Available online at www.sciencedirect.com
SCIENCE DIRECT®
ACADEMIC PRESS
Brain and Cognition 52 (2003) 129–132
Brain and Cognition
www.elsevier.com/locate/b&c

Commentary

Seven sins in the study of emotion: Correctives from affective neuroscience

Richard J. Davidson*

Laboratory for Affective Neuroscience, University of Wisconsin-Madison, 1202 West Johnson Street, Madison, WI 53706, USA

Accepted 26 September 2002

Abstract

This brief commentary highlights seven sins in the study of emotion that are explicitly treated in contemporary affective neuroscience. These sins are (1) Affect and cognition are subserved by separate and independent neural circuits; (2) Affect is subcortical; (3) Emotions are in the head; (4) Emotions can be studied from a purely psychological perspective; (5) Emotions are similar in structure across age and species; (6) Specific emotions are instantiated in discrete locations in the brain; and (7) Emotions are conscious feeling states. Each of these is briefly discussed and evidence from affective neuroscience that bears on these sins is noted. The articles in this Special Issue underscore the vitality of research in affective neuroscience and illustrate how some of these sins can be addressed and rectified using concepts and methods from affective neuroscience.

The contributions in this Special Issue attest to the growing maturity of affective neuroscience as a serious, established area of inquiry within the neurosciences. The extraordinary convergence over the past five years between research at the animal level on the neural substrates of basic emotional processes and at the human level using neuroimaging and lesion methods has helped to establish a solid foundation upon which to build this emerging subdiscipline. Having read the contributions for this Special Issue, I have decided to extract seven sins in the study of emotion that the emerging research in affective neuroscience should eventually address and help to correct. Each of these issues was touched upon by at least one of the invited contributions in this Special Issue and many of them were considered in several of the contributions.

1. *Sin 1: Affect and cognition are subserved by separate and independent neural circuits.* Many of the contributions in this collection underscore the overlap between circuitry involved in cognitive and affective processing. We now understand that emotion is comprised of many different subcomponents and is best understood not as a single monolithic process but rather as a set of differentiated subcomponents that are instantiated in a distributed network of cortical and subcortical circuits. Included within these subcomponents are different types of emotional cue recognition processes, processes involved in the production of behavioral, autonomic, and subjective changes associated with emotion, processes that serve to regulate emotion and processes that are required for remembering and retrieving emotional events. This is not meant to be an exhaustive, but rather merely an illustrative list. The Schulkin et al., the Erickson and Schulkin, and the Adolphs et al. articles all underscore this basic issue.
2. *Sin 2: Affect is subcortical.* There is a tendency among some investigators to regard emotions as largely subcortical and to sometimes also assume that cognitions are cortical. Panksepp makes this claim in his article. However, other articles that are based more on findings from humans clearly show that affect is both subcortical and cortical, and to some extent, it depends upon what the specific affect process is that is under study. The human neuroimaging literature is replete with examples that unambiguously demonstrate that affective stimuli (in comparison with appropriate control stimuli that are matched on basic physical characteristics)

* Fax: 1-608-265-2875.
E-mail address: rjdavids@facstaff.wisc.edu (R.J. Davidson).

0278-2626/03/$ - see front matter © 2003 Elsevier Science (USA). All rights reserved.
doi:10.1016/S0278-2626(03)00015-0

activate a broad network of both subcortical *and* cortical regions (see Davidson & Irwin, 1999; Davidson, Jackson, & Kalin, 2000, for reviews). A related tendency is to regard the cortical variations that are observed during emotion, e.g., in brain electrical activity measures, to derive from subcortical inputs. For example, in their article Pérez-Edgar and Fox (this issue) suggest that individual differences in temperament in young children that are manifested in behavioral, cortical EEG, and autonomic differences are fundamentally caused by variations in amygdala function. Yet Kalin, Shelton, Davidson, and Kelley (2001) have demonstrated that lesions of the amygdala in rhesus monkeys that preserve fibers of passage have no effect on behavioral, autonomic, and hormonal measures of temperament. Moreover, these authors also recorded EEG in the monkeys and found no systematic effect of amygdala lesions on measures of EEG frontal asymmetry. These findings underscore the caution that is needed in interpreting this complex literature. Certainly the amygdala may be required for the initial learning of these affective styles, but it is clearly not required once these tendencies have been learned. These suggestions are not in any way meant to marginalize the very significant contributions of subcortical processes to emotion, but rather to simply help correct the view that emotions are exclusively subcortical.

3. *Sin 3: Emotions are in the head.* There are some scientists who pay little attention to the body of subjects in whom emotion is induced. Many of the contributions in this Special Issue make the important point that emotion involves peripheral and visceral components and these are crucial in understanding its nature. The article by Adolphs et al. highlights the role of certain central circuits in the processing of visceral information or somatic states such as the insula and somatosensory structures. Hagemann et al. also illustrate the parallel changes in brain and viscera that occur in response to emotional stimuli. Porges et al. describe the adaptive changes permitted by autonomic adjustments associated with emotion in different species.
4. *Sin 4: Emotions can be studied from a purely psychological perspective.* This group of articles illustrates the power to be gained by considering the brain in the analysis of emotion. Anatomy is destiny in the study of the neural substrates of complex psychological functions. The brain's architecture and anatomy place important constraints upon psychological theory that facilitate the development of more powerful theories. For example, Schulkin et al. illustrate how visceral information can feedback upon the brain in one or two synapses and modulate activity in circuitry crucial for emotional information processing. Adolphs et al. show how the emotion of disgust may be critically dependent upon the insula, a brain region that is involved in the processing of somatic states. The critical distinction between liking and wanting is something that is directly suggested by the analysis of underlying brain circuitry as the article by Berridge reveals, though these states are often confounded in the psychological literature on positive affect.
5. *Sin 5: Emotions are similar in structure across both age and species.* There is some tendency among investigators to assume that the same basic incentive conditions will elicit the same basic emotional process in individuals at different ages. This is a particularly important but frustratingly difficult issue. For example, it is not entirely clear that stimuli that are standardized in adults as eliciting a certain type of emotion will necessarily evoke the same kinds of emotions in younger children or infants (e.g., see article by Schmidt et al. in this issue). There are important maturational and experience-induced changes in the circuitry subserving emotion and emotion regulation. Moreover, the developmental changes that occur in cognitive function shape the appraisal process and thereby influence the quality and quantity of elicited emotion. A related issue concerns the implicit assumption that the neural substrates of emotion gleaned from studies in rodents will apply to understanding human emotion (e.g., Panksepp, this issue). This is a very thorny problem. On the one hand, the rodent data have been essential in establishing some of the fundamental facts concerning the basic subcortical circuitry of emotion as Panksepp (1998) has illustrated. The convergence of aspects of the rodent work with studies in humans has been crucial for progress in this field. However, we now know that many of the anatomical details of crucial components of this circuitry are different in rodents and primates. The organization and connectivity of amygdala nuclei are different (Amaral, Price, Pitkanen, & Carmichael, 1992), the anatomy of the prefrontal cortex is fundamentally different (Goldman-Rakic, 1987) and the connectivity and functional status of the anterior cingulate is also different (Bush, Luu, & Posner, 2000). These differences in anatomy imply differences in the nature, function, and complexity of emotions across species.
6. *Sin 6: Specific emotions are instantiated in discrete locations in the brain.* This claim is often used to support the notion of "basic emotions" and the concept of "affect programs" first proposed by Silvan Tomkins (Tomkins, 1984). This latter concept holds that there are different routes to the activation of a basic emotion but once activated, there is a circuit in the brain that orchestrates a cascade of responses that are specific to each basic emotion. Most of the articles in this Special Issue reflect the view that affect is represented in distributed neural systems (e.g., Panksepp; Schulkin

et al.; Adolphs et al.; Berridge). The challenge that faces the study of emotion is similar to that once faced by investigators studying cognition—the decomposition of complex emotional phenomena into more elementary constituents. Cognitive scientists do not study "cognition" as a whole. Rather, they have developed specific paradigms to isolate more elementary stages of information processing. It is these more elementary components that will most likely yield to an analysis in terms of underlying neural systems. This view highlights the complexity of emotion and the multiple processes that are activated when emotions are elicited in the laboratory. For example, in response to a commonly used elicitor such as film clips or pictures, there are in the coarsest of distinctions, perceptual, and appraisal processes on the input side, processes that affect attentional deployment, processes involved in the generation of the somatic, visceral, and experiential features of emotion, and processes involved in the regulation of emotion. Each of these different subcomponents is implemented in different, but overlapping and interconnected circuitries. Thus when we use stimuli to arouse emotion in humans or in animals that have a fairly complex brain, it is important that we not unwittingly assume that we are activating a single process or program. The subcomponents that get triggered vary as a function of many different processes including the nature of the elicitor, and the context in which the emotion gets elicited (Davidson et al., 2000).

7. *Sin 7: Emotions are conscious feeling states.* Much of the psychological literature on emotion implicitly assumes that emotions are conscious feeling states. A vast number of studies depend upon self-report measures to make inferences about the presence of emotional states. Such self-report measures are often outcome variables in studies on emotion and they often serve as "manipulation checks" to confirm the presence of an intended emotional manipulation. Failure to find detectable change on self-report measures is sometimes offered as evidence that emotion was not elicited, and more frequently, the presence of self-reported emotion is taken as evidence that emotion has been activated. While the experiential side of emotion is unquestionably important and provides useful information to an individual that can be harnessed for adaptive functioning (e.g, Damasio, 1994), it is also clear that much of the affect that we generate is likely to be non-conscious. The article by Berridge in this issue underscores this point and reviews both behavioral and neuroscience evidence that strongly supports this conclusion. In their SPECT study reported in this issue, Tankard et al. provide evidence of only very modest relations between measures of blood flow and self-reported anxiety. Such evidence, which is representative of a plethora of findings in the literature on anxiety and depression (e.g., Davidson, Pizzagalli, Nitschke, & Putnam, 2002), underscores the importance of complementing self-report measures with other objective behavioral indices that may reflect aspects of emotion that are not adequately represented in conscious experience and thus amenable to self-report. Moreover, there are likely to be components of emotional processing that are simply opaque to conscious report. For example, I have argued (e.g., Davidson, 2000) that there are regulatory processes in emotion that occur automatically and that modulate the onset and recovery function of emotion. These regulatory processes that affect affective chronometry are likely not represented in conscious experience. They certainly affect the conscious products of affective information processing but they are not in themselves represented in conscious experience. Thus, the development of objective laboratory methods to probe affective chronometry and other aspects of emotional processing that are unlikely to be directly represented in conscious experience is crucial for the development of this field.

It is my hope that the brief description of these seven sins in the study of emotion and the role of affective neuroscience in addressing and even correcting these sins is helpful in considering some of the key issues that now face our field and challenge us in future research. The contributions in this Special Section underscore the vitality of affective neuroscience. These articles also illustrate the differences of opinion that are still pronounced, though they also suggest a number of lines of convergence that are beginning to provide a common foundation for future research. The contributions to this Special Issue also underscore the extraordinary range with which research on affective neuroscience touches the broad community of biobehavioral research. It is relevant to all areas of psychology, to much of psychiatry and to behavioral neurology. Affective neuroscience will be crucial in helping to understand development, psychopathology, personality and health. It is an emerging discipline with extraordinary vitality that should continue to grow over the coming years.

References

Amaral, D. G., Price, J. L., Pitkanen, A., & Carmichael, S. T. (1992). Anatomical organization of the primate amygdaloid complex. In J. P. Aggleton (Ed.), *The amygdala: Neurobiological aspects of emotion, memory, and mental dysfunction* (pp. 1–66). New York: Wiley-Liss.

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Science, 4*, 215–222.

Damasio, A. R. (1994). *Descartes error*. New York: Avon Books.

Davidson, R. J. (2000). Affective style, psychopathology, and resiliance: Brain mechanisms and plasticity. *American Psychologist, 55*, 1196–1214.

Davidson, R. J., & Irwin, W. (1999). The functional neuroanatomy of emotion and affective style. *Trends in Cognitive Science, 3*, 11–21.

Davidson, R. J., Jackson, D. C., & Kalin, N. H. (2000). Emotion, plasticity, context, and regulation: Perspectives from affective neuroscience. *Psychological Bulletin, 126*, 890–906.

Davidson, R. J., Pizzagalli, D., Nitschke, J. B., & Putnam, K. M. (2002). Depression: Perspectives from affective neuroscience. *Annual Review of Psychology, 53*, 545–574.

Goldman-Rakic, P. S. (1987). Circuitry of primate prefrontal cortex and regulation of behavior by representational memory. In V. B. Mountcastle (Ed.), *Handbook of physiology: 5* (pp. 373–417). Bethesda, MD: American Physiological Society.

Kalin, N. H., Shelton, S. E., Davidson, R. J., & Kelley, A. E. (2001). The primate amygdala mediates acute fear but not the behavioral and physiological components of anxious temperament. *Journal of Neuroscience, 21*, 2067–2074.

Panksepp, J. (1998). *Affective neuroscience*. New York: Oxford University Press.

Tomkins, S. S. (1984). Affect theory. In K. R. Scherer, & P. Ekman (Eds.), *Approaches to emotion* (pp. 163–196). Hilldale, NJ: Lawence Erlbaum Associates.