Because the author's main interest is neuropsychological (i.e., mapping of affects and emotions onto the brain), he should not attempt (as he envisages at the end of section 4.4) to abandon explicit psychological definitions and replace them, perhaps in a piecemeal manner, with neurological structures and pathways, even if he uses the metatheory of dynamic systems. A piecemeal way of relating psychological to neurological processes is invalid and detrimental. This common error of directly imputing psychological meaning to discrete parts of the brain organization without passing by a theory of the psychological organism has been called a *mereological fallacy*, because it violates the logical relations of parts to wholes (Bennet & Hacker 2003).

What is needed is a neuropsychological substantive theory: an *organismic* (i.e., general, causal, and interpretable in the brain) *theory* defined at the macro-level of performance, which can facilitate process and task analysis. The author unwittingly is reinforcing the tendency of neuroscientists to work only with fragmented (i.e., regional, not organismic) theories, such as discrete theories of emotional appraisal, working memory, declarative memory, perception, learning, and so on. This is problematic because the brain works as an integrated totality constituted by subsystems that dynamically interact in complex ways.

ACKNOWLEDGMENTS

Preparation of this commentary was supported by a grant from the Social Sciences and Humanities Research Council of Canada. I thank Dr. Janice Johnson for her advice.

The role of frontocingulate pathways in the emotion-cognition interface: Emerging clues from depression

Diego A. Pizzagalli

Department of Psychology, Harvard University, Cambridge, MA 02138.
dap@wjh.harvard.edu **http://www.wjh.harvard.edu/~daplab/**

Abstract: By emphasizing nonlinear dynamics between appraisal and emotions, Lewis's model provides a valuable platform for integrating psychological and neural perspectives on the emotion-cognition interface. In this commentary, I discuss the role of neuroscience in shaping new conceptualizations of emotion and the putative role of theta oscillation within frontocingulate pathways in depression, a syndrome in which emotion-cognition relations are dysfunctional.

In the target article, Lewis provides a wide-ranging and timely theoretical formulation of emotion-cognition relations. By emphasizing (a) bidirectional interactions between appraisal and emotion; (b) lower-order psychological and neural constituents underlying the emergence of emotion–appraisal processes; and (c) large-scale functional coupling through oscillatory neurophysiological mechanisms, Lewis offers a multilevel account of appraisal-emotion interactions, fostering a better integration of emotion theory and neurobiology.

In this commentary, I elaborate on two important points raised in the target article. First, I emphasize how a brain-based approach to emotion and appraisal can uniquely inform and constrain theoretical models of these complex constructs. Second, I comment on Lewis's assertion that "phase synchrony in the theta range may underpin the functional integration of systems mediating appraisal–emotion processes" (sect. 5.4). To this end, I review recent event-related potential (ERP) findings of action monitoring (Luu et al. 2004) and electroencephalographic (EEG) findings highlighting disrupted functional connectivity within frontocingulate pathways in depression (Pizzagalli et al. 2003a).

With respect to brain-based approaches to emotion and appraisal, Lewis discusses definitional problems that have hindered the development of comprehensive theories of emotion. Here, I would like to emphasize two points. First, as Lewis argues, definitions of "appraisal" and "emotion" often overlap substantially, causing formidable conundrums to theoretical approaches based on the assumption that these two constructs have distinct functions and are governed by simple, linear, and unidirectional causal processes (e.g., appraisal as a temporal and causal antecedent of emotion; Roseman & Smith 2001). Second, and more important, the definitional overlap between emotion and appraisal mirrors substantial anatomical and functional overlap among brain regions subserving affective and cognitive processes (see Davidson 2003b, for an extended discussion). That is, many brain regions subserving appraisal processes also participate in emotional functions, and vice versa. This evidence forcefully contradicts assertions that affect and cognition are subserved by separate and independent neural circuits, and speaks against the notion that affect and appraisal are subcortically and cortically mediated, respectively (e.g., Panksepp 2003). As suggested by Lewis and others (e.g., Davidson 2003b; Pizzagalli et al. 2003b), emotion is not a monolithic process but comprises different subcomponents encompassing a distributed network of cortical and subcortical systems. Acknowledging empirical data consistent with this assertion (Phan et al. 2002) has important theoretical consequences, because, as appropriately stated by Lewis, "brain function prohibits any real independence between appraisal and emotion" (sect. 5). In sum, although Lewis's overview of neural substrates underlying appraisal and emotional processes is neither comprehensive nor new, a reconceptualization of these substrates in terms of dynamic systems is indeed useful for stressing that the brain's anatomy places important constraints upon psychological theories of emotion and its relations to cognition. Emerging brain-based approaches to the study of depression have similarly underscored not only the synergy between emotional and appraisal processes, but also the utility of a neurobiological framework to parsing the clinical heterogeneity of the disorder (Davidson et al. 2002; Pizzagalli et al. 2004).

My second set of comments pertains to the hypothesis that phase synchrony in the theta range may play a critical role in the functional integration of appraisal–emotion processes. Specifically, Lewis predicts that theta synchronization across the amygdala, hippocampus, anterior cingulate (ACC), orbitofrontal (OFC), and prefrontal (PFC) cortices may "underpin the functional integration of systems mediating appraisal–emotion processes" (sect. 5.4). In humans, empirical evidence for this hypothesis is very limited, but recent findings provide promising support. First, a recent ERP study has shown that the error-related negativity (ERN) – an ERP peak occurring 50–100 msec after the commission of an error – was largely explained by transient phase-locking of midline theta activity to the error responses within distinct frontocingulate regions (Luu et al. 2004). This finding replicated and extended a prior report that error monitoring and evaluative feedback engaged dorsal and rostral ACC sources oscillating within the theta range (Luu et al. 2003). As Luu et al. (2003) proposed, these findings indicate that action regulation mediated by the ACC is associated with entrainment of frontocingulate pathways, consistent with the general framework of Lewis's model.

A second, albeit more indirect, line of evidence suggesting that large-scale corticolimbic synchronization is crucially involved in the emergence of emotion-appraisal processes can be derived from recent findings in major depression, a clinical condition in which coordination of these states is dysfunctional (Mineka et al. 2003). In a recent study, Pizzagalli et al. (2003a) found that baseline theta activity within ACC and PFC/OFC regions was functionally coupled for control, but not depressed, subjects. In healthy controls, this functional connectivity within frontocingulate pathways is in line with anatomical data suggesting that the ACC has reciprocal connections with the dorsolateral PFC and OFC (Barbas 1992; Petrides & Pandya 1999). Disrupted functional connectivity within frontocingulate networks in depression is intriguing, particularly in light of evidence reviewed in the target article and elsewhere (Bush et al. 2000) indicating that the ACC is critically implicated in monitoring conflicting response de-

mands, detecting errors, and evaluating the emotional significance of events, and may thus be a site of convergence and integration between affective and cognitive processes. The fact that functional connectivity within frontocingulate pathways emerged for the theta band (6.5–8 Hz) is consistent with the hypothesis that theta may serve a gating function for the information processing flow in corticolimbic limbic regions (Vinogradova 1995; Luu et al. 2003; 2004), thereby providing the necessary neurophysiological substrates for the emergence of adaptive emotion-appraisal processes, as Lewis discusses.

In sum, using a theoretical framework inspired by emerging neurobiological concepts and findings, Lewis proposes a reconceptualization of emotion-cognition relations that emphasizes nonlinear interactions between their psychological and neural constituents, ultimately giving rise to a unitary phenomenon. Large-scale corticolimbic theta synchronization is proposed as a putative neurophysiological substrate giving rise to a coordinated integration of emotion and cognition. Because the strength of any theoretical account lies mainly in its predictive validity, empirical work is now needed to test hypotheses derivable from this model, including its extension to psychopathology.

ACKNOWLEDGMENT

Preparation of this article was supported by NIH Research Grant R01MH68376 funded by the National Institute of Mental Health.

Characteristics of anger: Notes for a systems theory of emotion

Michael Potegal

Department of Pediatrics, MMC 486, University of Minnesota Medical School, Minneapolis, MN 55455. **poteg001@umn.edu**

Abstract: Although emotion may subserve social function, as with anger-maintaining dominance, emotions are more than variant cognitions. Anger promotes risk-taking, attention-narrowing, and cognitive impairment. The proposition that appraised "blameworthiness" is necessary for anger excludes young children's anger as well as adults' pain-induced anger. To be complete, any systems model of anger must account for its temporal characteristics, including escalation and persistence.

Lewis's ambitious and thought-provoking overview interweaves the psychology and affective neuroscience of emotion. This commentary advances the discourse by focusing specifically on the emotion of anger.

1. Emotion is not cognition. Emotional processes are not just another cognitive problem-solving option. The term "emotion" stems from the same medieval French root as "motion" and connotes the experience of movement; emotion can move someone to incur risk that would not otherwise be tolerated and to ignore pain that might not otherwise be endured. Although anger can function to maintain social dominance, this is not the same as, for example, a coolly plotted political strategy. Anger provides the motivation for the "commitment to aggression" (in Bronstein's [1981] felicitous phrase), that is, for the ability to sustain the costs, but it does so at the price of reducing self-control, restricting attention, and degrading cognition (cf. Zillman 1994). Cross-culturally, men see anger as a way to seize control of a situation whereas women experience anger as a loss of control (e.g., Astin et al. 2003; Campbell & Muncer 1994; Ramirez et al. 2001). The danger in viewing emotion as just another cognitive process lies not in the potential unemployment of some emotion theorists, but in obscuring emotion's special nature.

2. Appraising appraisal. Lewis's account of appraisal in generating Mr. Smart's road rage is so persuasive that it might convince Mr. Smart himself. However, such accounts may be "just so" afterthoughts. Some evidence suggests that anger can arise first and the angry individual then looks for someone or something to blame (Keltner et al. 1993; Quigley & Tedeschi 1996). The proposition that true anger occurs only in response to a provocation that has been appraised as "blameworthy" (Ortony et al. 1988) can be challenged through reductio ad absurdum because it would exclude anger that, for example, arises from acute or chronic pain (e.g., Bruehl et al. 2002; Gelkopf 1997).

The claim that attribution of blame is a necessary aspect of anger is particularly troublesome in throwing out the angry baby with the bathwater. The expression (and presumably experience) of anger begins in the first year of life. Mothers perceive "hard" or "forceful" cries, red face, arching and undirected kicking as indicating anger in infants by 3 months of age (Klinnert et al. 1984). Similarly, naïve judges reliably identify infants' anger expressions in the absence of contextual information (Stenberg & Campos 1990; cf. Oster et al. 1992). There is general agreement that facial expressions of anger are distinguishable from more generalized distress between 4 and 6 months of age (e.g., Stenberg et.al.1983). Izard and Malatesta's (1987) claim that anger can be distinguished as early as age 2 to 3 months is supported by observations that infants as young as 2 months who learned to pull a lever for pleasant stimulation significantly increased their angry facial expressions in the extinction phase of the task (Lewis et al. 1990).

3. Autonomic activation and subjective experience in anger. Autonomic activation also differentiates primary emotions from cognitive processes. Anger is associated with rises in heart rate and diastolic blood pressure (the latter distinguishes anger from fear; e.g., Levenson 1992). Earlier claims of anger also being signaled by a rise in finger temperature have not been consistently replicated (e.g., Sinha & Parsons 1996), but more recent evidence suggests a strong association with increased forehead temperature (Drummond & Quah 2001; Stemmler et al. 2001). This association is entirely consistent with the recognition, dating to antiquity, that facial flushing can signal anger (Potegal 2000). Many people experience anger as rising heat, often in the face, which may help explain the consistent reference to a hot liquid under pressure as a metaphor for the subjective experience of anger (Lakoff & Kovecses 1987). Autonomic activation also actively augments the experience of anger and increases the probability of aggression (Zillman 1994). Because hypothalamically controlled autonomic activation is so integral a part of emotion, the hypothalamus should be included in the motivated action loop of the target article's Figure 3.

4. Anger intensity and time course: Escalation and persistence. The anger induced by sudden pain can be almost reflexively rapid. In the domain of social provocation, conflicts between strangers may escalate slowly, but anger between parties known to each other flares quickly (Cairns et al. 1994). Anger's rapid rise is just one aspect of its general tendency to escalate. Even when provocation remains at the same level, anger frequently escalates (e.g., Pruitt et al. 1997). Moreover, once anger has been provoked, it often persists for some time after the provocation has stopped (consult any parent who has unsuccessfully tried to mollify a child throwing a tantrum by offering him whatever it was he initially craved). The term "aggressive arousal" (AA) denotes provocation-induced, centrally mediated increases in attack probability in other animals (Potegal 1994). AA can be induced quickly (e.g., by briefly presenting a same-sex conspecific) and persists well beyond the withdrawal of the provoking stimulus. Like anger, AA has a cost in a maladaptive reduction in anti-predator vigilance. AA may be the anlagen of the action tendency associated with anger in humans.

Any thorough model of emotion must account for time course. Temporal persistence is a motif of amygdala function, even at the neuronal level (Potegal et al. 1996). However, the rapid rise and slower fall of anger may be shaped by processes beyond the usual neuronal interactions; for example, yet-to-be-investigated forms of potentiation may underlie the escalation and persistence of AA (Potegal et al. 1996). The amygdala regulates and prolongs motivated behavior through the hypothalamus, which controls not only