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Four Systems for Emotion Activation: Cognitive and Noncognitive Processes

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The significant role of emotions in evolution and adaptation suggests that there must be more than 1 mechanism for generating them. Nevertheless, much of current emotion theory focuses on cognitive processes (appraisal, attribution, and construal) as the sole, or primary, means of eliciting emotions. As an alternative to this position, the present model describes 4 types of emotion-activating systems, 3 of which involve noncognitive information processing. From an evolutionary–developmental perspective, the systems may be viewed as a loosely organized hierarchical arrangement, with neural systems, the simplest and most rapid, at the base and cognitive systems, the most complex and versatile, at the top. The emotion-activating systems operate under a number of constraints, including genetically influenced individual differences. The hierarchical organization of the systems for generating emotions provides an adaptive advantage.

Rafe's friends always describe him as a happy person. He likes to play tennis and finds great enjoyment in watching the top professionals play the game. After watching his favorite player win in the semifinals of a grand prix tennis tournament, Rafe contentedly stood in line under a hot August sun waiting to get a cool drink. As the glow of his vicarious victory faded, the heat and humidity became more and more oppressive. Suddenly, Rafe felt a piercing pain from a blow to his lower back. Rafe turned rapidly with an angry expression and clenched fist. Rafe saw that he had been hit by Rebecca, a person with hemiplegia whose wheelchair had gone out of control and caused her to crash into Rafe and to spill her drink on her dress. Rafe's understanding that the cause of his pain was an uncontrollable event that had embarrassed Rebecca immediately changed his anger to sadness and sympathy. Though still in pain, his happy nature surfaced, and he began helping Rebecca recover from the accident.

According to the present model, Rafe's normally happy disposition is a function of a genetically influenced neural system that operates more or less continuously to generate his characteristic emotional state. The background neural activity responsible for Rafe's trait emotionality (i.e., happy mood), the changes in his emotions system that were due to neurochemical activity instigated by the ambient temperature, the affective/pain processes that led to anger, and the amplification of his anger by sensorimotor/expressive actions required no cognitive mediation. All of these emotion-activating and regulatory systems can operate in very young infants, probably before they are capable of engaging in cognition based on representational processes and memory (Harris, 1983b).

Rafe's perception of the visual scene engaged the emotion-activating processes of his cognitive systems, resulting in appraisals, causal attributions, judgments, and appropriate emotion responses. In the present scenario, his perception of the visual scene led him to attribute the cause of his pain to an uncontrollable event that had actually embarrassed and hurt the causal agent, Rebecca. These appraisal and attributional processes changed his anger to sadness and sympathy. The cognitive systems for emotion activation accounted for the third emotion that Rafe experienced. His characteristically happy mood and the pain-activated anger were products of noncognitive emotion-activating systems, which together with cognition are capable of generating emotion responses for all contingencies.

The nascent idea of alternative routes to emotion activation has been in the shadows of a growing set of theories that focus primarily on cognitive processes. Yet, emotions are so basic to the evolution and adaptation of human beings (Darwin, 1872/1965; Hamburg, 1963; Izard, 1989; Nesse, 1990; Plutchik, 1980; Zajonc, 1980) that it would seem obvious that evolution and the brain's penchant for redundancy (Mayr, 1988; Valenstein, 1973) would have guaranteed the existence of multiple processes for generating them. This compelling notion has not yet motivated a comprehensive theory that attempts to specify the various emotion-activating processes. This is true despite the fact that there are a number of theorists who acknowledge different types of emotion activators and a growing body of relevant data (for reviews, see Izard, 1990; Zajonc, Murphy, & Inglehart, 1989). The increasingly wide acceptance of emotions as motivational processes that influence cognition and action (Anderson, 1989; Harris, 1989; Lazarus, Kanner, & Folkman, 1980; G. A. Miller & Johnson-Laird, 1976; Neisser, 1976) underscores the need for a theoretical framework for understanding their activation.

Another reason for a comprehensive model of emotion acti-

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vation is the need to gain some perspective on the importance of cognition in generating emotions. There is a growing interest in research on emotion–cognition relations, but it does not seem possible to analyze or predict such relations until there is a framework for examining the relative contributions of cognitive and noncognitive elicitors of emotions. If, on the one hand, cognition were always the cause as well as a constituent of emotion (Lazarus, 1991; Ortony, Clore, & Collins, 1988), there would be some restrictions on the ways that cognition and emotion could relate. Emotions could not be considered as qualitatively distinct independent variables (Izard, 1965; Zajonc, 1980). On the other hand, if emotion can be defined as something other than a cognition-dependent phenomenon and if cognition is but one of several types of emotion-activating processes, then there can be a greater range of relations between emotion and cognition. For example, noncognitively activated emotions could generate and interact with cognition.

A prominent emotion theorist (Lazarus, 1991) has continued to maintain that "cognitive mediation is a necessary condition of emotion" (p. 356). He argued that even drug-induced emotions and emotions processed entirely through subcortical structures and pathways are mediated by cognition. Indeed, Lazarus held that discussion of noncognitive activators of emotion "keeps us from asking the right questions, which have to do with the kinds of cognitive activities that are causally involved in the emotion process" (p. 357). To maintain this position, Lazarus (1984) held that the simplest perceptual phenomenon, inputs without full-fledged denotations, can constitute cognitive appraisal that generates meaning, evaluative judgment, and emotion.

Another cognitive theory of emotion takes a stand similar to that of Lazarus (1984, 1991). It phrases the causal process a bit differently, maintaining that emotions are a function of the person's construal of salient features of the context (Ortony et al., 1988). The authors of this theory held that of four types of data relating to emotions, only language and self-report are to be considered in the generation of emotion experiences. They "largely ignore behavioral and physiological evidence" on the grounds that they are "consequences or concomitants of emotion states" (p. 14). A similar position is that "emotions result from meanings, and meanings, to a large extent, from inferred consequences or causes" (Frijda, 1986, p. 310; also see Frijda, 1988).

Although Frijda (1986) and other cognitively oriented emotion theorists have acknowledged that brain stimulation can elicit emotion, neither this nor any other noncognitive variable plays a significant role in their treatment of emotion activation. For example, Frijda discussed "neurophysiological mechanisms involved in emotional phenomena" as " 'conditions' for emotion" (p. 379) rather than as a neural system capable of independently activating emotions.

These strong views on the peremptory role of cognition in emotion activation raise two critical questions. First, can cognition be defined by some conceptual boundaries such that it can be understood as something more specific to mental representation and memory than the very broad concept of information processing? Second, if cognition is conceived as antecedent to emotion, then what drives cognition?

Information Processing Versus Cognition in Emotion Activation

Contrary to Lazarus's (1984, 1991) position, some theorists have maintained that it is not heuristic to define cognition so broadly as to include all information processing (Hoffman, 1985; Izard, 1989, 1992; Zajonc, 1984). Several kinds of information processing are going on in all organisms all the time. Surely it is not helpful to conceive of information processing in DNA molecules as cognition. Although all cases of emotion activation involve information processing, in some cases, according to the present model, the information processing that generates emotion is noncognitive.

A challenge for cognitive scientists is to draw some working boundaries around the domain of cognition. Without such boundaries and without clear specification of the mechanism for cognitive mediation, arguments regarding cognition as *the* necessary cause of emotion will be without strong foundation.

Hoffman (1985) has attempted to resolve the question of the necessity of cognition in emotion activation by delineating several different modes of information processing that mediate affect arousal. He discussed these modes in the order in which they emerge in development. The first mode to emerge, direct response to the physical features of a stimulus, may not be a function of cognition if any restrictions are put on that term. This first mode of emotion activation, according to Hoffman, requires only "registration of the stimulus event and a minimum of perceptual organization" (p. 246).

Although only a few theorists take the extreme position that cognition is a necessary antecedent of all emotion experiences, most theories of emotion activation and most of the related empirical research are concerned only with cognitive concepts. From the existing literature, students of emotions can gain the impression that an understanding of cognitive operations such as appraisal and attributional processes gives them an adequate knowledge of the causes of emotions. There are several theories that provide a good description of these processes (e.g., Frijda, 1986; Scherer, 1988; Smith & Ellsworth, 1985). Some of these theories allow for the possibility of other avenues to emotion, but they inspire only studies of cognitive causes and thus give the impression that cognition accounts for virtually all the emotions of human experience or, at least, the most important ones.

Weiner's (1985) social–cognitive (attribution) theory of emotions is an exception to the foregoing characterization of cognitive theories of emotions. His theory explains the generation of emotions as a function of causal ascriptions, but it acknowledges that certain emotions (e.g., excitement, joy, and disgust) are not necessarily, or even primarily, mediated by perceptions of causality.

Perhaps the most significant issue here is not whether cognition is the exclusive determinant of emotions but whether there are viable alternative hypotheses for emotion activation and whether noncognitive activators account for a substantial amount of the emotions experienced in daily life. I shall argue that there are numerous specific activators of emotions and that they fall into four broad classes: neural (noncognitive evaluative processes), sensorimotor, motivational, and cognitive. The classes of activators constitute four separate but highly interac-

tive systems. Thus, cognitive operations constitute one of the four classes of emotion-activating processes, but whether cognition accounts for more emotions than any other class of activators, or more than all the other classes combined, is an empirical question.

I shall discuss possible limitations on cognition as the primary cause of emotion. This discussion will involve a consideration of individual differences that might be expected to influence appraisal processes and emotionality through noncognitive information processing.

On the Need for a Concept of Noncognitive Information Processing

For the purpose of discussing cognition as an emotion-activating system and for studying emotion–cognition relations, I suggest some working boundaries or distinctions among varieties of noncognitive and cognitive information processes. Some such distinctions are necessary to consider the possibility that emotions are generated by truly different processes.

Surely, part of the difference between cognitive and biosocial theories on the issue of emotion activation is a function of their different conceptions of cognition and their different views on the heuristic value of conceptually separating emotion and cognition, information processing and cognition, and noncognitive and cognitive information processes. The problem of definition or boundary setting is made more complex by the existence of some seemingly overinclusive definitions of cognition (see the Lazarus-Zajonc debate, Lazarus, 1984; Zajonc, 1984). Furthermore, there are many different types of cognition: automatic as contrasted with deliberate (Schneider & Shiffrin, 1977; Shiffrin & Schneider, 1977); conscious as contrasted with unconscious, preconscious, and subconscious (Kihlstrom, 1987; Kihlstrom & Hoyt, 1990); cognition at different levels of awareness (Bower, 1990); and cognition in repression and dissociation (see Singer, 1990). Building on earlier work on subliminal perception and the distinctions between forms of episodic memory, Kihlstrom (1990) has made a case for implicit cognition (implicit perception, implicit learning, and implicit memory) that might lead to "implicit emotion." There is impressive evidence and argument for all the foregoing types of cognitive processes, and, considered together, they make for a very broad conception of cognition.

Even with cognition defined to include all the foregoing types of mental activities, however, it is misleading to equate it with information processing. The study of information processing in the genes of *Drosophila melanogaster* has made great contributions to understanding the human genome, but it has not enlightened us on human mental life. Not only does it seem proper to exclude the decoding of information in amino acids, enzymes, and genes from the cognition of cognitive psychology, it may also be appropriate to exclude that which guides reflexive, instinctive, and biologically prepared or genetically disposed behavior. Thus, a central thesis of the present article is that cognition, defined as dependent on some form of learning- or experience-based memory, does not include all the forms of information processing that lead to emotion.

Information Processing as a Continuum or Chain of Types

Clearly, information processing consists of several types or levels of data decoding and synthesis, ranging from that which leads to the color of an eye to that which produces a Mona Lisa or a theory of relativity. All these kinds of information processing may lie along a single continuum. To paraphrase Sinnott (1966), who can say when the information processing involved in the chick's embryological development ends and that which mediates its behavior and mental life begins? When it starts pecking its way out of the shell? When it perceives the mother hen and imprinting results in following behavior? When it imitates her scratching movements in search of food? When it flees from the shadow of a hawk? When its behavior is guided primarily by sensory data from the environment?

Some of the foregoing chick activities, including fear behavior and flight, are guided, in part, by information encoded in the genes and, in part, by sensory input from the environment. Therein may lie a clue to a boundary or distinction between noncognitive and cognitive information processing. One possibility for marking the boundary is to identify behaviors that are instinctive, that are instinctlike, or that are elicited by particular environmental features (Schneirla, 1965), biologically prepared or genetically disposed stimuli (Rozin & Fallon, 1987; Seligman, 1970), or "natural clues" (Bowlby, 1973). Such information leads to a number of emotion responses in animals, young infants, and children (Bowlby, 1973; Gray, 1971; Izard, Hembree, & Huebner, 1987; Stenberg & Campos, 1990). Infants of 3 weeks smile at a human face and display anger in pain at 8 weeks and anger at restraint at 16 weeks. In all of these conditions, infants undoubtedly process information that leads to the emotion response, but such processing does not constitute cognition as defined here. The infants detect a pattern of light, lines, or neurochemical changes and experience an emotion-eliciting pattern of stimulation (which is probably mediated subcortically), but these phenomena do not require cognitive representation, memory, matching, or comparison processes. Infants of 3 to 8 weeks of age are probably incapable of such mentation (Harris, 1983b).

The arbitrariness of any answer to Sinnott's (1966) question as to where embryological growth ends and cognition and behavior begin suggests the reasonableness of a concept of a single continuum of information processing. Whether the underlying phenomena are points on a continuum or discrete classes, for purposes of discussing cognitive versus noncognitive processes and considering cognition as an activator and correlate of emotion, I propose four differentiable sorts of information processing: cellular, organismic, biopsychological, and cognitive. These may be conceived as segments on the grand continuum, but because my goal here is to show significant differences among them, I refer to them as categories or types.

The first three of the foregoing categories involve types of noncognitive information processing. The cellular category is exemplified by information processing in enzymes and genes. Such processing has no direct relation to sensory input or the cognitive processes that depend on sense data. The information processed at the cellular level was encoded through natural selection in the evolution of the species. Cellular processing

does not directly affect cognition and action. Cellular information processing does play a significant role in determining emotion thresholds and proneness to experience certain emotions, and thus it is a major determinant of an individual's characteristic mood. Furthermore, as I argue later, the individual differences rooted in cellular information processing place constraints on organismic, biopsychological, and cognitive information processing.

Organismic information processing that leads to emotion also involves biological information coded in the genes during the course of evolution. In some cases, biological information is processed with little or no sensory information from exteroceptors. However, organismic information processing often involves sense data from interoceptors, and the primary sources of such information are physiological drive states. Examples of emotion activation through organismic information processing are pain-activated anger (Berkowitz, 1990; Izard et al., 1987) and taste-elicited disgust (Steiner, 1979).

Biopsychological information processing that activates emotions involves an interaction of biological information processing and the processing of information derived through learning or experience, that is, an interaction between organismic information processing and acquired knowledge (cognition). The processes involved in the interaction of these two types of information and the subsequent effect on emotion activation may be roughly analogous to the processes involved in the interaction of implicit and explicit memory and the subsequent effect on conscious processes (Kihlstrom, 1980; Kihlstrom & Hoyt, 1990). Kihlstrom showed that memorized words that were rendered nonrecallable (or relegated to implicit memory) through posthypnotic suggestion were still available for use in response to words previously associated with them. In similar fashion, genetically coded information relevant to evolutionarily significant stimuli, though unavailable to consciousness, interacts with information derived through cognitive processes to determine the specific emotion response.

Although biopsychological information processing requires cognitive evaluation of sensory input from the environment, the critical database is biological information from evolution. (For a discussion of genetic constraints on "what can be learned, hence what can be attended to, and thence what can be perceived," see Shepard, 1984, p. 432). This category of information processing is exemplified in biologically prepared or genetically disposed fear and disgust responses (Lanzetta & Orr, 1986; Mineka, Davidson, Cook, & Keir, 1984; Rozin & Fallon, 1987; Seligman, 1970).

Evidence suggests that it is the critical contribution of biological information to prepared or evolutionarily influenced fear responses that makes their acquisition more likely and their extinction slow and difficult. Although McNally (1987) argued that the data relating to acquisition and extinction are inconclusive, he acknowledged that fears of evolutionary significance are overrepresented in human phobias. Following Bennett-Levy and Marteau (1984), he concluded that humans may be biologically prepared to learn to fear certain perceptual features embedded in phobic stimuli. This suggests that emotions activated by such stimuli might require only feature detection. Even when more complex cognition is involved in biopsychological information processing that leads to emotion, cognition may not be the dominant determinant of the emotion response. The primary cause may be a genetically determined predisposition.

Biopsychological information processing could be considered a special subtype of cognition. It can involve a variety of cognitive processes. Perhaps the choice of category label should follow a determination of which type of information is dominant in determining a specific emotion response. In any case, emotion activation through the interaction of biological information processing and the processing of individually acquired information invokes the idea of an information-processing continuum.

The cognitive category includes all mental processes that depend on acquired representations, those representations derived from the learning and experience of the individual. Cognition, as defined in differential emotions theory (Izard & Malatesta, 1987) and in the present model of emotion activation, begins at that point on the information-processing continuum where learning or experience produces mental representations and memory sufficient to mediate comparison processes and discrimination. It is when these, as well as higher order mental processes, are involved in assessing stimulus information that cognition can, though not necessarily, play a dominant role in emotion activation. Possibly the cognitive category (or cognitive segment of the information-processing continuum), as defined here, is sufficiently broad to include the types or levels of information processing of interest to cognitive psychologists—whether simple discrimination or complex symbol manipulation; automatic or deliberate; conscious or unconscious, preconscious, or subconscious; and whether in repression or dissociation.

Such a range of differentiable types of noncognitive information processing has not been discovered (or much sought), but it is known to vary from simple to complex. Something of the extent of its complexity is illustrated by Lorenz's (1965) description of the instinctive behavior of the young swift. Although reared in a cave too narrow for it to extend its wings and too shallow for it to obtain a sharp retinal image, the swift can, on its first flight, assess distances by the parallactic shift of the objects' images; cope with air resistances, upcurrents, and air pockets; recognize and catch prey; and make a precise landing in a suitable place.

I emphasize that the foregoing treatment of cognition is a special-purpose one. Its primary function is to facilitate the discrimination of cognitive and noncognitive information processing and the discussion of emotion–cognition relations and cognition as an emotion-activating system.

On Defining Emotion

Before presenting a multisystem–multimodal model of emotion activation, it is necessary to say something about what is being activated. This brings us to a very thorny issue, defining emotion, and it will not be resolved to everyone's satisfaction. Although an attempt at a complete definition would certainly create controversy, it is possible to identify basic characteristics of emotion on which there is considerable agreement.

Everyone agrees that emotions involve particular neural processes. Several neuroscientists have identified mechanisms,

pathways, and neurotransmitters for emotion in general or for specific emotions (e.g., Aggleton & Mishkin, 1986; Kling, 1986; LeDoux, 1987; Panksepp, 1986; Rolls, 1986). They are not in complete agreement, but they share considerable common ground. They agree that the neural substrates of emotions are best described as circuits or networks of structures and not as localized brain centers. Two pathways for the activation of fear are depicted in Figure 1. The important point here is that particular neural substrates can be identified and that an emotion is not so general a process that all brain structures and neurotransmitters are equally involved. Thus, as research technology improves, it should become possible to index emotions in terms of increasingly specific neural systems.

A substantial number of emotion researchers agree that emotion includes an expressive or motor component or, at least, an efferent activity in the central nervous system (e.g., Ekman, 1984; Izard, 1991; Lang, 1984; Leventhal, 1984; Plutchik, 1980; Tomkins, 1962). If an action tendency is conceived as involving expressive or efferent processes, several other theorists could be included in the consensus on this point (e.g., Arnold, 1960; Bull, 1951; Cacioppo, Martzke, Petty, & Tassinary, 1988; Frijda, 1986; Lang, 1984). In any case, it is clear that many emotion researchers agree that particular expressive movements or action tendencies help define emotion. Furthermore, some of these investigators have developed precise and objective methods, including observational (Ekman & Friesen, 1978; Izard, 1979) and psychophysiological techniques (Cacioppo & Tassinary, 1990; Lang, 1979) for identifying this component. Various aspects of the efferent–expressive component of emotion are identified in the following list:

Neuromuscular Activity
Expressive Behavior
1. Central nervous system efferent activity
2. Prototypical facial expressions
3. Expression components
4. Posture
5. Vocal expression
6. Head and eye movement
7. Muscle action potentials.

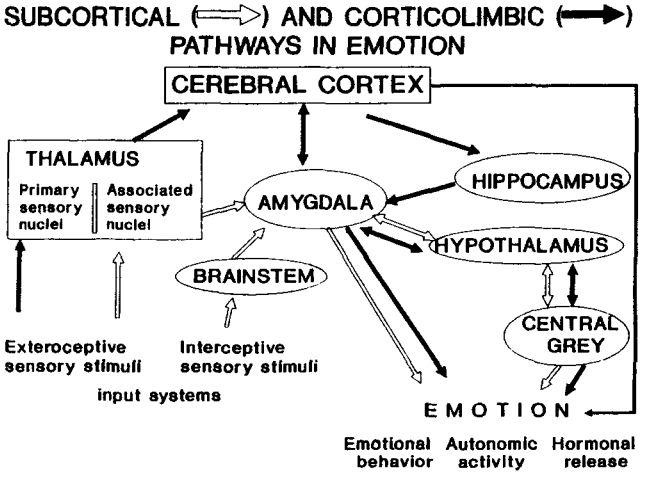

Figure 1. Neural substrates of emotion: subcortical and corticolimbic pathways in emotion activation.

Finally, all researchers, from clinical investigators to neuroscientists, agree that emotions register in consciousness. Indeed, since William James (1884) identified the feeling of certain bodily changes as the quintessence of emotion, many writers use the terms emotion, feeling, and emotion experience interchangeably. Although James made a persuasive case for considering the experiential component as the quintessence of emotion, the use of the term emotion when referring only to emotion experience ignores the other components and is, thus, imprecise and misleading.

There is disagreement on the nature of the experiential component of emotion, but most definitions include one or more of the following:

1. Motivation
2. Action readiness
3. Action tendency
4. Perceptual selectivity
5. Cues for cognition and action
6. Feeling state.

Although all these terms may not apply equally to all emotion experiences, I propose that all of them are acceptable ways of identifying aspects of the third component of emotion. One could argue that the common denominator among the descriptors of emotion experience is a noncognitive motivational condition or process in consciousness that normally, after language acquisition, can be accessed and verbally reported, albeit imprecisely. However, when the cognition associated with an emotion experience has been repressed or relegated to a lower level of awareness, retrieval (accessing or labeling) may require the aid of appropriate cues (Bower, 1990).

One possible exception to the foregoing reportability rule is the individual with a repressor personality type, who is apparently capable of dissociating the conscious aspects of emotions, especially the negative emotions in anxiety, even when psychophysiological indexes indicate that there is emotion arousal (see Schwartz, 1990; Weinberger, 1990). There are two possibilities here. There may be no specific emotion experience because the arousal is undifferentiated, or an emotion experience is present but special retrieval cues and procedures are necessary to obtain a veridical verbal report.

Although there may be exceptions, it seems reasonable to stay with the notion that emotion experience, or the third component of emotion, can be identified as a motivational condition or process in consciousness that manifests itself as action readiness, action tendency, a biasing of perception, cues for perceptual–cognitive processes, or a feeling state. In this context, motivational condition is conceived as a property inherent in the activity of the neural substrates of emotion or as a direct product of neural processes.

In differential emotions theory (Izard, 1971, 1977), the theory underlying the present model of emotion activation, an emotion is distinguished from a drive state. As Tomkins (1962) has argued, drives, such as hunger, thirst, sex, and the need to eliminate, are cyclical in nature, and each one is associated with and satisfied by a relatively restricted range of stimuli. The satisfaction of a drive state requires activity in peripheral organs

dedicated to, or at least associated with, the drive system. Drives provide specific information regarding the time and place that something needs to be done and cues a relatively specific set of responses. In contrast, an emotion has no temporal cycle, is not dependent on peripheral physiological processes, can be associated with a virtually limitless variety of phenomena, and can motivate an equally wide range of cognitions and actions (Tomkins, 1962). In contrast to emotion feelings, feelings that derive from drive states are influenced by cyclical input from visceral organs innervated by the autonomic nervous system (ANS). Time is a stable source of variance in the intensity of drive states, whereas any number of variables (e.g., stage of development, nature of the eliciting stimulus, unconscious cognitive processes, or personality defense mechanisms such as repression) may, independently of time, contribute to variations in the intensity of emotions. Mood is substantively synonymous with emotion but may be used to refer to emotion that is sustained over a relatively long period of time (cf. Mandler, 1984). In differential emotions theory, affect and motivation are interchangeable terms that refer to all motivational phenomena—emotions, drives, and affective–cognitive structures.

On Distinguishing Emotion and Cognition

Fundamentally, emotion is about motivation—positive and negative feelings, readiness or tendency to cope, and cues for cognition and action. Cognition is about knowledge—learning, memory, symbol manipulation, thinking, and language. Perception, when it leads to representation and memory, is clearly part of cognition. Subcognitive perception, or perception that does not result in memory (cf. Zajonc, 1980), may operate primarily in the service of the emotions system.

The foregoing working definitions of emotion and cognition describe them as distinct systems. An important aspect of the distinction between these two domains was recognizing the motivational state that follows from neurochemical activity as the outer boundary of the emotion process, a boundary that separates the qualitatively distinct phenomena of emotion and cognition. Because emotion experience has motivational and cue-producing functions and because cognition is often essential in guiding person–environment transactions, the interaction of emotion and cognition is common. Emotion–cognition interactions occur in all the many coping activities that require stimulus appraisal and judgment before action. Such interactions lead to affective–cognitive structures, the frequency and nature of which are limited only by the joint capacities of the emotions and cognitive systems (Izard, 1977, 1992).

However, despite the readiness with which emotion and cognition interact, some emotion-eliciting stimuli (e.g., odors) never form strong links to cognition. Odors produce a range of emotion responses in animals, including humans (Ehrlichman & Bastone, 1992; Moncrieff, 1966). Yet, Engen (1987) and others have shown that odors are not processed and stored in memory in a semantically cohesive way but rather in essentially nonverbal terms. "There is no pure recall of odor perceptions using a name as paired associate. . . . The main function of the sense of smell, then, is not to recall odors for cognitive reasons, but to respond to odors actually encountered" (p. 503).

Thus, olfaction provides an example of how the brain, specifically the primitive smell-brain, can process and store information in memory in a nonverbal, and perhaps in some cases noncognitive, fashion. Richardson and Zucco (1989) have suggested that memory for odors may be encoded in a rudimentary sensory form, such as sensory traces, and may be processed in a system that is functionally independent of systems of visual images and verbal representations. It may be essentially through noncognitive processes that odor plays a significant role in mother–infant attachment and maternal nurturance of the young of a number of species (see Schneirla, 1965).

Although emotions and cognition are highly interactive and have reciprocal causal relations, there are heuristic advantages in distinguishing between them (Hilgard, 1980; Izard, 1965; Zajonc, 1980). A principle advantage is a theoretical framework that enables the study of cognition as a distinct independent variable in emotion activation and emotion as an independent variable in emotion–cognition relations.

As argued elsewhere, both evolutionary and developmental considerations support the notion that the emotions system preceded the cognitive system in evolution and outpaces it in ontogeny (Izard, 1972; Izard & Malatesta, 1987; Zajonc, 1980). It is highly adaptive for animals to be able to feel before they think, as in the case where pain elicits withdrawal or pain-induced anger motivates defensive actions. It is equally adaptive for the preverbal infant (as young as 3 weeks) to smile at caregivers and begin establishing attachment bonds that greatly increase the chances of survival. The weight of cross-cultural and developmental data indicates that a number of emotion expressions are innate and universal and emerge before the infant is capable of cognition as defined here (Ekman, Sorenson, & Friesen, 1969; Izard, Huebner, Risser, McGinnes, & Dougherty, 1980; Izard & Malatesta, 1987). One can argue that these expressions are not necessarily connected to emotion experiences, particularly when emotion experience is defined to include cognition (Lewis & Michalson, 1983), but with emotion experience as defined in differential emotions theory it is also possible to make a case for innate expression–feeling congruence (Izard & Malatesta, 1987). That the young infant's smile is accompanied by positive feeling is suggested by the dramatically different expression and behavior that follow pain or restraint and by the fact that young infants' facial expressions are morphologically the same as those of adults (Izard et al., 1980).

Four Systems for Emotion Activation: Synopsis of the Model

The multisystem model of emotion activation is depicted in Figure 2. The four types of systems that activate emotions are neural, sensorimotor, motivational, and cognitive. All emotion activation processes necessarily involve a neural system, but neural systems can activate emotions independently of the other types of activating systems. In the neural systems, the generation of emotions can be explained in terms of the activity of certain neurotransmitters and brain structures. The involvement of specific neurotransmitters is well established for the complex patterns of emotions involved in depression and anxiety (Redmond, 1985; Whybrow, Akiskal, & McKinney, 1984).

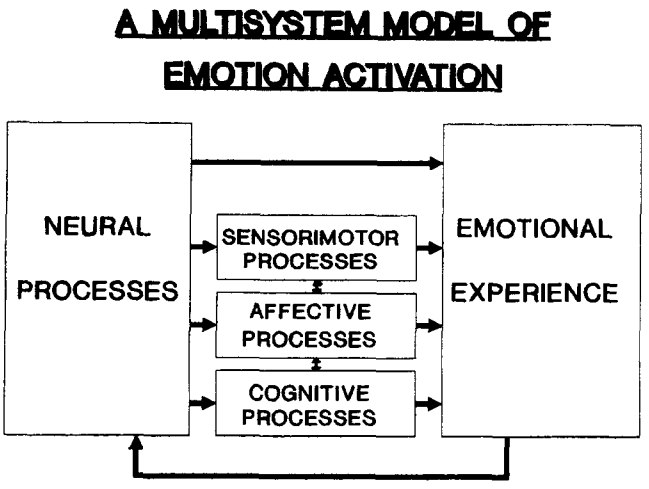

Figure 2. A multisystem model of emotion activation.

In the sensorimotor systems, emotions are activated by efferent or motor messages, and the process may include afferent feedback from muscle activity, muscle spindles, or cutaneous receptors. In the motivational systems, defined here to include physiological drives and emotions, the sensory processes involved in a drive state, such as pain, activate an emotion. Similarly, an emotion may activate another emotion to which it is innately linked or associated through learning. In the cognitive systems, processes such as appraisal and attribution lead to emotions.

Causal explanations of emotions in terms of the different activating systems (e.g., the neural and cognitive) involve not only different levels of analysis but also different mechanisms and processes. In all systems of emotion activation, however, the generation of emotions lies ultimately in the particular substrates involved in the neural evaluation of the affective significance of information. The likelihood that a particular emotion will be activated depends on the pattern of stimulation, the individual's threshold for that emotion, and other individual differences spawned by both genetic and experiential factors.

I argue that from an evolutionary–biological perspective, the four types of emotion-activating systems can be viewed as a loosely organized hierarchical system of emotion activators. It is not a rigid hierarchical arrangement, except that processing at the neural (simplest) level is primal and always necessary, and emotion activation through inference or attribution always requires the cognitive (highest level) system. It is hierarchical in a developmental sense, in that the neural and sensorimotor systems participate in emotion activation before some types of motivation (e.g., heterosexual drive) and such motivational processes before some types of cognition (e.g., higher order formal operations and propositional reasoning). It is a also hierarchical in terms of complexity of information that can be processed at each level. In terms of capacity for independently processing increasingly complex emotion-eliciting information, the hierarchy of activating systems is as follows: neural, sensorimotor, motivational, and cognitive.

Neural processes are the only type of activator that is both a necessary and sufficient cause of emotion. Although the sensorimotor and motivational systems can operate independently of cognition, they frequently interact with cognitive processes in activating emotions. In general, the more complex the emotion-eliciting conditions, the more likely the highest order activation system, cognition, will be called into play.

The model is placed in context by considering constraints or factors that influence the operation of the four emotion-activating systems. These include individual differences, social factors, and certain stimulus characteristics. Finally, the adaptiveness of hierarchically arranged systems for generating emotions are examined as a feature of the model.

Emotion regulation is not a specific focus of this article, but it should be noted that all four types of emotion-activating systems also function as emotion regulatory systems (cf. Izard & Kobak, 1991). The vast literature on the biochemistry of anxiety and depression (see Thompson, 1988, for a review) testifies to the efficacy of neural processes (endogenous neurotransmitters and exogenous drugs) to regulate emotions. A considerable body of evidence indicates that emotions are also regulated by sensorimotor processes (see Izard, 1990, for a review) and by cognitive processes (e.g., Beck, 1976).

Neural Systems for the Activation of Emotions

Although neural processes are part of all emotion-activating systems, I propose that there are neural systems that can activate emotions independent of cognition and, in some cases, independent of all the other emotion-activating systems. Structures and pathways involved in subcortical and corticolimbic systems of emotion activation were identified in Figure 1. Aggleton and Mishkin (1986), Panksepp (1986), and others have discussed versions of these systems, and LeDoux (1987) has shown that a subcortical pathway can activate emotions independent of the neocortex. Some broad categories of neuroactive agents that can operate on the neural systems to generate emotions are endogenous hormones and neurotransmitters, exogenous drugs, and direct electrical or chemical stimulation of the brain.

There are two types of data that suggest that neural processes can lead to emotion without cognitive mediation. The first comes from psychopharmacological research on emotion-related neurotransmitters and psychoactive drugs. The second derives from research on the effects of direct stimulation of brain structures that constitute the neural substrates of emotions.

There is wide agreement among neuroscientists who study anxiety and depression that certain hormones and neurotransmitters are involved in the etiology and course of these clinical phenomena (Gray, 1982; Meltzer, Arora, Baber, & Tricou, 1981; Redmond, 1985). Probably none of them would claim that these chemical substances are the sole cause of these psychological disorders, but they maintain that biochemical factors are significant determinants. Furthermore, there is substantial evidence that the biochemistry of depression has significant genetic determinants (Hill, Wilson, Elston, & Winokur, 1988; Schlesser & Altshuler, 1983).

Although it is recognized that environmental stress and other experiential factors contribute to the etiology of depression

(Anisman & Zacharko, 1982), Lazarus's (1991) claim that cognition is necessary for the mediation of genetic and biochemical influences on depression seems overinclusive. Although Lazarus and others may be correct in noting that in some cases cognition mediates emotion responses to psychoactive drugs, the evidence suggests that this is not always the case. Cognitive appraisal of drug-induced sensations might well be sufficient to activate emotions, but an appraisal of the sensations simply as negative would not account for the efficacy of some drugs to activate (or alleviate) depression-related emotions and other anxiety-related emotions. Possibly, different drugs recruit different appraisals and hence different emotions, but to confirm this hypothesis it would be necessary to show that the pattern of sensations induced by a depressogenic drug, for example, is logically related to the pattern of appraisals that induce sadness (Izard, 1972). If one found a pattern of drug-induced sensations that was capable of eliciting a sadness pattern of appraisals, would one not have to consider the possibility that this pattern of sensations was the emotion of sadness?

The critical point here is that the weight of the evidence indicates that naturally occurring or experimentally manipulated changes in brain levels of certain biochemical substances can activate emotions or alter emotion experiences without cognitive mediation. The question is not whether neural processes are always, or even typically, the sole mediators of emotions. No one would advocate this extreme position.

The evidence reviewed in the next section indicates that neural processes do indeed influence emotion. I suggest that hormones and neurotransmitters affect emotions directly and that the emotions in turn affect cognitive processes.

Emotions Induced by Changes in Levels of Neurotransmitters

A great deal of evidence has shown that decreased levels of norepinephrine or serotonin are associated with depression (Meltzer et al., 1981; Schildkraut, 1973). It is difficult to rule out a role for cognition in bringing about these biochemical changes, and in some cases stress and stress-related cognition may be an important factor. However, there is substantial evidence that physiological mechanisms—hormones, diet, sleep—coupled with a genetic predisposition, can trigger changes in brain levels of these neurotransmitters (Whybrow et al., 1984).

Another type of relevant evidence comes from the numerous studies that have shown the capacity of antidepressant drugs to ameliorate depressive mood (for a review, see Thompson, 1988). This evidence is far from definitive. Some drugs work better on some types of patients than on others, and all types are generally thought to work better with concomitant psychotherapy. Nevertheless, few students of the biochemistry of depression doubt the efficacy of antidepressant drugs to alter mood.

Biochemical Induction of Anxiety

In general, the relations between neurotransmitters and anxiety are nearly as well established as those for neurotransmitters and depression. For anxiety, as in the case of depression, there is still incomplete agreement as to precisely what neural systems are involved, but there is substantial evidence of the efficacy of a number of antianxiety drugs. A number of experts do agree that the noradrenergic system is central in anxiety (Gray, 1982; Lader, 1974; Redmond, 1985), but some of them acknowledge that other systems may contribute to anxiety directly, or indirectly, by influencing the noradrenergic system.

Of particular interest here are a number of experiments on the biochemical induction of anxiety. Although much remains to be learned about the neural mechanisms involved, there can be little doubt that several chemical agents produce anxiety in humans. For example, carbon dioxide, yohimbine, and amphetamine induce anxiety (see Charney & Redmond, 1983, for a review), and all these agents activate the noradrenergic system (Grant & Redmond, 1982). Other substances, for example pentylenetrazol, induce anxiety by acting, at least initially, at nonadrenergic sites.

Research on the experiential component of depression and anxiety has shown that the key emotions in these phenomena are sadness and fear, respectively (Izard, 1972). The biochemistry of anxiety and depression undoubtedly relates to their key discrete emotions, but there is the possibility that other emotions involved in anxiety and depressive disorders may complicate the search for underlying neural mechanisms. Nevertheless, the point at issue is whether emotions of any sort can be activated biochemically, and the weight of the evidence suggests that the emotions in anxiety and depression can be induced by chemical agents.

Emotion Induction by Electrical Stimulation

Since the early studies of neurosurgical patients by Penfield (1958; Penfield & Jasper, 1954) and Hess (1957), a number of researchers have reported eliciting various emotions by direct stimulation of the brain. In pioneering investigations that led to a Nobel Prize, Hess stimulated various areas of the diencephalon and observed emotion responses. After a series of such experiments, Hess concluded that direct stimulation of specific neural structures produced four types of emotion responses: somatic motor responses (e.g., facial expression and postural changes), responses mediated by the autonomic nervous system (e.g., changes in cardiac activity and pupillary dilation), a "psychic response" or mental state, and instrumental behavior (e.g., flight or attack).

Hess's (1957) experiments and those of later investigators (e.g., Bandler, 1982; Flynn, 1967) indicated that the somatic and autonomic responses and the internal state were elicited directly by brain stimulation, independently of information from the environment. Hess inferred that the stimulation elicited a mental state because expressive and autonomic signs emerged independent of external information input, and the instrumental behavior (attack) followed immediately on the animal's perception (biased interpretation) of some environmental object as the enemy. Hess's work implies that the selectivity or bias in the animal's perception and the organization of instrumental behavior into an effective attack was determined by the emotion experience—motivation, action tendency or readiness, cues for perception and cognition, and feeling state.

Delgado (1969) described numerous clinical investigations in humans in which electrical stimulation of the brain (ESB) was used for diagnostic or therapeutic purposes. In these cases, ESB

in different structures produced subjective experiences and behavioral signs of joy or pleasure, anger, and fear.

More recent clinical investigations of patients with intractable temporal lobe epilepsy suggest an essential role for the limbic forebrain in the generation of conscious experience, including experiential emotion (Gloor, Olivier, Quesney, Andermann, & Horowitz, 1982). Gloor et al. observed emotion (and other) experiences resulting from both spontaneous seizures and electrical stimulation that was being used for diagnostic or therapeutic purposes. By monitoring brain activity with depth electroencephalography, they noted that the patients reported experiential phenomena only when the seizure or electrical stimulation affected areas of the limbic forebrain. Seizure activity or electrical stimulation of the amygdala frequently resulted in the patients reporting fear experiences.

In a series of ingenious ESB studies in cats, Flynn (1967) not only successfully elicited anger or rage states but also showed that these states and their accompanying expressive manifestations could be elicited independently of aggressive actions. More important, he confirmed Hess's (1957) conclusion that the somatic-expressive and autonomic responses and the presumed accompanying subjective state were centrally controlled, whereas subsequent instrumental or aggressive behavior was controlled by sensory input.

With electrodes implanted in the cat's hypothalamus, and in some cases in the amygdala and the hippocampus, Flynn (1967) stimulated free-ranging cats under various environmental conditions. First, he showed that electrical stimulation of the hypothalamus of an otherwise normal cat elicited a display of rage. Second, he found that when various objects, ranging from an anesthetized rat to a styrofoam block, were available, most of the stimulated cats would selectively attack the rat and ratlike objects. Seven of 9 cats in the experiment never attacked the styrofoam block, and only 1 of them attacked the items indiscriminately. Clearly, the animals perceived and appraised the stimuli selectively. It is reasonable to assume that Flynn's work confirms Hess's (1957) inference that the cat's psychic response or emotion experience influenced perceptual and attentional processes and motivated the attack. The instrumental behavior (aggressive attack) was channeled by information from the environment.

Of particular relevance to the hypothesis that central neural processes are a sufficient cause of emotion, Flynn (1967) examined the role of the relevant sensory systems (olfaction, touch, and vision) and information input from the environment in the stimulation–emotion–attack sequence. Removal of the olfactory bulb had no effect on the somatic expressive behavior or on aggressive attack. Blindfolding the cats had no effect on the somatic expressive behavior but reduced the frequency of attack from 100% to 40%. Sectioning the mandibular branches of the sensory fibers of the trigeminal (eliminating perioral sensation) had no effect on the somatic expressive behavior but substantially reduced the frequency of attack; in almost half the cats deafferentation completely eliminated attack behavior. When both perioral sensation and vision were blocked, none of the cats exhibited instrumental or attack behavior.

Flynn (1967) concluded that the somatic expressive behavior (facial–postural display, sniffing, and walking) was a direct effect of brain stimulation independent of information from the environment. He called Hess's (1957) psychic response, or internal state, a motor disposition, a term equivalent to the action tendency or action-readiness aspects of the present definition of emotion experience. When a range of stimuli are present, this motor disposition plus sensory input lead to selective attack on the appropriate stimulus.

Bandler (1982) extended the work of Hess (1957) and Flynn (1967) by using microinjections of glutamate, an excitatory amino acid transmitter. The advantage of this procedure is that glutamate affects only the cell bodies at the injection site and not the axons passing through to other areas. Bandler located a population of neurons in midbrain central gray where glutamate elicited the rage display and, when adequate stimuli were present, aggressive attack. Basically, he reaffirmed the conclusion of Hess and Flynn. He concluded that the glutamate activated central processes that elicited rage expressive behavior independent of cognitive processes based on environmental information. Like the earlier investigators, he saw sensory input from the environment as the information that guided the attack behavior. He rejected Hess's concept of an elicited psychic response, describing the attack motivation "as a product of level setting in sensory and motor systems" (Bandler, 1982, p. 390). This notion of altered thresholds for sensorimotor responses may be related to the action readiness or action tendency component of the present definition of emotion experience.

Although the terminology differs across investigators, the conclusion is essentially the same. All agree that direct stimulation of neural substrates of emotion produce emotion expressive behavior, ANS activity, and some kind of motivational, experiential state independent of environmental information. Conceivably, the set of responses to direct stimulation might vary across different emotions. The investigators also agree that it is the sensory input from the environment that guides the emotion-related instrumental or goal-directed behavior. It is the transformation and integration of the environmental information necessary to the attack, escape, or other emotion-related behavior that requires higher order cognitive processes.

Emotion Induced by Changes in Cerebral Blood Temperature and Subsequent Neurochemical Processes

In a series of experiments, Zajonc and his colleagues (Zajonc et al., 1989) found that changes in subjective states of pleasantness or unpleasantness could be induced by expressive and nonexpressive facial muscle contractions that altered cerebral blood flow, the blood-cooling capacity of the cavernous sinus, cerebral blood temperature, and, presumably, subsequent neurochemical processes that mediate subjective feelings. Although the first statement of this emotion activation model (Zajonc, 1985) was challenged on evolutionary and neurological grounds (Burdett, 1985; Fridlund & Gilbert, 1985; Izard, 1985), Zajonc has modified his position, and his experimental findings have not been challenged by other investigators.

Emotion Without Neocortical Involvement

A series of experiments by LeDoux and his colleagues (LeDoux, 1987, 1989) has demonstrated that acoustically and visually conditioned fear can be established in rats after the

acoustic or visual cortex, respectively, has been ablated. LeDoux identified subcortical structures that mediated the conditioned fear response. The circuit consists of interoceptors or exteroceptors, thalamic nuclei, and amygdaloid nuclei. The relatively small number of synapses in this thalamoamygdaloid pathway makes transmission very rapid and enables an animal to make an immediate response. This mode of responding is highly adaptive when information-processing demands are simple and the animal has to respond rapidly to the broad physical features of the stimulus. When the situation requires more complex evaluation, processing occurs in a thalamo–cortico–amygdaloid pathway.

LeDoux (1987, 1989) concluded that the amygdala is the key structure for the immediate evaluation of the emotional significance of stimuli and the releasing of emotion responses. It is involved in both the subcortical and the cortico-limbic pathways. LeDoux inferred that these dual pathways for emotion activation constitute separate systems for affective and cognitive information processing. Although LeDoux's evidence does not provide direct support for the idea that neural processes are sufficient causes of emotion, it does indicate that neural-evaluative processes lead to emotion without higher order cognition or cognition that requires cortical processing.

Sensorimotor Systems for the Activation of Emotions

Since the beginnings of the scientific study of emotions, theorists have recognized a relation between the motor system and subjective states. Darwin (1872/1965) observed that "the free expression by outward signs of an emotion intensifies it" (p. 365), and James (1890/1950) said, "Refuse to express a passion and it dies" (p. 463). Whereas these statements suggest only that sensorimotor processes can regulate ongoing emotion experiences, James attributed even greater powers to expressive behavior. He indicated that the quality of feeling states could be altered from undesirable to desirable if "we . . . assiduously . . . go through the outward movements of those contrary dispositions which we wish to cultivate" (James, 1890/1950, p. 463). In these statements, the effect of expressive behavior is confounded with that of intention and cognitive self-instruction. However, Darwin's and James's view on the role of somatic responses in emotion experiences is not inconsistent with the data showing that the motor system responds to direct stimulation of emotion substrates (Bandler, 1982). Of the various aspects of the sensorimotor-activating systems, facial expression and posture have been studied empirically. The sensorimotor processes are as follows:

1. Central efferent activity
2. Facial expressions
3. Posture
4. Instrumental behavior
5. Muscle action potentials.

Facial Expression and Emotion

A number of theorists have extended the thinking of Darwin (1872/1965) and James (1890/1950) and postulated that sensory feedback from facial and postural movement is a sufficient, though not a necessary, cause of emotion experience (Izard, 1971; Tomkins, 1962). Laird (1974, 1984) went a step further and proposed that experimenter-manipulated emotion expression can generate emotion experiences, and some investigators suggested that such emotion activation is independent of cognitive processes.

Theories in the tradition of Darwin and James and the hypothesis of Laird (1974, 1984) have led to about 30 published studies on facial feedback as a determinant of subjective states of emotion. These include several critical reviews and meta-analyses of the empirical studies (Adelmann & Zajonc, 1989; Izard, 1990; Matsumoto, 1987; Winton, 1986). The various forms of the hypotheses suggest that expressive behavior can (a) alter ongoing emotion experience, (b) generate positive or negative emotion experiences, and (c) activate a specific emotion experience corresponding to the manipulated expression.

There is a substantial body of evidence supporting the first and second forms of the facial feedback hypothesis and some favoring the third form, the notion that expressive behavior can activate emotion-specific experiences (e.g., Duclos et al., 1989). Consistent with the Duclos et al. finding of the specificity of expression generated experiences, Ekman, Levenson, and Friesen (1983) reported that experimenter-directed facial action produced emotion-specific patterns of ANS activity. Furthermore, in a follow-up study, Levenson, Ekman, and Friesen (1990) found that directed facial action elicited emotion-specific patterns of ANS activity and specific subjective experiences without the subjects seeing their faces in a mirror or the face of a model. The data from the Levenson et al. replication did not appear quite as robust as that of the original study, and emotion-specific ANS patterns from directed facial action have not yet been reported from other laboratories.

Although the effects of expressive-behavior manipulations on emotion experience have been replicated many times, some skepticism remains as to whether the relevant experiments have completely controlled for cognitive influences (Zajonc et al., 1989). However, Zajonc et al. used procedures that seem to eliminate cognitive influences, and they reported that facial muscle contractions generated pleasant or unpleasant feelings by increasing or decreasing cerebral blood flow and thereby decreasing or increasing brain-blood temperature and neurochemical activity.

Body Posture and Emotion

Both Darwin and James included posture when they described the effects of expressive behavior on emotion experience. James (1932) was quite specific. He described the posture of *expansion*—erect head and trunk, expanded chest, and raised shoulders—as associated with positive states like pride and self-esteem as well as negative states like contempt. He described the posture of *contraction,* forward trunk, bowed head, and drooping shoulders, as characteristic of states of sadness, depression, and abasement.

In the tradition of Darwin and James, Bull (1951) proposed a motor-attitude theory of emotion activation. In her model, the sequence is as follows: Stimulus → neural organization → bodily readiness, action set (motor attitude) → oriented feeling or mental attitude → action. For Bull, the motor attitude is the proximal cause of a feeling state or "oriented awareness."

Despite the observations of Darwin (1872/1965), James (1932), and Bull (1951), only a few empirical studies have examined the effects of posture in emotion. In a longitudinal study, Weisfeld and Beresford (1982) showed that boys, ranked by peers in early grade school as tough or dominant, were characterized by erect posture in high school. In another study, they found that erectness of posture in college students on receiving the results of a test was positively correlated with their test performance.

Although the foregoing findings suggest only a correlation between posture and mood, Riskind and Gotay (1982) and Riskind (1984) showed that manipulations similar to James's (1932) expansion and contraction postures altered emotion experiences, expectations, and task persistence in the expected directions. In a series of three studies, Riskind (1984) showed that the appropriateness of the posture to the situation influenced the effects of the posture. For example, a slumping posture increased ongoing sadness or discouragement, but it also helped the person to recover from the discouragement more quickly. Riskind concluded that appropriateness of posture or posture–mood congruence enhanced the person's effectiveness in coping with both rewarding and stressful situations.

Because Riskind's (1984) studies always involved the posture manipulation in interaction with a success–failure manipulation, he did not show that posture alone activated emotion experience. The studies did show, however, that posture could have an effect seemingly independent of, and contrary to, cognitive influences, as in the case of an induced (and inappropriate) slumping posture having an adverse effect following a success experience.

In the study demonstrating the emotion-specific effects of facial expressions, Duclos et al. (1989) examined the direct effects of emotion-specific postures corresponding to sadness, anger, and fear. The selection of movements for encoding the postures was guided by descriptions in emotion theories. After assuming a given posture for 15 s, subjects rated their feelings on the target emotion and seven others. The results showed that the postures of sadness, anger, and fear elicited the highest ratings for feelings of sadness, anger, and fear, respectively. The authors concluded that emotion postures had clear and specific effects on emotion feelings.

Motivational Systems for the Activation of Emotions

I use the term *motivation* to include emotions (e.g., joy, sadness, anger, and fear) as well as drive states rooted in cyclical physiological processes (e.g., hunger, thirst, fatigue, and sex) or tissue damage (pain). As already noted, Tomkins (1962) has made convincing arguments for distinguishing emotions and drives and for the ability of drives to activate emotions. Indeed, Tomkins argued that drives are effective in influencing behavior only by virtue of their capacity to recruit the motivational power of emotions.

On the ability of drives to activate emotions, Tomkins (1963) wrote,

> One of the most important and somewhat neglected sources of distress [sadness and anguish] is the low-grade pain or discomfort of the low energy state. . . . Frequently, as the state of fatigue deepens, it is sufficient to activate distress, and then the combination of fatigue and distress is often sufficient to activate anger. (p. 20)

I return to Tomkin's description of the mechanism for one emotion activating another emotion.

Taste, Odor, and Emotion

Studies by Steiner (1973) and others (Fox & Davidson, 1986; Rosenstein & Oster, 1988) have shown that the basic tastes elicit a range of emotion expressions in infants. Some investigators have reported that sweet tastes elicit expressions of interest, whereas bitter tastes elicit expressions of disgust (Fox & Davidson, 1986). Regardless of whether there is agreement on the specificity of the elicited responses, no one disputes that the different tastes activate expressions that vary along the positive–negative or pleasant–unpleasant dimension. Although one can question whether taste-elicited expression means that emotion experience has also been activated, the infant's effort to eject the distasteful substance suggests that some sort of motivational state or action tendency has been instigated. There are theoretical arguments and convergent evidence for innate expression–experience concordance (Izard & Malatesta, 1987).

Steiner (1979) summarized a series of studies with neonates and concluded that odor-elicited emotion expressions reflected changes in hedonic states. Although Gilbert, Fridlund, and Sabini's (1987) study of odor-elicited facial expressions in adults failed to confirm Steiner's reflexive-hedonic interpretation, there is substantial evidence that odor elicits changes in feeling states (Ehrlichman & Bastone, 1992). Furthermore, Berkowitz (1983, 1990) reported that foul odors elicited anger and aggression. As already noted, odors produce effects on feeling states and behavior despite their weak relation to verbal labels and cognition.

Pain-Elicited Emotion

Many people can remember crashing into something in the dark and experiencing an immediate unreasoned outburst of anger. Such anecdotal evidence is only suggestive of a connection between pain and anger, but data from many animal studies indicate that there is an innate connection between aversive stimulation, anger, and aggression (see Moyer, 1976, for a review). This research shows that when animals are caged together and subjected to aversive stimulation they frequently begin to fight. Whether they fight or flee can be influenced by, among other things, the size and status of the other animal. Consistent with the findings of Hess (1957) and Flynn (1967), cognitively processed environmental information apparently influences the direction of the motivation or action tendency. The dominant or larger of the two aversively stimulated animals regularly begins to fight.

Berkowitz (1983, 1990) has extended the study of pain–anger–aggression relations to human adults. He reviewed the literature showing that immersion in cold water, exposure to high temperatures, and foul odors can instigate aggression. Although he recognized that cognitive processes could contribute to the intensification of reactions, he concluded that pain or aversive stimulation was sufficient cause for anger and aggression. Berkowitz proposed that the mechanism for the activation

of anger and aggression was the stimulus-elicited negative affect (e.g., pain).

Extending Berkowitz's (1983, 1990) suggestion and considering LeDoux's (1987) identification of a subcortical pathway for emotion activation, it is possible to suggest a mechanism whereby pain elicits anger without cognitive mediation. Pain receptors send afferent messages to the thalamus and on to the amygdala, which releases the emotion responses directly. LeDoux's data indicate that the neocortex and cognition requiring cortical processing need not be involved.

Cross-sectional and longitudinal studies have assessed the emotion expressions of human infants to the unanticipated pain of diphtheria–pertussis–tetanus (DPT) inoculations (Izard, Hembree, Dougherty, & Spizzirri, 1983; Izard et al., 1987). Analysis with an anatomically based, objective coding system (Izard, 1979) showed that in infants under 7 months of age, the injection elicited a physical distress expression that was regularly followed by an anger expression, which was sometimes followed by a sadness expression. The peremptory physical distress or pain expression began to diminish after 7 months, and by 19 months of age 25% of the infants responded immediately with the anger expression. The authors reasoned that the physical distress expression was adaptive in the relatively defenseless young infant. At this stage of development, it is appropriate for all available energy to be channeled into an emergency cry for help. However, in the 19-month-old toddler, who is more capable of defensive behavior, the more adaptive response was energy-mobilizing anger.

Emotion-Elicited Emotion

Tomkins's (1962) theory of innate activators of emotions holds that three variants of a single principle, density of neural firing, can explain the activation of all emotions. The three variants are stimulation increase, stimulation level, and stimulation decrease. For example, sadness and anger are stimulation-level emotions, with anger activation requiring a higher density of neural firing. Tomkins maintained that a continued unrelieved level of stimulation adequate to elicit sadness could, as a function of time, increase beyond the threshold for anger. As already noted, he held that a stimulation increase could occur as a result of other noncognitive processes, such as fatigue. Tomkins's view of the neurological relations among emotions and their stimulation gradients led him to conclude that sadness was an innate activator of anger.

Tomkins's (1962) interesting model of emotion activation has not been empirically tested, but clinical observations and some empirical work is consistent with his notion that sadness is a sufficient cause of anger. The *Diagnostic and Statistical Manual of Mental Disorders* of the American Psychiatric Association (1987) recognizes "irritable mood" as a symptom of depression, and clinicians frequently describe depressed individuals as "sad–mad" (Berkowitz, 1983; Carlson & Cantwell, 1980). Systematic studies of the affective symptomatology of depressed adults and children have revealed that sadness and anger are the first and second most prominent emotions in depression (Blumberg & Izard, 1985; Izard, 1972).

Although the foregoing evidence only shows a correlation in the occurrence of sadness and anger, some studies have shown that the experimental induction of depressive feelings generates anger and hostility (Finman & Berkowitz, 1989; I. W. Miller & Norman, 1979). These data and relevant clinical observations led Berkowitz (1990) to conclude that there is very good reason to believe "that the depressed mood in itself produces angry feelings and hostile inclinations" (p. 496).

The study of young infants' emotion responses to unanticipated pain also suggests causal relations among emotions (Izard et al., 1987). Following the DPT injection, the most typical sequence of expressions was physical distress followed by anger. The anger expression was often followed by sadness expression. That sadness followed anger, in this instance, could be explained, at one level, by Tomkins's (1962, 1963) principle of emotion activation by density of neural firing. As the pain subsided, the level of stimulation decreased to the range that produces sadness. Also, a brief separation of 13-month-old infants from their mothers often produced a sequence of anger–sadness expressions (Shiller, Izard, & Hembree, 1986). In a longitudinal study using the same procedure with infants at 13 and 18 months of age, the anger–sadness pattern occurred on both occasions, and the individual differences in the amount of anger and sadness expressions were stable over time (Hyson & Izard, 1985).

An alternative explanation of the pain- and separation-elicited anger–sadness sequence is that the emotions are organized as a system and that specific emotions within the system are dynamically related for adaptive purposes and thus tend to form patterns or clusters (Izard, 1972; Watson & Clark, 1992). The co-occurrence of anger and sadness in the distressed infant can be viewed as adaptive. The anger mobilizes energy for coping with the situation and the sadness expression appeals for help.

In any case, whenever a particular emotion is activated, whether by affective process (pain), sensorimotor process (facial expression), or by cognitive process, other emotions are also activated (Duclos et al., 1989; Hansen & Hansen, 1988; Izard, 1972; Izard et al., 1987). The occurrence of the other emotions is nonrandom, and the pattern is replicable. The same specific emotions tend to occur together. The evidence suggests that motivation-induced emotion, and, more specifically, emotion-induced emotion, is a viable hypothesis. It can be further tested through cross-cultural research. The hypothesis would be strongly supported by finding, for example, that imagining an anger-eliciting situation elicited a similar pattern of emotions in different cultures.

Another alternative explanation of the co-occurrence of a set of emotions is the array of emotion-eliciting stimuli in the environment. This seems an improbable solution. Anxious and depressed persons, regardless of their environment or circumstances, tend to report the same pattern of emotions. An even more telling blow against the stimulus–response or contextual explanation of the regularity of the co-occurrence of certain emotions is the evidence from the studies of infants' emotion responses to pain and separation (Hyson & Izard, 1985; Izard et al., 1987). In the pain study, the mother and the nurse followed a prepared protocol, and there were no differences in the interpersonal interactions during the interval that the pain–anger–sadness expression sequence was observed.

In the separation studies, the anger–sadness pattern was ob-

served during a child-alone episode in which the environment remained constant. In this study, the child was 13 months of age, and memory retrieval could play a part in the sequence of emotions. The flow of memories, however, does not suggest an explanation of the tendency of anger to precede sadness. In the pain study, the anger–sadness expression sequence was observed as early as 2 months, an age when long-term memory, particularly semantic memory, is nonfunctional.

The idea of one emotion eliciting another can generate hypotheses for further research. For example, assuming that sadness and anger are more strongly associated than sadness and other emotions, one could induce sadness by external stimulation (e.g., appropriate film) and then test the effects of manipulating appraisals and attributions or other activating processes relevant to anger and other emotions.

Cognitive Processes in the Activation of Emotions

As noted at the outset, there is a substantial body of literature on emotion activation that stems from cognitive theories. This section reviews only a brief sample of the available material. Generally, this line of research has been concerned with determining which set of cognitive appraisals or attributions elicits, or is associated with, what specific emotion. The typical conclusion is that the cognitive determinants and correlates of particular emotions can be specified (e.g., Frijda, Kuipers, & ter Schure, 1989; Smith & Ellsworth, 1985; Weiner & Graham, 1984). The usual experiment requires subjects to self-induce emotions through imaginal or recall processes and then to rate the emotions on appraisal dimensions. Alternatively, subjects read vignettes in which appraisal dimensions are manipulated and then predict the emotion the character in the story will feel. As Smith and Ellsworth (1985), Frijda et al. (1989), and others have shown, these studies show coherent correlations among particular appraisal dimensions and specific emotions. Types of cognition that activate emotions are as follows:

1. Appraisal and evaluation
2. Comparison, categorization, inference, and judgment/decision
3. Attribution and belief
4. Memory and anticipation.

The data on cognitive processes—appraisals, attributions, beliefs, and desires—considered as correlates of emotion appear robust (Harris, 1989; Roseman, 1984; Scherer, 1988; Weiner, 1985). Harris (1983a, 1989) has made an elegant case in theory and empirical research for the way in which children's beliefs and desires index their understanding of emotions. His argument is based on the assumptions that even preschool children are aware of their own mental states and are capable of pretense, distinguishing pretense from reality, and, through pretend play, of understanding other people's emotions. His studies show that if children know a person or animal character's beliefs and desires about an object, they can predict the character's emotion response to receiving or failing to receive that object. The correlation between beliefs and desires on the one hand and emotions on the other seems indisputable, and, as Harris maintains, this relation helps us understand the child's theory of mind.

Drawing on the work of several cognitive theories of emotion, Smith and Ellsworth (1985) derived six orthogonal dimensions of appraisal. Their studies showed that cognitively induced emotions varied systematically in terms of the subjects' ratings of the emotions on the six dimensions. These data provide good verbal descriptions of some of the cognitive concomitants of discrete emotions. For example, the data show that the dimension of other responsibility/control is associated with surprise and anger and that the dimension of self-responsibility/control is associated with shame and guilt.

Roseman (Roseman, 1984; Roseman, Spindel, & Jose, 1990) has shown that each of 15 emotional states are significantly related to a particular set of his appraisal dimensions. However, his position is not altogether typical of cognitive theories. Although several theorists (Frijda, 1986; Lazarus, 1991; Scherer, 1988) hold that motivation influences appraisal processes, Roseman is the only one to argue that all emotions are a function of both motivation and cognition, and he seems sanguine to the possibility that in some cases the motivation (defined to include physiological drives and motives) may be noncognitive. Thus, his theory occupies a middle ground between some cognitive views of emotion activation and that of the present model. An example from one of Roseman's studies provides a basis for examining the similarities and differences between his model and mine.

One of Roseman's (1984) vignettes described a girl (Susan) who wanted badly and worked hard to pass an exam but failed because the instructor was uncommonly strict. This story manipulated the dimensions that Roseman et al. (1990) now calls motivational state (Susan wanted to pass the exam), motive inconsistent/thwarted (she got an F), legitimacy (the F was undeserved), and agency (the instructor's hard grading caused her to fail). Roseman confirmed his hypothesis that subjects would predict that Susan would be angry.

What Roseman (1984) calls motivation includes what I have termed physiological drives and affective–cognitive structures (Izard, 1977). Thus inserting my terminology into Roseman's scheme, drive states, ongoing emotions, or the affective component of affective–cognitive structures may influence appraisal and the generation of new emotion. Our theories are alike in recognizing noncognitive determinants of emotions. They differ in emphasis on this point. Roseman sees noncognitive factors as contributing input to the appraisal process (motivation plus cognition generates emotion), whereas I propose that motivation is capable of generating emotions independently of cognition or appraisal. Finally, some of the phenomena that Roseman views as emotions (e.g., hope and pride) are seen in differential emotions theory as affective–cognitive structures, phenomena that include both affect and cognition.

Although there is substantial evidence of cognition functioning as an emotion-activating system, two questions can be raised for investigators of cognitive antecedents of emotions. The first question is that of clearly specifying cognition as an independent cause. The second, perhaps more fundamental question is what drives cognition when it is operating as a causal process.

On Identifying Cognition as Cause of Emotion

The first problem in specifying cause–effect relations for cognition and emotion is that of definitional boundaries for

these two domains. This problem, and possibly that of tautological argument stemming from lack of such boundaries, is illustrated in Frijda's (1988) laws of emotion that describe the cognitive processes that dictate emotions. For example, one of his laws is that "emotions arise in response to events that are important to the individual's goals, motives, or concerns" (Frijda, 1988, p. 351). Alternatively, one can say that goals, motives, and concerns are themselves vested with emotions. How can a goal be important or meaningful if one has no feelings about it?

Another of Frijda's (1988) laws that assumably determines emotion experiences is that emotions arise out of one's concerns. However, does one not have concerns for people and objects because one has already invested emotions in them? I suggest that Frijda's concepts of goal, motive, and concern could be considered as affective–cognitive structures, feelings plus cognitions. Goals, motives, and concerns are virtually always part of the contents of consciousness, and if it is acknowledged that they have an affective component, they could be seen as part of ongoing affect or emotion that influences appraisals and attributions.

A challenge for researchers basing their work on cognitive theories of emotion activation is to determine whether and to what extent the presumed antecedents of emotion are themselves independent of emotion. A next step would be to determine whether and to what extent appraisal, attribution, desire, and the memory on which these phenomena depend are influenced by noncognitive factors such as drive states and individual differences. Then it would be desirable to develop procedures for assessing the relative contribution of cognitive and noncognitive activators of emotions.

Another problem in specifying cause–effect relations in studies of cognitive antecedents of emotions is methodological in nature. In the empirical studies, emotion experiences are often induced by giving subjects verbal descriptions of events or agentic processes that they relive through imagination. Determining the causes of the emotions then consists of having the subjects identify the emotions in two ways. Subjects give the emotions names and provide verbal descriptions of emotions by rating them on dimensions of appraisal or causal attributions. Both of these procedures are cognition and language dependent, and both simply require the subjects to identify the emotions verbally. There is the possibility of spuriously inflated correlations between appraisal dimensions and emotion labels because of method variance—data on both variables are obtained by the same (self-report) method. For this reason, it is important that the language of the appraisal dimensions or other cognitive antecedents be free of emotion connotation.

As already suggested, another possible problem for cognitive explanations of emotion activation is that they ignore or neglect to treat systematically individual differences that might place constraints on appraisal processes and emotionality. For example, compared with introverts, extraverts have a lower threshold for interest and enjoyment in social situations (Eysenck & Eysenck, 1985). Similarly, compared with Type B personalities, persons of Type A personality have a lower threshold for anger or aggressive behavior (Matthews & Angulo, 1980).

In summary, the critical issue here is not whether cognition is a sufficient cause of emotions. There can be no doubt that in any situation where an appropriate response requires the person to access memory, make comparisons, categorizations, judgments, or decisions, cognitive processes may constitute a sufficient cause of emotion. The question, at least for some theorists, is whether a clear separation has been made between the two languages (one being emotion labels and the other being appraisals, attributions, and construals) used in assessing causal processes.

What Drives Cognition? Is Selective Perception and Attention Focusing a Function of Emotions?

In contrast to the present model, some cognitive theories seem to assume, at least for purposes of experimental investigations, that appraisal and attributional processes occur in an emotionless or affectively neutral mind. This raises the question as to what motivates and guides the perceptual and evaluative processes involved in appraisal.

Differential emotions theory has answered this question by postulating emotion as a fixed characteristic of consciousness (Izard, 1977, 1989). Thus, emotions are viewed as the motivational basis for selective perception and the focusing of attentional activity (cf. James, 1890/1950). This is consistent with the common observation that in joy one sees the world through rose-colored glasses (Meadows, 1975) and in fear one experiences tunnel vision (cf. Easterbrook, 1959). In a similar vein, Buck (1986, 1990) proposed that subcortical systems, which constitute the neural substrates of motivation and emotion, serve as filters that guide perception and determine input to the appraisal process. Thus, "we feel *before* we know, and in an important sense, feeling *determines what we know*" (Buck, 1986, p. 363).

The issue of the continual presence in consciousness of some emotion at some level of intensity will not be settled here and cannot be easily settled in the future. Volumes have been devoted to the issue as to whether emotions are still operative even in repression and dissociation (see Singer, 1990), at lower levels of awareness (Bower, 1990), or in unconscious processes (Kihlstrom, 1987; Kihlstrom & Hoyt, 1990). Clinician and experimentalist alike seem to agree that emotions operate outside of focal awareness and working memory, but much remains to be learned about this fascinating aspect of emotion–cognition relations. Nevertheless, the question of emotion as a characteristic of consciousness or of neural substrates of emotions serving as filters for sensory input has significant implications for the problem of the activation of emotions and should not be ignored.

If Buck (1986) is correct in assuming that feelings determine what we know or even significantly influence sensory input to the neocortex, then we need to reexamine the issue of the relative contribution of appraisal processes and ongoing affect in the activation of emotion experiences. If emotion is continuously influencing cognition at either the conscious or the unconscious level (cf. Bower, 1990; Kihlstrom & Hoyt, 1990), the whole problem of emotion activation may be more precisely framed in terms of activating a new or different emotion or of effecting qualitative changes in ongoing states of consciousness. The notions of neural and motivational filtering of sensory input and a continuously emotional mind as normal and typical

in human beings are particularly relevant to arguments that assume an affectless appraisal system and to those relating to emotion activation independent of motivational processes.

The work of Isen and her colleagues (e.g., Isen & Means, 1983; Isen, Shalker, Clark, & Karp, 1978) bears on the question as to what motivates and guides perceptual–cognitive processes. They maintain that emotion can serve as a retrieval cue. After reviewing the research on the influence of affect on cognition, Isen (1984) concluded that "if feeling state serves as a retrieval cue, then material in memory must be encoded according to how that material makes one feel. . . . This suggests a much more intimate relationship between affect and cognition than was ever suspected previously" (p. 218).

Isen's position seems to complement the notion that some emotion is continually present in consciousness. The two ideas together suggest that emotion experience guides ongoing perceptual–cognitive processes and the storage and retrieval of memories. Although these ideas need a stronger empirical foundation, they challenge one to think more seriously about emotion–cognition relations. They also invite one to reconsider the nature of emotion experiences and their activation. Viewing emotion experiences as varying widely in intensity and subtlety (cf. James, 1890/1950) and viewing the activation of subtle emotion as requiring only minimal changes in internal or external processes make it easier to accept the idea of their continual presence in consciousness. As already noted, the idea that the virtually omnipresent phenomena of goals, motives, and concerns have an affective component is consistent with the hypothesis of a continually emotional mind.

Constraints on the Functioning of the Emotion-Activating Systems

The present model assumes that in addition to ongoing motivation, the emotion-activating systems are influenced by three types of constraints: individual differences, social variables, and environmental conditions, or, more specifically, stimulus characteristics.

Individual Differences and Emotion Activation

The studies of infants' emotion responses to pain and separation clearly revealed individual differences (Hyson & Izard, 1985; Izard et al., 1987). Infants differed in the amount of pain, anger, and sadness that they expressed. As already noted, these individual differences were stable over time. Studies from a number of other laboratories have confirmed the existence of stable individual differences in emotion-expressive behavior (Malatesta, Culver, Tesman, & Shepard, 1989; Sullivan, Lewis, & Alessandri, in press). In the experiment of Sullivan et al., stability was demonstrated in the first 4 months of life. Although the foregoing studies indexed only expressive behavior, other research has found stable individual differences in emotion experiences in adults (Diener & Emmons, 1984; Izard, Libero, Putnam, & Haynes, 1991; Tellegen, 1985). The work of Diener and Emmons, Tellegen, and their colleagues showed stability of positive and negative emotionality. The Izard et al. (1991) study showed stability of discrete emotion experiences.

Several studies have shown that individual differences in emotion experiences are significantly correlated with dimensions of temperament in infants (Izard, Brown-Lawler, Haynes, Simons, & Porges, 1991) and to traits of personality in adults (Watson & Clark, 1992). These findings suggest that personality, including temperament, can be considered as a complex supersystem that includes emotion-based traits and that should be expected to influence the emotion-activating systems.

Additional evidence of individual differences in emotionality has been found in research with biologically prepared stimuli. Mineka et al. (1984) studied observational conditioning of snake fear in rhesus monkeys. Several of their interesting findings are relevant here. Wild-reared monkeys that had not seen a snake for as long as 15 years immediately displayed fear behaviors and avoidance on exposure to a snake or snakelike objects. Laboratory-reared juvenile monkeys showed no observable fear responses to the stimuli. When laboratory-reared juveniles observed a wild-reared adult's responses to the snake or snakelike stimuli, most of them quickly learned and long retained fear of snakes and snakelike stimuli. However, even though the effects of modeling were so immediate and strong that they suggested that snakes were biologically prepared stimuli, there were individual differences in emotion responses among the laboratory-reared juveniles. A few of them continued to show no fear of snakes after observing the fear behaviors of the wild-reared adults. Environmental information had a very different effect on the emotions system of those who displayed fear and those who reached over the snake to obtain a food reward. It seems an incomplete explanation simply to say that the fearless juveniles appraised the snake as nonthreatening. Is not one's understanding of the emotion responses of these animals increased if one considers the possibility that genetically determined individual differences in the threshold for fear or fear conditioning altered appraisal and other emotion-activating systems?

Kagan and his colleagues (e.g., Kagan, Reznick, & Snidman, 1988) have identified an extremely inhibited group of children who have a very low threshold for shyness or fearfulness, particularly in novel social situations. These severely inhibited children were identified as early as 21 months of age, and their scores on several indexes of inhibition showed a significant amount of stability through the last assessment at 7.5 years.

Although Kagan et al. (1988) did not think that the trait of extreme shyness or fearfulness was completely genetically determined, he concluded that the children with this trait are temperamentally or predispositionally prone to extreme shyness or fearfulness. Apparently, compared with normal children, the inhibited children's appraisal systems are much more sensitive to threat in novel social situations. As in the case of observationally learned snake fear in rhesus monkeys, it is an apparently incomplete explanation to say that these children's extreme shyness or fearfulness is a function only of their appraisals. It seems a more satisfactory explanation to say that their appraisal systems have been influenced by a genetically determined predisposition and that emotion activation in novel social situations is a matter of biopsychological information processing or a joint function of biological information (a genetic proneness to extreme shyness) and cognitive processes. Analysis of both genetically influenced individual differences and learned appraisal skills appears to be necessary for a more

complete understanding of the emotional responses of these inhibited children.

Furthermore, the biological disposition to socially inhibited behavior seems to affect not only the appraisal system but neural processes as well. During the assessments at several ages, Kagan and his colleagues obtained a number of physiological measures, including levels of cortisol, a hormone known to be a correlate of stress and negative emotions. Even during sleep, these children had above-normal levels of cortisol. The predisposition to inhibition apparently lowered the threshold for shyness in both neural and cognitive emotion-activating systems.

Evidence from research on emotional development in atypical children lends further support to the notion that biological conditions influence appraisal and other emotion-activating processes (see Cicchetti, 1990, for a review). Cicchetti and Sroufe (1978) did a series of studies that compared the emotion responses of normal infants with those of infants with Down's syndrome. They used a variety of stimuli designed to elicit positive and negative emotions. Even when matched in mental age, Down's syndrome infants showed less smiling and laughter to positive stimuli than did normal children. Similarly, with the cognitive-developmental level the same in the two groups, negative emotion elicitors, such as looming object and visual cliff, produced significantly less negative emotion in Down's syndrome infants.

These data led Cicchetti (1990) to conclude that emotions and cognition are "indeed separate developmental systems" (p. 276). It does not seem fruitful to explain differences in the emotion responses of cognitively matched Down's syndrome and normal children simply in terms of stimulus appraisal. Of course, matching on cognitive level does not mean matching on cognitive content, and content could influence appraisal. However, the evidence suggests that the cause of the differences in emotional responding is rooted, at least in part, in Down's syndrome, and this disorder cannot be explained in terms of learning and environmental factors. Down's syndrome is a genetic anomaly, apparently one that alters emotion thresholds as well as cognitive appraisal processes.

Salovey and Mayer (1990) have discussed a concept of *emotional intelligence* that they interpret as a set of individual differences that influence emotionality. They defined emotional intelligence as a constellation of emotion-related abilities or traits, including such dispositional variables as emotion expressiveness, emotion perception (expression decoding ability), and empathic responsiveness. They reviewed evidence supporting the validity of instruments designed to measure individual differences in these characteristics. For example, the Profile of Nonverbal Sensitivity has been well documented as an index of the ability to detect emotion cues in the face, body, and voice (Rosenthal, Hall, Archer, DiMatteo, & Rogers, 1979), and Buck (1984) has demonstrated individual differences in people's ability to send and receive emotional cues. A number of investigators have found wide individual differences in empathic responding (see Eisenberg & Strayer, 1987), and theorists have suggested that differences in empathy may be accounted for by differences in emotion thresholds or in emotion information processing (see Hoffman, 1984). The evidence suggests that individual differences in such emotion-related traits as emotion expressiveness, emotion perception, and empathy reflect the person's ability to detect and process emotion information, and, therefore, they place constraints on the emotion activation systems.

In view of the foregoing evidence for genetically and biosocially based individual differences in thresholds or predispositions for emotion information processing and emotion experiences, it is clear that emotion activation is not fully explained by cognitive processes. Furthermore, it may be more accurate in some cases to conceive of appraisal or attributional processes not as the necessary or sufficient cause, but rather as one of several factors in the chain of conditions and events that influence the generation of emotion.

To say that cognition is only one of several factors involved in emotion activation and emotion responsiveness in no way denigrates the important work of the researchers who have focused on appraisal and attributional processes. Cognition as cause and concomitant of emotion is undoubtedly quite common, and the study of cognitive antecedents (and consequences) has made a significant contribution to the understanding of the functions of emotions and emotion–cognition relations.

Social and Interpersonal Factors

A number of social or relationship variables might be expected to influence the emotion-activating systems. An example, drawn from extensive developmental research, is infant–mother attachment. Some of the studies in this large body of literature have focused on discrete emotions, and they suggest that the quality of the infant–mother attachment influences emotion responsiveness and the generation of emotions.

In a longitudinal project on emotional development, emotion responses (facial expressions and heart rate variability) indexed in early infancy predicted quality of attachment, and quality of attachment, in turn, predicted later emotion responses (Izard, Haynes, Chisholm, & Baak, 1991; Termine, 1990). In the reunion episode of the Strange Situation procedure (Ainsworth, Blehar, Waters, & Wall, 1978) at 13 months, insecure–ambivalent infants expressed significantly more anger and sadness and less joy than either insecure–avoidant infants or secure infants (Shiller et al., 1986; Termine, 1990). Additionally, longitudinal studies have shown that compared with secure infants, infants identified as insecure at 13 months show more negative emotions and less positive emotions in a variety of situations during preschool years and later childhood (Egeland & Farber, 1984; Sroufe, Schork, Motti, Lawroski, & LaFreniere, 1984).

The foregoing findings suggest that the socioemotional bond of infant–mother attachment influences the emotion-activating systems. Compared with secure infants, insecure infants, particularly insecure and resistant infants, apparently have lower thresholds for negative emotions, and this, in turn, is associated with lower social competence and greater vulnerability to psychological disorders (Egeland & Farber, 1984; Sroufe et al., 1984). In the case of insecure infants, the difference in emotion-activating systems is reflected in neural systems (as indicated by higher heart rate variability) and presumably in the cognitive system as well.

Environmental Factors: Stimulus Characteristics

Tomkins's (1962) model of emotion activation suggests that certain parameters of stimuli, intensity and rise time, influence emotion activation independent of appraisal or attribution. Bowlby's (1973) concept of innate (natural) clues to fear is consistent with this idea. He proposed that strangeness, height, rapid approach, and sudden changes in stimulation were natural clues to fear.

Bowlby (1973) made his strongest case for a natural clue to fear in relation to the condition of being alone. This condition might elicit fear at different ages and in many situations if, as a child, the individual had an insecure attachment to a primary caregiver who was insensitive and emotionally unavailable. In this case, an innate predisposition to vigilance or wariness on being alone is reinforced by psychological isolation, and the net effect is a lowering of thresholds for negative emotions in many situations. This is another example of biopsychological information processing, with genetically encoded information (or predisposition) interacting with cognitive evaluation. If one accepts the ideas of Tomkins (1962) and the theoretical and empirical work of Bowlby, then it follows that environmental factors and social-relationship variables may interact with (or constrain) appraisal processes in the generation of emotions.

Consistent with Tomkins's (1962) and Bowlby's (1973) notions of stimulus characteristic and natural clues, Tinbergin (1951, 1973) and other ethologists have shown that many different species of animals have innate dispositions to respond emotionally to certain stimuli. An example is appeasement behavior in the face of threat.

Similar in some respects to the ethological concept of innate releasers is the previously discussed notion of biologically prepared stimuli (see McNally, 1987; Rozin & Fallon, 1987; Seligman & Hager, 1971). After 20 years of research on this topic, some of it controversial, it is generally agreed that some human fear and disgust responses are based on environmental stimuli that successfully elicited avoidant or rejection responses during the evolution of the species. The evidence suggests that the information stored in the genes has a significant impact on the emotion responses elicited by evolutionarily or biologically prepared stimuli and that the role of cognition may not be a sufficiently dominant one to consider it a sufficient cause. Although the number of fear and disgust responses to such stimuli in contemporary societies may be limited, the number of easily derived or conditioned responses may be virtually limitless (Bowlby, 1973; Rozin & Fallon, 1987).

Adaptiveness of a Hierarchical Organization of Emotion-Activating Systems

At the outset, I suggested that from an evolutionary–developmental perspective the emotion-activating systems could be viewed as a loosely organized hierarchical arrangement: neural systems, sensorimotor systems, motivational systems, and cognitive systems. Through the selection and development of these systems, evolution guaranteed that there would be an emotion-activating process for all contingencies. Each of the emotion activation systems is particularly suited to certain of the contingencies involved in survival and adaptation.

Neural systems work continuously to maintain the background emotional experiences that are manifested in stable individual differences such as positive emotionality and negative emotionality. The sensorimotor systems operate in early infant–mother interactions to facilitate social communication and strengthen socioemotional bonds. The motivational systems activate emotions whenever drive states become sufficiently intense to disturb homeostatic and autonomic processes or when one emotion activates another to change motivational conditions and increase behavioral alternatives. The cognitive systems activate emotions when interoceptive or exteroceptive input requires appraisal, comparison, categorization, inference, attribution, or judgment.

Continuously Active Neural Systems as the Base of the Hierarchy

The evidence already reviewed suggests that neural systems are capable of generating emotions without engaging any of the other emotion-activating systems. I propose that spontaneous activity in the neural systems may periodically activate certain of the emotions or alter emotion thresholds in the absence of effective stimuli for the other emotion-activating systems. An example might be the activation of the emotion of interest to organize and motivate exploration, when exploration is adaptive but not immediately necessary for survival. Such exploration might lead to the discovery of new resources or previously unknown threats to well-being. Alternatively, interest might not be activated spontaneously but may be activated by environmental stimuli, which would indicate the involvement of the sensorimotor system.

The independent activation of emotions by neural systems may help explain some of the variance, within and across subjects, in positive and negative emotionality and emotion-related traits of personality (Diener, Larsen, Levine, & Emmons, 1985; Watson & Clark, 1992). Individual differences on these dimensions have not been effectively explained by other theories. The idea of emotions resulting from periodic changes in the spontaneous activity of the neural systems might help explain periodic depression that is apparently influenced by cyclic phenomena such as circadian rhythm and seasonal changes (cf. Bunney, 1987).

The main argument here is that in animals (including humans) that are proactive as well as reactive, neural systems at the base of the hierarchy of emotion-activating systems must be prepared to activate emotions even when environmental pressures do not demand them. This is in keeping with the assumption that emotions constitute the primary motivational system for all levels of behavior, including drive-free exploration and creativity. It is also consistent with the notion that the functions of emotions in organizing and motivating adaptive and creative behavior require some independence of other systems and of information from the environment. Finally, it is in keeping with the previously reviewed evidence that revealed individual differences in emotionality.

Sensorimotor Systems as the Second Tier

From a developmental perspective, it is logical that the sensorimotor systems of emotion activation should be second in

the hierarchy. Evidence reviewed earlier showed that sensory feedback from facial expressions could activate and regulate emotion experiences. Facial expressions are a basic component of the sensorimotor system, and a number of them are innate and universal (Ekman et al., 1969; Izard, 1971; Izard et al., 1980). Their neural substrates are in evolutionarily old parts of the brain, and at least some expressions can be elicited in anencephalic neonates (Steiner, 1973). Facial expressions are preadapted to form the basis for social communication between infant and caregiver (Izard & Malatesta, 1987), and they constitute a major aspect of infant–mother interactions (Field & Fogel, 1982; Malatesta et al., 1989; Malatesta & Haviland, 1982; Termine & Izard, 1988).

Apparently, the capacity of the infant to imitate the expressions of the caregiver is also innate or biologically prepared (Darwin, 1872/1965; Field, 1982). Even specific emotion expressions can be imitated or empathically triggered in 10-week-old infants (Lelwica & Haviland, 1981). On the basis of the previously reviewed evidence supporting expression-generated emotion experiences and the significance of expressive behavior in infant–mother interaction, it seems reasonable to conclude that sensorimotor expressive processes are a fundamental source of emotion experiences, particularly in infancy. Another reason for ascribing adaptiveness to a sensorimotor-activating system is that expression imitation and motor mimicry may facilitate the development of empathy and altruism (Hoffman, 1978), traits that may favor survival and adaptation (Hamilton, 1976; Trivers, 1971; Wilson, 1975).

Although I have placed the sensorimotor-activating systems at the second level of the hierarchy, clearly there are exceptions to the rule. Whenever any affect reaches a certain level of intensity, it can override the neural systems involved in generating background or trait emotions and the sensorimotor systems and elicit the emotion appropriate to the affect and the behavior required for adaptation. The study of infants' responses to the acute pain of DPT inoculations demonstrated the peremptory nature of this intense affect (Izard et al., 1987). However, given the cyclical nature of all the drive states except pain and the relatively brief periods that they remain at peak intensity without being satisfied, it still seems reasonable in developmental perspective to place the sensorimotor systems at the second level of the hierarchy.

Motivational Systems at Level Three

As just explained, the position of the drives and emotions (as activators of other emotions) in the hierarchy may be largely a function of their intensity. Because these states are so essential in motivating and guiding the individual's functioning, it is adaptive that they have the power to preempt the emotion-activating channels. Drive states may often interact or conflict with other emotion-activating systems, and, as described in the next section, the emotions frequently operate in conjunction with cognitive processes. The presence of emotion adds the advantage of focusing and facilitating appraisal and attribution.

Cognition at the Top of the Hierarchy

Human beings are the most complex of living systems, and they are continually confronted with an array of complex information. Adaptive responses, including emotion responses, to such information frequently require several types of cognitive processes, from simple or even unconscious appraisal to inference and propositional reasoning. One of the assumptions of the present model is that these cognitive processes are focused and motivated by ongoing emotion that is always present in consciousness. A new emotion appropriate to a change in incoming information may be activated by the joint effect of ongoing emotion and any one of the foregoing cognitive processes or any combination of them.

In the adult, cognition can transform a physically simple stimulus into a symbol for a highly complex array of information. Consider, for example, the cognitive processes and emotion experiences that followed from patient Z's ongoing interest and his perception of a few digits on a medical report. The digits, according to the doctor, indicated an abnormally high white cell count. Z quickly remembered that this was a symptom of the lymphocytic leukemia that caused the deaths of his father and one of his brothers. Other symptoms of leukemia experienced by his father and brother flashed through his mind, and he searched his memory to determine whether he had them too. He remembered one or two that seemed to belong in the category of leukemia symptoms. This combination of thoughts led Z to infer that he might have leukemia. The fear that probably began when he heard the doctor's comment on the white cell count was now intensified. After a moment, he felt anger over his bad luck. He then remembered the success his father and brother had had in coping and living with leukemia. His keen memories of their courage and happy lives brought a mix of sadness and joy. The adaptiveness of cognition at the top of the hierarchy is illustrated by the way in which his accessing and rehearsing of these memories caused his fear to subside, his interest to return to the doctor's comments on several possible causes of what might be a transient high white cell count, and his return to a more tranquil state.

Through an equally diverse set of cognitive processes, an initially complex array of information can be reduced to a simple emotion-eliciting event. Such is the case when an author's mental computations lead to the conclusion that a long list of suggestions for revision from an editor and reviewers is actually a manageable bit of work that will improve the article.

Illustrations of the Model

An idea of how the hierarchical firing of the motivational and cognitive activating systems might work in evolutionary–developmental perspective can be inferred from the studies of human infants' responses to the acute pain of DPT inoculations (Izard et al., 1983). Because the pain was unexpected, the cognitive appraisal system cannot have had a role in the immediate pain response. On the basis of the previously reviewed evidence for a pain–anger connection, there is no reason to believe that cognition mediated the initial anger response.

As soon as the infant has the capacity to associate the pain of a medical procedure with relevant features of the context and to remember them until the next trip to the clinic, cognition will surely play a prominent role in determining the emotion response. In this case, the emotion is very likely to be fear (Bowlby, 1973), not anger, and the action tendency would most

likely relate to escape behavior. Thus, once cognitive development and experience enable long-term memory and anticipation, cognitive processes, interacting with ongoing affect, mediate the appropriate emotion experience.

At the outset of this article, the description of Rafe's characteristic emotionality and his experiences of pain, anger, sadness, and sympathy on being struck from behind by Rebecca's wheelchair showed how multiple emotion-activating systems can operate hierarchically in adulthood. In this example, Rafe's trait emotion was a function of a genetically disposed neural system, and his anger was activated by event-elicited motivational processes (pain). Then, when the same physical event that brought pain and anger was cognitively appraised as the uncontrollable act of a person with hemiplegia who was embarrassed by the situation, sadness and sympathy ensued, and Rafe engaged in altruistic behavior.

Concluding Remark

In summary, the model suggests that four emotion-activating systems are continually operating and interacting to maintain the background or trait emotionality characteristic of a given personality and to activate new emotions appropriate to behaviorally relevant changes in input from any point on the information-processing continuum. This suggests that studies of emotion activation might well begin with an assessment of the state or ongoing emotion. The model and the relevant supporting data also indicate that an effective understanding of the emotional aspects of personality and behavior and the processes involved in generating emotion experiences require attention to all four modes of emotion activation: neural, sensorimotor, motivational, and cognitive.

In describing the four-systems model of emotion activation, it has been necessary to identify a number of important differences among emotion theories. This does not mean that the field of emotion is in conceptual disarray. Most major theories of emotion agree that cognitive processes are a very significant source of emotions and that emotions constitute a powerful motivational system that influences perception, cognition, coping, and creativity in important ways. Furthermore, all would agree that a science of behavior dominated by the study of cognition to the neglect of the emotions would be seriously incomplete, as some psychologists have cautioned for more than 200 years (see Hilgard, 1980).

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