Child Development, March/April 2004, Volume 75, Number 2, Pages 361 – 365

Disambiguating the Components of Emotion Regulation

H. H. Goldsmith and Richard J. Davidson

Affective neuroscience and cognitive science approaches are useful for understanding the components of emotion regulation; several examples from current research are provided. Individual differences in emotion regulation and a focus on the context of emotion experience and expression provide additional tools to study emotion regulation, and its development, from a biobehavioral perspective.

As a point of departure, we provisionally accept the definition of emotion offered in the lead article by Cole, Martin, and Dennis (this issue). We suggest that emotion regulation (ER) can be approached profitably from three complementary perspectives, which are compatible with many of Cole et al.'s points. First, we acknowledge that ER is a difficult topic because it taps into one of the enduring problems in developmental psychology: the conceptualization and measurement of change. Regulation implies change, and in this domain the change is likely to be dynamic and dependent on complex processes. We also note the conceptual and methodological conundrum of separating emotion from ER processes. Emotion processes and ER processes overlap temporally, which presents challenges for studying ER and might even lead some to question whether they are separable. However, we believe that the distinction between emotion and ER, even if it artificially divides processes that lie along a continuum, is heuristically useful and might require developmental psychologists to augment their typical methods. The three perspectives that we wish to highlight are the affective neuroscience approach, the cognitive science perspective, and the interplay of individual differences and context.

Endophenotypes From Affective Neuroscience

Fully distinguishing emotion from ER with behavioral methodology alone might border on the impossible, which is a stronger assertion than Cole et al.'s (this issue) call for multiple, converging measures to predict the organization of ER. We think that the evidence for neural substrates of ER is stronger than Cole et al. mentioned, but the relevant human evidence is mostly recent and from adults. The evidence from rodents and nonhuman primates is more substantial although harder to assimilate to the child development literature.

Investigation of genetic factors is a salient issue when endophenotypes are implicated. Despite assumptions in the literature that the roots of ER lie only in learning, Goldsmith and colleagues have demonstrated that ER is a partially heritable characteristic. Using samples of young twins, Goldsmith, Buss, and Lemery (1997) demonstrated, and later replicated (Goldsmith, Lemery, & Essex, in press), that identical twins were more similar than fraternal twins on parentally reported ER measures. These ER reports have antecedents in earlier temperament and later correlates in symptoms similar to attention deficit hyperactivity disorder (Nigg, Goldsmith, & Sachek, 2004).

Especially in the fear, anxiety, internalizing domain, multiple endophenotypes of emotion can be viewed as intermediate between the levels of the gene and the behavior. These endophenotypes can be viewed as reflecting either the ''regulated'' or the ''regulatory'' aspect of emotion (in the terminology of Cole et al., this issue), and still other endophenotypic measures might reflect the product of reactive and regulatory processes. Some of these endophenotypes can be measured on a second-by-second basis (e.g., electrodermal response, response to startle probes, cardiovascular measures), and others summarize changes occurring over longer intervals (e.g., fMRI measures of neural activation, cortisol reactivity). With the caveat that a modern neuroscience approach to ER is in its early stages (Davidson, Jackson, & Kalin, 2000; Ochsner, Bunge,

H. H. Goldsmith and Richard J. Davidson, Department of Psychology, University of Wisconsin-Madison.

The authors acknowledge support from National Institute of Mental Health to the Wisconsin Center for Affective Science (P50-MH069315; director, Richard Davidson).

Correspondence concerning this article should be addressed to H. H. Goldsmith or Richard J. Davidson, Department of Psychology, 1202 W. Johnson St., University of Wisconsin, Madison, WI 53706. Electronic mail may be sent to hhgoldsm@wisc.edu or rjdavids@wisc.edu.

Gross, & Gabrieli, 2002; Schaefer et al., 2002), we mention some examples of this approach from our own research.

It is first important to distinguish between voluntary and automatic ER (Davidson, Jackson, et al., 2000). Many regulatory processes are presumably invoked automatically as soon as emotion itself is elicited (or even before a punctate emotion is elicited). Other regulatory processes are more voluntary. Davidson and his colleagues have developed experimental paradigms to probe both voluntary and automatic ER. For example, Jackson, Malmstadt, Larson, and Davidson (2000) instructed participants to voluntarily enhance, suppress, or maintain the emotion they were experiencing in response to unpleasant and neutral pictures and found that when participants were voluntarily suppressing their emotion, there was a reliable diminution in the magnitude of eyeblink startle to an acoustic probe delivered after the instruction was presented.

Using a variant of this paradigm in the MRI scanner, Davidson and colleagues demonstrated that reliable changes in amygdala activation occur in response to instructions to voluntarily regulate emotion (Schaefer et al., 2002). Ochsner et al. (2002) also used a variant of this voluntary ER paradigm and replicated the amygdala findings, finding that activation in a ventral prefrontal region varied inversely with activation in the amygdala.

Chronometric Approaches Inspired by Cognitive Science

Investigation of ER within contemporary affective science can benefit from cognitive science methods. For example, chronometric paradigms can distinguish between inhibition and decay in memory processes and language comprehension. Because parallel issues exist for ER (e.g., does fear decrease because it is dampened by regulatory processes or does it simply decay in strength without regulatory processes being invoked?), we need similar chronometric paradigms in the emotion field. If our interests were in the comprehension of emotion, the differential reaction-time measures of cognitive psychology would be applicable for studying processes that inhibit the activation of emotion concepts (Gernsbacher, Goldsmith, & Robertson, 1992). However, developmental psychologists' more central interests lie in the experience and expression of emotion. That is, how do the experience and expression of emotion diminish? Does dampening of, say, the experience of fear always reflect ER processes, or can fear simply decay? Such questions require chronometric approaches, with measures other than reaction time. As Cole et al. (this issue) and others (Gross, 2001) emphasize, the temporal patterning of emotion behaviors, affective endophenotypes, and regulatory behaviors is crucial. Our earlier study (Buss & Goldsmith, 1998), which was reviewed by Cole et al., used a temporal analysis of behavioral contingencies, but we now believe that such analyses should be even more fine-grained and should interrogate the biological processes involved.

Among other salient questions about ER that can be approached from a cognitive science perspective are these:

1. Are ER processes continuous or punctuated?
2. Are ER processes anticipatory or reactive?
3. Do different ER strategies compete?
4. Does ER involve automatic or conscious, voluntary, or strategic processing (as mentioned earlier)?

The general answers to these questions are probably both, both, yes, and both. Thus, the questions are more usefully framed about specific ER processes, such as, ''Is the dampening of negative affect after the introduction of a strange person into the social context automatic or strategic?'' The questions are also more usefully framed from a developmental perspective, such as, ''Can voluntary ER processes become relatively automatic as a child matures and gains experience with the relevant incentive contexts?'' This question about automatic versus strategic inhibitory processes has been approached empirically in cognitive psychology using proportionality manipulations and dual-task procedures with adult participants. The field has not extended such techniques to the affective domain or to children on a large scale.

However, we have begun to approach this issue in studies with adults. For example, Jackson et al. (2003) developed a paradigm to study automatic ER by examining the chronometry of affective reactivity. This was operationalized as the rapidity of recovery of startle magnitude following the offset of a negative emotion elicitor. We felt comfortable classifying the recovery following a negative event as a component of regulation rather than natural decay because studies with animals reveal that lesions to particular territories of prefrontal cortex (PFC) result in a prolongation of negative reactivity in certain paradigms (see Davidson, Jackson, et al., 2000, for review). This implies that there is a descending regulatory signal from PFC to certain limbic structures, particularly the amygdala, that attenuate or regulate responsivity. Jackson et al. found large

individual differences in recovery speed; measures of prefrontal activation asymmetry obtained prior to the startle experiment predicted this recovery speed. Increased left prefrontal activation predicted more rapid recovery of startle magnitude. This effect was demonstrated following the removal of variance associated with startle magnitude during the stimulus itself, thus operationally disentangling the impact of emotion from the presumed regulatory component (in this case, the poststimulus offset recovery). These examples illustrate the potential of chronometric approaches to elucidate the components of ER when coupled with biological measures.

Individual Differences and Context

Individual differences in ER are salient and significant. The typical individual differences questions about ER—concerning its structure or organization, its biological substrates, its stability and consistency, its antecedents in experience, and its adaptive function—all require more investigation, although our knowledge is accumulating at an accelerating pace (see Kopp & Neufeld, 2003, for a developmentally oriented review). We think that these individual differences interact strongly with context to affect behavior.

Cole et al. (this issue) emphasized the need to study contrasting conditions as an aid to inferring ER, and they provided various examples of successful use of that strategy. Of course, contrasting conditions vary the context; thus, we strongly agree with Cole et al. However, we wish to highlight the interplay of individual differences in ER with the situational context of emotion and to do so in ways that incorporate endophenotypes from affective neuroscience. The boundary between typical and disordered, the nature of ER, and the importance of context in determining the significance of affective responding are three highly interrelated issues. Dysfunction in ER can be conceptualized as expression of emotion outside its typical incentive contexts. To the extent that typical incentive contexts can be specified, varying incentives can be a powerful method for studying ER, especially when combined with endophenotypic measures (Kalin & Shelton, 2000). Concretely, this perspective claims that high fear under threatening incentives has a different meaning, and thus has different correlates, than high fear in nonthreatening contexts. The same claim would hold for other emotions and their typical contexts.

We recently demonstrated that parentally reported out-of-context fear in 540 young children was a better predictor of later internalizing symptoms than was the strength of in-context (i.e., temperamental) fearful reactions (Lemery & Goldsmith, 2003). Other recent research from our laboratory concerning the frequencies and correlates of out-of-context fearful reactions is based on second-by-second coding of videotaped reactions of infants (Locke & Goldsmith, 2003). Depending on the exact criteria used, 5% to 15% of infants showed consistent negative affect in two highly standardized contexts that typically elicit pleasure. The patterns of correlates in other behavior for out-of-context anger and out-of-context fear differed, suggesting that ER processes may differ according to discrete affect categories.

An affective neuroscience approach to the study of context is now feasible. Ample evidence suggests that contextual processing depends on specific neural substrates, particularly in the PFC and hippocampus (see Davidson, Pizzagalli, Nitschke, & Kalin, 2003, for review). For example, studies of rodents implicate the hippocampus in contextual fear conditioning. Lesions of the hippocampus abolish contextual fear conditioning while preserving fear cue conditioning. The hippocampus and PFC are the brain regions exhibiting the highest densities of glucocorticoid (GC) receptors (see Davidson et al., 2003, for review). Chronic exposure to stressful life events that results in elevated cortisol may, over time, result in neurotoxicity in the regions with high densities of GC receptors (Sapolsky, 2000). This process would impair functional activity in these regions, including emotion-relevant contextual processing. Future developmental work on this topic should include behavioral measures of context-appropriate and context-inappropriate emotional behavior; measures of hypothalamic-pituitary-adrenocortical (HPA) function, particularly cortisol; and morphometric or functional measures of PFC and hippocampus. Because the PFC in particular is late to mature and does not reach its adult functional status until at least puberty, developmental changes in particular regions of PFC may play an important role in developmental changes in context-dependent emotional responses. In developmental studies in rhesus monkeys, Kalin, Shelton, and Takahashi (1991) found systematic changes in context-sensitive affective reactions to standardized emotion elicitors. Although that study did not simultaneously track changes in PFC function, such studies are now possible using noninvasive imaging methods.

The PFC also plays an important role in modulating activation in the amygdala (Davidson, Putnam, & Larson, 2000). Some aspects of behavioral inhibition may be conceptualized as reflecting difficulty in the

regulation of negative affect and anxiety. The underregulation of negative affect may be reflected in accentuated activation of the amygdala in response to unfamiliar stimuli. In a recent report, Schwartz, Wright, Shin, Kagan, and Rauch (2003) studied a group of individuals in their 20 s who had been classified as behaviorally inhibited or uninhibited when they were toddlers. They found significantly greater activation of the amygdala in response to unfamiliar faces in participants previously classified as inhibited compared with those previously classified as uninhibited. These examples illustrate the potential utility of applying concepts and methods from the emerging area of affective neuroscience to the study of developmental issues in ER.

Critical Considerations to Advance the Study of ER

In concluding, we offer three questions and observations—ones consistent with the suggestions of Cole et al. (this issue)—that developmental researchers who wish to study ER might take into consideration in the design of studies and interpretation of results.

First, can ER accounts be empirically confirmed against plausible alternative accounts for the same behavioral phenomena? For example, an alternative to an ER explanation might be a multiple-activation account, in which different emotion systems with mutually inhibitory influences might be activated in close temporal proximity under the influence of complex incentive stimuli. More generally, hypotheses about ER should be posed so that the presence of ER is not the only plausible outcome of studies. Thoroughly testing such hypotheses is likely to require endophenotypes from affective neuroscience.

Second, and more generally, developmental research on ER must make use of modern concepts from affective and cognitive neuroscience to dissect the subcomponents of emotion and ER in a way that honors the distinctions made by the brain. Theoretical accounts of ER must be consistent with known biological constraints. Combining psychophysiological, neuroendocrine, and neuroimaging methods promises to yield considerable new information on this important topic.

Finally, a truly developmental approach to ER should eventually explain why affective states are apparently so labile during childhood and so resistant to modification during some psychopathological states. This may well be related to developmental changes in neural circuitry underlying ER.

References

Buss, K. A., & Goldsmith, H. H. (1998). Fear and anger regulation in infancy: Effects on the temporal dynamics of affective expression. *Child Development*, *69*, 359–374.

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development*, *75*, 317–333.

Davidson, R. J., Jackson, D. C., & Kalin, N. H. (2000). Emotion, plasticity, context and regulation: Perspectives from affective neuroscience. *Psychological Bulletin*, *126*, 890–906.

Davidson, R. J., Pizzagalli, D., Nitschke, J. B., & Kalin, N. H. (2003). Parsing the subcomponents of emotion and disorders of emotion: Perspectives from affective neuroscience. In R. J. Davidson, K. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 8–24). New York: Oxford University Press.

Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation—A possible prelude to violence. *Science*, *289*, 591–594.

Gernsbacher, M. A., Goldsmith, H. H., & Robertson, R. R. W. (1992). Do readers mentally represent characters' emotional states? *Cognition and Emotion*, *6*, 89–111.

Goldsmith, H. H., Buss, K. A., & Lemery, K. S. (1997). Toddler and childhood temperament: Expanded content, stronger genetic evidence, new evidence for the importance of environment. *Developmental Psychology, 33*, 891–905.

Goldsmith, H. H., Lemery, K. S., & Essex, M. J. (in press). Roles for temperament in the liability to psychopathology in childhood. In L. F. DiLalla (Ed.), *Behavior genetics principles—Perspectives in development, personality, and psychopathology*. Washington, DC: American Psychological Association.

Gross, J. J. (2001). Emotion regulation in adulthood: Timing is everything. *Current Directions in Psychological Science*, *10*, 214–219.

Jackson, D. C., Malmstadt, J. R., Larson, C. L., & Davidson, R. J. (2000). Suppression and enhancement of responses to emotional pictures. *Psychophysiology, 37*, 515–522.

Jackson, D. C., Mueller, C. J., Dolski, I., Dalton, K. M., Nitschke, J. B., & Urry, H. L., et al. (2003). Now you feel it, now you don't: Frontal brain electrical asymmetry and individual differences in emotion regulation. *Psychological Science*, *14*, 612–617.

Kalin, N. H., & Shelton, S. E. (2000). The regulation of defensive behaviors in rhesus monkeys: Implications for understanding anxiety disorders. In R. J. Davidson (Ed.), *Anxiety, depression, and emotion* (pp. 50–68). New York: Oxford University Press.

Kalin, N. H., Shelton, S. E., & Takahashi, L. K. (1991). Defensive behaviors in infant rhesus monkeys: Ontogeny and context-dependent selective expression. *Child Development*, *62*, 1175–1183.

Kopp, C. B., & Neufeld, S. J. (2003). Emotional development during infancy. In R. J. Davidson, K. Scherer, & H.

H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 347–374). New York: Oxford University Press.

Lemery, K. S., & Goldsmith, H. H. (2003). *Predicting internalizing and externalizing symptoms from context-inappropriate affect.* Manuscript in preparation.

Locke, R., & Goldsmith, H. H. (2003). *Context regulation of infant affect.* Manuscript submitted for publication.

Nigg, J. T., Goldsmith, H. H., & Sachek, J. (2004). Temperament and attention-deficit/hyperactivity disorder: Preliminary convergence across child temperament and adult personality data may aid a multi-pathway model. *Journal of Clinical Child and Adolescent Psychology, 33*, 42–53.

Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. E. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience*, *14*, 1215–1229.

Sapolsky, R. M. (2000). Glucocorticoids and hippocampal atrophy in neuropsychiatric disorders. *Archives of General Psychiatry, 57*, 925–935.

Schaefer, S. M., Jackson, D. C., Davidson, R. J., Aguirre, G. K., Kimberg, D. Y., & Thompson-Schill, S. L. (2002). Modulation of amygdalar activity by the conscious regulation of negative emotion. *Journal of Cognitive Neuroscience*, *14*, 913–921.

Schwartz, C. E., Wright, C. I., Shin, L. M., Kagan, J., & Rauch, S. L. (2003). Inhibited and uninhibited infants ''grown up'': Adult amygdalar response to novelty. *Science*, *300*, 1952–1953.