavior problems in previous work, and a decrease in magnitudes of this component was hypothesized to reflect a reduction in the effort required to maintain inhibitory control in stressful situations.

The findings showed differences in the N2 and the frontal P3 between the nonclinical and clinical groups. The N2, which showed larger magnitudes for the clinical group, was found to be highly sensitive to treatment-based improvement: improvers showed a sharp reduction in activation whereas activation for non-improvers stayed the same from pre- to post-treatment. Improvers reached levels of activity similar to those of the normal controls, whereas this was not the case for the non-improvers. Additional analysis revealed that N2 magnitudes correlated with the degree of improvement, lending further credence to the assumption that these neural indicators are related to the capacity for emotion regulation.

These ERP results were consistent with those of the source analysis. Similar to Lewis et al. (2008), and as expected from N2 localization studies, reduced activation was found in ventromedial PFC (indicative of the ventral ACC and OFC) regions for improvers only. In addition, a reduction in activation was
also found in limbic regions such as the bilateral anterior medial temporal lobe. These results suggest changes in fronto-limbic systems associated with reactive emotion regulation. However, in contrast to Lewis et al. (2008), the dorsomedial PFC (dorsal ACC region) also showed a significant reduction in activation. The reduction in dorsal activation for improvers with treatment may indicate a reduction in deliberate control, which seems counterintuitive, since we would expect deliberate regulation to be bolstered with treatment. It is possible, however, that the dorsal ACC plays a supportive role, and that a decrease in this particular system could reflect a reduced need of “deliberate” support when ventral systems are doing their job more effectively in the first place. Bilateral regions in the dorsolateral as well as ventrolateral PFC regions, implicated in reappraisal strategies, did not show changes with treatment.

We concluded that children with externalizing problems mostly demonstrated an overactivity in neural circuits indicative of a reactive style of emotion regulation. The activation decreases in these systems for improvers could reflect a reduction in the rigid, reactive style of emotion regulation which is characteristic of children with behavior problems.

The Anxiety Hypothesis of Aggression

How could these results increase our understanding of children’s externalizing problem behavior and stimulate future research directions?

We concluded that a reduction in this ventrally mediated fronto-limbic activity in improvers could indicate that this neural system of reactive regulation, and the socio-emotional behaviors it mediates, has normalized. Intervention studies with a neuroimaging component, conducted with populations diagnosed with externalizing problems, are rare, so it is difficult to draw comparisons. However, it is remarkable that treatment studies using brain imaging with internalizing populations have shown reductions in similar brain systems. The overactivity in those fronto-limbic systems in our clinical sample of externalizing children seems to be in line with the results of imaging studies in anxious populations. Both research programs find overactive limbic circuits to be associated with rapid, threat-focused attentional biases (Bishop, 2007; Ressler & Mayberg, 2007). In this light, it’s interesting to note that an extremely large proportion of children in our sample showed high (clinical) levels of internalizing problems. This comorbidity was reported to be a general phenomenon in a recent review by Bubier and Drabick (2009), and it may be key in interpreting our neural results as well. These findings support the idea that ventrally mediated fronto-limbic overactivity in our children could underlie their internalizing problem behavior and could explain the tension these children bring to social situations—a tension that eventually manifests in aggressive outbursts.

In our work, we have fostered a hypothesis of aggression based on comorbid anxiety that can serve as the basis for further research. Similar to Dollard (1939), the hypothesis proposes that aggression occurs because blocked goals would lead to a state of frustration which could lead to aggression in order to resolve
this uncomfortable state. In our hypothesis, we propose that anxiety leads to an increased risk of frustration that can underlie externalizing behavior problems in a subset of children. Anxious individuals are known to cling to predictable behavioral patterns and thoughts that provide them with a sense of control. This perceived control can become so important that it becomes a goal in itself. But since social life is full of events that are beyond one’s own control, such as other people’s opinions about you, rapidly shifting group dynamics, and unexpected mood swings, it is easy to feel a loss of control, particularly for anxious children. Together with a negative threat-focused bias, anxious children could have a greater propensity for becoming frustrated because more events are perceived as threatening, blocking goals of safety and control. Frustration can then build up and may result in aggression (see Figure 1, for illustration of the model). Whether aggression occurs may depend on a child’s learned behavioral response style, the intensity of the frustration, and a child’s self-regulatory strength. The overactivity in ventrally mediated fronto-limbic systems could thus be seen as a neural marker of this ineffective reactive regulation. The response patterns developed due to these negative biases could then not only manifest themselves in maladaptive avoidance behavior (e.g. the development of anxious behavior problems), but also in maladaptive approach behavior (e.g. the development of aggressive behavior problems).

![Figure 1](image.png)

Figure 1  An anxiety hypothesis of aggression: a path through which anxiety can lead to externalizing problem behavior.

The quest for many clinical neuroscientists is to find neural markers that relate to the syndrome of interest. Neural markers can inform diagnosis, suggest prognosis, and help decide on the most effective type of treatment, and they can also increase understanding of neurocognitive mechanisms that translate into general models of psychological functioning. The implications of our hypothesis for the development and treatment of externalizing problem behavior may prove to be valuable. Interventions could improve their effectiveness by directly targeting anxious behavior for a significant subset of children. Whether we continue to validate this hypothesis, or turn our attention to other approaches, we hope to have shown how translational research that applies neuroscientific principles to clinical problems can contribute to the discussion of hypotheses that benefit child-clinical practice.
References