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.

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Characteristics of anger: Notes for a systems theory of emotion

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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

autonomic concomitants of aggression, but some of its motor patterns and motivational aspects in humans, as well (e.g., Weissenberger et al. 2001). These are additional reasons for including the hypothalamus in the motivated action loop of Figure 3 in the target article. According to Lewis, temporal characteristics might also arise from the "self-amplifying" positive feedback among amygdala, anterior temporal, and orbitofrontal cortices. If so, the reciprocal inhibition between amygdala and dorsolateral frontal cortex (Drevets & Raichle 1998) may explain the decline in dorsolateral frontal cortex-mediated cognition during high levels of anger. To explore these ideas, a reliable, moment-to-moment measure of anger intensity is required (cf. sect. 2.2 of the target article).

5. Quantifying anger. Although the intensity of angry facial expressions can be estimated reliably (Hess et al. 1997), their dynamic range is unknown and they are methodologically difficult to capture. Even here in the 21st century, psychologists still estimate anger from subjective self-reports (e.g., Hoeksma et al. 2004). Peihua Qiu and I have been able to model the overall trajectory of anger based on the time courses of the individual angry behaviors objectively observed in tantrums (Potegal & Davidson 2003). The single latent variable, Momentary Anger, which drives all the individual angry behaviors, would be a suitable output variable in a dynamic systems model (Qiu et al., submitted).

Amalgams and the power of analytical chemistry: Affective science needs to decompose the appraisal-emotion interaction

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Abstract: The issues addressed in this commentary include: (1) the appropriate conceptualization of "appraisal"; (2) the nature and unfolding of emotional episodes over time; (3) the interrelationships between the dynamic elements of the appraisal process and their effects on other emotion components, as well as repercussions on ongoing appraisal in a recursive process; and (4) the use of brain research to constrain and inform models of emotion.

In this BBS target article, an admirable tour de force of scholarship, Lewis presents a formal model of appraisal-emotion relationships and reviews relevant evidence from neurobiology. We found many points in this article with which we agree wholeheartedly, but there are a few major issues on which we beg to disagree. For example, we feel that Lewis unduly equates the psychology of emotion with narrow conceptions of appraisal theory published more than a decade ago and fails to recognize the contribution of cognitive neuroscience to emotion theory (see Davidson et al. 2003; Kosslyn & Koenig 1995; Lane & Nadel 2000; Scherer 1993a; Scherer & Peper 2001). Although Lewis acknowledges that several emotion theorists have proposed appraisal-emotion interactions based on nonlinear dynamics and bidirectional causality, he suspects that the protagonists treat this as "an interesting diversion from more classical modeling" (sect. 2.2 of the target article). It is true that attempts to describe emotions as episodes of subsystem synchronization driven by nonlinear appraisal processes (Scherer 2000), and to specify hysteresis functions in integration models (Scherer 2004), have not progressed beyond a preliminary stage of modeling. Unfortunately, much of nonlinear dynamics theorizing, including the current target article, does not lend itself readily to designing appraisal experiments and analyzing multimodal data. Here we focus on four major issues:

1. The conceptualization of the appraisal process. Google finds 6,700,000 entries for the word "appraisal." Undoubtedly, Lewis's components of appraisal (perception, attention, evaluation, and reflection; see his Fig. 1) are involved in many of these instances. In contrast, appraisal theorists use the term in a more restricted fashion, specifying the *criteria or dimensions* which are constitutive for emotion elicitation and differentiation through event appraisal. These essential elements of appraisal theory are lacking from Lewis's account and readers unfamiliar with the appraisal literature are unlikely to fully comprehend what the discussion is all about. Evidently, the appraisal of these criteria involves *cognitive structures and mechanisms* such as attention, memory, problem solving, and self-representation (Scherer 2001), including multiple levels of processing (Leventhal & Scherer 1987). Appraisal theorists will need to pay greater attention to these cognitive mechanisms – in particular to the executive functions (see Fig. 5.3 in Scherer 2001) – but Lewis's rather general discussion of such "appraisal components" as "evaluation" adds little to our understanding.

2. The definition of emotion. Lewis adopts the componential view of emotion as advocated by appraisal theorists (Frijda 1986; Scherer 1984). However, the components he identifies in his "skeletal model" in Figure 1 and in the text – such as, "arousal," "feeling tone," or "attentional orientation" – are hardly consensual as representative emotion components. The component of *motor expression* is conspicuously absent. The most serious problem of Lewis's account is the lack of a specification on when an emotion begins and when it ends, as well as of the difference between an emotion episode and the non-emotional background of an individual's experience. Lewis (at the end of sect. 2.3) claims that "a process account should demonstrate how constituent processes give rise to a whole appraisal in the first place," and suggests that such an account is presented in his Figure 1. We have trouble understanding how his Figure 1 explains the unfolding of an emotional episode. If appraisal-emotion relationships are to be explored with respect to their circular causality, there must be a way of delimiting the respective episodes in order to avoid the rather unsatisfactory statement that everything interacts with everything else all the time. One solution is Scherer's (1984; 2000; 2001) suggestion to define the onset of an emotion episode as a certain degree of synchronization of emotion components driven by specific appraisal outcomes.

3. The nature of the appraisal-emotion relationship. Appraisal theorists have never denied that motivation and affect have a strong influence on appraisal. Most theories explicitly integrate the motivational state of the individual as one of the major determinants of appraisal outcomes. Obviously this includes emotion components such as action tendencies that have been produced by prior appraisal. A process-oriented account (see Scherer 2000; 2001), assuming constantly changing appraisal due to new information, would seem to cover bidirectional causality over time. Lewis's "skeletal model," lacking concrete mechanisms and predictions, does not provide a viable alternative to existing models. His terminology, with vague concepts such as appraisal-emotion "amalgam" or "whole," and the absence of suggestions for operationalization or experimental designs for empirical study, raises concerns about the epistemological status of the proposal. One senses an underlying reticence to engage in analytical procedures designed to take the amalgam apart in order to understand its nature. Yet, we need to decompose the appraisal-emotion interaction to understand its nature (just as we require analytical chemistry to study metal amalgams). As an alternative model of the dynamic elements of the appraisal process and their effects on other emotion components, as well as repercussions on ongoing appraisal in a recursive process, we suggest the Component Process Model proposed by Scherer (1984; 2000; 2001; 2004). Our Figure 1 presents a combination of Figures 5.1 and 5.2 in Scherer (2001). We feel that this model is sufficiently well specified to allow posing concrete questions about bidirectional appraisal-emotion interactions.

Contrary to Lewis's model, this model allows a detailed consideration of the effects of emotional processes on attention, memory, and other cognitive processes. In particular, it suggests a distinction between (i) an effect of particular *appraisal criteria* on

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