

Developmental Neuroscience Perspectives on Emotion Regulation

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ABSTRACT—*Because individual differences in emotion regulation are associated with risk for childhood behavioral problems, multidisciplinary investigation of the genetic and neural underpinnings of emotion regulation should be a research priority. This article summarizes research findings from 3 independent laboratories to demonstrate the ways in which a variety of developmental human neuroscience-based approaches can address critical conceptual issues in the emergence of emotion regulation. To do so, the authors present 3 perspectives on how developmental neurobiology constrains and enriches theories of emotion regulation. The 3 perspectives of (a) genetics, (b) brain structure and function, and (c) plasticity of development are illustrated with empirical results derived from both typical and atypical samples of children and adults. These perspectives are complementary and sometimes represent different levels of analysis of the same question.*

KEYWORDS—*emotion regulation; developmental neuroscience; genetics; plasticity*

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Among the most consequential behavioral and neural changes that have occurred over the course of phylogeny is the capacity to regulate emotion. Humans acquire complex capabilities to regulate their emotions. Indeed, lack of competence in emotion regulatory skills is associated with a variety of behavioral problems. Much recent scholarly activity has been devoted to the definition and operationalization of emotion regulation (ER), as well as to the clarification of the myriad ways in which dysregulated emotion is associated with psychopathology (Cole, Martin, & Dennis, 2004). This article describes how various neuroscience-based approaches can identify and measure processes involved in the development of ER. We pose some conceptual issues in the study of ER and then summarize selected results from three independent laboratories that demonstrate how these issues can be made empirically tractable. The authors do not share a specific theoretical orientation toward the issue of ER, nor do we rely on a single definition of what constitutes ER. Rather than address definitional issues, we aim to highlight the utility of integrating across behavioral, neuroscience, and genetic research traditions as a way to better understand ontogenetic processes in ER. Thus, we offer three perspectives on how developmental neurobiology constrains and enriches theories of ER: the perspectives of (a) genetics, (b) brain structure and function, and (c) plasticity of development. These perspectives are complementary and sometimes represent different levels of analysis of the same question.

GENETICS OF ER

An understanding of genetics has become critical for understanding the link between children's adaptive ER and the emergence of behavioral problems. The development of all human characteristics is genetically influenced, in the sense that the genetic code carries transmissible information to assemble and regulate the organism (Lamb et al., 2006). However, individual differences and atypicalities in behavior might or might not be influenced by differences in allelic

variants among humans. For instance, differences in infant–mother attachment appear to be at most weakly influenced by genetic differences and thus necessarily primarily associated with nongenetic differences (Bokhorst et al., 2003; O'Connor & Croft, 2001). In contrast, most features of child temperament, adolescent and adult personality, and atypicalities that lead to psychopathology diagnoses are associated with genetic differences to some degree (Jang, 2005). The most common conception is that genetically based diatheses interact with environmental stressors to yield psychopathology. Progress in identifying specific genes and stressors has accelerated recently. Thus, the question naturally arises, “Are individual differences in ER heritable, and if so, which aspects of ER are most heritable? Do we have evidence implicating specific genes?”

Are Individual Differences in ER Associated With Genetic Differences Among Children?

One might hypothesize that differences in ER skills are due to learning differences, given that good ER skills are subject to socialization and seem prized by families and schools. On the other hand, evidence is strong that individual differences in impulsiveness and attention deficit hyperactivity disorder (ADHD), which are similar in behavioral manifestation to problematic ER, are moderately to strongly heritable (Ruf, Schmidt, Lemery-Chalfant, & Goldsmith, 2008; Waldman & Gizer, 2006). The best source of evidence for heritability in children is twin studies, and here, we focus on twin studies that

have investigated ER in the sense proposed by Rothbart and colleagues (Derryberry & Rothbart, 1997; Rothbart, Ellis, Rueda, & Posner, 2003), that is, effortful control of behavioral actions, often accomplished via allocating attention adaptively.

Table 1 summarizes twin studies directly relevant to effortful control. With the exception of the Chinese (Fan, Wu, Fossella, & Posner, 2001) and Japanese (Yamagata et al., 2005) studies, the data are confined to childhood. Twin studies imply genetic effects when monozygotic (identical) twin similarity exceeds dizygotic (fraternal) twin similarity, as it does for all studies, except Fan et al. (2001). Especially for sample sizes of fewer than 100 pairs, we must expect that random sampling variation will affect the correlation estimates substantially. Despite differences in age groups and assessment methods, the conclusion emerging from Table 1 is that individual differences in childhood effortful control are at least moderately heritable. The parent report questionnaire scales are clearly heritable, and the three laboratory and observer-based measures, considered as a whole, also appear to be heritable. Beyond that, the literature remains too sparse to draw conclusions about more nuanced behavior–genetic issues such as genetic bases of (a) continuity and change, (b) the association of effortful control with other temperament traits and clinical diagnoses, and (c) sex differences. However, all these issues are under investigation, with initial results in some of the articles cited in Table 1. The body of evidence implicating genetic effects is fully compatible with developmental plasticity in ER systems, as we describe below.

Table 1
Twin Similarity Indexed by Intraclass Correlations (R) for Selected Emotion Regulation Measures

Study; age range/mean age—measure(s)	MZ		All DZ	
	<i>R</i>	<i>N</i> pairs	<i>R</i>	<i>N</i> pairs
Goldsmith et al. (1997); 3–7 years				
CBQ Effortful Control Factor scores	.53	55	.35	64
Fan et al. (2001); 14–42 years				
Attentional Network Test (reaction time measures, conflict/executive control)	.73	26	.66	26
Yamagata et al. (2005); 17–32 years				
Effortful Control Scale, Adult Temperament Questionnaire (Japanese version)	.45	152	.21	73
Gagne & Saudino (2006); 2nd year				
TBAQ Inhibitory Control Scale	.83	66	.55	80
Lab-TAB measures of inhibitory control	.43	44	.00	52
Lemery-Chalfant et al. (2008); 7.6 years				
CBQ Attentional Focusing and Inhibitory Control scales (mother/father average)	.68 ^a	214	.07 ^a	349
Lemery-Chalfant et al. (2008); 5.5 years				
CBQ Attentional Focusing and Inhibitory Control scales (mother/father average)	.71 ^a	112	.13 ^a	171
Averaged observer ratings of attentional control	.82 ^a	112	.49 ^a	171
Gagne & Goldsmith (2007); older subsample, 3 years				
CBQ Inhibitory Control Scale	.56	144	.19	263
Gagne & Goldsmith (2007); younger subsample, 22 months				
TBAQ Inhibitory Control Scale	.71	129	.47	255

Note. DZ = dizygotic; MZ = monozygotic; CBQ = Children's Behavior Questionnaire; TBAQ = Toddler Behavior Assessment Questionnaire.

^a*Rs* from Lemery-Chalfant et al. (2008) are averaged across gender groupings.

What Specific Genes Are Associated With ER Differences?

The investigation of genetic variants associated with complex, polygenic medical and behavioral phenotypes is evolving rapidly. The first generation of molecular genetic studies typically analyzed one—or at most a handful—of the candidate genes suspected to increase risk for behavioral patterns related to ER, such as ADHD. Most of these studies examined genetic variants that regulate synaptic availability of the neurotransmitters serotonin or dopamine. For example, a single nucleotide polymorphism (SNP, or single base pair substitution in the DNA) in the catechol-*O*-methyltransferase (*COMT*) gene affects activity of an enzyme that degrades dopamine in prefrontal cortex (PFC). Individuals with the genetic code for valine on both chromosomes at the site of this SNP have decreased levels of dopamine in prefrontal areas and perform worse on various attention and executive function tasks than those who are homozygous for the methionine codon (e.g., Blasi et al., 2005) or are heterozygous (e.g., Wahlstrom et al., 2007). However, a review concludes that this SNP in the *COMT* gene usually accounts for only about 4% of the variance in task performance (Heinz & Smolka, 2006).

The second and current generation of human molecular genetic studies related to ER involves the simultaneous analysis of many genes. This work is represented by an international project to identify genes responsible for ADHD (Brookes et al., 2006a). The scale of this project—674 families containing 776 ADHD combined type cases along with parents and siblings, 1,038 SNPs covering 51 genes mostly related to regulation of neurotransmitter pathways, and 62 authors of the report—allows detection of genes likely to have been missed in studies conducted 5 or 10 years ago. This study replicated the association of a dopamine transporter (DAT1) and a dopamine receptor (DRD4) with ADHD and also identified associations with at least suggestive significance levels for 16 other genes, of which 8 showed stronger association. Findings for the *COMT* gene, discussed above, were negative, and all positive associations with ADHD were modest in strength. In a smaller study, Brookes et al. (2006b) showed that a sequence of markers (a haplotype) within the DAT1 gene were associated with ADHD. Moreover, children whose mothers drank alcohol during pregnancy accounted for the association between the DAT1 marker and the ADHD. This apparent interaction (DAT1 Risk Allele $\times$ Prenatal Alcohol Exposure) was complicated by the finding that mothers who themselves carried the DAT1 “risk” allele were more likely to drink alcohol during pregnancy.

Recent publications have examined differences between thousands of cases and controls in the frequency of thousands of SNPs covering virtually the entire human genome. The findings from these genomewide association studies promise to yield insight into hypertension, type II diabetes, celiac disease, bipolar disorder, and other common phenotypes (Wellcome Trust Case Control Consortium, 2007). Two early

generalizations are that new and unanticipated genes are being discovered and that single genes typically have small independent effects (less than 1% of the observed variance) on the phenotypes. It seems likely that insights about ER from genomewide association will first emerge from analyses of ADHD.

In summary, genetic research on ADHD foreshadows what we can expect from future molecular genetic studies of ER. A key problem will probably be heterogeneity in ER: Is there more than one process that can lead to problematic ER in a given context, and if so, do the different processes involve different genes? Another research challenge will be discovering interactions of risk alleles with environmental exposures. These interactions may actually reflect unsuspected gene–environment correlations and Gene $\times$ Gene interactions or they may be real and exist alongside these complicating factors. In any case, an understanding of genetic factors in ER will surely depend on analyzing endophenotypes from cognitive and affective neuroscience.

NEURAL BASES OF ER

A neuroscience approach to ER requires highly standardized assessment procedures that allow concurrent recording of neural or other physiological measures. Neuroscience approaches tend to focus on specific dimensions of ER. One key dimension along which ER strategies can be organized is voluntary and effortful versus automatic and uninstructed. The existence of automatic and uninstructed ER is predicated on the existence of neural circuits that modulate and attenuate certain forms of negative affect once they are elicited. Corresponding mechanisms that sustain positive affect may also exist. These mechanisms can be invoked automatically and then coactivated along with the generation of the emotion. The most basic forms of automatic ER are simple forms of emotion learning such as extinction. In extinction learning, a conditioned stimulus (CS) is presented without the accompanying unconditioned stimulus, and the responses previously associated with the CS (i.e., the conditioned response, e.g., electrodermal activity in the case of human autonomic conditioning) diminish in magnitude with repeated presentations. In rodents, such extinction learning is dependent on the medial PFC. Because simple cued-based emotional associative learning is thought to be amygdala dependent (Phelps & LeDoux, 2005), the extinction process is understood to depend on inhibitory pathways from PFC to amygdala that attenuate amygdala responsivity. Output pathways from the central nucleus of the amygdala directly control the autonomic outflow that indexes conditioned responding. Rodent studies have strongly confirmed the role of the medial PFC in modulating amygdala activity as the basic architecture for extinction learning (Quirk, Garcia, & Gonzalez-Lima, 2006). Human imaging studies with simple fear conditioning and extinction

are consistent with this rodent evidence (Phelps, Delgado, Nearing, & LeDoux, 2004).

In Addition to the Very Simple Form of Learning Expressed in Extinction, Are There Other Forms of Automatic or Uninstructed ER That Can Be Harnessed in the Laboratory?

We have operationalized one form of uninstructed ER using affective chronometry, specifically, the magnitude and rapidity of recovery following a negative emotion-eliciting stimulus. The principal conjecture underlying this work is that the regulation and generation of emotion overlap in time and that circuitry that regulates the negative affect is simultaneously and automatically activated when negative affect is elicited. To address this question, we characterized the time course of negative affect following the offset of a negative emotional stimulus. Using the emotion-modulated startle, in one study with adult participants (Jackson et al., 2003), we presented startle probes at various latencies following the offset of a negative picture to examine individual differences in the magnitude of startle potentiation after a negative stimulus ended. To ensure that we were measuring regulation (recovery) rather than reactivity differences, we measured startle magnitude *during* picture presentation as an index of reactivity and we regressed out this measure of reactivity from the measures of recovery, thus creating a “pure” residualized measure of recovery with reactivity removed. We predicted that participants with greater relative left prefrontal activation as assessed in a separate session with electroencephalography methods would show more recovery following the offset of a negative stimulus. The findings strongly supported our hypothesis and imply that variations in prefrontal function influence individual differences in automatic ER. The procedure that taps automatic or uninstructed ER could be adapted to examine developmental changes in ER using age-appropriate stimuli to evoke emotion. Our findings underscore three points: (a) the speed and magnitude of recovery following the offset of a negative stimulus appear to be automatic aspects of ER, (b) wide individual differences exist in this aspect of ER, and (c) variations in prefrontal function predict this aspect of ER.

Using Neuroimaging Methods to Interrogate the Brain Circuitry That Underlies Individual Differences in Voluntary ER

We have related individual differences in voluntary ER to other dimensions of affective style and psychopathology, as have others (Ochsner & Gross, 2005; Quirk et al., 2006). Davidson and colleagues (Urry et al., 2006) used functional magnetic resonance imaging (fMRI) to examine whether voluntary ER produced variations in amygdala activation and whether such changes in amygdala function were associated with variations in prefrontal function, as the anatomical circuitry would suggest. Using a voluntary ER task that we developed outside the

scanner (Jackson, Malmstadt, Larson, & Davidson, 2000), participants were trained prior to the scanning session to use cognitive strategies to reappraise a negative emotional picture. The reappraisals were expected to down- or upregulate negative emotion while brain function was measured using fMRI. The results revealed a linear ordering of amygdala activation, with the upregulate condition producing the highest levels of amygdala activation, a control condition producing an intermediate level of amygdala activation, and the downregulate condition producing the least amount of amygdala activation. More important for our purposes, however, was the identification of the prefrontal region that varied most closely with variations in amygdala function. We searched the entire brain volume and asked which regions were most strongly reciprocally related to the amygdala. The ventromedial PFC, including the ventral anterior cingulate cortex, was strongly reciprocally coupled with the amygdala such that participants who showed the largest decrease in amygdala activation during the downregulate condition (compared with the control condition) showed the greatest activation in the ventromedial PFC ($r_s > -.8$; Figure 1).

In this study, we also examined baseline cortisol. We collected six saliva samples per day for 7 consecutive days to calculate a robust measure of diurnal cortisol slope. Participants with a neural profile suggesting good ER (high levels of ventromedial PFC activation and low levels of amygdala activation) had the steepest cortisol slopes (Figure 1). These associations between brain function and cortisol slope were mostly driven by variations in cortisol at the end of the day. Our “good” emotion regulators had lower levels of evening cortisol (see details in Urry et al., 2006).

We recently extended these findings to patients with a diagnosis of major depressive disorder, all of whom were off medication for a minimum of 1 month, and compared them with an age- and sex-matched group of healthy controls (Johnstone, van Reekum, Urry, Kalin, & Davidson, 2007). In the control participants, we replicated the association we previously found between amygdala and ventromedial PFC activation during cognitive reappraisal to downregulate emotion. In the depressed patients, we found *opposite* relations, indicating that when depressed patients used cognitive strategies to effortfully downregulate negative emotion, they showed an increase in amygdala activation that was positively coupled with an increase in activation in ventromedial PFC. These findings raise the possibility that differences in the regulation of negative affect are one component of a core vulnerability in major depression. Recent evidence implicates genetic variations in serotonergic function in the modulation of amygdala reactivity (Hariri & Holmes, 2006), and some have explicitly suggested that such genetic variation modulates prefrontal–amygdala reactivity and as such affects vulnerability to depression (Pezawas et al., 2005). The voluntary ER paradigm used in these studies can also be easily adapted to study developmental changes and individual differences in ER in

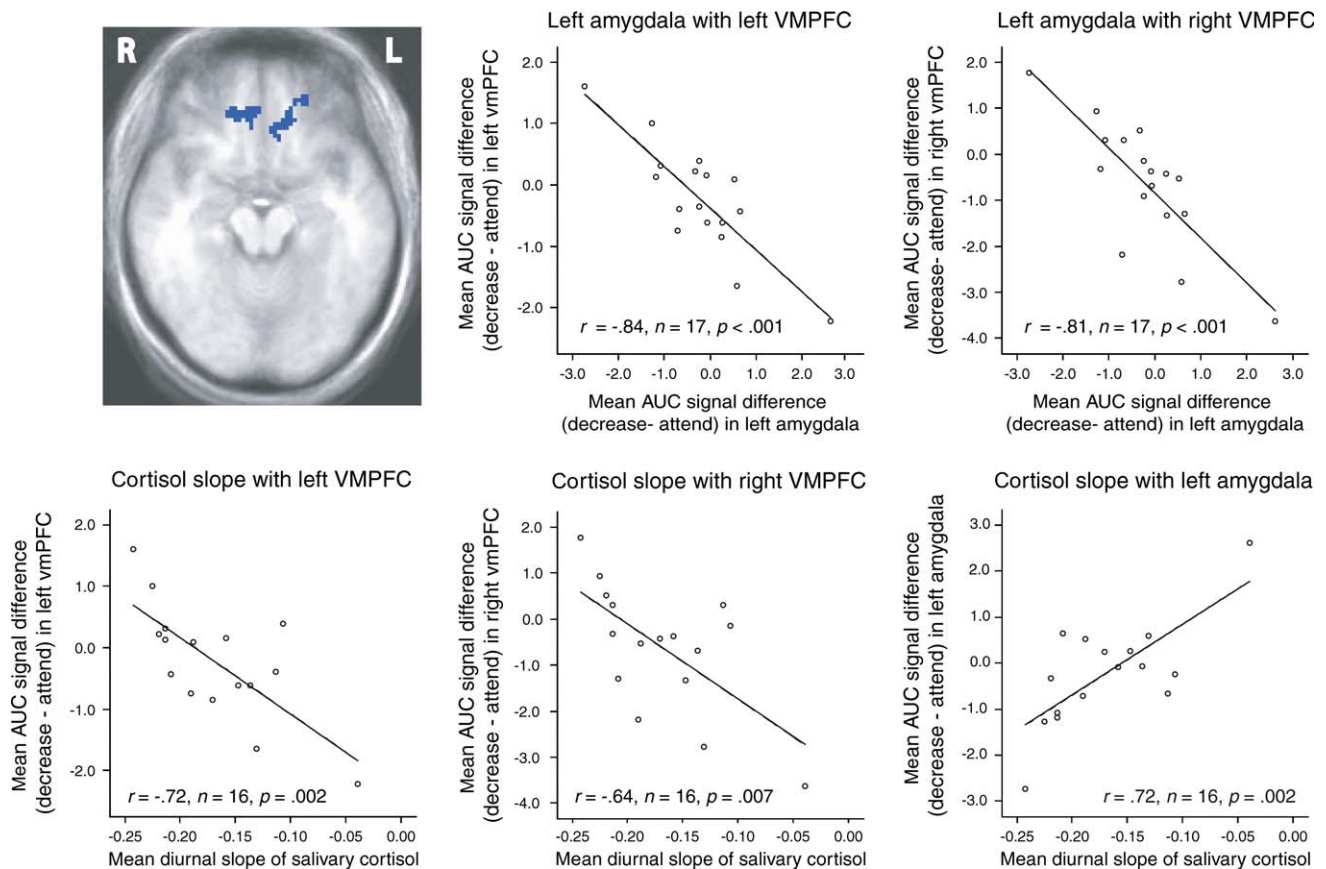


Figure 1. (a) Two regions in left (L) and right (R) VMPFC, maximum: $t(16) = -4.79$ at Talairach coordinates $x = -23$, $y = 43$, $z = -10$, and $t(16) = -5.28$ at $x = 5$, $y = 37$, $z = -12$, respectively, demonstrate an inverse across-subjects correlation with the left amygdala. For illustrative purposes, we extracted mean signal across all voxels for the decrease and attend conditions separately for the two VMPFC regions and for the left amygdala. We then computed zero-order correlations between the amygdala and the VMPFC across subjects and depicted those associations in the accompanying scatter plots. Subjects with lower activation in the amygdala (top middle and right; abscissa) exhibit higher activation in the right and left VMPFC (ordinate). Represented on all axes is the difference in AUC for percent signal change between the decrease and the attend conditions (decrease–attend). (b) We next computed zero-order across-subjects correlations between diurnal cortisol slope (absdssa) and the difference in AUC for percent signal change between the decrease and the attend conditions (decrease–attend) across all voxels in the left and right VMPFC (bottom left and middle) and the amygdala (bottom right; ordinate) clusters. Subjects with higher activation in VMPFC and lower activation in the amygdala exhibit the steepest declines in salivary cortisol over the course of the day.

Source. Urry et al. (2006). AUC = area under the curve; VMPFC = ventromedial prefrontal cortex.

children and adolescents. Moreover, because structural variations in ventromedial PFC predict extinction in humans (Milard et al., 2005) and because ventromedial PFC shows dramatic changes over the course of adolescence (Toga, Thompson, & Sowell, 2006), the study of relations between developmental changes in prefrontal structure and function and ER is ripe for examination. A key to examining developmental change is investigating mechanisms of plasticity.

DEVELOPMENTAL PLASTICITY

Over the course of development, for most children, emotional processes appear to interact seamlessly with the contingency structure of the social environment. However, underlying these complex behaviors are myriad skills—such as decoding and conveying emotional signals with caregivers—that reflect rapid and complex learning. These affective processes become

increasingly intricate as relevant neuroanatomical and neurophysiological systems mature, suggesting that more sophisticated emotional skills might rely solely on the growth of relevant neural substrates. However, the kinds of emotional experiences that the child encounters likely organize affective neural circuitry: For example, the nature of the early parental care that a child receives can have long-term repercussions for behavioral development. The mechanisms underlying the effects of early caregiving remain poorly understood, but data from nonhuman animals have provided insight into potential molecular mechanisms that alter healthy development.

Can We Coherently Translate From Nonhuman Animal to Human Studies?

Insight into the biological influences of parenting has come from studies showing that rodent maternal behavior can affect long-term changes in responses of the offspring to stress;

these changes reflect altered gene expression, so-called environmental programming (Meaney & Szyf, 2005). A very consistent body of evidence for these Early Environment $\times$ Gene interactions involves a neurotransmitter transporter called serotonin transporter (5-HTT) that fine-tunes transmission of serotonin by reuptaking it from the synaptic cleft. The gene comes in two common allelic variants: the long (l) allele and the short (s) allele, which confer higher and lower serotonin reuptake efficiency to the 5HTT, respectively. Animal studies have shown that in stressful conditions, those with two long alleles cope better. Mice with one or two copies of the short allele show more fearful reactions to stresses such as loud sounds (Murphy, Li, & Engel, 2001). In addition, monkeys with the short allele that are raised in stressful conditions have impaired serotonin transmission (Bennett, Lesch, & Heils, 2002).

Social interactions between young organisms and their caregivers appear to have downstream effects on systems such as the hypothalamic–pituitary–adrenal axis, functions associated with the orbital–ventral regions of the PFC (Schrijver, Pallier, Brown, & Wurbel, 2004), and neuropeptide systems that regulate social behavior (Carter, 2005). A critical question concerns how to examine the ontogenesis of these mechanisms in humans. As we noted in the previous section, recent studies with nonhuman animals are leading to new insights about the biological basis of emotions and creating models to motivate biologically informed human studies. However, translation between species is not always straightforward, and we must exercise caution in applying basic findings with nonhuman animals to human children—especially in the domain of emotion (for discussion, see Sanchez & Pollak, *in press*). As an example, it is not apparent how stressful events (such as handling or isolation) in nonhuman animals approximate the kinds of events that human children experience. Variations in the quantity and quality of parental care and stress exposure are operationally different across species. Another critical issue concerns the effects of developmental timing. The strongest effects of early experience on stress neurobiology in the rodent are observed during the first 2 weeks of the pup's life, but the timing and even the existence of a comparable period in human development are unclear. For these reasons, the phenomena of child abuse and neglect have begun to take center stage, both in questions about nature–nurture effects on human development and as a test case for translation between human and nonhuman models of neurobehavioral development. Indeed, behavioral genetic analyses suggest that many of the emotional problems observed in abused children are attributable to environmental effects, with vulnerability to experience modulated by genetic factors (Jaffee, Caspi, Moffitt, & Taylor, 2004; Kaufman et al., 2004).

How Might Emotional Systems Be Changed by Aspects of Social Experiences?

Children are confronted with many opportunities to attach emotional significance to cues in their environments. For this

reason, the central nervous system draws attention to important features in the environment and allows regulation of responses to change (Rueda, Posner, & Rothbart, 2005). Some experiences may heighten the salience of emotional cues and, conversely, the absence of some developmentally appropriate experiences may hinder emotional development because of insufficient learning opportunities. Consistent with this notion, we found that physically abused children, who likely experience high levels of interpersonal violence and hostility, responded differentially to angry, as opposed to other facial expressions of emotion. Children who had experienced various forms of psychological neglect and social isolation were delayed in learning to differentiate facial expressions of emotion (Pollak, Cicchetti, Hornung, & Reed, 2000; Wismer Fries & Pollak, 2004).

To better understand the processes underlying children's performance on simple ER tasks, we have employed paradigms that can capture specific aspects of perceptual processing. In these studies, we found that children who had experienced direct physical maltreatment from their parents had adjusted their sensory thresholds for perception of angry but not other facial expressions (Pollak & Kistler, 2002). We also found that abused children were able to accurately identify facial displays of anger on the basis of less sensory input than their peers (Pollak & Sinha, 2002). That is, their sensory systems appear able to act, accurately, on only partial affective information from the environment. These findings suggest a high degree of automaticity in children's perceptual and attentional processing. Although most extant research on attention and emotion has focused on visual processing and facial expressions, we are finding even more robust effects when abused children are presented with auditory emotional cues, such as angry voices (Shackman & Pollak, 2005). This may reflect the relatively faster development of the human auditory system compared with the visual system. These findings are consistent with nonhuman animal studies that implicate the role of circuitry involving the medial thalamic nucleus, amygdala, and orbitofrontal cortex in assigning emotional significance to stimuli (Kawagoe et al., 2007; Quirk et al., 2006).

If Children's Experiences Learning About Emotional Signals Can Influence the Development of Sensory Systems, How Might Such Plasticity Influence Regulatory Behaviors?

To assess how children come to understand emotional reactions in others, we presented preschoolers with a variety of emotional situations that varied in outcome and equivocality. We tested the association of maltreatment with children's ability to map emotions to their eliciting events by giving the children an emotional outcome and asking them to evaluate the likely causes of such outcomes. In contrast to typically developing preschoolers, abused children interpreted positive, negative, and ambiguous events as being equally plausible causes of anger

(Perlman, Kalish, & Pollak, 2008). This difference in maltreated children's reasoning about emotions suggests a critical role of experience in aiding children's mastery of the structure of interpersonal discourse. We also examined preschool children's autonomic nervous system responses to both benign and hostile interactions between adults. Children listened to a conversation between adult actors that became hostile over time. When anger was introduced into the dialogue, abused children showed and continued to maintain a state of anticipatory monitoring of the environment, even after the angry exchange was resolved. Non-abused controls were better able to regulate their attention and emotional responses once they had assessed that the anger was not personally relevant (Pollak, Vardi, Bechner, & Curtin, 2005). When we tested abused children's regulation of attention, we found that they exhibited increased voluntary attention toward both facial and vocal anger cues, but importantly, that abused children's attention also appeared to be *involuntarily* drawn to anger cues from their own parent. These features of children's ER were related to the manifestation of symptoms of anxiety (Shackman, Shackman, & Pollak, 2007). Taken together, these studies and others (such as Dodge, Pettit, Bates, & Valente, 1995) suggest that rather than using their attentional resources to attenuate emotional reactivity, abused children automatically attend to threatening cues, perhaps at the expense of other contextually relevant information.

In summary, developmental approaches to emotion are enriched by focusing on how individuals learn to respond to the affordances in their environments. When such an approach is combined with recent insights regarding the plasticity of neural systems, we can begin to understand risk and resilience in children. That is, we can elucidate individual differences in how children respond to their early experience. We can also study how psychological interventions can induce neural changes and thus foster recovery (Pollak, 2005). Breakdowns within cortilimbic circuitry could affect a child's learning about emotional cues in many ways. This circuitry influences not only how children respond to emotional signals in the world but how they perceive, interpret, and understand these signals as well. Perceptual and attention systems are relatively plastic and responsive to environmental input early in development. The relative plasticity of these mechanisms may serve an adaptive function: Children are prepared to learn about whatever contingencies are salient in their environments. But plasticity may also confer risk if emotional input or contingencies are aberrant, leading children to over- or underattend to certain emotional signals.

SUMMARY

Genetics, neuroimaging, and learning are complementary neuroscience-based approaches for understanding ER. Each of these approaches is grounded in animal research, although we emphasize human studies here. Common to all these approaches is the idea that a developmental perspective offers greater

understanding of the processes linking observations of overt social behavior with human biology. Such knowledge holds tremendous promise for conceptual advances and improvement of public health.

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