

# Neural Substrates of Implicit and Explicit Emotional Processes: A Unifying Framework for Psychosomatic Medicine

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There are two broad themes in psychosomatic medicine research that relate emotions to physical disease outcomes. Theme 1 holds that self-reported negative affect has deleterious effects and self-reported positive affect has salubrious effects on health. Theme 2 holds that interference with the experience or expression of negative affect has adverse health consequences. From the perspective of self-report these two traditions appear contradictory. A key thesis of this paper is that the foundational distinction in cognitive neuroscience between explicit (conscious) and implicit (unconscious) processes, corresponding to Themes 1 and 2, respectively, provides a unifying framework that makes empirical research on unconscious emotional processes more tractable.

A psychological model called “levels of emotional awareness” is presented first that places implicit and explicit emotional processes on a cognitive-developmental continuum. This model holds that the ability to become consciously aware of one’s own feelings is a cognitive skill that goes through a developmental process similar to that which Piaget described for other cognitive functions. Empirical findings using the Levels of Emotional Awareness Scale are presented. A parallel hierarchical model of the neural substrates of emotional awareness is presented next supported by recent neuroimaging and lesion work. The evidence presented in this review suggests that the neural substrates of implicit and explicit emotional processes are distinct, that the latter have a modulatory effect on the former, and that at the neural level Theme 1 and Theme 2 phenomena share critical similarities. The implications of this psychobiological model for research in psychosomatic medicine are discussed. **Key words:** neuroscience, health, implicit processes, emotional awareness, emotion regulation.

**ACC** = anterior cingulate cortex; **BA** = Brodmann Area; **dACC** = dorsal anterior cingulate cortex; **HF** = high frequency; **HRV** = heart rate variability; **IAPS** = International Affective Picture System; **IBS** = irritable bowel syndrome; **LEAS** = Levels of Emotional Awareness Scale; **PET** = positron emission tomography; **rCBF** = regional cerebral blood flow.

## INTRODUCTION

The physiology of emotion is arguably the cornerstone of psychosomatic medicine. The American Psychosomatic Society was launched by the publication of *Emotions and Bodily Changes* by H. Flanders Dunbar (1). Conferences were held on this same topic in 1936 and 1937 and the journal *Psychosomatic Medicine* was inaugurated in 1939 with Dr. Dunbar as Editor. The journal was to be “devoted not to the isolated problems of the diseased mind or the diseased body, but to the interrelationships between emotional life and bodily processes” (2). To this day, a majority of papers in this journal involve emotion and emotion regulation, broadly construed. Yet, although emotion and its physiology have been foundational for the field, the approaches taken have not been unified. There have been two distinct themes in how it has been approached over the past 70 years.

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The first theme, which is the dominant perspective within the field today, holds that aversive emotional states are associated with adverse health outcomes. There is now a long list of negative emotional states and traits for which this has been found to be true, including depression (3), hostility (4), worry (5), anxiety (6), hopelessness (7), and perceived stress (8), among others. According to this perspective, self-reports are assumed to provide a satisfactory way of identifying these states and traits. We now have unequivocal evidence, for example, that self-reported depression is associated with decreased survival in patients with coronary artery disease (9). Depression is also associated with increased mortality in the context of diabetes (10) and certain forms of cancer (11). A more recent variant on this hypothesis, for which data are accumulating, is that positive emotional states are associated with beneficial health outcomes (12,13).

The second theme, which predominated in the early years of the Society, is that interference with the experience or expression of negative emotion is deleterious for health (2). This perspective can be traced to the work of Breuer and Freud in 1895 on conversion disorder (14). Their core psychoanalytic hypothesis was that conversion disorder or hysteria arose because affect that was activated during the traumatic event could not be expressed at the time of the trauma. Symptoms were a result of this failure to express the emotions associated with the event, as the unexpressed emotions prevented the memory of the trauma from dissipating. The “strangled” affect was expressed indirectly, however, in a somatic symptom which symbolically represented that which had been repressed; e.g., paralysis of an arm was related to the unexpressed wish to strike out. This foundational concept of repressed emotion led to a variety of applications, one of which

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was to the mind-body problem and what came to be known as the modern field of psychosomatic medicine (15).

Franz Alexander at the Chicago Psychoanalytic Institute advanced psychosomatic theory by articulating the concept of visceral neurosis, which was based on the observation, modifying Freud's idea, that the symptom itself did not have symbolic meaning for the patient (16). Rather, a psychological conflict led to repression of affect, which continued to be expressed physiologically, resulting in dysregulated physiology and disease. Grinker and Spiegel (17), also from Chicago, observed in World War II veterans that psychosomatic conditions, such as peptic ulcer, arose when the physiological expression of emotion persisted in the absence of the conscious experience of emotional distress.

This classic psychoanalytic approach to psychosomatic disorders reached its zenith in the 1940s when Alexander formulated what came to be called "Specificity Theory" (16). Using the premier "science of the mind" at that time—psychoanalysis, he sought to emulate the successes of infectious disease by identifying the specific psychological contexts in which specific physical disorders occurred (2).

Alexander held that each of seven disorders, including essential hypertension, bronchial asthma, ulcerative colitis, peptic duodenal ulcer, neurodermatitis, rheumatoid arthritis, and thyrotoxicosis, was associated with a specific unconscious conflict (16). For example, those prone to hypertension had a conflict about expressing aggressive tendencies for fear of jeopardizing important relationships. Hypertensive individuals seemed to be meek and compliant and were thought to not be in touch with or consciously aware of their aggressive tendencies. Episodes of hypertension were thought to be elicited in contexts in which aggressive tendencies were activated but could not be consciously processed, resulting in the bodily expression of the emotion (i.e., hypertension). This was a visceral neurosis in the sense that the blood pressure elevation itself did not have symbolic meaning. It was recognized that such a conflict alone was not sufficient to bring on the disorder and that constitutional (e.g., genetic) and other factors contributed. Although the nature of the unconscious conflict was unique to each disease, all core conflicts were associated with repression of emotion. Alexander asserted that the essence of the analytic process in the treatment of psychosomatic patients was bringing into the patient's consciousness emotions and motivations of which he or she was unaware. Thus, although each disorder was postulated to be associated with a specific conflict, there was also a common denominator involving emotions that were not consciously processed that applied to all of the disorders.

Alexander's Specificity Theory has not stood the test of time. Although some empirical support for the theory was obtained (18), empirical tests using standardized measures and unbiased sampling methods typically failed to provide supportive evidence (19). These results were due, in part, to the fact that conditions such as hypertension had diverse etiologies and because the psychological profile of patients with a given disorder varied as a function of the stage of the disorder

(15). However, Theme 2 is still an accepted approach to understanding how psychological factors contribute to physical disease, as illustrated by constructs that are still current such as the repressive coping style (20), alexithymia (21), denial (22), suppression (23), Type C personality (24), and the Type D personality (25). All of these constructs have in common that negative emotions, particularly in the context of stressful life circumstances, are either not consciously experienced or are somehow stymied in their outward expression. Each is associated with findings demonstrating a linkage to adverse health outcomes.

One of the challenges associated with this line of research is demonstrating the presence of an emotional state or trait that is not reportable by the subject. An alternative variant of this approach is to assess specific emotion regulatory functions or skills, the impairment of which may be manifested as interference with emotional experience or expression. Examples include attention to emotion (26), emotional approach coping (27), acceptance of emotion (28), and levels of emotional awareness (29). When used in research on health outcomes, low scores on these variables are thought to reflect the phenomenon of interference inherent in Theme 2.

The continued interest in the Theme 2 approach is illustrated by the four successful international conferences on "The (non)Expression of Emotion in Health and Disease," held in Tilburg, Netherlands, in 1996, 1999, 2003, and 2007 (30–32). In Germany, there continue to be separate departments of Psychotherapy and Psychosomatics in which the psychoanalytic approach to understanding psychological factors in physical disease is the predominant model. Although these approaches remain viable because of their clinical and cost-effectiveness (33), they are clearly in the minority in the US today.

Given the extent of research associated with Themes 1 and 2, it is surprising to consider that there has been relatively little cross-talk between them. Reconciliation of the two perspectives is challenging because, at the level of self-reported experience, they seem to be contradictory. Theme 1 holds that self-report is a reliable and useful indicator of emotional state or trait. Theme 2 holds that what is most salient in the domain of emotions and health is that which cannot be experienced or expressed. Yet, given their demonstrated value over decades, each must contain at least some element of truth. How might these two approaches be reconciled?

A main thesis of this paper is that neuroscience provides a way to integrate Themes 1 and 2 to create a unified framework for psychosomatic medicine research on emotion. A neuroscientific approach reconciles these two perspectives and provides an explanation for how these different psychological constructs can lead to peripheral physiologic changes that are potentially pathogenic. Before turning to neuroscience, however, we consider how the relationship between Themes 1 and 2 can be understood from a psychological perspective.

### Psychological Model

In the mid-20<sup>th</sup> century, in the context of a growing appreciation within the psychoanalytic tradition of the importance

of preoedipal factors in psychopathology, the conceptualization of the relationship between affect and disease shifted from a focus on conflict to a focus on deficits in affective development (15). In addition, as a counterpoint to Specificity Theory, there was growing interest in common personality characteristics that contributed to psychosomatic disorders. Thus, in a landmark paper in *Psychosomatic Medicine* in 1948 titled "The Infantile Personality," Jurgen Ruesch (34) attributed the psychoneuroses to "pathological development" and psychosomatic conditions to "arrested development." He posited a host of potential causes for the personality structure associated with psychosomatic conditions, such as a lack of consistent parenting or trauma that overwhelms the child's sense of mastery. Ruesch proposed that these experiences caused deficits in social learning and an "infantile" form of "self-expression" that manifested as somatic symptoms. Similarly and independently, in the 1950s and early 1960s, Marty and de M'Uzan of the French Psychosomatic School observed a specific cognitive style ("pensée opératoire") in individuals prone to psychosomatic disorders, characterized by a lack of fantasy and a preoccupation with the concrete details of external events (35).

At about the same time, Nemiah and Sifneos (36) were interested in testing the hypotheses of Alexander and colleagues regarding the association between specific types of psychological conflicts and specific disease states. Rather than finding evidence for Alexander's Specificity Theory, they were struck by the marked difficulty that these individuals had in verbally expressing their feelings. Noting that others had observed similar characteristics, Sifneos coined the term "alexithymia" in 1972, which translates from Greek to mean "without words for emotion" (37).

Although it was hypothesized that alexithymia arose from an arrest in development, the developmental line along which arrest had occurred had not been described. The theory of "levels of emotional awareness" sought to describe the developmental line in question (38). It was inspired by observations of emotion processing deficits in patients in a medical setting such as the following case.

A 41-year-old unmarried woman was hospitalized for work-up of abdominal pain. Abdominal ultrasound and endoscopy were negative. A psychiatric consultation was requested. The patient reported that persistent pain had been present for 3 months. She also stated that 3 months previously her mother died.

This was actually her adoptive mother who took her in at the age of 8 years. Her biological parents were alcohol dependent and physically abusive. She was placed in a series of foster homes until she was adopted. Her adoptive mother was a very kind and nurturing person. She suffered from diabetes and peripheral vascular disease. During the last 5 years of the mother's life, the patient cared for her on a daily basis. One week before her death, after a medical setback, the patient contemplated the possibility of her mother's death and was overwhelmed with a feeling of grief.

A week later, mother died of a gastrointestinal vascular obstruction. After her mother's death, the patient experienced no feelings of grief or sadness. In addition to pain, she suffered from anhedonia, had neurovegetative symptoms of depression, and met criteria for major depressive disorder.

A case such as this, which is commonly seen, raises a number of intriguing questions. What happened to the grief that she had experienced 1 week before mother's death? Why was the absence of experienced grief concomitantly associated with pain? Was her apparent difficulty in experiencing grief related to her early childhood history of abuse?

Lane and Schwartz (38) proposed that an individual's ability to recognize and describe emotion in oneself and others, called emotional awareness, is a cognitive skill that undergoes a developmental process similar to that which Piaget described for cognition in general. A fundamental tenet of this model is that individual differences in emotional awareness reflect variations in the degree of differentiation and integration of the schemata (implicit programs or sets of rules) used to process emotional information, whether that information comes from the external world or the internal world through introspection.

Emotional awareness is considered to be a separate line of cognitive development that may proceed independently from other cognitive domains (39). The concept that development can proceed at different rates in different domains of knowledge is known as horizontal decalage (40). In principle, it is possible that a developmental arrest can occur in one domain whereas development in other domains of intelligence continues unabated.

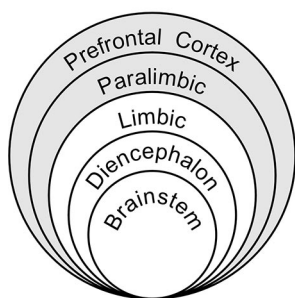
The model posits five "levels of emotional awareness" that share the structural characteristics of Piaget's stages of cognitive development (41). The five levels of emotional awareness in ascending order are awareness of physical sensations, action tendencies, single emotions, blends of emotions, and blends of blends of emotional experience (the capacity to appreciate complexity in the experiences of self and other).

The five levels therefore describe the cognitive organization of emotional experience. The levels are hierarchically related in that functioning at each level adds to and modifies the function of previous levels but does not eliminate them. For example, blends of emotion (Level 4 experiences), compared with action tendencies (Level 2 experiences), should be associated with more differentiated representations of somatic sensations (Level 1). The feelings associated with a given emotional response can be thought of as a construction consisting of each of the levels of awareness up to and including the highest level attained. The trait level of function is the level at which a given individual typically functions (Figure 1, right half).

Modern conceptions of cognitive development have refined Piaget's views but are still consistent with the model proposed here. Karmiloff-Smith (42), for example, holds that the development of knowledge proceeds through a process called "representational redescription." Cognitive development from this

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### Neuroanatomical



### Psychological

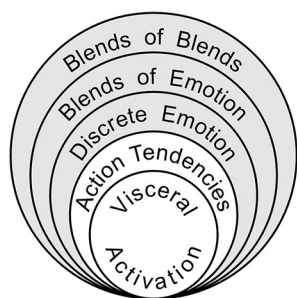


Figure 1. Parallels in the hierarchical organization of emotional experience and its neural substrates. The shell structure is intended to convey that each succeeding level adds to and modulates lower levels but does not replace them. Although each model contains five levels, a one-to-one correspondence between each level in the psychological and neuroanatomical models is not intended. Lower levels with white background correspond to implicit processes. Higher levels with gray background correspond to explicit processes.

perspective consists of the transformation of knowledge from implicit (procedural, sensorimotor) patterns to explicit (conscious thought) representations through use of language or other semiotic mode. This transformation renders thought more flexible, adaptable, and creative. This viewpoint is consistent with the theory that the way language is used to describe emotion modifies what one knows about emotion and how emotion is experienced consciously (43).

The five levels of emotional awareness can therefore be mapped onto the distinction between implicit and explicit processes (44). In cognitive science, the distinction between implicit and explicit roughly corresponds to the distinction between unconscious and conscious processes (45). Level 1 (bodily sensations) and Level 2 (action tendencies) phenomena, viewed in isolation, would not necessarily be considered indicators of emotion but are critical components of emotional responses. The peripheral physiological arousal and action tendencies associated with emotion are implicit in the sense that they occur automatically and do not require conscious processing to be executed efficiently. If one focuses conscious attention on a somatic sensation or action tendency in isolation, emotion is implicit in the sense that the quality of experience needed to call it an emotion requires processing at higher levels. Levels 3, 4, and 5 consist of conscious emotional experiences at different levels of complexity and by definition are explicit. The levels of emotional awareness framework therefore puts implicit and explicit processes on the same continuum and at the same time distinguishes between types of implicit (Level 1 versus Level 2) and explicit (Level 3 versus Level 4 versus Level 5) processes.

To illustrate with the clinical example above, the anticipatory experience of grief 1 week before the mother's death was a Level 3 experience. After the mother's death, the patient experienced abdominal pain, which was in her case a Level 1 (somatic) experience of grief. This also illustrates the distinction between explicit (grief) and implicit (abdominal pain) emotional processing. It deviates from psychodynamic think-

ing, however, by not attributing a motivational basis or mechanism (such as repression) that would explain why the level of processing changed, although such a motivational basis is possible.

### Levels of Emotional Awareness Scale (LEAS): Psychometric Findings

The LEAS is a written performance measure that asks a person to describe his or her anticipated feelings and those of another person in each of 20 vignettes described in two to four sentences (29). Scoring is based on specific structural criteria aimed at determining the degree of differentiation in the use of emotion words (the degree of specificity in the terms used and the range of emotions described) and the differentiation of self from other. The scoring involves essentially no inference by raters. Because the scoring system evaluates the structure of experience and not its content, individuals cannot easily enhance their scores or create a socially desirable impression in their responses, as is the case with some self-report instruments. A glossary of words at each level was created to guide scoring.

Each of the 20 vignettes receives a score of 0 to 5 corresponding to the cognitive-developmental theory of emotional awareness that underlies the LEAS (38). A score of 0 is assigned when nonaffective words are used, or when the word "feel" is used to describe a thought rather than a feeling. A score of 1 is assigned when words indicating physiological cues are used in the description of feelings (e.g., "I'd feel tired"). A score of 2 is assigned when words are used that convey undifferentiated emotion (e.g., "I'd feel bad"), or when the word "feel" is used to convey an action tendency (e.g., "I'd feel like punching the wall"). A score of 3 is assigned when one word conveying a typical, differentiated emotion is used (e.g., happy, sad, angry). A score of 4 is assigned when two (or more) Level 3 words are used in a way that conveys greater emotional differentiation than would either word alone. Respondents receive a separate score for the "self" response and for the "other" response ranging from 0 to 4. In addition, a total LEAS score is given to each vignette equal to the higher of the self and other scores. A score of 5 is assigned to the total when self and other each receive a score of 4 and are differentiated from one another; thus, a maximum total LEAS score of 100 is possible.

The LEAS has consistently been shown to have high interrater reliability and internal consistency (46). The test-retest reliability at 2 weeks has been shown to be good. Norms for age, gender, and socioeconomic status have been established.

A variety of studies (but not all) (47,48) support the construct validity of the LEAS. The LEAS correlates moderately positively with two cognitive-developmental measures—the Sentence Completion Test of Ego Development by Loevinger et al. (49,50) and the cognitive complexity of the description of parents by Blatt and colleagues (51). These results support the claim that the LEAS is measuring a cognitive-developmental continuum and that the LEAS is not identical to these other measures. Greater emotional awareness is associated

with greater self-reported impulse control, consistent with the theory that functioning at higher levels of emotional awareness (Levels 3–5) modulates function at lower levels (actions and action tendencies at Level 2) (44). Greater emotional awareness is also associated with greater openness to feelings (29) and greater emotion recognition ability (52,53). The LEAS correlates positively with empathy ability, certain subtests of a newer measure of emotional intelligence (54), the tendency to seek help for emotional problems, and the actual amount of social support that a person has. Additionally, individuals with lower LEAS scores rated their general sense of well-being lower during experimental induction of sad mood compared with baseline ratings, whereas those with higher LEAS scores had a stable sense of general well-being independent of their momentary mood (55).

Clinically, it has been shown that patients with borderline personality disorder score lower on the LEAS than age-matched control subjects (56) and that individuals with the “disorganized attachment style” have lower LEAS scores than those with the “organized attachment style” (57). Patients with irritable bowel syndrome (IBS) do not on average have lower LEAS scores than healthy controls but, among those with IBS, lower scores on the LEAS are associated with greater pain (58). Patients on a psychosomatic inpatient ward with somatoform disorders had lower LEAS scores than patients with disorders involving psychological distress, such as depression. This same study showed that somatoform patients showed significant increases in LEAS scores after 3 months of multimodal inpatient treatment that integrated body-based techniques with intensive group and individual psychotherapy (59). These findings support the theory that impairments in emotional awareness can occur developmentally, that lower emotional awareness is associated with a greater tendency to experience emotional distress as bodily symptoms, and that emotional awareness can improve with therapeutic interventions that facilitate the transition from implicit to explicit processing.

The LEAS has also yielded useful findings in a variety of other clinical settings. Patients with essential hypertension had lower LEAS scores than those with hypertension secondary to other medical conditions, such as renal disease (60). Patients with eating disorders (anorexia and bulimia) were observed to have lower LEAS scores than matched controls (61), consistent with Hilde Bruch’s classic observation that eating disorders are associated with an impairment in interoceptive awareness of one’s own emotions (62). Patients with posttraumatic stress disorder (PTSD) have lower LEAS scores than matched controls, and LEAS scores were inversely correlated with the severity of PTSD symptoms, particularly symptoms involving dissociation (63). Patients with morbid obesity were observed to have lower LEAS scores than controls, and it was also observed that, among the obese patients, the higher the LEAS scores the greater their social anxiety (64). The latter finding indicates that greater emotional awareness is associated with a greater awareness of the negative emotional responses that

morbid obesity elicits from others. A related finding is that individuals with generalized anxiety disorder have greater LEAS scores than matched controls (65), indicating that emotional awareness can be a double-edged sword. In contrast, patients with depression were found to have decreased awareness of the emotions of others (66,67), consistent with the pathological introspective focus that can occur with depression. Together, these findings indicate that the LEAS can detect variations in emotional awareness that have meaningful clinical correlations.

A key issue in the assessment of emotion-processing deficits is the need to distinguish between Theme 1 and Theme 2 phenomena. The LEAS typically does not correlate with self-reported negative affect, such as anxiety or depression, in the absence of anxiety or depressive disorders (29), unlike the 20-item Toronto Alexithymia Scale, which typically does (47). In three separate studies, including studies of essential hypertension (60), eating disorders (61), and somatoform disorders (59), it has been shown that associations between lower emotional awareness and the clinical condition were not altered by partial correlations removing variance due to negative affect, whereas control for self-reported negative affect rendered associations with the TAS-20 nonsignificant. These findings suggest that the LEAS may have an advantage in those contexts in which distinguishing between Theme 1 and Theme 2 phenomena is important.

### Brain Model—Early History

Scientifically credible models of how the human brain processes emotion have only been formulated in the past 100 years. The model to be presented here builds on important forerunners early in the 20<sup>th</sup> century.

In the early 1920s, Walter Cannon suggested that the physiology of emotion provided a key link between mental states and physical disease. He pointed out that subcortically generated emotion could be routed downstream to the hypothalamus associated with physiological expression or upstream to the neocortex for symbolic representation and expression (68). However, Cannon did not attempt to formulate a detailed model of how the brain mediates emotion (69).

In 1937, James Papez published a landmark paper in which he proposed the first complete neural circuit mediating emotion (70). This proposal postulated a role for brain structures, whose function was not understood, in the mediation of a function, emotion, whose locus in the brain was not understood. Papez proposed that emotion could be induced either through stimulus perception or thought by engaging a reverberating circuit that included the mamillary bodies of the hypothalamus, the anterior thalamic nucleus, the anterior cingulate cortex (ACC), and the hippocampus. Although current models of how the brain mediates emotion are very different from the Papez model, his hypothesis that the dorsal anterior cingulate cortex (dACC) was the seat of emotional experience was surprisingly prescient.

In the 1930s and 1940s, Kluver and Bucy (71) resected the temporal lobes of monkeys and observed a condition called

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“psychic blindness” associated with emotional blunting (loss of normal fear and anger responses), inappropriate sexual behavior, visual agnosia, and hyperphagia. In 1956, Weiskrantz (72) discovered that the amygdala, a structure in the medial temporal lobe, was the critical structure mediating these behavioral changes.

In 1949, Paul MacLean wrote a seminal paper entitled “Psychosomatic Disease and the Visceral Brain” that was published in *Psychosomatic Medicine* (73), in which he expanded on the Papez model of emotion, incorporating some of the findings of Kluver and Bucy and drawing on evidence implicating other structures such as the frontal lobes in the mediation of emotion. MacLean hypothesized that psychosomatic disorders arose when emotion-related neural activation in the visceral brain, which he renamed the “limbic system” in 1952 (74), did not get transferred to the neocortex for higher processing. As MacLean stated, “Emotional feelings, instead of finding expression and discharge in the symbolic use of words and appropriate behavior, might be conceived as being translated into a kind of organ language.”

MacLean had therefore proposed a neural basis for the interference or blockage of emotion processing inherent in Theme 2. He quoted the model by Ruesch (34) (described above) that itself was a forerunner of the alexithymia construct and the levels of emotional awareness model. Although MacLean’s model was consistent with the concept of interference or blockage of emotion processing, it did not require a motivated basis for the lack of transfer, consistent with Ruesch’s deficit model. This neural model lay dormant for many years, however, because the primary tools for studying the brain in humans at the time, such as the study of patients with brain lesions identifiable during surgery or postmortem studies, did not lend themselves to examining how brain function might contribute to the development of psychosomatic disorders in the typical context of individuals with intact brains. However, with the advent of modern techniques for functional and structural brain imaging, MacLean’s model becomes highly relevant and testable. One might therefore ask, “How do we currently understand the role of cortical and subcortical structures in the mediation of emotional awareness?”

### Neural Substrates of Emotional Awareness

To date, there have been two imaging studies in which the LEAS has been correlated with brain activity during emotion in healthy subjects. The first included 12 right-handed female volunteers who were free of medical, neurological, or psychiatric abnormalities. Happiness, sadness, disgust, and three neutral control conditions were induced by film and recall of personal experiences (12 conditions). Twelve 60-second positron emission tomography (PET) images of regional cerebral blood flow (rCBF) were obtained in each person, using intravenous bolus injections of <sup>15</sup>O-water (46).

We examined the correlates of rCBF due to emotion by subtracting the neutral conditions from the emotion conditions. Subtracting the neutral conditions removes brain activity due to the experimental paradigm that is not due to emotion. To

ensure sufficient statistical power, we combined the three emotion conditions together and did the same for the neutral conditions. We then correlated LEAS scores with the emotion-minus-neutral rCBF difference to identify regions of the brain that correlated with emotional awareness during emotional arousal.

Findings from this covariate analysis revealed one cluster for film-induced emotion with a maximum located in the right midcingulate cortex. For recall-induced emotion, the most statistically significant cluster was located in the right ACC. An analysis was then performed to identify areas of significant overlap between the two covariance analyses. Based on an a priori statistical threshold, a single cluster was observed in the right dACC (BA 24) (46).

Traditionally, the dACC has been thought to have a primarily affective function (70,75). However, in addition to emotion, it is now recognized to play important roles in attention, pain, conflict monitoring, response selection, maternal behavior, vocalization, skeletomotor function, and autonomic control (76). How can these different functions be reconciled with the present findings involving emotional awareness?

One answer is that these various functions of the dACC reflect its superordinate role in executive control of attention and motor responses (46). According to this view, emotion, pain, or other salient exteroceptive or interoceptive stimuli provide moment-to-moment guidance regarding the most suitable allocation of attentional resources for the purpose of optimizing motor responses in interaction with the environment. The findings from this study suggest that attentional resources are more readily engaged by one’s own emotional responses among those individuals who are more emotionally aware. To the extent that people who are more emotionally aware attend more to internal and external emotion cues, the subsequent cognitive processing of this information can contribute to ongoing emotional development. The fact that the dACC can be engaged by so many other kinds of nonemotion stimuli may mean that less emotionally aware individuals tend to use their attentional resources more readily for purposes other than processing internal or external emotional signals.

These findings were replicated and extended in a second PET study using the same radiotracer, <sup>15</sup>O-water (77). Pleasant, unpleasant, and neutral pictures from the International Affective Picture System (IAPS) (78) were presented in blocks so that we could obtain scans corresponding to high arousal pleasant, low arousal pleasant, high arousal unpleasant, low arousal unpleasant, and neutral conditions. This design enabled us to disentangle whether LEAS correlated with valence (pleasant, unpleasant), arousal (high, low arousal), or both. We also were able to consider sex differences as 22 men and 22 women were studied.

We observed a significant correlation between dACC activity and LEAS in the high arousal (pleasant and unpleasant) conditions relative to the low arousal (pleasant and unpleasant) conditions. This finding was observed in the combined sample of men and women but was greater in women. The association between LEAS and dACC was not observed as a

function of pleasant or unpleasant valence relative to neutral or in the low arousal conditions relative to neutral.

These results illustrate that engagement of the dACC differs as a function of individual differences in emotional awareness in the context of conditions that induce high arousal emotions. Individuals who are more emotionally aware are better able to tolerate and consciously process intense emotions than those who are less aware (79–81). Conversely, individuals functioning at a lower level are more likely to behave impulsively and be less aware of what they are feeling in the context of high arousal emotions (44). This may be understood as a greater ability among more highly aware individuals to be cognizant of their own emotional reactions in the context of high arousal and to anticipate and evaluate the consequences of their actions in advance of their behavioral expression. This greater ability may be mediated at least in part by the dACC, consistent with Paus' view that the dACC is fundamentally involved in translating intentions into actions in the context of emotional arousal (82), and is consistent with the role of the dACC in mediating regulated rather than automatic, prepotent responses (83).

These findings suggest an important role for the dACC in the conscious processing of emotional information. How does the brain process emotional information that is not conscious? This has been studied in the functional imaging context, using a technique called backward masking, in which the target stimulus, typically a picture of a facial expression such as fear or anger, is presented very briefly (typically <30 milliseconds) and prevented from being consciously processed by a "masking" stimulus, typically a neutral face, that follows immediately and is presented for a longer duration (typically 100 milliseconds). This approach has been used effectively to demonstrate amygdala activity when facial expressions of fear (84) or anger (85) are not consciously perceived. These imaging studies have not, however, attempted to simultaneously examine subjective emotional responses to these stimuli.

The most rigorously conducted study to date that examined whole-brain activity during implicit and explicit processing of emotional faces in the same subjects was published in 2006 by Leanne Williams and colleagues (86). Consistent with previous work (84,85,87), this study demonstrated amygdala activity during implicit processing of fearful faces. Explicit processing, compared with implicit processing, was associated with greater activity in the dACC, medial prefrontal cortex, and amygdala (the latter likely due to activation by both subcortical [implicit] and cortical [explicit] pathways). Implicit processing, compared with explicit processing, was associated with greater activity in the ventromedial prefrontal cortex.

Another feature of this study was that amygdala activity was examined as a function of implicit and explicit processing during the first half and second half of the experimental trials, which averaged 1300 milliseconds in total. Results showed that amygdala activity was greater during the first half relative to the second half. The decrease in amygdala activity during the second half, however, was greater with explicit rather than implicit processing. The small decrease from the first to the

second half during implicit processing is likely due to the inhibitory influence from the ventromedial prefrontal cortex. The greater decrease with explicit processing is likely due to the combined effect of ventral and dorsal medial prefrontal top-down inhibitory influences. Thus, conscious, relative to unconscious, processing is associated with greater inhibition of the amygdala, and late amygdala activity was numerically greater during unconscious than conscious processing ( $p < .098$ ). These findings are reminiscent of those with the LEAS in which higher levels of conscious processing were associated with greater attenuation of Level 1 and Level 2 phenomena.

### Model of the Neural Substrates of Implicit and Explicit Emotional Processes

The distinction between implicit (nonconscious) and explicit (conscious) processes is foundational in cognitive neuroscience because their neural substrates are dissociable (88). The distinction was first applied to memory. Explicit memory for facts and events requires participation of medial temporal lobe structures (such as the hippocampus) and diencephalon, whereas implicit memory requires structures such as the striatum (skills and habits), neocortex (priming), amygdala and cerebellum (classical conditioning), and reflex pathways (non-associative learning). Implicit processes have also been demonstrated in a variety of other cognitive domains, including attention, perception, and problem solving (88). This body of research has led to a growing recognition that consciousness is the tip of the cognitive iceberg in the sense that the vast majority of cognitive processing occurs outside of conscious awareness (89).

Antonio Damasio's distinction between primary emotion and feeling and their dissociable neural substrates (90) paved the way for the application of the implicit-explicit distinction to emotion. Primary emotion is the phylogenetically older behavioral and physiological expression of an emotional response. Primary emotion occurs automatically and without the necessity of conscious processing. Feeling, on the other hand, involves the conscious experience of that emotional state. According to Damasio, primary emotion and feeling are separable, both conceptually and neuroanatomically (90). Whereas primary emotion is necessary for successful adaptation to environmental challenges and the physiological adjustments needed to meet those challenges, a conscious feeling state enables previous emotional experiences to be consciously recalled or current experiences to be accessed and used for decision-making and navigation of the social world. In this paper, the term "affect" encompasses both primary emotion and feeling.

One might question whether a distinction applied in the cognitive domain applies to emotion. My colleagues at the University of Arizona and I hold that there is nothing about emotion that is not cognitive if one equates cognition with information processing (91). Others including Kihlstrom and colleagues (45), who were early investigators of implicit cognition (92), argued that the implicit-explicit distinction applies to emotion as well as cognition.

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Parallels between the corresponding neuroanatomical and psychological models are schematically depicted in Figure 1. Both models are hierarchical and show a similar architecture of concentric shells. A similar conception has been independently developed by Fellous (93). The concentric architecture means that each new level subsumes and modulates that of previous levels. Although both the psychological and neuroanatomical models designate five levels, there is no intention to suggest a direct correspondence between a given level in one model and that of the other model. In general, implicit functions at Levels 1 and 2 in the psychological model correspond to Levels 1 to 3 in the neuroanatomical model, and explicit functions at Levels 3 to 5 in the psychological model correspond to Levels 4 and 5 in the neuroanatomical model.

Although the neural substrates of each level of emotional awareness are not yet known, a rudimentary neuroanatomical model that distinguishes between implicit and explicit processes can be formulated (44). This model distinguishes between the neural substrates of implicit emotion, on the one hand, and three different aspects of the conscious experience of emotion: background feelings, focal attention to feelings, and reflective awareness (Figure 2). Implicit emotion includes those sensory and motor aspects of emotional responses that precede the emergence of an emotional feeling state. Background feelings are bodily states on the periphery of awareness that color conscious experience but are not noticed, as such, unless attended to. Focal attention to feelings refers to the condition in which one's own subjective emotional state is the object of directed attention. Reflective awareness involves thinking about the contents of conscious emotional experience, typically after it has been the object of focal attention. We will consider the neural substrates of each of these categories below.

Several fundamental principles characterize this model.

1. There are distinct neural substrates of implicit and explicit emotional processes.
2. The structures that are uniquely responsible for emotion generation operate implicitly, i.e., outside of conscious awareness.



Figure 2. Structures on the medial surface of the frontal lobe that participate in 1) background feelings, 2) attention to feelings and 3) reflective awareness of feelings.

3. The brain structures needed for conscious emotional experiences emerged more recently during phylogenesis compared with those required for implicit processes.
4. The structures that preferentially participate in the conscious experience of emotion are not unique to emotion but rather perform domain-general cognitive functions.
5. Emotions compete with other inputs for conscious processing.

### Implicit Aspects of Emotion

Brain imaging studies in recent years demonstrated that amygdala activation can occur in response to emotional stimuli in the absence of conscious awareness of the stimulus. This finding is consistent with the notion that the amygdala performs its functions implicitly and plays a key role in establishing whether exteroceptive stimuli have an emotional meaning. Echoing the distinction by Papez between emotion induced by perception versus thought (70), LeDoux has written widely about the distinction between the thalamo-amygdala pathway for processing exteroceptive stimuli rapidly and crudely in the absence of conscious awareness (69), and the neocortical-amygdala pathway that provides more precise and differentiated identification of the stimulus requiring an additional 12 milliseconds of processing time. The time saved by having behavior directed by implicit processes could potentially mean the difference between life and death in life-threatening situations. This is not inconsistent with the view that the capacity for explicit processes, such as reflection, deliberation, delay of impulses, planning and the like, are arguably more important to adaptive success in modern human cultures, in which success or failure rather than life or death is the more salient issue.

There are several additional lines of evidence that further support the hypothesis that the amygdala executes its functions implicitly. Studies of the subjective experience of emotion in patients with unilateral and bilateral amygdala lesions show that emotional experience is not appreciably altered in these patients compared with controls (94). During ictal fear, cortical structures such as the ACC and prefrontal cortex are activated in addition to the amygdala, whereas when amygdala activation alone is observed during seizures detected by depth electrodes, the experience of fear does not occur (95). Other structures that participate in implicit emotion processing include the thalamus, hypothalamus and pituitary, basal ganglia, and brainstem nuclei, the latter including those involved in autonomic regulation and ascending neurotransmitter systems.

An important new development is that “unconscious emotion” is now recognized as a legitimate topic of scientific inquiry in academic psychology (96). For example, studies have examined how emotional stimuli that are not consciously detected influence behavior and subjective experience. A dialogue between researchers in this area and researchers in psychosomatic medicine will likely be of mutual benefit as this work progresses.



### Background Feelings

Background feelings (97) are bodily states that color conscious experience but are not noticed, as such, unless attended to. They are generated by internal regulatory functions as well as external stimuli and provide information about one's current state of well-being. They are consistent with what Farthing (98) refers to as peripheral awareness—those mental contents that are on the periphery of focal awareness. They include stimuli that are being processed automatically as focal attention is directed elsewhere, as well as events that have been in focal awareness recently. This concept is intended to include both undifferentiated (e.g., feeling lousy) and differentiated (e.g., feeling sad) emotional states that are situated on the periphery of focal awareness, are part of conscious experience, and are easily attended to, if necessary. Thus, background feelings constitute the conscious experience of emotion without awareness.

A number of paralimbic and neocortical areas likely participate in background feelings. The subgenual ACC is an area known to participate in visceral regulation. Bechara and colleagues (99) have shown that the ventromedial prefrontal cortex biases behavior and decision-making without conscious awareness of an emotional state necessarily occurring, although the gut feelings associated with this structure that are central to the somatic marker hypothesis (90) clearly implicate it in some type of conscious bodily experience. In the same vein, the observation by Williams and colleagues (86) that subgenual ACC is more activated during implicit than explicit presentation of fearful faces is not inconsistent with its hypothesized role in background feelings. The so-called “affective division” of the ACC (subgenual and pregenual ACC) has dense inputs from the amygdala and other subcortical structures and may well participate in background feelings. This interpretation is consistent with the findings of Bush and colleagues (100) involving the Emotional Counting Stroop, in which the pregenual ACC was activated during a foreground cognitive task whereas background emotional stimuli served as distractors.

The insula is another cortical area that is thought to contribute to the representation of one's own bodily state (101,102). The insula contains a topographic representation of inputs from visceral, olfactory, gustatory, visual, auditory, and somatosensory areas and is proposed to integrate representations of external sensory experience and internal somatic state (103). The posterior insular cortex is the primary projection area for visceral sensation, whereas the anterior insula, particularly on the right side (102), is a higher association area for these bodily signals (104) and is involved in remapping these signals into conscious bodily feelings (105). The insula is therefore involved in the representation of bodily states that can provide positive or negative biases to cognitive decision-making (90,106). The insula also conveys cortical somatosensory information, such as imagined discomfort associated with fear, to the amygdala (107). The insula has been found to be

activated in a number of studies of emotional processing such as disgust (108), especially those involving emotion that is induced by imagery or recall (109), and emotional tasks that have a concurrent cognitive demand (106). Consistent with a role in background emotion, the insula was found to be more engaged when emotional stimuli were passively viewed than when attention was directed at one's own emotional state (110).

The right parietal cortex is known to be activated by emotional arousal (111). Activation has also been observed as subjects engage in a task that involves emotional stimuli that are not necessarily the focus of attention, such as a gender decision task (112). Somatosensory cortex plays a major role in the mapping of bodily sensations and thus contributes to background feelings as well (90,97). Thus, the ventromedial prefrontal cortex, ventral and pregenual ACC, anterior insula, somatosensory cortex, and right parietal cortex are all likely participants in the neural basis of background feelings.

### Focal Attention to Feelings

Two independent studies demonstrated that greater emotional awareness was associated with greater activity in the dACC (46,77). An almost identical area of the ACC was implicated in emotional experience in a study by Rainville and colleagues (113). That study involved manipulating the unpleasantness of a painful stimulus by hypnotic suggestion. As a result of the hypnotic suggestion, the subjects focused conscious attention on the unpleasantness of the painful stimulus and this mental task was associated with activation of dACC.

Lesions in the dACC are known to alter emotional experience (114) and meta-analytic data demonstrate activation of the dACC in several functional imaging studies of emotion (106). For example, dACC is activated when attention is directed to the feeling of sadness (115), during the experience of social rejection (116), and during awareness and monitoring of one's own emotional experiences (117–119). Together, these studies are consistent with the conclusion that this area of ACC is involved in that aspect of phenomenal awareness involved in focused attention on emotional experience. It bears repeating that the function of this region is not specific to emotion, but rather to attention.

### Reflective Awareness

Within the field of consciousness research, a distinction is made between phenomenal and reflective awareness. Phenomenal awareness refers to the actual content of consciousness (focal attention to feelings and background feelings) whereas reflective awareness involves attending to or performing a cognitive operation on the contents of conscious experience (98). Reflective awareness, or metacognition, requires the creation of a representation of experience, and this representation will affect how future emotional information is interpreted and experienced. The acquisition of reflective awareness is a key goal of treatment in patients with more primitive personality organizations (120).

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In another PET study, we examined the pattern of neural activation associated with attending to one's own emotional experience (121). To confirm that subjects were allocating their attention as we instructed, we had them indicate on a keypad how each emotion-evoking picture made them feel. In essence, we were examining an aspect of conscious experience involving commentary on that experience (122). By having subjects attend to and label their experience in this study, we were examining reflective awareness.

We studied 10 healthy men as they viewed 12 picture sets, each consisting of pleasant, unpleasant, and neutral pictures from the IAPS (78). Pictures were presented for 500 milliseconds every 3.0 seconds. Twelve PET-derived measures of rCBF were obtained in each subject, one for each picture set. During half the scans, subjects attended to their emotional experience (indicating on a keypad whether the picture evoked a pleasant, unpleasant, or neutral feeling); during the other half, they attended to spatial location (indicating whether the scene depicted was indoors, outdoors, or indeterminate). Across subjects, picture sets were counterbalanced across the two attention conditions.

During attention to subjective emotional responses, increased neural activity was elicited in rostral ACC (BA 32) and medial prefrontal cortex, right temporal pole, insula and ventral ACC. Under the same stimulus conditions when subjects attended to spatial aspects of the picture sets, activation was observed in parieto-occipital cortex bilaterally, a region known to participate in the evaluation of spatial relationships. Activation of dorsomedial prefrontal cortex during attention to and labeling of emotional experience and activation of parieto-occipital areas during the evaluation of spatial relationships have been replicated in other independent laboratories (123,124), using functional magnetic resonance imaging.

Frith and Frith (125) hypothesized that the ability to mentalize, i.e., the ability to understand the mental state of others or oneself, evolved from the action system for the purpose of identifying the intentions of others and anticipating their future actions. We know that emotional states may fundamentally consist of action tendencies (126), which can be construed as equivalent to the intentions of the self. This dovetails beautifully with the Piagetian perspective on cognitive development in that all mental representations at the conceptual level are fundamentally derived from action schemes. It is therefore reasonable to consider, as a first approximation, that the paracingulate sulcus, which includes the dorsomedial prefrontal cortex, is a substructure within the prefrontal cortex that participates in establishing the representations of mental states (including emotional states) of both self and other. As such, another aspect of emotional awareness, the ability to reflect on one's own emotional state, requires participation of a structure that serves a more general function (mentalizing) that is not specific to emotion.

This conclusion is supported by several findings. Johnson and colleagues (127) demonstrated activation of this region during self-reflective thought (considering whether statements or attributes pertained to the self). Vogeley and associates

(128) observed activation of this region during the process of evaluating what someone else is thinking (theory of mind), as have others previously (129).

The imaging research just reviewed, which focuses on the conscious experience of emotion, highlighted activity in paralimbic and neocortical structures that are not unique to emotion. Rather, these structures perform domain-general functions, such as executive control of attention (in the case of dACC) and mentalizing (in the case of dorsomedial prefrontal cortex). These findings suggest that the structures that preferentially participate in emotional awareness are not unique to emotion but rather are domain-general. To the extent that this is true, it provides powerful support for a cognitive neuroscientific approach to emotion (91). The domain-general function of these areas means that emotions compete with other inputs for conscious processing. This may help to explain why there are such vast individual differences in the extent to which people attend to and use emotional information, whether that information originates externally or internally.

### Top-Down Modulation

The focus up to this point has been on bottom-up phenomena—the transition from subcortical-implicit to cortical-explicit processing. The neuronal pathways between cortical and subcortical structures are bidirectional, indicating that top-down processes must be considered as well. To the extent that we are dealing with conscious processing, the question may be asked how conscious processing of emotional information alters the functioning of subcortical structures, and how top-down and bottom-up processes equilibrate. This is an area in which little work has been done.

One approach is to consider cortical influences on autonomic regulation. The dACC and dorsomedial prefrontal cortex have autonomic regulatory functions that are mediated by direct neural connections with subcortical visceromotor centers, such as the lateral hypothalamus (130). Drevets (131) observed in PET research that blood flow in medial prefrontal cortex is inversely related to heart rate. Stimulation of the medial prefrontal cortex is also known to produce decreases in heart rate and blood pressure in rabbits (132). We have similarly observed a positive correlation between the high frequency (HF), vagal component of heart rate variability (HRV) and activity in the medial prefrontal cortex (BA10) (133). Thus, these centers, when activated in conjunction with conscious emotional experience, have a tonic inhibitory effect mediated through the vagus nerve.

Two other studies have examined the neural correlates of vagal tone during the performance of stressful cognitive tasks. Gianaros and colleagues (134) observed a positive correlation between HF-HRV and ventral ACC activity during the n-back memory task. Matthews and colleagues (135) observed a positive correlation between HF-HRV during the Counting Stroop task performed outside the scanner and ventral ACC activity during the execution of the Counting Stroop Task in the scanner. Differences in the correlation between vagal tone and the specific locus of brain activity may be due to differ-

ences in the tasks performed in the studies as the emotional stress in the latter two studies probably occurred as background feelings. Given the dense interconnections between the dorsomedial and ventromedial prefrontal cortex (130) and the fact that the densest connections between subcortical autonomic regulation centers and the frontal lobe are to be found in the ventral ACC (136), the latter may be a final common pathway from the frontal lobe to subcortical autonomic regulation centers in all three studies.

The positive correlation between dorsomedial prefrontal activity and vagal tone is interesting in two respects. First, one aspect of the correlation is that higher levels of vagal tone are associated with greater activity in dorsomedial prefrontal cortex. Based on the findings reviewed above, it would seem that a greater degree of conscious processing of emotion is associated with greater vagal tone. This can be understood in terms of parallel levels of complexity in the experience of emotion and the associated autonomic substrate for that experience (79). Greater HRV constitutes greater variation and complexity in the patterning of autonomic responses. Greater emotional awareness is associated with greater differentiation and complexity of experience (38). Consistent with this perspective, patients who had surgical removal of the dorsomedial prefrontal cortex and ACC had significant increases in the intensity of their subjective emotional experiences compared with patients who had medial frontal lobe resections that spared these structures or those who had surgical removal of dorsolateral prefrontal cortex (137).

Second, in the case of lower levels of vagal tone, the positive correlation means that when vagal tone is low, activity in the dorsomedial prefrontal cortex is diminished. One way of understanding this is that in a state of emergency associated with high arousal (and reduced vagal tone), the more evolutionarily advanced frontal lobe mechanisms go offline (i.e., their activity becomes attenuated), leaving behavioral control to more automatic, evolutionarily more primitive, neural centers. Put another way, in an emergency situation, implicit processes take over as explicit, deliberative mechanisms go offline. This may be an automatic mechanism that ensures that, in a life-threatening crisis, mechanisms perfected during the course of evolution take over.

It therefore seems that when emotional experience is consciously attended to, a feedback process occurs whereby vagal tone is facilitated and emotional arousal is modulated. In contrast to a generalized state of arousal, in which a diffuse network of neural structures is mobilized to process sensory information, attention is selective for certain inputs and not others, and involves selecting certain actions as others are inhibited (79–82). Thus, through a process of equilibration of bottom-up and top-down interactions, bodily state is altered as diffuse arousal mechanisms become modulated and differentiated. One implication of this model is that becoming consciously aware of one's own emotional responses in itself has a physiological effect that is self-regulatory. Although this has not been tested directly, it has been shown that labeling one's emotions verbally inhibits activity in the amygdala (138), a

finding that suggests completion of a feedback loop from subcortical to cortical and back to subcortical. This model is entirely consistent with the clinical notion that to get through and recover from an episode of emotional distress, one must first allow oneself to experience the distress fully, as this will then lead to a process of recovery that includes putting the distress into words, creating new meaning, and thereby transforming emotional experience (139). In this sense, emotional awareness is a necessary but not sufficient step in the process of emotion self-regulation. It also highlights that simple expression of emotion (Freud's original model of catharsis) is typically insufficient whereas generating new meaning for the purpose of transforming experience often is.

### Common Neural Substrates of Theme 1 and Theme 2 Phenomena

The model just presented constitutes a modern update of MacLean's brain model of psychosomatic disorders arising from a failure of communication between subcortical emotion-generating centers and cortical emotion-processing centers. The mechanisms of top-down modulation are consistent with the hypothesis that failure to consciously process emotional arousal, as in Theme 2 phenomena, would be associated with a failure to modulate or inhibit sites of visceromotor activation that, in turn, could lead to autonomic dysregulation and disease.

These considerations led to the conceptualization of alexithymia as the emotional equivalent of blindsight (80). Blindsight is a phenomenon resulting from lesions in area V1 of the primary visual cortex (140,141), in which patients claim to be blind but respond with high accuracy on visual tracking and other select visual tasks. Thus, these patients behave as if they can see even though they are blind. Similar phenomena have been noted in other sensory modalities, such as touch (142) and olfaction (143). These cases have in common a lesion in or near the primary sensory cortex specialized to process that modality along with accurate behavioral responses, suggesting that some aspects of sensory perception remain intact.

My colleagues and I hypothesized that alexithymia is a disconnection syndrome (144) that results from disuse of fibers connecting affect-generating brain regions with those involved in attention and phenomenal experience of emotion, including the ACC (80). Analogous to blindsight, alexithymic individuals manifest implicit expressions of emotion in conjunction with little or no awareness of the emotions that have been activated. The absence of conscious processing of affect could arise due to disuse during early stages of development when such connections are still forming (145). Consistent with Ruesch's proposal (34) that psychosomatic disorders result from neglectful parenting or childhood trauma, disuse of such connections could result when parental figures fail to engage the child in forming such connections or when the child avoids acknowledging, representing, and processing arousal that is overwhelmingly aversive. In contrast to the functional commissurotomy model of alexithymia, for which there is some empirical support (146), an advantage of the blindsight model,

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which focuses on the vertical rather than horizontal axis, is that it inherently addresses the physiological dysregulation (resulting from the lack of top-down modulation) that may link alexithymia and physical disease.

Empirical support for this neural model of alexithymia was provided by Kano and colleagues (81), who observed that alexithymia was associated with decreased recruitment of dACC and anterior insula as subjects viewed pictures of facial affect and performed a gender identification task. Because gender identification was in the foreground and facial affect was in the background, this paradigm constitutes a “background feelings” stimulus that fails to recruit paralimbic structures needed for the conscious experience of feelings. A recent case report (147) of a 61-year-old woman who sustained a rare infarction in the perigenual region of the ACC and adjacent mesial prefrontal cortex and manifested features of alexithymia, including impaired ability to recognize and label pictures of facial affect and elevated scores on the TAS-20, provides corroborative evidence.

In this regard, it is interesting to note that rumination, a Theme 1 phenomenon, seems to involve a similar failure to recruit top-down modulatory mechanisms. Rumination is the tendency to focus on negative aspects of one’s self or negative interpretations of one’s life, thereby using thinking to amplify or upregulate negative emotion. Ray and colleagues observed that greater trait rumination during imaging was associated with greater increases in amygdala response when participants were asked to increase negative affect and with greater decreases in prefrontal regions (BA10) when participants were asked to decrease negative affect (148). Similarly, Siegle and colleagues observed that state rumination was associated both with greater amygdala activity bilaterally as well as reduced left dorsolateral prefrontal (149) and reduced rostral ACC activity (150), entirely consistent with the findings of Ray et al.

Together, these observations suggest that Theme 1 and Theme 2 phenomena may have similar neurobiological substrates, consisting of activation of subcortical affect-generating structures, such as the amygdala, with a concomitant relative deficit in top-down medial prefrontal influences that would serve to downregulate negative affect. As such, the apparent contradiction inherent in Theme 1 and Theme 2 at the psychological level is resolved when considering their neurobiological underpinnings.

### DISCUSSION

In the mid-20<sup>th</sup> century, a paradigm shift occurred in psychosomatic medicine from a psychoanalytic orientation, with its emphasis on the dynamic unconscious, to a more empirically oriented approach that focused on that which could be objectively measured (15). At the same time, theories of how the brain contributed to physical disease outcomes, such as MacLean’s, were formulated but not pursued further because of our technical inability at the time to study the intact human brain. As a result, we have come to rely almost exclusively on the utility of self-report measures of affective states, in part because this approach has proven to be very fruitful. However,

modern cognitive neuroscience has taught us that the vast majority of cognition is mediated in the brain in the absence of conscious awareness (89). With the discovery of implicit cognition, the traditional equation of cognition with conscious thought was no longer viable (91,92). Similarly, we now understand that undifferentiated emotional responses can occur in the absence of conscious, reportable feeling, and it could well be that emotions function in the brain in a similar way to cognition. To the extent that this is true, the framework presented here holds that both implicit and explicit emotional processes are important and that it is possible to rejuvenate research on unconscious (implicit) emotional processes without sacrificing our commitment to objective measurement by utilizing the conceptual framework of cognitive neuroscience.

Theme 2 originated with the psychoanalytic theory that mental contents were banished from conscious awareness due to repression. Freud conceptualized repression as a motivated process that minimized conscious conflict. Repression later came to be understood as an unconscious process, but a historical review of the concept demonstrated that Freud viewed repression as due to either conscious or unconscious processes (151). It is also noteworthy that the original use of the term “repression” in the early 19<sup>th</sup> century was based on the recognition that mental contents competed for limited processing space in consciousness (152). The term “repression” was coined by Herbart to refer to any mental process that led to the exclusion of some mental contents over others (151,152). This latter way of distinguishing between implicit and explicit emotional processes is the point of view that modern neuroscience supports. This is relevant to the unification of Theme 1 and Theme 2 because it places the Theme 2 tradition on an empirical footing that is separable from its psychodynamic origins in psychosomatic medicine. A failure to process emotions consciously could arise for a host of reasons, including both a motivation to exclude as well as a simple choice to attend to one type of content over another. From this perspective, alexithymia and the repressive coping style can be viewed as related on a severity continuum, with the former constituting a more severe or pervasive disturbance than the latter (53). As such, the present model untethers Theme 2 phenomena from psychodynamic concepts that can be exceedingly difficult to operationalize. It also facilitates the neuroscience agenda by inherently raising the question of where such competition might be happening in the brain.

Primary emotional responses have been preserved through phylogenesis because they are adaptive (90). They provide an immediate assessment of the extent to which goals or needs are being met in interaction with the environment, and they reset the organism behaviorally, physiologically, cognitively, and experientially to adjust to these changing circumstances (153). Feelings are just one aspect of this constellation of changes, and we know that there are vast individual differences in the extent to which people can access and report on their own emotional responses. The key factors for psychosomatic medicine are the physiological concomitants of emotional states and the extent to which emotion regulation strategies,

broadly construed to include voluntary and automatic processes, can be brought to bear to return states of peripheral physiological activation to a quiescent baseline. Such a return to baseline both interrupts or prevents sustained emotional states and permits appropriate responses to incoming stimuli. There are a host of ways in which people can be out of touch with or cannot express what they are feeling (in the Level 3 sense of feeling)—alexithymia (21), the repressive coping style (20), denial (22), suppression (23), Type C personality (24), and Type D Personality (25). What may be most relevant clinically is their association with sustained physiological expressions of emotion. As such, their pathogenic core may be shared with constructs that are readily reportable in the Theme 1 tradition, such as depression (3), anxiety (6), worry (5), and chronic caregiver stress (154). Our focus should therefore be on detecting and ameliorating clinically relevant states of prolonged, undifferentiated physiological activation in relationship to a broad conceptual and measurement approach to emotion regulation. To do so, we should be mindful of the importance of studying clinical populations and recognize the limitations of studying samples of convenience, such as healthy college students (47).

There has been little cross-talk between Theme 1 and Theme 2. One important reason for this may be that the Theme 1 tradition focuses on emotional *states*, whereas the Theme 2 tradition focuses on *traits* related to emotion processing. Although life-long patterns of use or disuse can affect the strength of neural connectivity within a circuit, it is also important to consider how transient states, such as strong emotional arousal, can alter patterns of functional activity within the brain. Both may be important, as illustrated by the case report of the 41-year-old woman who had been abused as a child, had a strong anticipatory grief response before her mother's death, and did not feel any grief but rather experienced abdominal pain after her mother had died. This corresponds well to the kind of clinical situation that Alexander was attempting to address when he observed that an enduring characteristic of the individual (which he labeled an unconscious conflict) made that person susceptible to pathological bodily changes in the context of new life circumstances that made it difficult to consciously process the emotions in question. Such considerations suggest that those who study Theme 2 phenomena should consider how contextual factors determine when the trait vulnerability leads to pathogenic physiological processes. Empirical support for the hypothesized association between alexithymia and physical disease may be limited, in part, because this interaction has not been adequately addressed. It is important to emphasize that we need to be able to detect contextual factors both in those who can and those who can't report them. Thus, objective contextual assessments of stress, as in the Life Events and Difficulties Schedule (155), which are not dependent on self-reported assessments of severity, may be especially useful; we need to recognize that if we rely exclusively on perceived stress (8), we will miss some individuals, perhaps those who may have particularly pronounced physiological changes. Particular at-

tention should also be paid to the phenomenon of background feelings in that individuals who accept, value, and are curious about their own emotions likely process them more extensively in consciousness, whereas those who are baffled by their own emotions and find them unsettling or intolerable may exclude them from conscious awareness. Given the evidence that conscious processing of emotion can have a modulatory effect on subcortical processing, the possibility exists that sustained implicit emotional states may be more pathogenic than sustained explicit emotional states, particularly if the duration of the former exceeds that of the latter.

Whereas this perspective extols the virtues of conscious processing, the long tradition of Theme 1 research makes it clear that reportable states of negative affect can be pathogenic. In order for the negative affect to be modulated by conscious processing, the feeling that is experienced and described should emanate from and correspond to a primary emotional state, using Damasio's terminology (90). Self-reports that do so are likely to be highly related to health, whereas those that do not may add more "noise" than "signal." The usefulness of self-reports as an indicator of underlying biology declines when defense mechanisms and other self-deceptive processes are used to conceal from oneself what one is feeling or the reasons for one's feelings. At the other extreme, conscious states, such as rumination or catastrophizing, are maladaptive and potentially pathogenic because they perpetuate and amplify negative affect rather than allow the meaning of the emotional state to be understood, the unmet needs to be identified, and adaptive action to be taken to resolve or accept the situation (140,156). Self-reports that focus on the trajectory of conscious processing of emotion in the context of stressful life events, such as emotion approach coping (27) and emotion acceptance (28), may facilitate greater cross-talk between the Theme 1 and Theme 2 traditions.

The empirical findings regarding the LEAS support its reliability, construct validity, and clinical applicability. However, there are a variety of ways in which this measurement approach can be advanced. The scale poses hypothetical situations and the scoring system does not attempt to determine whether the emotions described are appropriate to the situation. Wider use of the scale would be promoted by the development of vignettes that are sensitive to age, sex, race, and cultural context. Development of an orally administered version, in contrast to the current paper-and-pencil-approach, could broaden its applicability to a wider variety of contexts. It would also be important to determine if this measurement approach applies equally well to real-life circumstances. Perhaps there are domains of a person's life in which the schemata for processing emotional information are more developed than others. This approach may help to determine whether the various clinical conditions in which lower LEAS scores have been observed differ in the particular way that emotional awareness is reduced.

It has also been demonstrated in patients with PTSD that maladaptive emotional responses, such as reporting feeling guilt and shame when receiving a back rub from a loved one

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(one of the standard LEAS vignettes), can lead to inflated LEAS scores (63). Thus, adjustments to the scoring system may be needed in clinical contexts. Furthermore, there has been very little done to date on the psychophysiological correlates of emotional awareness. More generally, low LEAS scores imply lower level processing by virtue of the failure to use words indicative of higher level processing. A more direct measurement of implicit aspects of emotion, such as autonomic responses, would be particularly useful.

A key question is whether implicit emotional responses that are not associated with self-reported emotional experiences can influence disease pathophysiology in specific ways. An important implication of this model is to consider using implicit measures of emotion as independent rather than dependent variables. Given the challenges of capturing emotion processing deficits psychometrically, this model tells us that primary emotion that is activated but not experienced as feeling is manifested in somatomotor and visceromotor channels. Autonomic and endocrine variables are typically used as dependent measures in psychosomatic research. Perhaps variables within these domains could be used as independent variables (e.g., predictors of medical outcome). An example of this approach is the finding that resting heart rate is a predictor of all-cause mortality, with higher heart rates associated with higher mortality rates (157). A challenge for future research is to develop criteria that would enable researchers to distinguish visceromotor activity that is indicative of emotion from that which is not. Perhaps using somatomotor markers of emotion, such as facial expressions, nonverbal vocal expressions of emotion, gestures, and procedures or scripts (behavioral patterns, often ones that arose as adaptations in childhood and persist into adulthood but are maladaptive in the adult context (158) that are executed automatically and without conscious awareness), could be used in tandem with visceromotor variables for this purpose. Implicit emotion measures may be especially useful in those individuals who manifest affect processing deficits. Ultimately, a combination of modalities may be needed, including psychometric, psychophysiological, neuroendocrine, and neuroimaging techniques to capture implicit processes and their effect on relevant pathophysiology in a disease context.

Given the focus on brain mechanisms in this paper, what are the highest priorities for neuroscientific research in this area? One important focus would be on the separable neural substrates of implicit and explicit emotional processing and their bidirectional links with autonomic, neuroendocrine, and immune mediators of medical outcome. It would be important, for example, to more fully characterize what brain structures may be activated by emotional stimuli that are not associated with reportable emotional experience and those that are, and whether a neural signature for different levels of emotional awareness can be identified. The neural substrates of background feelings also need to be further elucidated to determine whether the neuroanatomical model depicted in Figure 1 is valid or whether Level 1 or Level 2 experiences involve

paralimbic structures, which would require designation of some paralimbic structures as involved in implicit processes. Much more also needs to be done to understand the cortical processes involved in amplifying versus attenuating emotional responses generated by subcortical structures. Patterns of brain structure, activity, and/or circuitry detectable with modern brain imaging techniques may potentially be considered “endophenotypes” that can be useful in risk profiling and could be targets of intervention for primary, secondary, or tertiary prevention.

Finally, this model provides a principled way to integrate body-based and mind-based treatments. According to the theory of levels of emotional awareness, emotions that are not experienced as feelings are experienced as bodily states. According to Piaget, one must meet the individual at the level that they are at (159). “Focusing” (160) and deriving meaning from bodily sensations, or other nonverbal therapies, are needed that facilitate the transition from implicit to explicit processing. More generally, psychosomatic medicine is currently struggling with the question of whether to include, and how to include, body-based treatment approaches that fall within the domain of complementary and alternative medicine, such as massage, yoga, acupuncture, tai chi, dance therapy, art therapy, movement therapy (e.g., Feldenkreis), and other nonverbal techniques. There is also the question of how mind-based psychotherapies, including but not limited to manualized treatments, such as cognitive-behavioral therapy (161), emotion-focused therapy (139) as well as mindfulness meditation, should be used in coordination with body-based treatments. In addition, there is also a need to incorporate psychoeducational techniques that instruct people about emotion recognition and emotion regulation skills, such as those used in dialectical behavior therapy, originally designed for the treatment of borderline personality disorder (162). According to the model presented here, body-based therapies may be particularly useful for mobilizing implicit affect and making it available for explicit processing, whereas mind-based treatments, in conjunction with psychoeducational methods, may be used to facilitate explicit processing. The integrated multimodal treatment approach used on the inpatient psychosomatic unit in Germany, in which patients diagnosed with somatoform disorders improved in their level of emotional awareness (59), used exactly this kind of approach. Although such integrated treatment approaches differ from those of our psychoanalytic predecessors, they are an outgrowth of their fundamental insights about the conscious and unconscious vicissitudes of emotion without which the newer, empirically based model presented in this paper would not have been possible.

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## REFERENCES

- Dunbar H. Emotions and Bodily Changes—A Survey of Literature on Psychosomatic Interrelationships 1910–1933. New York: Columbia University Press; 1935.
- Levenson D. Mind, Body and Medicine. A History of the American Psychosomatic Society. USA: American Psychosomatic Society; 1994.
- Stephoe A, editor. Depression and Physical Illness. Cambridge, UK: Cambridge University Press; 2006.
- Smith T, Glazer K, Ruiz J, Gallo L. Hostility, anger, aggressiveness, and coronary heart disease: an interpersonal perspective on personality, emotion, and health. *J Pers* 2004;72:1217–70.
- Brosschot J, Gerin W, Thayer J. The perseverative cognition hypothesis: a review of worry, prolonged stress-related physiological activation, and health. *J Psychosom Res* 2006;60:113–24.
- Kubzansky L, Kawachi I, Weiss S, Sparrow D. Anxiety and coronary heart disease: a synthesis of epidemiological, psychological, and experimental evidence. *Ann Behav Med* 1998;20:47–58.
- Stern S, Dhanda R, Hazuda H. Hopelessness predicts mortality in older Mexican and European Americans. *Psychosom Med* 2001;63:344–51.
- Vitaliano P, Scanlan J, Ochs H, Syrjala K, Siegler I, Snyder E. Psychosocial stress moderates the relationship of cancer history with natural killer cell activity. *Ann Behav Med* 1998;20:199–208.
- Frasure-Smith N, Lesperance F. Reflections on depression as a cardiac risk factor. *Psychosom Med* 2005;67(Suppl 1):S19–S25.
- Katon W, Rutter C, Simon G, Lin E, Ludman E, Ciechanowski P, Kinder L, Young B, Von Korff M. The association of comorbid depression with mortality in patients with type 2 diabetes. *Diabetes Care* 2005;28:2668–72.
- Onitilo A, Nietert P, Egede L. Effect of depression on all-cause mortality in adults with cancer and differential effects by cancer site. *Gen Hosp Psychiatry* 2006;28:396–402.
- Danner D, Snowdon D, Friesen W. Positive emotions in early life and longevity: findings from the nun study. *J Pers Soc Psychol* 2001;80:804–13.
- Pressman S, Cohen S. Does positive affect influence health? *Psychol Bull* 2005;131:925–71.
- Breuer J, Freud S. Studies on Hysteria. London: Hogarth Press; 1895.
- Taylor G. Psychosomatic Medicine and Contemporary Psychoanalysis. Madison, CT: International Universities Press; 1987.
- Alexander F. Psychosomatic Medicine. New York: Norton; 1950.
- Grinker RR, Spiegel, JP. War Neuroses. Philadelphia: Blakiston; 1945.
- Alexander F, French TM, Pollock GH. Psychosomatic Specificity. Vol I. Chicago: University of Chicago Press; 1968.
- Weiner H. Psychobiology and Human Disease. New York: Elsevier; 1977.
- Jammer L, Leigh H. Repressive/defensive coping, endogenous opioids and health: how a life so perfect can make you sick. *Psychiatry Res* 1999;85:17–31.
- Taylor G, Bagby R. New trends in alexithymia research. *Psychother Psychosom* 2004;73:68–77.
- Ketterer M, Denollet J, Chapp J, Thayer B, Keteyian S, Clark V, John S, Farha A, Deveshwar S. Men deny and women cry, but who dies? Do the wages of “denial” include early ischemic coronary heart disease? *J Psychosom Res* 2004;56:119–23.
- Scheier M, Bridges M. Person variables and health: personality predispositions and acute psychological states as shared determinants for disease. *Psychosom Med* 1995;57:255–68.
- Temoshok L, Dreher H. The Type C Connection. New York: Random House; 1992.
- Denollet J, Pedersen S, Vrints C, Conraads V. Usefulness of type D personality in predicting five-year cardiac events above and beyond concurrent symptoms of stress in patients with coronary heart disease. *Am J Cardiol* 2006;97:970–3.
- Goldman S, Kraemer D, Salovey P. Beliefs about mood moderate the relationship of stress to illness and symptom reporting. *J Psychosom Res* 1996;41:115–28.
- Stanton A, Kirk S, Cameron C, Danoff-Burg S. Coping through emotional approach: scale construction and validation. *J Pers Soc Psychol* 2000;78:1150–69.
- Politi M, Enright T, Weihs K. The effects of age and emotional acceptance on distress among breast cancer patients. *Support Care Cancer* 2007;15:73–9.
- Lane R, Quinlan D, Schwartz G, Walker P, Zeitlin S. The levels of emotional awareness scale: a cognitive-developmental measure of emotion. *J Pers Assess* 1990;55:124–34.
- Vingerhoets A, Van Bussel F, Boelhouwer A, editors. The (Non)-Expression of Emotion in Health and Disease. Tilburg, Netherlands: Tilburg University Press; 1997.
- Nyklicek I, Temoshok L, Vingerhoets A, editors. Emotional Expression and Health—Advances in Theory, Assessment and Clinical Applications. New York: Brunner-Routledge; 2004.
- Denollet J, Nyklicek I, Vingerhoets A, editors. The Third International Conference on the (Non) Expression of Emotions in Health and Disease, Tilburg, Netherlands, 2003.
- Reuddel H, Kreuznach B. Psychosomatic rehabilitation and psychotherapy in German hospitals and rehabilitation centers. In: Dimsdale J, chair. Psychosomatic Disorders in DSM-V and ICD-10. Annual Meeting of the American Psychosomatic Society, Budapest, Hungary, 2007.
- Ruesch J. The infantile personality. *Psychosom Med* 1948;10:134–44.
- Marty P, de M’Uzan M. La pensée opératoire. *Revue Française Psychoanalyse* 1963;27(Suppl):1345–56.
- Nemiah J, Sifneos P. Affect and fantasy in patients with psychosomatic disorder. In: Hill OW, editor. Modern Trends in Psychosomatic Medicine. Vol 2. New York: Appleton-Century-Crofts; 1970:26–34.
- Sifneos PE. Short-Term Psychotherapy and Emotional Crisis. Cambridge, MA: Harvard University Press; 1972.
- Lane R, Schwartz G. Levels of emotional awareness: a cognitive-developmental theory and its application to psychopathology. *Am J Psychiatry* 1987;144:133–43.
- Lane R, Pollerman B. Complexity of emotion representations. In: Feldman Barrett L, Salovey P, editors. The Wisdom in Feeling. New York: Guilford; 2002.
- Fischer K. A theory of cognitive development: the control and construction of hierarchies of skills. *Psychol Rev* 1980;87:477–531.
- Piaget J. La Construction du Réel. Neuchâtel, Switzerland: Delachaux et Niestlé; 1937.
- Karmiloff-Smith A. Beyond Modularity: A Developmental Perspective on Cognitive Science. Cambridge, MA: MIT Press; 1992.
- Werner H, Kaplan B. Symbol Formation: An Organismic-Development Approach to Language and the Expression of Thoughts. New York: John Wiley & Sons; 1963.
- Lane R. Neural correlates of conscious emotional experience. In: Lane R, Nadel L, Ahern G, Allen J, Kaszniak A, Rapcsak S, Schwartz G, editors. Cognitive Neuroscience of Emotion. New York: Oxford University Press; 2000:345–70.
- Kihlstrom JF, Mulvaney S, Tobias BA, Tobis IP. The emotional unconscious. In: Eich E, Kihlstrom J, Bower G, Forgas JP, Niedenthal PM, editors. Cognition and Emotion. New York: Oxford University Press; 2000.
- Lane R, Reiman E, Axelrod B, Yun L, Holmes A, Schwartz G. Neural correlates of levels of emotional awareness. Evidence of an interaction between emotion and attention in the anterior cingulate cortex. *J Cogn Neurosci* 1998;10:525–35.
- Lumley M. Alexithymia and negative emotional conditions. *J Psychosom Res* 2000;49:51–4.
- Waller E, Scheidt CE. Somatoform disorders as disorders of affect regulation: a study comparing the TAS-20 with non-self-report measures of alexithymia. *J Psychosom Res* 2004;57:239–47.
- Loevinger J, Wessler R. Measuring Ego Development. Construction and Use of a Science Completion Test. Vol I. San Francisco: Jossey-Bass; 1970.
- Loevinger J, Wessler R, Redmore C. Measuring Ego Development. Vol. II. Scoring Manual for Women and Girls. San Francisco: Jossey-Bass; 1970.
- Blatt S, Wein S, Chevron E, Quinlan D. Parental representations and depression in normal young adults. *J Abnorm Psychol* 1979;88:388–97.

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52. Lane R, Sechrest L, Reidel R, Weldon V, Kaszniak A, Schwartz G. Impaired verbal and nonverbal emotion recognition in alexithymia. *Psychosom Med* 1996;58:203–10.
53. Lane R, Sechrest L, Riedel R, Shapiro D, Kaszniak A. Pervasive emotion recognition deficit common to alexithymia and the repressive coping style. *Psychosom Med* 2000;62:492–501.
54. Barchard K, Hakstian A. The nature and measurement of emotional intelligence abilities; basic dimensions and their relationships with other cognitive abilities and personality variables. *Educ Psychol Meas* 2004; 64:437–62.
55. Ciarrochi J, Caputi P, Mayer, J. The distinctiveness and utility of a measure of trait emotional awareness. *Pers Individ Dif* 2003;34: 1477–90.
56. Levine D, Marziali E, Hood J. Emotion processing in borderline personality disorders. *J Nerv Ment Dis* 1997;185:240–6.
57. Subic-Wrana A, Beetz M, Paulussen J, Wiltink J, Beutel M. Relations between attachment, childhood trauma, and emotional awareness in psychosomatic inpatients. Presented at the Annual Meeting of the American Psychosomatic Society, Budapest, Hungary, March 2007.
58. Lackner J. Is IBS a problem of emotion dysregulation? Testing the levels of emotional awareness model. Presented at the Annual Meeting of the American Psychosomatic Society, Vancouver, British Columbia, Canada, March 2005.
59. Subic-Wrana C, Bruder S, Thomas W, Lane R, Kohle K. Emotional awareness deficits in inpatients of a psychosomatic ward: a comparison of two different measures of alexithymia. *Psychosom Med* 2005;67: 483–9.
60. Consoli S, Roch B, Plouin P. Patients suffering from essential versus secondary hypertension are characterized by lower levels of emotional awareness. Presented at the Annual Meeting of the American Psychosomatic Society, Budapest, Hungary, March 2007.
61. Bydlowski S, Corcos M, Jeammet P, Paterniti S, Berthoz S, Laurier C, Chambry J, Consoli S. Emotion-processing deficits in eating disorders. *Int J Eat Disord* 2005;37:321–9.
62. Bruch H. *Eating Disorders: Obesity, Anorexia Nervosa and the Person Within*. New York: Basic Books; 1973.
63. Frewen P, Lanius R, Lane R, Neufeld R, Densmore M. Neural correlates of individual differences in levels of emotional awareness during trauma script-imagery. *Psychosomatic Medicine* 2008;70:27–31.
64. Consoli S. Social anxiety is associated with higher levels of emotional awareness in obese patients waiting for gastric banding surgery. Presented at the Annual Meeting of the American Psychosomatic Society, Vancouver, British Columbia, Canada, March 2005.
65. Novick-Kline P, Turk C, Mennin D, Hoyt E, Gallagher C. Level of emotional awareness as a differentiating variable between individuals with and without generalized anxiety disorder. *J Anxiety Disord* 2005; 19:557–72.
66. Donges U-S, Kersting A, Dannlowski U, Lalee-Mentzel J, Arolt V, Suslow T. Reduced awareness of others' emotions in unipolar depressed patients. *J Nerv Ment Dis* 2005;193:331–7.
67. Berthoz S, Ouhayoun B, Parage N. Etude préliminaire des niveaux de conscience émotionnelle chez des patients déprimés et des contrôles. (Preliminary study of the levels of emotional awareness in depressed patients and controls.) *Ann Med Psychol (Paris)* 2000;158:665–72.
68. Cannon W. The mechanism of emotional disturbance of bodily functions. *N Engl J Med* 1928;198:877–84.
69. LeDoux JE. *The Emotional Brain: The Mysterious Underpinnings of Emotional Life*. New York: Simon & Schuster; 1996.
70. Papez J. A proposed mechanism of emotion. *Arch Neurol Psychiatry* 1937;38:725–34.
71. Kluver H, Bucy P. "Psychic blindness" and other symptoms following bilateral temporal lobectomy in rhesus monkeys. *Am J Physiol* 1937; 119:352–3.
72. Weiskrantz L. Behavioral changes associated with ablation of the amygdaloid complex in monkeys. *J Comp Physiol Psychol* 1956;49:381–91.
73. MacLean P. Psychosomatic disease and the "visceral brain": recent developments bearing on the Papez theory of emotion. *Psychosom Med* 1949;11:338–53.
74. MacLean P. Some psychiatric implications of physiological studies on frontotemporal portion of limbic system (visceral brain). *Electroencephalogr Clin Neurophysiol Suppl* 1952;4:407–18.
75. Vogt B, Finch D, Olson C. Functional heterogeneity in cingulate cortex: the anterior executive and posterior evaluative regions. *Cereb Cortex* 1992;2:435–43.
76. Vogt BA, Gabriel M. *Neurobiology of Cingulate Cortex and Limbic Thalamus*. Boston: Birkhauser; 1993.
77. McRae K, Reiman E, Fort C, Chen K, Lane R. Response of "cognitive" division of anterior cingulate cortex to arousal dimension of affective responses. Presented at the 11th Annual Meeting of the Organization for Human Brain Mapping, Toronto, Ontario, Canada, June 2005.
78. Lang PJ, Bradley MM, Cuthbert BN. *International Affective Picture System (IAPS). Instruction Manual And Affective Ratings*. Technical Report. Gainesville, Florida: University of Florida: Center for Research in Psychophysiology; 2001.
79. Thayer J, Lane R. A model of neurovisceral integration in emotion regulation and dysregulation. *J Affect Disord* 2000;61:201–16.
80. Lane R, Ahern G, Schwartz G, Kaszniak A. Is alexithymia the emotional equivalent of blindsight? *Biol Psychiatry* 1997;42:834–44.
81. Kano M, Fukudo S, Gyoba J, Kamachi M, Tagawa M, Mochizuki H, Itoh M, Hongo M, Yanai K. Specific brain processing of facial expressions in people with alexithymia: an H<sub>2</sub><sup>15</sup>O-PET study. *Brain* 2003;126: 1474–84.
82. Paus T. Functional anatomy of arousal and attention systems in the human brain. *Prog Brain Res* 2000;126:65–77.
83. Procyk E, Tanaka Y, Joseph J. Anterior cingulate activity during routine and non-routine sequential behaviors in macaques. *Nat Neurosci* 2000; 3:502–8.
84. Whalen P, Rauch S, Etkoff N, McInerney S, Lee M, Jenike M. Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *J Neurosci* 1998;18:411–8.
85. Morris J, Ohman A, Dolan R. Conscious and unconscious emotional learning in the human amygdala. *Nature* 1998;393:467–70.
86. Williams L, Liddell B, Kemp A, Bryant R, Mearns R, Peduto A, Gordon E. An amygdala-prefrontal dissociation of subliminal and supraliminal fear. *Hum Brain Mapp* 2006;27:661–2.
87. Etkin A, Klemenhagen K, Dudman J, Rogan M, Hen R, Kandel R, Hirsch J. Individual differences in trait anxiety predict the response of the basolateral amygdala to unconsciously processed fearful faces. *Neuron* 2004;44:1043–55.
88. Gazzaniga M, Ivry RB, Mangun GR. *Cognitive Neuroscience—The Biology of the Mind*. New York: Norton; 2002.
89. Gazzaniga M. *The Mind's Past*. Berkeley: University of California Press; 1998.
90. Damasio AR. *Descartes' Error: Emotion, Reason, and the Human Brain*. New York: G.P. Putnam's Press; 1994.
91. Lane R, Nadel L, Allen J, Kaszniak A. The study of emotion from the perspective of cognitive neuroscience. In: Lane R, Nadel L, Ahern G, Allen J, Kaszniak A, Rapcsak S, Schwartz G, editors. *Cognitive Neuroscience of Emotion*. New York: Oxford University Press; 2000.
92. Kihlstrom J. The cognitive unconscious. *Science* 1987;237:1445–52.
93. Fellous J. The neuromodulatory basis of emotion. *Neuroscientist* 1999; Summer:1–15.
94. Anderson A, Phelps E. Is the human amygdala critical for the subjective experience of emotion? Evidence of intact dispositional affect in patients with amygdala lesions. *J Cogn Neurosci* 2002;14:709–20.
95. Biraben A, Taussig D, Thomas P, Even C, Vignal J, Scarabin J, Chauvel P. Fear as the main feature of epileptic seizures. *J Neurol Neurosurg Psychiatry* 2001;70:186–91.
96. Winkelman P, Berridge K. Unconscious emotion. *Current Directions in Psychological Science* 2004;13:120–3.
97. Damasio AR. *Looking for Spinoza: Joy, Sorrow, and the Feeling Brain*. New York: Harcourt; 2003.
98. Farthing GW. *The Psychology of Consciousness*. Englewood Cliffs, NJ: Prentice Hall; 1992.
99. Bechara A, Damasio H, Tranel D, Damasio A. Deciding advantageously before knowing the advantageous strategy. *Science* 1997;275:1293–5.
100. Bush G, Luu P, Posner M. Cognitive and emotional influences in anterior cingulate cortex. *Trends Cogn Sci* 2000;215–22.
101. Augustine J. Circuitry and functional aspects of the insular lobe in primates including humans. *Brain Res Brain Res Rev* 1996;22:229–44.
102. Craig AD. Interoception: the sense of the physiological condition of the body. *Curr Opin Neurobiol* 2003;13:500–5.
103. Mesulam M, Mufson E. Insula of the old world monkey. III: Efferent cortical output and comments on function. *J Comp Neurol* 1982;212: 38–52.
104. Rolls E. Neurophysiology and functions of the primate amygdala. In: Aggleton JP, editor. *The Amygdala: Neurobiological Aspects of*



- Emotion, Memory, and Mental Dysfunction. New York: Wiley-Liss; 1992.
105. Critchley H, Weins S, Rotshtein P, Ohman A, Dolan R. Neural systems supporting interoceptive awareness. *Nat Neurosci* 2004;7:189–95.
  106. Phan K, Wager T, Taylor S, Liberzon I. Functional neuroanatomy of emotion: a meta-analysis of emotion activation studies in PET and fMRI. *Neuroimage* 2002;16:331–48.
  107. Phelps E, O'Connor K, Gatenby J, Gore J, Grillon C, Davis M. Activation of the left amygdala to a cognitive representation of fear. *Nat Neurosci* 2001;4:437–41.
  108. Phillips M, Young A, Senior C, Brammer M, Andrew C, Calder A, Bullmore E, Perrett D, Rowland D, Williams S, Gray J, David A. A specific neural substrate for perceiving facial expressions of disgust. *Nature* 1997;389:495–98.
  109. Reiman E, Lane R, Ahern G, Schwartz G, Davidson R, Friston K, Yun L, Chen K. Neuroanatomical correlates of externally and internally generated human emotion. *Am J Psychiatry* 1997;154:918–25.
  110. Hariri A, Mattay V, Tessitore A, Fera F, Weinberger D. Neocortical modulation of the amygdala response to fearful stimuli. *Biol Psychiatry* 2003;53:494–501.
  111. Tranel T. Electrodermal activity in cognitive neuroscience: Neuroanatomical and neuropsychological correlates. In: Lane R, Nadel L, Ahern G, Allen J, Kaszniak A, Rapcsak S, Schwartz G, editors. *Cognitive Neuroscience of Emotion*. New York: Oxford University Press; 2000.
  112. Idaka T, Omori M, Murata T, Kosaka H, Yonekura Y, Okada T, Sadato N. Neural interaction of the amygdala with the prefrontal and temporal cortices in the processing of facial expressions as revealed by fMRI. *J Cogn Neurosci* 2001;13:1035–47.
  113. Rainville P, Duncan G, Price D, Carrier B, Bushnell M. Pain affect encoded in human anterior cingulate but not somatosensory cortex. *Science* 1997;277:968–71.
  114. Cohen RA, Paul R, Zawacki T, Moser D, Sweet L, Wilkinson H. Emotional and personality changes following cingulotomy. *Emotion* 2001;1:38–50.
  115. Drevets WC, Raichle M. Reciprocal suppression of regional cerebral blood flow during emotional versus higher cognitive processes: implications for interactions between emotion and cognition. *Cogn Emot* 1998;12:353–85.
  116. Eisenberger NI, Lieberman M, Williams K. Does rejection hurt? An fMRI study of social exclusion. *Science* 2003;302:290–2.
  117. Hutcherson CA, Goldin P, Ochsner K, Gabrieli J, Feldman Barrett L, Gross J. Attention and emotion: does rating emotion alter neural responses to amusing and sad films? *NeuroImage* 2005;27:656–68.
  118. Ochsner K, Bunge S, Gross J, Gabrieli J. Rethinking feelings: an fMRI study of the cognitive regulation of emotion. *J Cogn Neurosci* 2002;14:1215–29.
  119. Taylor S, Phan K, Decker L, Liberzon I. Subjective rating of emotionally salient stimuli modulates neural activity. *Neuroimage* 2003;18:650–9.
  120. Fonagy P, Gergely, G, Jurist E, Target M. *Affect Regulation, Mentalization and the Development of the Self*. New York: Other Press; 2002.
  121. Lane R, Fink G, Chua P, Dolan R. Neural activation during selective attention to subjective emotional responses. *Neuroreport* 1997;8:3969–72.
  122. Weiskrantz L. Blindsight: implications for the conscious experience of emotion. In: Lane R, Nadel L, Ahern G, Allen J, Kaszniak A, Rapcsak S, Schwartz G, editors. *Cognitive Neuroscience of Emotion*. New York: Oxford University Press; 2000.
  123. Gusnard D, Akbudak E, Shulman G, Raichle M. Medial prefrontal cortex and self-referential mental activity: relation to a default mode of brain function. *PNAS* 2001;98:4259–64.
  124. Ochsner K, Knierim K, Ludlow D, Hanelin J, Ramachandran T, Glover G, Mackey S. Reflecting upon feelings: an fMRI study of neural systems supporting the attribution of emotion to self and other. *J Cogn Neurosci* 2004;16:1746–72.
  125. Frith C, Frith U. Interacting minds—a biological basis. *Science* 1999;286:1692–5.
  126. Frijda N. *The Emotions*. Cambridge, UK: Cambridge University Press; 1986.
  127. Johnson S, Baxter L, Wilder L, Pipe J, Heiserman J, Prigatano G. Neural correlates of self-reflection. *Brain* 2002;125:1808–14.
  128. Voegeley K, Bussfeld P, Newen A, Herrmann S, Happe F, Falkai P, Maier W, Shah N, Fink G, Zilles K. Mind reading: neural mechanisms of theory of mind and self-perspective. *Neuroimage* 2001;14:170–81.
  129. Happé F, Ehlers S, Fletcher P, Frith U, Johansson M, Gillberg C, Dolan R, Frackowiak R, Frith C. “Theory of mind” in the brain. Evidence from a PET scan study of Asperger syndrome. *Neuroreport* 1996;8:197–201.
  130. Price J, Carmichael S, Drevets W. Networks related to the orbital and medial prefrontal cortex; a substrate for emotional behavior? *Prog Brain Res* 1996;107:523–36.
  131. Drevets W. Prefrontal cortical-amygdalar metabolism in major depression. *Ann N Y Acad Sci* 1999;877:614–37.
  132. Buchanan S, Valentine J, Powell D. Autonomic responses are elicited by electrical stimulation of the medial but not lateral frontal cortex in rabbits. *Behav Brain Res* 1985;18:51–62.
  133. Lane R, Reiman E, Ahern G, Thayer J. Activity in medial prefrontal cortex correlates with vagal component of heart rate variability during emotion. *Brain Cogn* 2001;47:97–100.
  134. Gianaros P, Van Der Veen F, Jennings J. Regional cerebral blood flow correlates with heart period and high-frequency heart period variability during working-memory tasks: implications for the cortical and subcortical regulation of cardiac autonomic activity. *Psychophysiology* 2004;41:521–30.
  135. Matthews SC, Paulus MP, Simmons AN, Nelesen RA, Dimsdale JE. Functional subdivisions within anterior cingulate cortex and their relationship to autonomic nervous system function. *Neuroimage* 2004;22:1151–6.
  136. Ongur D, An X, Price J. Prefrontal cortical projections to the hypothalamus in macaque monkeys. *J Comp Neurol* 1998;401:480–505.
  137. Hornak J, Bramham J, Rolls ET, Morris RG, O'Doherty J, Bullock PR, Polkey CE. Changes in emotion after circumscribed surgical lesions of the orbitofrontal and cingulate cortices. *Brain* 2003;126:1691–712.
  138. Lieberman M, Eisenberger N, Crockett M, Tom S, Pfeifer J, Way B. Putting feelings into words: affect labeling disrupts amygdala activity to affective stimuli. *Psychol Sci* 2007;18:421–8.
  139. Greenberg L. *Emotion-Focused Therapy: Coaching Clients to Work Through Their Feelings*. Washington, DC: American Psychological Association; 2002.
  140. Cowey A, Stoerig P. The neurobiology of blindsight. *Trends Neurosci* 1991;14:140–5.
  141. Weiskrantz L. *Blindsight*. Oxford, UK: Oxford University Press; 1986.
  142. Paillard JF, Michel F, Stelmach G. Localization with content: a tactile analogue of “blindsight.” *Arch Neurol* 1983;40:548–51.
  143. Schwartz GE, Bell IR, Dikman ZV, Fernandez M, Kline JP, Peterson JM, Wright KP. EEG responses to low-level chemicals in normals and caecemics. *Toxicol Ind Health* 1994;10:633–43.
  144. Geschwind N. Disconnection syndromes in animals and man. *Brain* 1965;88:237–94.
  145. Munakata Y, Pfaffly J. Hebbian learning and development. *Dev Sci* 2004;7:141–8.
  146. Tabibnia G, Zaidel E. Alexithymia, interhemispheric transfer, and right hemispheric specialization: a critical review. *Psychother Psychosom* 2005;74:81–92.
  147. Schäfer R, Popp K, Jörgens S, Lindenberg R, Franz M, Seitz R. Alexithymia-like disorder in right anterior cingulate infarction. *Neurocase* 2007;13:201–8.
  148. Ray R, Ochsner K, Cooper J, Robertson E, Gabrieli J, Gross J. Individual differences in trait rumination and the neural systems supporting cognitive reappraisal. *Cogn Affect Behav Neurosci* 2005;5:156–68.
  149. Siegle G, Steinhauer S, Thase M, Stenger V, Carter C. Can't shake that feeling: event-related fMRI assessment of sustained amygdala activity in response to emotional information in depressed individuals. *Biol Psychiatry* 2002;51:693–707.
  150. Siegle G, Steinhauer S, Carter C, Thase M. Neural correlates of cardiac reactivity during emotional information processing in unipolar depression: a tale of 3 cingulates. In: Gianaros P, chair. *Neuroimaging of Autonomic-Cardiac Reactivity in Health and Disease*. Annual Meeting of the American Psychosomatic Society, Denver, Colorado, March 2006.
  151. Erdelyi M. The unified theory of repression. *Behav Brain Sci* 2006;29:499–511.
  152. Herbart J. *Psychologie als Wissenschaft neu gegründet auf Erfahrung, Metaphysik und Mathematik*, Vols 1 and 2. Unzer; 1824–1825.
  153. Levenson RW. Human emotion: a functional view. In: *The Nature of*

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- Emotion—Fundamental Questions. In: Ekman P, Davidson RJ, editors. New York, Oxford University Press; 1994.
154. Pinquart M, Sorensen S. Differences between caregivers and non-caregivers in psychological health and physical health: a meta-analysis. *Psychol Aging* 2003;18:250–67.
  155. Brown G, Harris T. *Life Events and Illness*. New York: Guilford; 1989.
  156. Williams V, Williams R. *Life-Skills*. New York: Times Books; 1997.
  157. Habib G. Reappraisal of heart rate as a risk factor in the general population. *Eur Heart J Suppl* 1999;1:H2–H10.
  158. Clyman RB. The procedural organization of emotions: a contribution from cognitive science to the psychoanalytic theory of therapeutic action. *J Am Psychoanal Assoc* 1991;39(Suppl):349–82.
  159. Lane R, Schwartz G. Levels of emotional awareness: implications for psychotherapeutic integration. *Journal of Psychotherapy Integration* 1992;2:1–18.
  160. Gendlin ET. *Focusing*. New York: Bantam Books; 1978.
  161. Teasdale J. Emotional processing, three modes of mind and the prevention of relapse in depression. *Behav Res Ther* 1999;37(Suppl 1): S53–S77.
  162. Linehan M. *Skills Training Manual for Treating Borderline Personality Disorder*. New York: Guilford Press; 1993.