damental thesis, which breaks from centuries of academic tradition, taken seriously.

In our neurovisceral integration model we too have proposed dynamical systems as a unifying framework in which the boundaries between emotion and cognition are brought down (Thayer & Lane 2000). Lewis certainly has incorporated many aspects of our model into his work. Thus, our models share many similarities including the integration of emotion theory with neurobiology, and the use of a dynamical systems framework. However, there are some important differences as well. One important difference is our emphasis on the role of inhibitory processes. Whereas we share the idea that emotions may be viewed as attractors or points of stability in an emotional state-space, we argue that inhibitory neural processes are critical for the phase transitions that allow a system to move adaptively from one attractor or emotion to another in the state-space. In fact, we would propose that inhibitory processes are crucial for all of the phases that Lewis states make up an emotional interpretation. As noted above, inhibitory processes are associated with phase transitions and are therefore involved in Lewis's trigger phase. We have noted previously that what Lewis calls the self-amplification phase is a result of disinhibition, that is, a release or sensitization of excitatory processes as a result of decreased inhibition. Lewis clearly notes the importance of inhibition for his self-stabilization phase and we have noted elsewhere the importance of inhibition for learning (Thayer & Friedman 2002). Therefore, to complete the connection between emotion theory and neurobiology we feel that an understanding of the role of inhibitory processes is essential. Inhibitory processes provide for the sculpting of neural action at all levels of the neuraxis. The features that make inhibitory processes critical have been progressively explored in neurobiology.

Constantinidis et al. (2002) have recently detailed the role of inhibition in the temporal flow of information in the prefrontal cortex. Using simultaneous single cell recordings in monkeys, they demonstrated inhibitory interactions between neurons active at different time points during the course of a complex working memory task. They noted that the influence of inhibition was particularly evident at transition points in the action sequence, thus supporting the idea that inhibitory neurons are critical for behavioral state changes. Similarly, it has recently been demonstrated in humans that enhancement of GABA-related inhibition may be a very efficient mechanism for synchronizing larger neuronal populations (Fingelkurts et al. 2004). These findings and others (Waldvogel et al. 2000) suggest that a little inhibition at the right time can have a large influence on the behavior of the organism, highlighting the nonlinear nature of the inhibitory control.

At the psychological level, we have also argued for the importance of inhibitory processes. We have noted that perseverative behavior, including worry and rumination, may represent the breakdown of inhibitory processes (Thayer & Lane 2002). Again, neurobiology supports such an idea. For example, in a murine model of anxiety, decreased GABAA-receptor clustering was associated with harm-avoidance behavior and an explicit memory bias for threat cues (Crestani et al. 1999). Mice with reduced GABAA-receptor clustering showed enhanced reactivity to threat stimuli (an effect that was reversed by diazepam), a facilitation of trace conditioning in a fear conditioning paradigm, and a deficit in ambiguous cue discrimination. These findings are remarkably similar to the HR acceleration to and explicit memory bias for threat words, and failure to habituate to neutral words, found in generalized anxiety disorder patients in a conditioning paradigm (Friedman et al. 2000; Thayer et al. 2000).

It should also be noted that whereas GABA is usually an inhibitory neurotransmitter and Lewis states that "GABA is always inhibitory" (sect. 5.2, para. 2), GABA like many neurotransmitters is functionally complex and hence can have excitatory actions (Köhling 2002). Therefore, recognition of the complexity of the neurobiology is also needed and is in fact called for in dynamical systems models.

Taken together, however, it appears that an understanding of the role of inhibition is critical if one is to fully integrate emotion theory, or behavior in general, with neurobiology. In the end we feel that Lewis has made an important contribution by outlining this general framework. It will definitely serve as a catalyst for additional theoretical and empirical work.

Mechanisms of the occasional self

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Abstract: Considered in relation to the component brain systems of appraisal-emotion interactions, dynamical systems theory blurs the divisions that seem obvious in a psychological analysis, such as between arousal, emotion, and appraisal. At the same time, the component brain mechanisms can themselves be seen to be incomplete as units of analysis, making sense only in the context of the whole organism.

In a time when powerful new methodologies are applied to studying human brain activity, the growing evidence base calls for more complex theoretical models. It is time to begin training theoreticians, generalists who forego methodological or empirical specialization to acquire the scholarship, intellectual discipline, and conceptual flexibility necessary for understanding both psychological and neural mechanisms. In this target article, Lewis explores the form that a comprehensive theoretical analysis might take when it is applied to cognition-emotion interactions in the brain.

Perhaps the major point of the article is that the evidence points to complexity in causal relations among the psychological functions of emotion and cognition, and a corresponding complexity in the causal relations among the brain mechanisms underlying those functions. Dynamic systems theory provides metaphors for complex cybernetics, including positive and negative feedback, self-stabilization, and emergent properties. Perhaps more important is that, through Haken's (1977) insights, this line of reasoning shows that the causality in part-whole relations is not always best understood through reductionism, toward mechanistic parts from superordinate wholes. Rather, the functional role that a mechanism plays within an integrated system becomes the embedding context that is also a kind of explanation. Certainly there are proximal causes that can only be understood as originating from the body's physico-chemical substrate. Yet, in a systems explanation, this functional role of a mechanism's operation is as important an explanation as the more elementary physiological and physico-chemical processes from which it emerges. In the psychological analysis of appraisal and emotion, Lewis provides important examples of the causal complexity that makes one-sided accounts (emphasizing linear cognitive or emotive causality) unsatisfying.

In the application to neural systems, the theoretical analysis faces a more daunting challenge. The brain systems currently understood to be integral to motivation, emotion, and cognition are not only complex but multiple. With patient scholarship, Lewis surveys the relevant landscape of brainstem, diencephalic, striatal, and corticolimbic circuits, and even here the review is illustrative rather than comprehensive. Nonetheless, it soon becomes apparent that, in every circuit or system surveyed, we find no separation, causal or otherwise, between emotional and motivational functions and cognitive functions. Apparently, psychological function and physiological function are not aligned in any simple harmony, at least not in the way we approach them in psychological theory. The conclusion, then, must be unsettling for psychologists. Whereas the separation of emotion and cognition seems to be obvious to a functional analysis, the complexity of interactions among multiple systems, for arousal, for specific action tendencies, or for more general attentional and memory biases, leads to great difficulty in saying what is cognition and how it differs from emotion. Is this what we expect from a theoretical analysis of complexity,

that we begin to lose the meaning of the functional questions that seemed so clear in the beginning?

Maybe it is. Maybe it could even become a necessary step toward sophistication in neuropsychological theory.

More than the loss of familiar functional distinctions, neurophysiology shows us the scope of constituent mechanisms. Lewis's review of neural circuits and processes leads us to confront a scope of phenomena – arousal, drives, memory organization, attentional control – that is much broader than the mental functions that were considered relevant in psychological appraisal theory. Even in his selective illustration of the brain's control systems, each system seems to cross multiple functional levels, leading to the remarkable conclusion that functions such as motives or emotions that we would isolate so clearly in a psychological analysis turn out to be embedded within a larger neurophysiological landscape.

What if we take this embeddedness of mechanisms back to the psychological theory? We would have to conclude that our isolation of emotions as separable functions, or of cognitions as distinct causal entities, may be psychological fictions – fictions that may be useful for academic psychological theory, but are of limited use for a neuropsychological theory that attempts to span both brain and mind of actual people. Rather, we need to fit any mechanism within the appropriate part-whole relations, where the organism-in-environment is the context, the whole that explains the mechanisms. Neither cognitions nor emotions are discrete causal agents that can be separated from the whole of the biological context. This context is formed both by the immediate physiological exigencies, such as environmental threats or visceral need states, and by the enduring residuals of the person's developmental history. In neural terms, the whole of the organism's cognitive-emotive matrix is achieved by vertical integration of multiple systems of the neuraxis. In psychological terms, the embedding whole represents the superordinate construct of the personality, the self.

On the other hand, when we instantiate an organismic construct, like the self, within neurophysiological terms, this construct becomes more tentative than when expressed only in psychological terms. Both cognitive and emotional components of the self are dependent upon their constituent physico-chemical substrates. As a result, the self cannot be assumed as an organizing principle for all mental or neural processes. Rather, it forms a context for only those processes that operate when the constituent self mechanisms are activated. Again, the discipline of thinking in both psychological and neurophysiological terms raises new challenges for the theorist. Not only does it complicate familiar functional distinctions, but it makes clear that dynamical psychophysiological systems are indeed dynamic, such that the embedding context of the ongoing self is an occasional state, emerging only to the extent that the constituent mechanisms are recreated in the continual flux of psychophysiological processes.

Dynamic brain systems in quest for emotional homeostasis

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Abstract: Lewis proposes a solution for bridging the gap between cognitive-psychological and neurobiological theories of emotion in terms of dynamic systems modeling. However, an important brain network is absent in his account: the neuroendocrine system. In this commentary, the dynamic features of the cross-talk between the hypothalamic-pituitary-adrenal (HPA) and gonadal (HPG) axes are discussed within a triple-balance model of emotion.

Lewis's dynamic systems approach on the interaction between brain, emotion, and cognition provides a timely contribution to heuristic reasoning in the field of affective neuroscience. However, his notion that psychologists and biologists cannot communicate on the issue of emotion misses ground. Admittedly, theories are still in their infancy but the first steps towards psychobiological theories of emotion have been set (e.g., Damasio 1998; Davidson 2003a; Panksepp 1998a).

This commentary mainly concentrates on a pivotal emotional network underexposed in Lewis's framework: the endocrine system. Attention is given in particular to the dynamic cross-talk between the hypothalamic-pituitary-adrenal (HPA) and the hypothalamic-pituitary-gonadal (HPG) axes (Viau 2002) and the antagonistic effects of their end-products, cortisol and testosterone, on motivation and emotion (e.g., Van Honk et al. 2003; 2004). Our discussion is framed in a triple balance model (TBM) of emotion, a heuristic which suggests that reverberating neurodynamic affective maps, created on different anatomical levels of the brain, depend in their continuous quest for emotional homeostasis on the fine-tuned action of the steroids cortisol and testosterone (Van Honk & Schutter, in press).

Emotional homeostasis is crucial for survival and a prerequisite for balanced reactions to reward and punishment (Ressler 2004). This homeostasis depends on (1) *Subcortical balance*: The primordial responses of reward and punishment are approach or withdrawal, and in simple animals they are classically illustrated by fight or flight, which is initiated in subcortical affective circuits and controlled by endocrine-autonomic nervous system interactions (Decatanzaro 1999). Millions of years of evolution have sculptured these primordial flight or fight machines into primates with highly complex social emotional brains. (2) *Cortical balance*: In humans, approach and withdrawal provided the rudimentary building blocks for the development of the emotions anger and anxiety. These occur in the behavioral hiatus when actions are delayed and provide for more flexible behavioral tendencies in which the neocortex is heavily implicated. In particular, the left and right prefrontal cortices are subsequently involved in these sophisticated forms of behavioral approach and withdrawal (Davidson 2003a). (3) *Subcortical-cortical balance*: Finally, to secure complete homeostatic emotion regulation, this layered subcortical-cortical system necessarily needed integration, therefore the expansion of the neocortex was accompanied by the emergence of one of evolution's finest yet most vulnerable adaptations, a loosely-coupled brain communication pathway (MacLean 1990). This TBM of emotion is an evolutionary inspired psychobiological heuristic that not only aims to scrutinize the neurobiological mechanisms behind adaptive homeostasis in human social-emotional functioning, but also sets out to predict the maladaptive, pathological consequences of particular imbalances in emotion (Van Honk & Schutter, in press). A crucial hypothesis in the model is that the end-products of the HPA and the HPG axes, the steroid hormones cortisol and testosterone, are pivotally involved in homeostatic emotion regulation through their *antagonistic* action on the balance between the sensitivity for punishment and reward.

This antagonism begins with the mutually inhibitory functional connection between the HPA and HPG axes (Viau 2002). Cortisol suppresses the activity of the HPG axis at all its levels, diminishes the production of testosterone, and inhibits the action of testosterone at the target tissues (Johnson et al, 1992). Testosterone in turn inhibits the stress-induced activation of the HPA axis at the level of both the hypothalamus and the pituitary gland (Viau 2002). The same steroids are also suggested to act by binding to amygdaloid-centered steroid-responsive neuronal networks (Wood 1996) where they regulate and facilitate neuropeptide gene-expression, which changes the likelihood of approach (testosterone) or withdrawal (cortisol) when confronted with particular emotional stimuli (Schulkin 2003).

The antagonistic involvement of cortisol and testosterone in the sensitivity for punishment and reward can be traced on the three balances of our psychobiological model of emotion. (1) *Subcortically*, animal evidence demonstrates that at the amygdala, cortisol-