

Consciousness, Emotional Self-Regulation and the Brain



3 2815 00865 0049

Edited by
Mario Beauregard

Advances in Consciousness Research





Digitized by the Internet Archive
in 2024

Consciousness, Emotional Self-Regulation and the Brain

Advances in Consciousness Research

Advances in Consciousness Research provides a forum for scholars from different scientific disciplines and fields of knowledge who study consciousness in its multifaceted aspects. Thus the Series will include (but not be limited to) the various areas of cognitive science, including cognitive psychology, linguistics, brain science and philosophy. The orientation of the Series is toward developing new interdisciplinary and integrative approaches for the investigation, description and theory of consciousness, as well as the practical consequences of this research for the individual and society.

Series B: Research in progress. Experimental, descriptive and clinical research in consciousness.

Editor

Maxim I. Stamenov
Bulgarian Academy of Sciences

Editorial Board

David Chalmers
University of Arizona

Gordon G. Globus
University of California at Irvine

Ray Jackendoff
Brandeis University

Christof Koch
California Institute of Technology

Stephen Kosslyn
Harvard University

Earl Mac Cormac
Duke University

George Mandler
University of California at San Diego

John R. Searle
University of California at Berkeley

Petra Stoerig
Universität Düsseldorf

† Francisco Varela
C.R.E.A., Ecole Polytechnique, Paris

Volume 54

Consciousness, Emotional Self-Regulation and the Brain
Edited by Mario Beauregard

Consciousness, Emotional Self-Regulation and the Brain

Edited by

Mario Beauregard

Université de Montréal

John Benjamins Publishing Company

Amsterdam/Philadelphia



TM The paper used in this publication meets the minimum requirements of American National Standard for Information Sciences – Permanence of Paper for Printed Library Materials, ANSI Z39.48-1984.

Library of Congress Cataloging-in-Publication Data

Consciousness, emotional self-regulation and the brain / edited by Mario Beauregard.

p. cm. (Advances in Consciousness Research, ISSN 1381-589X ; v. 54)

1. Neuropsychiatry. 2. Consciousness. 3. Brain. 4. Emotions. I. Beauregard, Mario. II. Series.

RC343.C725 2003

616.8-dc22

2003062910

ISBN 90 272 5187 8 (Eur.) / 1 58811 458 9 (US) (Hb; alk. paper)

ISBN 90 272 5188 6 (Eur.) / 1 58811 459 7 (US) (Pb; alk. paper)

© 2004 – John Benjamins B.V.

No part of this book may be reproduced in any form, by print, photoprint, microfilm, or any other means, without written permission from the publisher.

John Benjamins Publishing Co. · P.O. Box 36224 · 1020 ME Amsterdam · The Netherlands
John Benjamins North America · P.O. Box 27519 · Philadelphia PA 19118-0519 · USA

Table of contents

List of contributors	VII
Introduction	IX
CHAPTER 1	
Emotion self-regulation	1
<i>Maren Westphal and George A. Bonanno</i>	
CHAPTER 2	
Temperament and emotional regulation: Multiple models of early development	35
<i>Susan D. Calkins</i>	
CHAPTER 3	
Emotion dysregulation and psychopathology	61
<i>Kimberly Shipman, Renee Schneider, and Amy Brown</i>	
CHAPTER 4	
Neural substrates of conscious emotional experience: A cognitive-neuroscientific perspective	87
<i>Richard D. Lane and Kateri McRae</i>	
CHAPTER 5	
Self-regulation by the medial frontal cortex: Limbic representation of motive set-points	123
<i>Phan Luu and Don M. Tucker</i>	
CHAPTER 6	
Neural basis of conscious and voluntary self-regulation of emotion	163
<i>Mario Beauregard, Johanne Lévesque, and Vincent Paquette</i>	

CHAPTER 7

The volitional influence of the mind on the brain, with special
reference to emotional self-regulation 195

Jeffrey M. Schwartz, Henry P. Stapp, and Mario Beauregard

CHAPTER 8

EEG biofeedback ("Neurofeedback") and affective disorders 239

J. Peter Rosenfeld and Elsa Baehr

CHAPTER 9

Consciousness, emotional self-regulation, and the psychosomatic
network: Relevance to oral biology and medicine 253

*Francesco Chiappelli, Paolo Prolo, Elaina Cajulis, Scott Harper,
Elaine Sunga, and Edna Concepcion*

Name index 275

Subject index 279

List of contributors

Elsa Baehr

Department of Psychology and Institute
for Neuroscience Northwestern University
2029 Sheridan Road, Evanston, Illinois
60208, USA.

Mario Beauregard

Département de psychologie, Université
de Montréal
C. P. 6128, succursale Centre-Ville,
Montréal, Québec, Canada, H3C 3J7.

George A. Bonanno

Department of Counseling and Clinical
Psychology, Teachers College, Columbia
University
525 West 120th Street New York, New
York 10027, USA.

Amy Brown

Department of Psychology, University of
Georgia, Athens
Georgia 30602-3013, USA.

Elaina Cajulis

Division of Oral Biology & Medicine,
UCLA School of Dentistry
CHS 63-090 Los Angeles, California
90095-166815, USA.

Susan D. Calkins

Department of Psychology
P. O. Box 26164, University of North
Carolina at Greensboro
Greensboro, North Carolina 27402-6164,
USA.

Francesco Chiappelli

Division of Oral Biology & Medicine,
UCLA School of Dentistry
CHS 63-090 Los Angeles, California
90095-166815, USA.

Edna Concepcion

Division of Oral Biology & Medicine,
UCLA School of Dentistry
CHS 63-090 Los Angeles, California
90095-166815, USA.

Scott Harper

Division of Oral Biology & Medicine,
UCLA School of Dentistry
CHS 63-090 Los Angeles, California
90095-166815, USA.

Richard D. Lane

Department of Psychiatry, University of
Arizona Tucson
1501 N. Campbell Ave
P. O. Box 245002, Tucson, Arizona
85724-5002, USA.

Johanne Lévesque

Département de psychologie, Université
de Montréal
C. P. 6128, succursale Centre-Ville,
Montréal, Québec, Canada, H3C 3J7.

Phan Luu

Department of Psychology, University of
Oregon and Electrical Geodesics, Inc.
1600 Millrace Dr. Suite 307, Eugene, OR
97401, USA.

Kateri McRae
Department of Psychiatry, University of
Arizona Tucson, 1501 N. Campbell Ave
P. O. Box 245002, Tucson Arizona
85724-5002, USA.

Vincent Paquette
Département de psychologie, Université
de Montréal
C. P. 6128, succursale Centre-Ville,
Montréal, Québec, Canada, H3C 3J7.

Paolo Prolo
Division of Oral Biology & Medicine,
UCLA School of Dentistry
CHS 63-090 Los Angeles, California
90095-166815, USA.

J. Peter Rosenfeld
Department of Psychology and Institute
for Neuroscience Northwestern University
2029 Sheridan Road, Evanston, Illinois,
60208, USA.

Rene Schneider
Department of Psychology, University of
Georgia, Athens
Georgia 30602-3013, USA.

Jeffrey M. Schwartz
UCLA Neuropsychiatric Institute

760 Westwood Plaza, C8-619 NPI Los
Angeles, California 90024-1759, USA.

Kimberly Shipman
Department of Psychology, University of
Georgia, Athens
Georgia 30602-3013, USA.

Henry P. Stapp
Theoretical Physics Mailstop 5104/50A
Lawrence Berkeley Laboratory University
of California Berkeley, California
94720-8162, USA.

Elaine Sunga
Division of Oral Biology & Medicine,
UCLA School of Dentistry
CHS 63-090 Los Angeles, California
90095-166815, USA.

Don M. Tucker
Department of Psychology, University of
Oregon and Electrical Geodesics, Inc.
1600 Millrace Dr. Suite 307, Eugene, OR
97401, USA.

Maren Westphal
Department of Counseling and Clinical
Psychology, Teachers College, Columbia
University
525 West 120th Street New York, New
York 10027, USA.

Introduction

During the last decade, the study of emotional self-regulation has blossomed in a variety of sub-disciplines belonging to either psychology (developmental, clinical) or neuroscience (cognitive and affective neurosciences). This emerging domain currently attracts several innovatory researchers and has become a major field of scientific inquiry. Although recent from a scientific viewpoint, the notion of emotional self-regulation was originally tackled, in the Western civilization, by the Stoics, the first philosophers to propose that it was not only possible but necessary for human beings to use the intellectual component of the mind in order to control passions. Since Plato, Aristotle and the Stoics, many philosophers (e.g., Spinoza, Smith, Hume, etc.) have vigorously argued over the relative importance and influence that should be given to reason versus emotion. Despite many ideological differences, there is one thing on which most philosophers agreed when they reflected about this important question (the same could be said of contemporary philosophers, psychologists, and sociologists): individually and collectively, negative emotions are one of the foremost causes of suffering and dysfunction on planet Earth. This is why it is absolutely crucial for human beings to learn how to adequately regulate their emotions. Hence the importance of the new scientific field investigating emotional self-regulation.

In a relatively short span of time, this field has allowed to gather an impressive amount of data and has led to the elaboration of many interesting models. In this context, one of the main goals of this book is to present theoretical and developmental perspectives about emotional self-regulation (and dysregulation), and to provide cutting edge information with regard to the neural basis of conscious emotional experience and emotional self-regulation. Another major goal of this book is to expound theoretical models susceptible of explaining how healthy individuals are capable of consciously and voluntarily modifying the neural activity underlying emotional processes and states. Related to this issue, a particular attention was paid in the conception of this volume to the capacity of human consciousness to volitionally influence brain's electrical activity and modulate the impact of emotions on the psychoneuroendocrine-immune network.

To attain these goals and contribute to the advancement of the scientific study of emotional self-regulation (whether from the perspective of psychology or neuroscience), we have collected contributions from leading researchers in this field.

As the reader will discover, diverging perspectives are expressed in certain chapters. The resolution of these litigious points will undoubtedly be paramount in order to propel the emerging field of emotional self-regulation into a new era.

Overview of chapter contents

In the first chapter of the book, Westphal and Bonanno take the stance that emotions are multi-dimensional dynamic systems that coordinate and interrelate biological and psychological organismic subsystems, and organize interactions with the environment by way of goal-directed regulatory processes. They then examine emotional self-regulation in the larger context of psychological self-regulation and emphasize automated or non-conscious types of regulatory processes. They also argue that the main objective of emotional self-regulation is the continuous maintenance of emotional homeostasis. Westphal and Bonanno finally examine different categories of regulatory processes in emotion.

In Chapter 2, Calkins approaches the construct of emotional self-regulation from a broader perspective about self-control. She describes normative developments with regard to emotional self-regulation. She also considers the various roles that temperament might play in the normative development of emotional self-regulatory skills and the apparition of individual differences in emotional regulatory capacities. Calkins finally examines a multiple-level approach to the study of emotional self-regulation as a theoretical tool to consolidate what is known regarding the behavioral and biological processes involved in emotional self-regulation.

In the following chapter, Shipman, Schneider, and Brown analyze the relation between emotion dysregulation and psychopathology within the context of the functionalist theory. This theory views emotion dysregulation as involving normal emotion regulatory processes that are operating in a dysfunctional fashion. Shipman and her colleagues argue that patterns of emotion regulation that disrupt functioning may result in symptoms of psychopathology. They then review the empirical literature that examine the links between emotion dysregulation and psychological symptomatology in children.

In Chapter 4, Lane and McRae discuss the neural substrates of conscious emotional experience from the point of view of cognitive neuroscience. In the first section they consider individual differences in the conscious experience of emotion, and then present the supportive evidence for a psychological model called “levels of emotional awareness”. In the second section, Lane and McRae review the neural substrates of emotional experience, and present a neuroanatomical model that is parallel to the psychological model. In the last sections, they examine the implications of this model for emotion self-regulation and dysregulation.

In the next chapter, Luu and Tucker first discuss how self-regulation may be conceptualized in relation to homeostasis, allostasis, and executive control. They then review findings of electrophysiological and functional neuroimaging studies showing how emotional variables alter the process of action monitoring activity in the limbic networks of the prefrontal cortex when subjects detect an error, when they receive feedback on their actions, or when they discriminate good from bad targets. Finally, Luu and Tucker propose a cybernetic model of self-regulation that integrates cognitive and emotional/motivational influences, as well as neurophysiological and neuroanatomical evidence.

In Chapter 6, Beauregard, Lévesque, and Paquette first introduce a few central concepts related to the emotional self-regulation construct and then present the results of recent functional neuroimaging studies that have been conducted by their group as well as by other researchers to identify the neural circuitry underlying emotional self-regulation. Based on their findings, Beauregard, Lévesque, and Paquette propose a putative neural circuit that may underlie emotional self-regulation. They also argue in favour of a new paradigm in cognitive psychology and cognitive neuroscience that acknowledges the human agentic capacities and consider these capacities and self-consciousness in order to understand the relationship between mind and brain processes.

In the following chapter, Schwartz, Stapp, and Beauregard review findings that demonstrate that humans are capable of consciously and voluntarily changing the neural activity associated with emotional processes and states. They argue that these findings cannot be accounted for by the principles of classical physics, which only recognizes interactions between material factors to be causally efficient. These authors further claim that a mentalistic terminology is absolutely and critically necessary to understand these findings. In the second portion of this chapter, Schwartz, Stapp, and Beauregard demonstrate how the Von Neumann formulation of quantum theory allows to integrate mentalistic and neurophysiological terminology, and explain the neuropsychology of emotional self-regulation.

In Chapter 8, Rosenfeld and Baehr first define neurofeedback, and briefly discuss its underlying principles. Using the example of the conditioning of evoked potential amplitude, they also review the evidence demonstrating that the voluntary control of brain waves is not trivially mediated by familiar phenomena. Then, Rosenfeld and Baehr give a presentation of examples of the functional significance of neurofeedback. In particular, they present a clinical application of neurofeedback, the treatment of depression via the operant conditioning of left-right frontal cortical activation.

In the last chapter of the book, Chiappelli, Prolo, Cajulis, Harper, Sunga, and Concepcion explore the relationship between consciousness, volitional control, and psycho-emotional stress with respect to the fit of the individual within the environment. Regarding this issue, Chiappelli and his colleagues discuss how

consciousness of self and others, as well as volitional control seem to influence cellular and humoral immunity. They also present evidence indicating that relaxation, meditation, and cognitive-behavioral stress management exert a beneficial influence upon psychoneuro-immune regulation. They then provide examples of psychoneuroendocrine-immune regulation in the oral cavity.

Acknowledgements

We are most grateful to Maxim I. Stamenov who, as the editor of the *Advances in Consciousness Research* series (published by the John Benjamins Publishing Company), invited us to write a book about consciousness, emotional self-regulation and the brain. Second, we want also to thank all the contributors for their beautiful and timely chapters. Finally, we thank Ms. Bertie Kaal, acquisition editor for the John Benjamins Publishing Company, for her assistance in bringing the volume to print.

CHAPTER 1

Emotion self-regulation

Maren Westphal and George A. Bonanno

Introduction

Emotions play a central role in human lives. They help people respond adaptively to environmental demands and opportunities (Frijda 1988; Levenson 1994; Nesse 1990), and cope with ongoing challenges, stressful transitions, and acute crises. The myriad ways that people regulate emotion thus have important implications for both short and long-term adjustments. Before we can begin to understand emotion self-regulation, however, we first need to specify what we mean by emotion (Gross 1998b). In this chapter, we will consider emotion and emotion self-regulation from a functional perspective. Whereas early philosophical treatises of emotion tended to view emotion as a disruptive force that is inferior to reason (Salomon 2000), the tide turned in the early eighties to a conception of emotion as essentially adaptive. This view is encapsulated in Levenson's definition of emotions as "short-lived psychological-physiological phenomena that represent efficient modes of adaptation to changing environmental demands" (Levenson 1994: 123). As evolutionary theorists have argued, biologically based genetically encoded emotions are vital to human survival by preparing and triggering rapid motor responses to potential threats. Emotions facilitate decision-making (Isen, Daubman, & Nowicki 1987; Isen 2000), promote learning (Stein, Trabasso, & Liwag 2000), and alert us to mismatches between our goals and the environment (Schwartz & Clore 1983). Emotion expression serves important ontogenetic functions by helping the infant maintain proximity to and receive nurture from caregivers, and by providing a channel for social learning (Izard & Ackerman 2000). The expression of emotions continues to structure interactions throughout adulthood (Averill 1980) and helps maintain social order (Keltner 1995; Rozin, Lowery, Imada, & Haidt 1999) by influencing judgments about other people, and by providing information about their needs, desires, and behavioral intentions.

The relational and organizing aspects of emotions in inter- and intra-organismic functioning are expressed in Campos' definition of emotions as "processes of establishing, maintaining, or disrupting the relations between the person and the internal or external environment, when such relations are significant to the individual" (Campos, Campos, & Barrett 1989). This definition also reminds us of the fact that functional does not necessarily translate as adaptive – whereas emotions always fulfill some kind of function, depending on the context, their experience and expression can have adverse consequences. How emotion is regulated within the individual and the ways in which people consciously and unconsciously regulate their internal experience and external expression of emotion in response to exigencies and on a regular basis are significant factors in the cost-benefit analysis of emotion. If we regard function as a "consequence of goal-directed action" (Keltner & Gross 1999:469), then investigating emotion self-regulation from a functional framework entails both examining the nature of regulatory processes and the goals (the 'why' of regulation) that drive them.

The view of emotion guiding this chapter is that emotions are multi-componential dynamic systems that organize and couple biological and psychological organismic subsystems (Mayne & Ramsey 2001; Levenson 1994) and structure interactions with the environment via goal-directed regulatory processes. In the following pages, we will outline the components of emotion and briefly describe a widely accepted current appraisal-based model of emotion self-regulation. Drawing on Izard's multi-system model of emotion activation, we will make the case for an alternative approach to emotion self-regulation that allows greater consideration of automated or non-conscious types of regulatory processes. For this purpose, we will consider emotion self-regulation in the broader context of psychological self-regulation and discuss how control theory principles may be applied to emotion regulatory processes. We integrate this material using the tripartite distinction of control regulation, anticipatory regulation, and exploratory regulation (Bonanno 2001). We will refer to recent insights of evolutionary theorists to demonstrate how our model may help us understand in vivo dyadic processes in emotion self-regulation. We conclude by suggesting why and how future research might benefit from integrating developmental and adult emotion research in studying the relation between intrapsychic and interpersonal aspects of emotion self-regulation.

Components of emotion

Although the majority of contemporary emotion theorists agree that emotions serve important functions, there is some divergence among theorists on the structure and necessary components of emotion (Mayne & Ramsey 2001). On the one end of the spectrum are those who view emotions as a constructed set of social categories that are culturally relative (Averill 1980; Shweder 1994). At the other end are those who insist on the existence of discrete, innate, and universal emotions with unique experiential, behavioral, and physiological attributes. For example, Ekman and colleagues have proposed that facial actions produce emotion-specific patterns of autonomic nervous system (ANS) activity that shape the quality of emotional experiences (Ekman, Levenson, & Friesen 1983; Ekman 1984). Yet other investigators prefer to view emotions in terms of dimensions of affect that are located on orthogonal continua of positive to negative valence and low to high arousal (Feldman 1995).

Many differences in the conceptualization of emotion are due to the fact that researchers have studied emotion from different levels of analysis (Ochsner & Feldman-Barrett 2001). Focusing on experiential data of emotion, such as obtained by self-report, may lead one to conceive of emotion as dimensional, whereas studying physiognomic configurations in the face associated with the expression of emotions such as anger or joy draws attention to individual discrete emotions. As we shall discover below, this same tension is evident in the study of emotion self-regulation.

Gross' process model of emotion self-regulation

In a highly influential paper, Gross defined emotion self-regulation as referring to "the processes by which we influence which emotions we have, when we have them, and how we experience and express them" (Gross 1998b:275). The central feature of Gross' model is the distinction between different emotion self-regulation strategies by their location in the emotion-generative process. Antecedent-focused regulatory processes influence the generation of emotion whereas response-focused regulatory processes alter an emotional response tendency when it has already begun. Examples of antecedent-focused strategies include the selection of situations (involving avoidance or seeking out of people, places, or things), actively changing a situation to modify its (potential) emotional impact, selectively focusing on or distracting oneself from attending to specific aspects of a situation, or cognitively reappraising the meaning

of a potentially emotion-evoking situation. Response-focused emotion self-regulation strategies include enhancing or suppressing either the experience or the expression of an emotional response.

Theoretical and empirical challenges in measuring antecedent emotion self-regulation

Common to all antecedent-focused strategies is the act of selecting situations and meanings, which presupposes conscious awareness and decision-making based on knowledge of linkages between situations and emotional responses. Conceptualizing emotion self-regulation in this way may have limited heuristic value, because often situations are emotion-provoking due to high levels of ambiguity. Thus, many emotional episodes present 'muddy terrain' in which it is difficult to discern any clear causal relations. Because gaining insight into causal pathways requires at least one if not multiple prior experiences with the emotion-provoking situation(s), it may be difficult to distinguish empirically between antecedent and response-focused emotion self-regulation.

Measuring selection of meaning (i.e., appraisal) as an antecedent emotion regulatory strategy also runs the risk of confounding emotion self-regulation with emotion-understanding or verbal knowledge of emotion. Because appraisal has predominantly been measured by self-report, most of the available data on emotion self-regulation concerns changes in peoples' construal of their emotional experiences, which may or may not be accompanied by changes in their actual experience (Robinson & Clore 2002). Furthermore, it is difficult to establish whether such verbally reported changes in experience or construal also reflect changes in peoples' ability to regulate their emotions *in vivo*.

As for situation selection, this strategy may not be a feasible option for the majority of emotional events, because it requires that opportunities exist to select-out those situations. In daily life, however, the kind of situations that generate intense emotions most frequently tend to be interactions with partners, spouses, and superordinates at work that are difficult to avoid. In most cases, we cannot choose our families, though we can try to change how often we interact with them (situation selection), and learn to modify our behavior in these interactions (response-focused emotion self-regulation). Nonetheless, the extent to which we can modify our emotional responses by means of antecedent or response-focused regulation in these familial interactions might be rather limited, because these responses are likely to present the product of

a complex and robust mix of personality traits and quasi-automatic patterns that have co-developed over a period of time.

Izard's multi-system model of emotion generation

The considerations outlined above highlight the important but under-studied role of automatic or non-conscious types of regulatory processes. Accordingly, we next consider Izard's multi-system model, which offers an alternative position on the emotion generation process by proposing three additional emotion-activating processes that are presumed not to involve cognitive appraisal (Izard 1993). Questioning the heuristic value of defining cognition as including all information-processing (e.g., Lazarus 1984, 1991a), Izard argued that information-processing should be seen as a continuum that ranges from cellular processing (enzymes and genes) to the kind of sophisticated mental operations that produced the theory of relativity. Cognition is defined as a sub-category of information-processing that is "dependent on some form of learning or experience-based memory" and non-cognitive categories include processes that guide reflexive, instinctive, biologically prepared or genetically disposed behavior (Izard 1993:70). Although all emotion activation processes involve neural activity and information processing, Izard argued convincingly that neural, sensorimotor, and motivational systems can activate emotions independently of cognitive processes.

In support of the proposition that neural processes can directly activate emotions, Izard pointed to studies documenting (a) the effect of changes in neurotransmitters levels on depression and anxiety elicited by pharmaceutical drugs and physiological mechanisms (diet, sleep, and hormones) interacting with genetic predisposition (e.g., Whybrow, Akiskal, & McKinney 1984; Thompson 1988), (b) the biochemical induction of anxiety (e.g., Charney & Redmond 1983), (c) changes in emotion expressive behavior, ANS activity, and experiential states produced by electrical stimulation of neural structures (e.g. Hess 1957), and (d) the mediation of fear response via sub-cortical structures (LeDoux 1989, 1996).

Emotion activation via the sensory system may involve one or several of the following processes: central efferent activity, instrumental behavior, muscle action potentials, posture, and facial expressions. There is increasing evidence that facial feedback may alter ongoing emotion experience and generate positive or negative emotion experiences (see Izard 1990, for a review). For instance, Zajonc, Murphy, and Inglehart (1989) showed that specific facial

muscle movements alone, independently of cognitive mediation, can change subsequent affective experiences, presumably by altering the flow of blood to the brain. Another example is Ekman and Davidson's (1993) striking finding that deliberately induced smiles could produce patterns of regional brain activity similar to those observed in relation to genuine positive emotion. Although considerably less empirical research has been conducted on the effects of posture on emotion than facial action, there is some data indicating that experimentally manipulated postures of sadness, anger, and fear may influence verbal ratings of correspondent feelings (Duclos, Laird, Schneider, Sexter, Stern, & Van Lighten 1989) and that induced slumping postures can adversely affect emotion seemingly independent of, and contrary to cognitive influences, such as experimentally manipulated success experiences (Riskind 1984).

Conceptualized as an emotion-activation system, motivation includes both emotions and drive states that arise from cyclical physiological processes (hunger, thirst, fatigue, and sex) or tissue damage (pain). To show the role of motivation in emotion generation, Izard cited studies indicating that (a) taste can elicit emotion expressions in neonates (e.g., Fox & Davidson 1986), (b) odor can generate changes in feeling states and behavior in both adults and neonates (Ehrlichman & Bastone 1992), and (c), that pain or aversive stimulation can instigate anger and aggression in animals and humans (e.g., Berkovitz 1990). Drawing on Tomkin's principle of emotion activation by density of neural firing (Tomkins 1962), Izard hypothesized that emotions (e.g., sadness) may elicit other emotions (e.g., anger) via variations in intensity of stimulation over time. Thus, emotion-elicited emotion may explain anger-sadness sequences in depressed patients described by clinicians and infants' emotional responses to unanticipated pain and separation (e.g., Izard et al. 1987).

The fourth emotion activating system in Izard's model involves four types of cognitive processes: (a) appraisal and evaluation, (b) comparison, categorization, inference, and judgment, (c) attribution and belief, and (d) memory and anticipation. Although Izard acknowledged that situations requiring the use of cognitive processes may constitute a sufficient cause of emotion, he questions whether or to what extent the presumed cognitive antecedents of emotions are themselves independent of emotion. He postulated that emotions have a continual presence in consciousness at varying levels of intensity and subtlety and constitute the motivational basis for selective perception and attentional focus.

According to Izard, the four emotion-activating systems should be viewed as a loosely organized hierarchical arrangement: at the base of the hierarchy are constantly active neural systems that produce the background emotional expe-

periences underlying stable individual differences such as positive affectivity and negative affectivity. Sensorimotor systems of emotions drive socio-emotional development by facilitating communication in early infant-mother interactions. Motivational systems of emotions come into action when drive states reach an intensity that interferes with homeostatic or automatic processes, or when emotions elicit other emotions, altering motivational conditions or broadening the range of behavioral response options. Located at the top of the hierarchy, cognitive systems activate emotions when interoceptive or exteroceptive input indicates the need for appraisal, comparison, categorization, inference, attribution, or judgment.

Implications of Izard's multi-system model of emotion activation for emotion self-regulation

Gross and Munoz (1995) argued for the merit of separating regulation by emotion and regulation of emotion. In Izard's multi-system model of emotion activation, however, each emotion activation system also functions as an emotion regulatory system. Thus, Gross and Munoz's distinction may be valid for intentional appraisal-driven types of emotion self-regulation, but becomes problematic when we try to understand automatic self-initiated processes in emotion self-regulation. Although the hypothesis of a continuously emotional mind has not yet been empirically verified, Izard's multi-system model of emotion activation alerts us to potential limitations of cognitive theories of emotion highlighted poignantly in the following quote:

Some cognitive theorists seem to assume, at least for purposes of experimental experimentation, that appraisal and attributional processes occur in an emotionless or affectively neutral mind. (Izard 1993:81)

Indeed, the dominant theoretical position is that emotion generation is essentially an appraisal-driven process; emotional response programs are set off whenever an individual evaluates a situation as offering important challenges or opportunities (Lazarus 1991b; Tooby & Cosmides 1990). Similarly, Frijda stated that "emotions result from meanings, and meanings, to a large extent, from inferred consequences or causes" (Frijda 1986:310). This view makes it difficult if not impossible, however, to map the type of non-cognitive processes included in Izard's multi-system model. The discrete emotions perspective proposes a direct pathway whereby specific appraisal processes trigger a matching set of "loosely coupled" response tendencies (Bonanno & Keltner, in press)

including experiential or felt responses, expressive or behavioral responses, and associated physiological components (e.g., neural processes, sympathetic and parasympathetic systems, and various hormonal pathways). Although appraisal processes do sometimes elude conscious awareness (Lazarus 1991), the rapid and specific nature of discrete emotional responses generally makes their antecedent appraisals readily identifiable and more amenable to experimental observation (see Bonanno & Keltner, *in press*). In comparison, it is relatively difficult to identify the antecedent appraisal processes that produce affective states. Affect states tend to be relatively global, diffuse, and long-lasting. One way to integrate these different aspects of emotional experience, proposed recently by Russell (2003), is to view affective states as both free-floating (mood) and as possible precursors of appraisal processes that, in turn, may activate discrete emotional states.

Izard's emphasis on non-cognitive pathways to emotion is accordant with recent non-linear dynamic systems approaches that view sensitivity to initial conditions and linkage between individual emotions as integral to emotion processes (Mayne & Ramsey 2001). Echoing Izard's earlier criticism, Mayne and Ramsey (2001) pointed out that affect scientists have defined emotion essentially in stimulus-response terms. Different definitions of emotion as "ever ready monitoring of event relevance and concomitant adjustment readiness" (Frijda 1986:477), "short-lived psychological-physiological phenomena that represent efficient modes of adaptation to changing environmental demands" (Levenson 1994:123), and "relevance detection and response preparation system" (Scherer 1994:128) all conceptualize emotion as a response system. In contrast, Mayne and Ramsey view emotions as multi-componential dynamic systems that serve to organize and couple myriad organismic subsystems (Mayne & Ramsey 2001:20). Time is regarded as a crucial element in these organizational processes, because current emotional activity, like any other event in dynamic systems, tends to be influenced by past activity, as well as being likely to affect subsequent emotions.

As we will show in the following sections, applying control theory principles to emotion self-regulation helps to understand how cultural, individual, and social systems may influence the occurrence and outcome of regulatory processes operating at different levels of automaticity and conscious control.

Emotion self-regulation and emotional homeostasis

Living systems, including *Homo sapiens*, are characterized by complex self-regulating mechanisms designed to maintain a relatively constant biological balance. Similarly, human beings have different thresholds for and tolerance of emotional arousal that are determined both by congenital factors (e.g., temperament) and ontogenetic factors (the interaction of temperament and external influences, such as caregiver practices). We propose that conceptualizing emotion self-regulation as a variant of more general self-regulatory processes can help to integrate these structural features and dynamic processes. Our approach is based on the premise that the central goal of emotion self-regulation is the ongoing maintenance of emotional homeostasis (Bonanno 2001). Using a control theory framework, Bonanno (2001) conceptualized emotional homeostasis in terms of schematized goals that pertain to ideal frequencies, intensities, or duration of experiential, expressive-behavioral, or physiological channels of emotional responding. As we illustrate later in this chapter, emotional homeostasis will differ for specific kinds of emotion, and across individual, cultural, and situational variation.

Homeostasis is maintained via different types of regulatory activity that are instigated by the operation of open- and closed-loop control systems. Open-loop control systems, also called feed-forward systems, are programmed to launch specific actions that run to completion regardless of their consequences. Applied to human beings, examples of actions governed by feed-forward systems include reflexes, such as withdrawing one's hand from a hot surface. Such behavior programs are tightly organized and thus allow for little flexibility and adaptation. In situations in which it is necessary to quickly react to avoid damage, they prove more adaptive than behavior that is based on thorough cognitive reflection and analysis of multiple aspects of the situation. In other cases, however, inflexibility and insufficient sensitivity to contextual details is maladaptive, even in a situation that is as seemingly straightforward as touching a hot surface. For example, if the hot surface concerned is, say, the handbrakes of a car, and immediately withdrawing one's hand would result in the car crashing into the parking wall, it might be wiser to temporarily endure the heat. Doing that would require overriding a prepotent response, withdrawing one's hand, in favor of a more long-term goal, maintaining control over the car.

A second control system that allows for more flexibility in behavioral regulation is the closed-loop system or negative feedback loop. Negative feedback loops involve the operation of two control components, a reference value that designates a desirable set point (e.g., temperature), and a comparator that

monitors the relation between internal and external functioning and the reference value. Discrepancies or mismatches set off regulatory processes that continue until the reference point is met. A thermostat exemplifies the functioning of negative feedback loops by continuously regulating fluctuations in temperature via heating or air conditioning to match a pre-set optimal range.

Several affect theorists have applied the thermostatic control system model to mood regulation in humans (e. g., Larsen 2000; Carver & Scheier 1981, 1982, 1990). In Carver and Scheier's control model of positive and negative affect (1990), the set point in the control system consists of a goal, and what is monitored is progress toward this goal. If progress towards the goal is perceived as too slow, negative affect results, whereas fast progress produces positive affect.

An important limitation of Carver and Scheier's model is its conceptualization of affect as a by-product of behavioral self-regulation. Their model is more concerned with the regulation of goal directed behavior than with affect *per se* (Larsen 2000). Goal-directed behavior, however, is only one component of affect and of emotion. Larsen's application of control theory of mood regulation addresses this limitation and describes the role of six individual difference mechanisms that interact with affect-relevant environmental information and affect regulation mechanisms within the control loop sequence (Larsen 2000). These individual difference mechanisms include (a) attention to affect-relevant stimuli in the environment, (b) the degree to which regulation strategies effect changes within the person or in the environment, (c) temperament-based affective reactivity, (d) attention to or perception of current state, (e) sensitivity to one's current state, and (f) desires, beliefs and values regarding the optimal mood state. These individual difference subcomponents are assumed to affect the regulatory processes with varying degrees of automaticity and effort.

Larsen's model of mood regulation can be usefully applied to emotion self-regulation in illustrating how regulatory processes are intertwined with different aspects of information processing stages. There are two aspects of this model, however, that are problematic: first, beliefs, desires, and values, rather than constituting a subcomponent of regulatory processes may play a more central role in adult emotion self-regulation processes than in mood regulation. This is because emotion tends to be directed towards an object, whereas mood is a more diffuse state. Second, Larsen describes the regulatory process as motivated by the desire to "create and maintain generally pleasant or positive subjective states" (p. 131). Such an assumption of hedonistic pleasure is an oversimplification and may not pay justice to the complexity of human motivation. Emotional homeostasis should not simply be equated with the achievement of a hedonistically positive state.

To gain a more comprehensive understanding of emotional homeostasis, it is important to take a closer look at what the reference values in the control system signify. The principles and models of control theory were formulated in the fields of mechanics and computer science. In the course of the cognitive revolution, many psychologists turned to computer language and concepts in an attempt to model specific cognitive processes. Although this led to great advances in many areas in the field, likening human behavior to mechanistic metaphors can also hamper scientific progress by oversimplifying complex processes and neglecting contextual factors. To some extent, the thermostat analogy is flawed, because emotional homeostasis in human beings denotes a dynamic process of striving towards an ideal state rather than achieving a set point that can be measured. Rather than limiting the meaning of reference value in a model of human emotion to a set point of a desired mood state, we propose that the construct may be used to understand the way in which complex psychological constructs such as idealized self-images and other subjective cognitive schemata and abstract goals impinge upon regulatory processes (cf. Carver & Scheier 1990).

Extending the meaning of reference values in this way helps to understand situations in which a person's behavior may appear emotionally unintelligent, irrational, or even self-destructive (Bonanno 2001). When evaluating the operations of control systems, it is important to realize that the output of the negative feedback control system – that is, the actual behavior that might be observed from an objective perspective, is irrelevant. Feedback control systems are organized to keep their inputs (the feedback) matching the reference values that are driving regulatory processes. Thus, the feedback does not come directly from any specific behaviors performed, but rather, from their consequences. Therefore, to understand the operation of control systems we have to identify the end results of behaviors, from which we may infer the content of the reference value.

In this inductive process, it is imperative to consider that control systems are hierarchically organized, with subordinate loops embedded within superordinate loops (Powers 1973). Superordinate loops tend to be linked to longer-term abstract goals (e.g., succeeding at one's career), whereas subordinate loops are associated with proximal mechanisms (e.g., regulation of chronic back pain). Level of abstraction and corresponding status in the control systems hierarchy may increase the likelihood of some feedback loops overriding the regulatory processes of other feedback loops. This can occur whenever there is competition between simultaneously operating feedback loops that are driven by reference values or goals that are either permanently or temporarily incom-

patible. For example, workplace pressure imposed by an impending deadline may escalate the conflict between career ambitions and health concerns. Regulatory behaviors instigated by the control loop concerned with regulating back pain, such as taking a long break or spending less time sitting at the desk will satisfy the short-term goal of relieving back pain, but may hinder one in meeting the deadline and thus impede progress towards the long-term career goal.

Baumeister and colleagues have defined self-regulation in terms of higher processes overriding lower processes and consider the reverse a failure of self-regulation that can be attributed to insufficient strength or will-power (Baumeister, Heatherton, & Tice 1994; Leith & Baumeister 1996). This may, in part, be true for addictive behaviors, which is the main focus of research on self-regulation in adults. If we take a broader view of self-regulation that includes emotion self-regulation as a sub-type, however, it becomes clear that this definition is too narrow if not misleading. Although couched in the language of systems theory, it assumes a unidirectional relation between the two regulatory processes that does not sufficiently capture the dynamic nature of self-regulation. In the example of meeting a workplace deadline, optimal self-regulation in the long run does not uniformly involve adhering to work demands (the higher process) and overriding the signals that instigate pain-regulatory processes as the proximal process. Rather, the abstract goal of career success needs to be aligned with the proximal goal of health protection, so that their associated feedback loops work in unison, one enhancing the functioning of the other.

Applied to emotion self-regulation processes, regulatory failures may indeed occur when internal or external emotional stimuli set off biologically programmed automated emotional processes that override more abstracted self-regulatory processes (e.g., as in the fear response). If we remind ourselves of the adaptive value of emotions, however, it is conceivable that the reverse could also constitute a self-regulation failure. Take the well-documented gender difference in the marriage-health connection, for example. Married men, in general are healthier than unmarried men, irrespective of reported marital satisfaction, whereas married women, in general, are less healthy than unmarried women, and derive health benefits only from being in a good marriage (e.g., Levenson, Carstensen, & Gottman 1993; Levenson, Carstensen, & Gottman 1994).

This pattern may be due to the way in which men and women typically deal with marital conflict: wives engage, husbands withdraw (e.g., Carstensen, Gottman, & Levenson 1995; Carstensen, Graff, Levenson, & Gottman 1996).

Both experience similar levels of physiological arousal during conflict, but women tend to disregard their aversive emotional states in trying to maintain the relationship. From an emotion self-regulation perspective, we hypothesize that women's failure to access and reduce their emotional arousal in marital conflict (i.e., self-regulate) may be due to higher-level abstract self-regulatory loops (e.g., driven by a reference goal associated with self representations as a socially connected and responsive wife) consistently overriding proximal feedback loops concerned with protecting the organism from aversive emotional stimulation.

This example underscores the complexity of emotion-regulatory processes in illustrating how cultural factors (socialization of gender roles) may impact on the operation of different feedback loops. It is important to realize that control systems seldom operate in isolation from other control systems, which on a broader level may include societal institutions. Thus, our main empirical data, the consequences of regulatory processes, may constitute the joint product of multiple interacting control systems. Another factor that complicates the examination of specific emotion self-regulation processes is that reference values in humans do not represent fixed entities. Whereas in mathematics the term value refers to results produced by the application of mathematical functions, the reference values involved in different emotion regulatory feedback loops may overlap, cross, or change over time, depending on their level of resistance towards competing reference values and external pressures.

A sequential model of emotion self-regulation

We introduced the concept of emotional homeostasis as central premise underlying our model of emotion self-regulation, explained a number of principles assumed to characterize the operation of regulatory mechanisms, and discussed some previous applications of control theory to affect regulation. We will now explicate three categories of regulatory processes in emotion and link them to a number of behaviors and processes that are relevant to emotion self-regulation.

Control regulation

Control regulation encompasses automatic processes and instrumental behaviors associated with the immediate regulation of emotional responses that have

already been instigated. This category corresponds to the class of emotion self-regulation processes that Gross describes as response-focused. Control regulation “includes mechanisms that dampen or short-circuit emotion responses, such as emotional dissociation and suppression, as well as mechanisms that serve to accentuate and/or intensify emotional responses” (Bonanno 2001: 15).

Emotional dissociation

Although discrete emotions are generally thought to produce a coordinate set of emotional responses or response programs involving experiential, expressive-behavioral, and physiological components, a large body of research attests to the co-occurrence of contradictory or inconsistent emotional responses in different response channels (Buck 1988; Ekman 1992; Izard 1977, 1992; Lang 1979; Lang, Kozak, Miller, Levin, & McLean 1980; Levenson 1994; Leventhal 1984, 1991; Schwartz, Fair, Greenberg, Freedman, & Klerman 1974; Schwartz, Fair, Salt, Mandel, & Klerman 1976). An everyday example for this phenomenon is a sales person who might eagerly compliment a customer while simultaneously expressing disgust in his face. Conversely, subjective emotion responses may be experienced in the absence of overt behavioral expression, for instance, when a speaker feels anxious when giving a speech, but appears confident and relaxed to the audience.

Another type of experiential-expressive discrepancy that appears to arise from at least partially automatic processes rather than conscious regulatory efforts is emotional dissociation. Processes of this nature are characterized by a transient shift in the focus of attention away from awareness of a particular, and usually negative, emotional response toward other, more benign content. This attentional shift may serve a regulatory function by attenuating or diminishing the subjective experience of aversive emotions that would normally accompany the physiological arousal elicited by potentially stressful situations.

Viewed from a control theory framework, the attentional shifts operative in emotional dissociation may regulate emotions by either short-circuiting or undoing the undesirable emotional response output, or instigating a competing emotional response that might over-ride the undesired response (Bonanno 2001). Although such dissociative shifts have been linked to over-arching, long-term goals and personality traits (e.g., Bonanno & Singer 1990), they manifest at a subordinate point in the regulatory hierarchy, close to the output end of the chain of control responses, and thus function in a relatively automated fashion (Bonanno, Keltner, Holen, & Horowitz 1995).

One of the most widely used measures of emotional dissociation is affective-autonomic response dissociation, which is observed when subjective emotional experience is disproportionately low compared to the intensity of the observed autonomic response (Newton & Contrada 1992). Research has shown that individuals categorized as repressors are particularly likely to obtain high affective-autonomic discrepancy scores (Weinberger, Schwartz, & Davidson 1979) and show a general propensity to selectively attend away from threatening information (e.g., Bonanno, Davis, Singer, & Schwartz 1991).

Shifting awareness away from distressing emotion has traditionally been viewed as a form of maladaptive denial that may incur deleterious physical and emotional costs over the long-term (Shedler, Mayman, & Manis 1993). The hierarchical organization of regulatory feedback loops described in the preceding section, however, leads us to expect that emotional dissociation may render subsequent self-control efforts more likely than regulatory processes that require identifying the sources of distress (e.g., appraisal), because automatic control processes may put less demands on limited cognitive resources. This view contrasts with an ego depletion model that implies that any sort of self-regulation depletes cognitive resources (e.g., Muraven, Tice, & Baumeister 1998).

Support for the view that emotional avoidance may potentially serve an adaptive function comes from studies linking affective-autonomic response dissociation with better overall adjustment among sexually abused adolescent girls (Bonanno, Noll, Putnam, O'Neill, & Trickett, *in press*) and middle-aged adults coping with the recent death of a spouse (Bonanno, Keltner, Holen, & Horowitz 1995). One of the possible ways in which affective-dissociation may have assisted these individuals in their coping efforts is by allowing them to minimize the disruptive impact of intense negative emotions.

Affective-autonomic dissociation has also been linked to the generation of positive states such as genuine or Duchenne laughter (Bonanno *et al.*, *in press*; Keltner & Bonanno 1997). For example, Keltner and Bonanno (1997) found that bereaved individuals who showed genuine laughter while talking about their loss were significantly more likely to evidence autonomic-affective dissociation than those who did not laugh. Similar to autonomic-affective dissociation, laughter often arises in negative contexts that seem incongruous with the experience of positive emotions. By creating mental distance to the negative stimuli, autonomic-affective dissociation may help to preserve or restore the cognitive flexibility that is needed to recognize novel aspects of a situation that afford humorous interpretations. As the individual begins laughing, a new chain of regulatory processes is set into motion with relaxing

effects that may spill over into the laughing individual's social environment and generate further opportunities for experiencing positive emotions. The expression of positive emotions might also offset the physiological arousal caused by autonomic-affective response-dissociation and benefit psychosomatic health by eliciting positive responses from other people that help maintain social support networks (Keltner & Bonanno 1997).

Enhancing and suppressing emotional expression

Our discussion of the potential role of autonomic-affective dissociation in the course of bereavement illustrates the general point that the adaptive value of autonomic-affective response mechanism or any other regulatory mechanism depends on a myriad of factors, including timing, chronicity, cultural norms, and situational demands. Rather than asking whether a particular mechanism is beneficial or maladaptive *per se*, a more ecologically valid approach to studying emotion regulatory processes may be to consider them as skills that involve trade-offs in different aspects of human functioning. These include an individual's mental and physical health, as well as his or her expectations, values, and goals concerning various economic, social, and cultural variables that are relevant to overall quality of life – that is, what we refer to as reference values.

Adopting such a systemic framework may help to qualify the polarized perception of expression as healthy and expressive suppression as unhealthy that seems to dominate the literature on adult emotion self-regulation and related areas in psychology (e.g., Salovey 2001; Consedine, Magai, & Bonanno 2002). Defined as conscious inhibition of emotion while emotionally aroused (Gross & Levenson 1993), suppression has been found to result in poorer self-reported and objective memory (Richards & Gross 2000), increased sympathetic activation (Gross 1998a; Gross & Levenson 1993, 1997), and intrusive, ruminative thought (e.g., Pennebaker & Susman 1988; Pennebaker 1990, 1993, 1995; Pennebaker & Beall 1996; Pennebaker & Seagal 1999). As a personality-related habitual behavior, suppression has been associated with increased vulnerability to stress-related diseases and adverse outcomes in cancer and cardiovascular disease (e.g., Cooper & Faragher 1992, 1993; Pennebaker, Kiecolt-Glaser, & Glaser 1988; Ornish et al. 1990; Sapolsky 1998, 1999; Stein & Spiegel 2000; Giese-Davis & Spiegel 2001).

From a control theory perspective, suppression may be conceptualized as control regulatory mechanism that aims to minimize emotion response

outputs that are incompatible with an individual's reference goals or values. Although probably driven by similar reference goals as emotional dissociation, suppression is likely to operate at a more superordinate level in the feedback control hierarchy and appears to have opposite effects on the expression and experience of emotion: as a study using disgust-eliciting film material with undergraduates showed, suppression was effective in reducing the overt display of disgust (Gross & Levenson 1993), but did not change the self-reported experience of emotion. In contrast, affective-autonomic response dissociation appears to be fairly effective in reducing the experience of self-reported (negative) emotions, but clinical observers have rated repressors as actually showing more distress than expressors (Asendorpf & Scherer 1983; Fox, O'Boyle, Barry, & McCreary 1989).

Conceptualizing suppression as a regulatory strategy that serves to maintain emotional homeostasis may cast cultural and individual variations in emotional expressiveness in a different light. Thus, in Japan or China, inhibiting the expression of negative emotions is a socially desirable behavior that is consistent with the value placed on harmonious interpersonal relationships in these interdependent cultures (Markus & Kitayama 1991; Mesquita 2001). If reference values center on the avoidance of social conflict, the apparent ineffectiveness of suppression in controlling or reducing the experience of emotion may be irrelevant. On the other hand, if the goal is to reduce the experience of emotion, then suppression may be a relatively ineffective, and potentially harmful strategy.

To understand when emotion expression may contribute to or interfere with emotional homeostasis, it is important to consider how cultural factors may interact with individual differences and self-evaluations. For example, a recent study on personality and emotion found that the experience of negative emotion was positively correlated with the expression of negative emotion in dispositionally high-expressive people, but not in low-expressive people (Gross, John & Richards 2000). This suggests that the relation between emotional experience and expression may be moderated by dispositional expressivity.

Expression can carry very different meanings for different people (e.g., Kitayama & Markus 1996; Mesquita & Frijda 1992; Porter & Samovar 1998; Surgenor & Joseph 2000); some people may experience involuntary expression as shameful lapse in self-control, while others might feel very comfortable with overtly expressing emotions (Kennedy-Moore & Watson 1999). The regulatory functions of emotional expressions may vary not only among different people, but also across different types, intensities, and frequencies of emotion. For

instance, it is a well-known observation that expressing anger can magnify and prolong the experience of anger (Ebbesen, Duncan, & Konecni 1975).

Chronicity is likely to be an important factor in predicting when the expression of negative emotion is beneficial (e.g., by bringing about insight), or produces emotional dysregulation. As research on patients suffering from post-traumatic stress disorder (PTSD) indicates, the unrestrained or chronic expression of negative emotions associated with traumatic events may impede psychological recovery (Van der Kolk 1994). Studying reactions to bereavement in a normal population, Bonanno and Keltner (1997) found that facial expressions of negative emotions, in particular anger, predicted a more protracted grief course, even when initial levels of grief and the self-reported experience of emotion were statistically controlled (Bonanno & Keltner 1997). In contrast, the expression of positive emotions while relating distressing information about the loss of one's spouse predicted reduced levels of grief at subsequent assessments.

Perhaps a more important issue in determining the relative costs of expressing or suppressing emotions is whether or not these regulatory processes can be employed in a flexible manner when enacting different roles to match situational demands and opportunities. From a control theory perspective, one may view situational constraints as interacting with an individual's goals and values through a multi-faceted self concept (Higgins 1987; Markus & Nurius 1986; Singer & Salovey 1993). For example, a person occupying a position of authority (e.g., supervisor of a large group of employees) may have a sense of self in this role as being agreeable, committed, and fair-minded. Upon activation, this self-structure may drive the regulatory system toward the suppression of negative emotion in some situations (e.g., dealing with a troubled employee) and to the enhanced expression of emotion in others (e.g., delivering a passionate speech to motivate production). By the same token, situations that are unrelated to the supervisory role (e.g., a lunch date with an old friend) may access other features of self (e.g., that of a fun-loving and gregarious person) that instigate different forms of expressive regulation.

To examine the regulatory functions of expressing negative emotions in therapy, it may be helpful to distinguish between primary and secondary classes of emotional expression (Greenberg & Safran 1987). Primary emotional reactions arise from biologically based, basic emotions that provide adaptive information for functioning. As Kennedy-Moore and Watson (2001) have pointed out, patients entering therapy often are unaware of these emotions and their recognition and expression in therapy may elicit insight and lead to therapeutic changes. In contrast, expressing secondary emotional reactions is likely to be

maladaptive, because it involves recycling highly accessible and well-rehearsed feelings and corresponds to passive rumination.

Whether or not we accept Greenberg and Safran's distinction between primary and secondary emotional reactions, it is important to consider both the risks and benefits that emotional expression may pose outside the safety of the therapy setting. Although verbal disclosure of emotionally charged information can be helpful in eliciting social support, it may have adverse interpersonal consequences depending on what is expressed, to whom, and how (Kelly & McKillop 1996; Kennedy-Moore & Watson 2001). For example, the listener may misread the expresser's intent or minimize or dismiss the importance of the communication, leaving the communicator feeling rejected and humiliated (e.g., Lehman, Ellard, & Wortman 1986; Major, Cozzarelli, Sciacchitano, Cooper, Testa, & Mueller 1990; Silver, Boon, & Stones 1983; Silver & Wortman 1980; Silver, Wortman, & Crofton 1990). Distress expression in close relationships can instigate or intensify negative escalation cycles, particularly if the communication is perceived as implying blame (Gottman & Levenson 1986; Julien, Markman, & Lindahl 1989).

Anticipatory regulation

Anticipatory regulation of emotion involves identifying and implementing different strategies for maintaining emotional homeostasis. Anticipatory regulatory activity is dependent upon the achievement of a relative state of emotional homeostasis in which immediate needs for control regulation are minimal. At this point in the regulatory sequence the next form of evaluation pertains to goals oriented around anticipated challenges to homeostasis. These strategies encompass the deliberate, antecedent-focused processes described by Gross. We will not review those processes here, but instead refer readers to Gross' (1998a, 1998b, 1999, 2002) insightful reviews. Our view of anticipatory regulation complements and expands earlier models by considering in more detail processes that do not necessarily require deliberate conscious activation.

We would argue, for example, that changing the meaning of potentially emotion-eliciting events via reappraisal, a process typically studied using a deliberate conscious manipulation (e.g., Gross & Levenson 1993), might also be enacted in a relatively automated manner. Individuals who characteristically engage in overly positive or self-enhancing biases in perception and attribution may exemplify such automated forms of reappraisal. These self-enhancers appear to utilize numerous forms of reappraisal and reattribution, such as

downward social comparisons (Helgeson & Taylor 1993; Taylor, Kemeny, Reed, & Aspinwall 1991), or positive reframing (Taylor, Wood, & Lichtman 1983). One of the defining characteristics of dispositional self-enhancers is their absence of insight and awareness into their use of these mechanisms (Paulhus 1991). Self-enhancers' habitual and nonconscious use of avoidant strategies may explain, at least in part, why they experience and express reduced levels of negative emotion when confronted with highly aversive situations and cope unusually well (Bonanno 2002; Bonanno, Field, Kovacevic, & Kaltman 2002).

We might also consider that newly acquired regulatory skills could become habitual or even automated over time. For example, an individual who has overcome physical dependence on substance use in rehabilitation and has begun to understand his psychological dependence may plan to attend a support group to receive continued reinforcement of his intent to stay sober. Outside the group setting, he may consciously avoid social settings that are likely to trigger a relapse into addictive behaviors. Participating in support groups or individual therapy may also provide opportunities for developing or improving emotion control skills; for instance, exposure treatment and stress inoculation have as their explicit goal helping patients gain skills to confront and manage fear (Rothbaum & Foa 1994). Similarly, popular press psychology books, workshops, Yoga, and especially Meditation present further resources for learning about different ways to regulate one's emotions. With practice, the application of these acquired skills in everyday life may cease to require conscious effort.

Social approaches to emotional expression offer multiple examples of how emotional expressions help to maintain both individual and social homeostasis by reinforcing predictable interactions and consolidating social hierarchies. A recent application of the selfish-gene hypothesis to the evolution of emotional expression highlights the survival value of smiling and laughter (Owren & Bachorowski 2001). According to Owren and Bachorowski's analysis, smiling and laughter can instigate positive feedback loops between two individuals that foster the mutual growth of positive affect. If enacted repeatedly, such feedback loops can transform an initially mild positive emotional stance between two genetically unrelated individuals into a stable affective bond that provides the basis for reciprocal cooperative behaviors. In this way, smiling and laughter not only serves to regulate present interactions, but also anticipates and ensures the fulfillment of future material, physical, and emotional needs.

Exploratory regulation

Exploratory regulation encompasses behaviors that foster the development of skills, build knowledge, or help to access new resources that incidentally contribute to the maintenance of emotional homeostasis. Exploratory regulations may come into play when immediate or anticipated control needs are perceived to be absent, and can occur through vicarious experience of emotional situations depicted in movies, folk-tales, or novels. By observing other people grapple with moral and social dilemmas, or by imagining themselves in the role of the protagonist, individuals may get a feel of different experiential realities and learn about possible solutions and their associated emotions. Many people seek out certain genres of film such as thrillers or comedies or activities (e.g., roller coasters) to experience specific emotions in an intensity that may be undesirable, rare, or impossible to find in their everyday lives. The safety of the context combined with the strength of the stimulus provides the opportunity to explore, observe, and learn about these intense emotional responses. Rock climbing, parachuting, and other extreme sports may also promote future emotion self-regulation, for example, by honing concentration skills, practicing patience, and allowing experimentation with novel ways to manage negative emotions or accentuate positive emotions.

All creative activities have the potential for serving as a form of exploratory regulation through which we may explore possible solutions to emotional control needs. For example, exploratory writing has been found to be a health-promoting tool for therapeutic inquiry, self-understanding, and self-expression (e.g., Pennebaker 1989, 1990, 1993, 1995; Pennebaker, Colder, & Sharp 1990; Pennebaker & Seagal 1999; Rabinor 1998), not only when used as part of a personal journal, but also when describing imaginary reactions to another person's trauma (Greenberg, Wortman, & Stone 1996). As Greenberg and colleagues argued, imagining another person's trauma "may foster self-empathy by allowing participants to observe their own emotional pain in a context uncontaminated by knowledge of failed coping efforts and associated self-derogation" (p. 599). Linking observed health gains from writing to the opportunity to construct "resilient possible selves" is consistent with the earlier mentioned idea of self-schemas presenting superordinate reference values that drive regulatory processes.

Because exploratory regulation typically does not have an "agenda" and is driven by reference goals that tend to be more diffuse and abstract than those underlying control and anticipatory regulation, it allows for greater cognitive flexibility and openness to novelty. These qualities are integral to any kind of

creative activity, including children's play. Whereas anticipatory emotion self-regulation often draws on relatively advanced analytical skills in utilizing past and ongoing experience to find solutions to anticipated problems, exploratory regulation can be a multi-sensory process that does not require a specific level of cognitive sophistication. Indeed, it could be argued that exploratory regulation may be more pervasive in childhood than in any other stage of human life.

Consistent with the suggested link between play and emotion self-regulation, Winnicott defined mental health as the ability to play. Play requires a tolerance of ambiguity or, as Winnicott puts it, an acceptance of "...the precariousness of the interplay of personal psychic reality and the experience of control of actual objects" (Winnicott 1971:55). Play spurs the development of children's regulatory abilities by allowing "the experience of magical control" or "omnipotence." Viewing play and creative activities as essential aspects of emotion self-regulation may present a rationale for including art therapies in the treatment of various psychopathological symptoms, such as communicative disfluency and rumination in depression, stereotyped repetitive behaviors in autism, and compulsions in Obsessive-Compulsive Disorder (OCD).

Emotion self-regulation as a contextually-bound process

As this chapter demonstrated, considering emotion self-regulation in the broader context of psychological self-regulation has several benefits in trying to understand the functions and consequences of different emotion regulatory processes. The concept of emotional homeostasis highlights the biological underpinnings of emotion self-regulation, but does not limit our scope of inquiry to proximal processes aimed at self-preservation. Conceptualizing regulatory processes as feedback loops that are hierarchically organized around reference goals of varying complexity points to ways in which culture may influence the use of emotion self-regulation strategies. Focusing on underlying reference goals can help to explain under what circumstances it may be adaptive to inhibit emotional expression and experience, and when up regulating emotions fulfils important functions. Many researchers seemed to equate self-regulation with self-control in presuming that conscious awareness is a prerequisite for emotion self-regulation (e.g., Baumeister, Heatherton, & Tice 1994; Tice & Bratslavsky 2000). Distinguishing between control, anticipatory, and exploratory regulation helps to understand the role of automatic and unconscious regulatory processes, as well drawing attention to types of emotion

self-regulation such play and creative activities that have not been addressed by previous models.

Viewing emotional homeostasis as the organizing principle of regulatory processes underscores the dynamic nature of emotion self-regulation and is compatible with the position that emotion is inherently regulatory and regulated (Cole, Michel, & Teti 1994). Many models of emotion generation or emotion self-regulation do not sufficiently address the fact that emotion regulatory processes are not steered solely by the individual, but rather are often influenced by others in an ongoing fashion. This may, at least in part, be due to the way these processes have been studied: as Parkinson has pointed out, most experimental studies of emotion self-regulation have employed a stimulus presentation paradigm that implies and reinforces a split between emotion sender and receiver (Parkinson 1995). It may be argued, however, that detecting another person's emotional expressions and responding to these expressions is an inherently social process that should not be studied exclusively in terms of individual behavior. In contrast, for example, observations of autonomic linkage in couples discussing conflictual topics (e.g., Gottman & Levenson 1986) show how interactive processes shape an individual's emotion self-regulation at multiple physiological and psychological levels. As part of a systemic framework, the concept of emotional homeostasis is not tied to intrapsychic aspects of regulatory processes, but can be used to explain the complex interplay of sender and receiver interests in emotion self-regulation. As an example, we mentioned Owren and Bachorowski (2001) evolutionary account of smiling and laughter as arising out of emotional communication in mutually constituted social feedback loops.

Developmental studies of mother infant-interaction also highlight the intimate reciprocal relation between self-regulation and social processes. This is especially apparent in the synchronized interactions of mother-infant dyads in which infants learn to modify their emotional expressions to engage or maintain their mother's attention (Feldman, Geenbaum, & Yirmiya 1999). Social referencing (Bretherton 1992; Walden 1991) is another example of socially based regulation, as infants reduce their anxiety by using their attachment figure's reactions as a model for dealing with novel or ambiguous situations. The strange situation paradigm (Ainsworth, Blehar, Waters, & Wall 1978) essentially examines self-regulatory behavior in infants that is driven by the biological need to maintain proximity with the attachment figure. Although the reference goal is a biological given, the infant's choice of specific strategies or regulatory techniques is influenced by intra-organismic variables, such as negative or positive affectivity, arousal threshold, and other constituents

of temperament, and by external influences, including the caregiver's physical and emotional availability and, on a broader level, the surrounding "emotion culture" (Gordon 1989).

Observing regulatory behavior in attachment situations from a functionalist perspective in terms of goal-oriented action helps to prevent confounding normative desirable personality organization with optimal emotion self-regulation (Thompson 1994). For example, for a child that has abusive parents, avoidant or withdrawn behavior may be adaptive in sustaining the relationship with his or her caregivers and averting injury. Conceiving of attachment schemata as reference values that guide the operation of regulatory feedback loops may help to understand how intrapsychic and interpersonal aspects of emotion self-regulation intersect.

Summary and future research directions

The study of emotion self-regulation has advanced greatly in the past decade. Yet, it is still a relatively new area of study in which theories far outdistance the available empirical evidence. Many theoretical advances have been made in different disciplines under the umbrella of psychology (see Gross 1998b, for a review). There is an imperative need to identify linkages between these areas and to synthesize the evidence into more coherent models. In closing, we would like to highlight briefly several of the issues that we hope will receive greater empirical attention in the future.

As we have repeatedly pointed out in this chapter, automatic or non-conscious emotion regulatory processes in adults still await systematic investigation. So far, only a handful of studies have touched upon these processes (e.g., Bonanno et al. 1995; Bonanno et al., in press; Kneier & Temoshok 1984; Weinberger et al. 1979). Another important but neglected area of research is how different types of regulatory processes relate to each other. Jackson et al. (2000) made a promising advance by developing and validating a within-subjects paradigm for measuring different processes that regulate the experience of emotion. In our own lab (Bonanno et al., in press) we have recently developed a within-subjects paradigm for measuring expressive enhancement and expressive suppression. This paradigm enabled us to examine whether subjects' ability to flexibly engage in either form of expressive regulation predicts longitudinal adjustment. In both of these studies, however, the experimental manipulations were limited to conscious or deliberate regulatory processes. Although instructing people to regulate emotions in the laboratory perhaps

inevitably involves studying consciously driven processes, future studies could compare these data with findings from naturalistic studies that tap automated emotion self-regulation processes.

Such comparisons may help to identify different emotion regulatory profiles, including information on asymmetries in emotion self-regulation abilities. For example, it may be interesting to examine psychological and social functioning in individuals who score high on measures of conscious expressive regulation, but evidence dysregulated automated emotional regulatory processes, and vice versa. Further, one could investigate how different regulatory profiles affect related abilities such as empathic accuracy; that is, the ability to perceive accurately how another person is feeling (Levenson & Ruef 1992). Using attachment theory in interpreting data thus obtained may help to identify developmental origins of regulatory profiles and understand stability and discontinuity in relational patterns across the life cycle. In an applied context, such knowledge could then inform techniques modifying emotion regulatory skills in adults and children as part of therapeutic interventions designed to improve social competence in at-risk and clinical populations (e.g., children who have suffered sexual and physical abuse or neglect, and individuals diagnosed with borderline personality disorder, schizophrenia, and bipolar depression).

References

- Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation*. Hillsdale, NJ: Erlbaum.
- Asendorpf, J. B. & Scherer, K. R. (1983). The discrepant repressor: Differentiation between low anxiety, high anxiety, and repression of anxiety by autonomic-facial-verbal patterns of behavior. *Journal of Personality and Social Psychology*, 45, 1334–1346.
- Averill, J. R. (1980). A constructivist view of emotion. In R. Plutchik & H. Kellerman (Eds.), *Emotion: Theory, research, and experience* (pp. 305–339). Orlando, FL: Academic Press.
- Baumeister, R. F., Heatherton, T. F., & Tice, D. M. (1994). *Losing control: How and why people fail at self-regulation*. New York: Academic Press.
- Berkowitz, L. (1990). On the formation and regulation of anger and aggression: A cognitive-neoassociationistic analysis. *American Psychologist*, 45, 494–503.
- Bonanno, G. A. (2001). Emotion Self-Regulation. In T. J. Mayne & G. A. Bonanno (Eds.), *Emotions: Current issues and future directions* (pp. 251–285). New York: Guildford Press.
- Bonanno, G. A. (2002). Unpublished raw data.
- Bonanno, G. A., Field, N. P., Kovacevic, A., & Kaltman, S. (2002). Self-enhancement as a buffer against extreme adversity: Civil war in Bosnia and traumatic loss in the United States. *Personality and Social Psychology Bulletin*, 28, 184–196.

- Bonanno, G. A., Keltner, D., Holen, A., & Horowitz, M. J. (1995). When avoiding unpleasant emotion might not be such a bad thing: Verbal-autonomic response dissociation and midlife conjugal bereavement. *Journal of Personality and Social Psychology*, 46, 975–989.
- Bonanno, G. A. & Keltner, D. (1997). Facial expressions of emotion and the course of bereavement. *Journal of Abnormal Psychology*, 106, 126–137.
- Bonanno, G. A., Noll, J. G., Putnam, F. W., O'Neill, K. M., & Trickett, P. (in press). Predicting the willingness to disclose childhood sexual abuse from measures of repressive coping and dissociative experiences. *Child Maltreatment*.
- Bonanno, G. A. & Singer, J. L. (1990). Repressive personality style: Theoretical and methodological implications for health and pathology. In J. L. Singer (Ed.), *Repression and dissociation* (pp. 435–470). Chicago: University of Chicago Press.
- Bonanno, G. A., Znoj, H. J., Siddique, H., & Horowitz, M. J. (1999). Verbal-autonomic response dissociation and adaptation to midlife conjugal loss: A follow-up at 25 months. *Cognitive Therapy and Research*, 23, 605–624.
- Bonanno, G. A. & Keltner, D. (in press). The coherence of emotion systems: Comparing “on-line” measures of appraisal and facial expressions, and self-report.
- Bretherton, I. (1992). Social referencing, intentional communication, and the interfacing of minds in infancy. In S. Feinman (Ed.), *Social referencing and the social construction of reality in infancy* (pp. 57–77). New York: Plenum Press.
- Buck, R. (1988). *Human motivation and emotion* (2nd ed.). New York: Wiley & Sons.
- Campos, J. J., Campos, R. G., & Barrett, K. C. (1989). Emergent themes in the study of emotional development and emotion self-regulation. *Developmental Psychology*, 25, 394–402.
- Carstensen, L. L., Gottman, J. M., & Levenson, R. W. (1995). Emotional behavior in long-term marriage. *Psychology & Aging*, 10, 140–149.
- Carstensen, L. L., Graff, J., Levenson, R. W., & Gottman, J. M. (1996). Affect in intimate relationships: The developmental course of marriage. In C. Magai & S. H. McFadden (Eds.), *Handbook of emotion, adult development, and aging* (pp. 227–247).
- Carver, C. S. & Scheier, M. F. (1981). *Attention and self-regulation: A control theory approach to human behavior*. New York: Springer-Verlag.
- Carver, C. S. & Scheier, M. F. (1982). Control theory: A useful conceptual framework for personality-social, clinical and health psychology. *Psychological Bulletin*, 92, 11–135.
- Carver, C. S. & Scheier, M. F. (1990). Origins and functions of positive and negative affect: A control-process view. *Psychological Review*, 97, 19–35.
- Charney, D. S. & Redmond, D. E., Jr. (1983). Neurobiological mechanism in human anxiety. *Neuropharmacology*, 22, 1531–1536.
- Cole, P. M., Michel, M. K., & Teti, L. O. (1994). *The development of emotion self-regulation and dysregulation: A clinical perspective*. Monographs of the Society for Research in Child Development, 59 (2–3, Serial No. 240), 73–100.
- Consedine, N. S., Magai, C., & Bonanno, G. A. (2002). Moderators of the emotion inhibition-health relationship: A review and research agenda. *Review of General Psychology*, 6, 204–228.
- Cooper, C. L. & Faragher, E. B. (1992). Coping strategies and breast disorders/cancer. *Psychological Medicine*, 22, 447–455.

- Cooper, C. L. & Faragher, E. B. (1993). Psychosocial stress and breast cancer: The inter-relationships between stress events, coping strategies and personality. *Psychological Medicine*, 23, 653–662.
- Duclos, S. E., Laird, J. D., Schneider, E., Sexter, M., Stern, L., & Van Lighten, O. (1989). Emotion-specific effects of facial expressions and postures of emotional experience. *Journal of Personality and Social Psychology*, 57, 100–108.
- Ebbesen, E. B., Duncan, B., & Konecni, V. J. (1975). Effects of content of verbal aggression on future verbal aggression: A field study. *Journal of Experimental Social Psychology*, 11, 192–204.
- Ehrlichman, H. & Bastone, L. (1992). Olfaction and emotion. In M. Serby & K. Chobor (Eds.), *The science of olfaction* (pp. 410–438). New York: Springer-Verlag.
- Ekman, P. (1984). Expression and the nature of emotion. In K. Scherer & P. Ekman (Eds.), *Approaches to emotion* (pp. 319–344). Hillsdale, NJ: Erlbaum.
- Ekman, P. (1992). Are there basic emotions? *Psychological Review*, 99, 550–553.
- Ekman, P. & Davidson, R. J. (1993). Voluntary smiling changes regional brain activity. *Psychological Science*, 4, 342–345.
- Ekman, P., Levenson, R. W., & Friesen, W. V. (1983). Autonomic nervous system activity distinguishes among emotions. *Science*, 211, 1208–1210.
- Feldman, L. A. (1995). Valence focus and arousal focus: Individual differences in the structure of affective experience. *Journal of Personality and Social Psychology*, 69, 153–166.
- Feldman, R., Greenbaum, C. W., & Yirmiya, N. (1999). Mother-Infant Affect Synchrony as an Antecedent of the Emergence of Self-Control. *Developmental Psychology*, 35, 223–231.
- Fox, E., O'Boyle, C. A., Barry, H., & McCreary, C. (1989). Repressive coping style in stressful dental surgery. *British Journal of Medical Psychology*, 62, 371–380.
- Fox, N. A. & Davidson, R. J. (1986). Taste-elicited changes in facial signs of emotion and the asymmetry of brain electrical activity in human newborns. *Neuropsychologia*, 24, 417–422.
- Frijda, N. H. (1986). *The emotions*. Cambridge, England: Cambridge University Press.
- Frijda, N. H. (1988). The laws of emotion. *American Psychologist*, 43, 349–358.
- Giese-Davis, J. & Spiegel, D. (2001). Suppression, repression-defensiveness, restraint, and distress in metastatic breast cancer: Separable or inseparable constructs? *Journal of Personality*, 69, 417–449.
- Gordon, S. L. (1989). The socialization of children's emotions: Emotional culture, competence, and exposure. In C. Saarni & P. L. Harris (Eds.), *Children's understanding of emotion. Cambridge studies in social and emotional development* (pp. 319–349). Cambridge, UK: Cambridge University Press.
- Gottman, J. M. & Levenson, R. W. (1986). Assessing the role of emotion in marriage. *Behavioral Assessment*, 8, 31–48.
- Greenberg, M. A., Wortman, C. B., & Stone, A. A. (1996). Emotional expression and physical health: Revising traumatic memories or fostering self-regulation? *Journal of Personality and Social Psychology*, 71, 588–602.
- Greenberg, L. S. & Safran, J. D. (1987). *Emotion in psychotherapy*. New York: Guilford Press.

- Gross, J. J. (1998a). Antecedent and response-focused emotion self-regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology*, 74, 224–237.
- Gross, J. J. (1998b). The emerging field of emotion self-regulation: An integrative review. *Review of General Psychology*, 2, 271–299.
- Gross, J. J. (1999). Emotion self-regulation: Past present, future. *Cognition & Emotion*, 13, 551–573.
- Gross, J. J. (2002). Emotion self-regulation: Affective, cognitive, and social consequences. *Psychophysiology*, 39, 281–291.
- Gross, J. J. & Munoz, R. F. (1995). Emotion self-regulation and mental health. *Clinical Psychology: Science and Practice*, 2, 151–164.
- Gross, J. J. & Levenson, R. W. (1993). Emotional suppression: Physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology*, 64, 970–986.
- Gross, J. J. & Levenson, R. W. (1997). Hiding feelings: The acute effects of inhibiting negative and positive emotion. *Journal of Personality and Social Psychology*, 64, 970–986.
- Gross, J., John, O. P., & Richards, J. (2000). The dissociation of emotion expression from emotion experience: A personality perspective. *Personality and Social Psychology Bulletin*, 26, 712–726.
- Helgeson, V. S. & Taylor, S. E. (1993). Social comparison and adjustment among cardiac patients. *Journal of Applied Social Psychology*, 23, 171–195.
- Hess, W. R. (1957). *The functional organization of the diencephalon*. New York: Grune & Stratton.
- Higgins, E. T. (1987). Self-discrepancy theory: A theory relating self and affect. *Psychological Review*, 94, 319–340.
- Isen, A. M. (2000). Positive Affect and Decision Making. In M. Lewis & J. M. Haviland-Jones (Eds.), *Handbook of Emotions* (pp. 417–435). New York: Guilford Press.
- Isen, A. M., Daubman, K. A., & Nowicki, G. P. (1987). Positive affect facilitates creative problem solving. *Journal of Personality and Social Psychology*, 47, 1206–1217.
- Izard, C. E. (1977). *Human Emotions*. New York: Plenum.
- Izard, C. E. (1990). Facial expressions and the regulation of emotion. *Journal of Personality and Social Psychology*, 58, 487–498.
- Izard, C. E. (1992). Basic emotions, relations among emotions, and emotion-cognition relations. *Psychological Review*, 99, 561–564.
- Izard, C. E. (1993). Four systems for emotion activation: Cognitive and noncognitive processes. *Psychological Review*, 100, 68–90.
- Izard, C. E., Hembree, E. A., & Huebner, R. R. (1987). Infants' emotion expressions to acute pain: Developmental change and stability of individual differences. *Developmental Psychology*, 23, 105–113.
- Izard, C. E. & Ackerman, B. P. (2000). Motivational, organizational, and regulatory functions of discrete emotions. In M. Lewis & J. M. Haviland-Jones (Eds.), *Handbook of Emotions* (pp. 253–264). New York: Guilford Press.
- Jackson, D. C., Malmstadt, J. R., Larson, C. L., & Davidson, R. J. (2000). Suppression and enhancement of emotional responses to unpleasant pictures. *Psychophysiology*, 37, 515–522.

- Julien, D., Markman, H. J., & Lindahl, K. M. (1989). A comparison of a global and a microanalytic coding system: Implications for future trends in studying interaction. *Behavioral Assessment, 11*, 81–100.
- Kelly, A. E. & McKillop, K. J. (1996). Consequences of revealing personal secrets. *Psychological Bulletin, 120*, 450–465.
- Keltner, D. (1995). Signs of appeasement: Evidence for the distinct displays of embarrassment, amusement, and shame. *Journal of Personality and Social Psychology, 68*, 441–454.
- Keltner, D. & Bonanno, G. A. (1997). A study of laughter and dissociation: Distinct correlates of laughter and smiling during bereavement. *Journal of Personality and Social Psychology, 73*, 687–702.
- Keltner, D. & Gross, J. J. (1999). Functional accounts of emotions. *Cognition and Emotion, 13*, 467–480.
- Kennedy-Moore, E. & Watson, J. C. (1999). *Expressing emotion: Myths, realities, and therapeutic strategies*. New York: Guilford Press.
- Kennedy-Moore, E. & Watson, J. C. (2001). How and When Does Emotional Expression Help? *Review of General Psychology, 5*, 187–212.
- Kitayama, S. & Markus, H. R. (Eds.). (1996). *Emotion and culture*. Washington, DC: American Psychological Association.
- Kneier, A. W. & Temoshok, L. (1984). Repressive coping reactions in patients with malignant melanoma as compared to cardiovascular disease patients. *Journal of Psychosomatic Research, 28*, 368–378.
- Lang, P. J. (1979). Language, image, and emotion. In P. Pliner, K. R. Blankstein, & J. M. Spigel (Eds.), *Perception of emotion in self and others* (Vol. 5). New York: Plenum Press.
- Lang, P. J., Kozak, M. J., Miller, G. A., Levin, D. N., & McLean, A. (1980). Emotional imagery: Conceptual structure and pattern of somato-visceral response. *Psychophysiology, 17*, 179–192.
- Larsen, R. J. (2000). Toward a Science of Mood Regulation. *Psychological Inquiry, 11* (3), 129–141.
- Lazarus, R. S. (1984). On the primacy of cognition. *American Psychologist, 39*, 124–129.
- Lazarus, R. S. (1991a). Cognition and motivation in emotion. *American Psychologist, 46*, 352–367.
- Lazarus, R. S. (1991b). *Emotion and adaptation*. New York: Oxford University Press.
- LeDoux, J. E. (1989). Cognitive-emotional interactions in the brain. *Cognition and Emotion, 3*, 267–289.
- LeDoux, J. E. (1996). *The emotional brain. The mysterious underpinnings of emotional life*. New York: Simon & Schuster.
- Lehman, D. R., Ellard, J. H., & Wortman, C. B. (1986). Social support for the bereaved: Recipients' and providers' perspectives on what is helpful. *Journal of Consulting and Clinical Psychology, 54*, 438–446.
- Leith, K. P. & Baumeister, R. F. (1996). Why do bad moods increase self-defeating behavior? Emotion, risk-taking, and self-regulation. *Journal of Personality and Social Psychology, 71*, 1250–1267.

- Levenson, R. W. (1994). Human emotion: A functional view. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 123–126). Oxford, England: Oxford University Press.
- Levenson, R. W., Carstensen, L. L., & Gottman, J. M. (1993). Long-term marriage: Age, gender, and satisfaction. *Psychology & Aging*, 8, 301–3313.
- Levenson, R. W., Carstensen, L. L., & Gottman, J. M. (1994). Influence of age and gender on affect, physiology, and their interrelations: A study of long-term marriages. *Journal of Personality and Social Psychology*, 67 (1), 56–68.
- Levenson, R. W. & Ruef, A. M. (1992). Empathy: A physiological substrate. *Journal of Personality and Social Psychology*, 63, 234–246.
- Leventhal, H. (1984). A perceptual-motor theory of emotion. In L. Berkowitz (Ed.), *Advances in experimental social psychology*, Vol. 17 (pp. 117–182). New York: Academic Press.
- Leventhal, H. (1991). Emotion: Prospects for conceptual and empirical development. In R. G. Lister & H. J. Weingartner (Eds.), *Perspectives on cognitive neuroscience* (pp. 325–348). Oxford, UK: Oxford University Press.
- Markus, H. & Nurius, P. (1986). Possible selves. *American Psychologist*, 41, 954–969.
- Major, F., Cozzarelli, C., Sciacchitano, A. M., Cooper, M. L., Testa, M., & Mueller, P. M. (1990). Perceived social support, self-efficacy, and adjustment to abortion. *Journal of Personality and Social Psychology*, 77, 735–745.
- Markus, H. R. & Kitayama, S. (1991). Culture and the self: Implications for cognition, emotion, and motivation. *Psychological Review*, 98, 224–253.
- Mesquita, B. (2001). Emotions in collectivist and individualist contexts. *Journal of Personality and Social Research*, 80, 68–204.
- Mesquita, B. & Frjda, N. H. (1992). Cultural variations in emotions: A review. *Psychological Bulletin*, 112, 179–204.
- Muraven, M., Tice, D. M., & Baumeister, R. F. (1998). Self-control as a limited resource: Regulatory depletion patterns. *Journal of Personality and Social Psychology*, 74, 774–789.
- Nesse, R. (1990). Evolutionary explanations of emotions. *Human Nature*, 1, 261–289.
- Newton, T. L. & Contrada, R. J. (1992). Repressive coping and verbal-autonomic response dissociation: The influence of social context. *Journal of Personality and Social Psychology*, 62, 159–167.
- Ochsner, K. N. & Feldman Barrett, L. (2001). A multiprocess perspective on the neuroscience of emotion. In T. J. Mayne & G. A. Bonanno (Eds.), *Emotions: Current Issues and Future Directions* (pp. 1–81). New York: Guildford Press.
- Ornish, D., Brown, S. E., Scherwitz, L. W., Billings, J. H., Armstrong, W. T., Ports, T. A., McClanahan, S. M., Kirkeide, R. L., Brand, R. J., & Gould, K. (1990). Can lifestyle changes reverse coronary artery disease? The lifestyle heart trial. *Lancet*, 336, 129–133.
- Owren, M. J. & Bachorowski, J.-A. (2001). The evolution of emotional expression: A “selfish-gene” account of smiling and laughter in early hominids and humans. In T. J. Mayne & G. A. Bonanno (Eds.), *Emotions: Current issues and future directions* (pp. 152–191). New York: Guildford Press.
- Parkinson, B. (1995). *Ideas and realities of emotion*. London: Routledge.

- Paulhus, D. L. (1991). Measurement and control of response bias. In J. P. Robinson, P. Shaver, & L. S. Wrightsman (Eds.), *Measures of personality and social psychological attitudes* (pp. 17–59). San Diego, CA: Academic Press.
- Pennebaker, J. W. (1989). Confession, inhibition, and disease. *Advances in Experimental Social psychology*, 22, 211–244.
- Pennebaker, J. W. (1990). *Opening up: The healing power of confiding in others*. New York: Morrow.
- Pennebaker, J. W. (1993). Putting stress into words: Health, linguistic, and therapeutic implications. *Behavior Research and Therapy*, 31, 539–548.
- Pennebaker, J. W. (Ed.). (1995). *Emotion, disclosure, and health*. Washington, DC: APA Press.
- Pennebaker, J. W. & Beall, S. K. (1996). Confronting a traumatic event: Toward an understanding of inhibition and disease. *Journal of Abnormal Psychology*, 95, 274–281.
- Pennebaker, J. W., Colder, J., & Sharp, L. K. (1990). Accelerating the coping process. *Journal of Personality and Social Psychology*, 58, 528–537.
- Pennebaker, J. W., Kiecolt-Glaser, J. K., & Glaser, R. (1988). Disclosure of traumas and immune functions: Health implications for psychotherapy. *Journal of Consulting and Clinical Psychology*, 56, 239–245.
- Pennebaker, J. W. & Seagal, J. D. (1999). Forming a story: The health benefits of narrative. *Journal of Clinical Psychology*, 55, 1243–1254.
- Pennebaker, J. W. & Susman, J. R. (1988). Disclosure of trauma and psychosomatic health. *Social Science Medicine*, 26, 327–332.
- Porter, R. E. & Samovar, L. A. (1998). Cultural influences on emotional expression: Implications for intercultural communication. In P. A. Andersen & L. K. Guerrero (Eds.), *Handbook of communication and emotion: Research, theory, applications, and contexts* (pp. 451–472). San Diego, CA: Academic Press.
- Powers, W. T. (1973). *Behavior: The control of perception*. Chicago: Aldine.
- Powers, W. T. (1978). Quantitative analysis of purposive systems: Some spadework at the foundations of scientific psychology. *Psychological Review*, 85, 417–435.
- Rabinor, J. R. (1998). Journal writing: A guide to the inner world. *Eating Disorders. The Journal of Treatment and Prevention*, 6, 253–266.
- Richards, J. M. & Gross, J. J. (2000). Emotion regulation and memory: The cognitive costs of keeping one's cool. *Journal of Personality and Social Psychology*, 79, 410–424.
- Risikind, J. H. (1984). They stoop to conquer: guiding and self-regulatory functions of physical posture after success and failure. *Journal of Personality and Social Psychology*, 47, 479–493.
- Robinson, M. D. & Clore, G. L. (2002). Belief and feeling: Evidence for an accessibility model of emotional self-report. *Psychological Bulletin*, 128 (6), 934–939.
- Rothbaum, B. O. & Foa, E. B. (1994). Cognitive-behavioral therapy for Posttraumatic Stress Disorder. In B. A. van der Kolk, A. C. McFarlane, & L. Weisaeth (Eds.), *Traumatic stress: The effects of overwhelming experience on mind, body, and society* (pp. 491–509). New York: Guilford.
- Rozin, P., Lowery, L., Imada, S., & Haidt, J. (1999). The CAD triad hypothesis: A mapping between three moral emotions (contempt, anger, disgust) and three moral codes (community, autonomy, divinity). *Journal of Personality and Social Psychology*, 76, 574–586.

- Russell, J. A. (2003). Core affect and the psychological construction of emotion. *Psychological Review*, 110, 145–172.
- Salomon, R. C. (2000). The philosophy of emotions. In M. Lewis & J. M. Haviland-Jones (Eds.), *Handbook of Emotions* (pp. 3–15). New York: Guilford Press.
- Salovey, P. (2001). Applied emotional intelligence: Regulating emotions to become health, wealthy, and wise. In J. Ciarrochi, J. P. Forgas, & J. D. Meyer (Eds.), *Emotional intelligence in everyday life: A scientific inquiry* (pp. 168–184). Psychology Press.
- Sapolsky, R. M. (1998). *Why zebras don't get ulcers. An updated guide to stress, stress-related diseases, and coping*. New York: Freeman & Company.
- Sapolsky, R. M. (1999). The physiology and pathophysiology of unhappiness. In D. Kahneman, E. Diener, & N. Schwarz (Eds.), *Well-being: The foundations of hedonic psychology* (pp. 453–469).
- Scheier, M. F. & Carver, C. S. (1988). A model of behavioral self-regulation: Translating intention into action. In L. Berkowitz (Ed.), *Advances in the experimental study of social psychology*, Vol. 21 (pp. 303–346). New York: Academic Press.
- Scherer, K. R. (1994). Emotion serves to decouple stimulus and response. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion* (pp. 127–130). New York: Oxford University Press.
- Schwartz, G. E. (1982). Physiological patterning and emotion: Implications for the self-regulation of emotion. In K. R. Blankstein & M. Polivy (Eds.), *Self-control and self-modification of emotional behavior* (pp. 13–27). New York: Plenum.
- Schwartz, G. E., Fair, P. L., Greenberg, P. S., Freedman, M., & Klerman, J. L. (1974). Facial electromyography in the assessment of emotion. *Psychophysiology*, 11, 237.
- Schwartz, G. E., Fair, P. L., Salt, P., Mandel, M. R., & Klerman, J. L. (1976). Facial muscle patterning to affective imagery in depressed and non-depressed subjects. *Science*, 192, 489–491.
- Schwarz, N. & Clore, G. L. (1983). Mood, misattribution, and judgments of well-being: Informative and directive functions of affective states. *Journal of Personality and Social Psychology*, 45, 513–523.
- Shedler, J., Mayman, M., & Manis, M. (1993). The illusion of mental health. *American Psychologist*, 48, 1117–1131.
- Shweder, R. (1994). You're not sick, you're just in love: Emotion as an interpretive system. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 32–44). New York: Oxford University Press.
- Silver, R. L., Boon, C., & Stones, M. H. (1983). Searching for meaning in misfortune: Making sense of incest. *Journal of Social Issues*, 39, 81–102.
- Silver, R. L. & Wortman, C. B. (1980). Coping with undesirable life events. In J. Garber & M. E. P. Seligman (Eds.), *Human helplessness: Theory and applications* (pp. 279–340). New York: Academic Press.
- Silver, R. L., Wortman, C. B., & Crofton, C. (1990). The role of coping in support provision: The self-representational dilemma of victims of life crises. In B. R. Sarason, I. G. Sarason, & G. R. Pierce (Eds.), *Social support: An interactional view* (pp. 397–426). New York: Wiley.
- Singer, J. A. & Salovey, P. (1993). *The remembered self: Emotion and memory in personality*. New York: Free Press.

- Stein, N. L., Trabasso, T., & Liwag, M. D. (2000). A goal appraisal theory of emotional understanding: Implications for development and learning. In M. Lewis & J. M. Haviland-Jones (Eds.), *Handbook of emotions* (pp. 436–457). New York: Guilford Press.
- Stein, S. & Spiegel, D. (2000). Psychoneuroimmune and endocrine effects on cancer progression. In K. Goodkin & A. P. Visser (Eds.), *Psychoneuroimmunology: Stress, mental disorders, and health*. Progress in psychiatry, No. 59 (pp. 105–151). Washington, DC: American Psychiatric Publishing, Inc.
- Surgenor, T. & Joseph, S. (2000). Attitudes to emotional expression and personality in predicting post-traumatic stress disorder. *Review of Personality and Social Psychology*, 5, 170–191.
- Taylor, S. E., Kemeny, M. E., Reed, G. M., & Aspinwall, L. G. (1991). Assault on the self: Positive illusions and adjustment of threatening events. In J. Strauss & G. R. Goethals (Eds.), *The self: Interdisciplinary approaches* (pp. 239–254). New York: Springer-Verlag.
- Taylor, S. E., Wood, J. V., & Lichtman, R. R. (1983). It could be worse: Selective evaluation as a response to victimization. *Journal of Social Issues*, 39, 569–575.
- Thompson, J. G. (1988). *The psychobiology of emotions*. New York: Plenum Press.
- Thompson, R. A. (1994). Emotion self-regulation: A theme in search of definition. In N. A. Fox (Ed.), *The development of emotion self-regulation: Biological and behavioral considerations*. Monographs of the Society for Research in Child Development, 59, (Serial No. 240), 25–52.
- Tice, D. M. & Bratslavsky, E. (2000). Giving in to feel good: The place of emotion self-regulation in the context of general self-control. *Psychological Inquiry*, 11 (3), 149–159.
- Tomkins, S. S. (1962). *Affect, imagery, consciousness: Vol. 1. The positive affects*. New York: Springer.
- Tooby, J. & Cosmides, L. (1990). The past explains the present: Emotional adaptations and the structure of ancestral environments. *Ethology and Sociobiology*, 11, 375–424.
- Walden, T. A. (1991). Infant social referencing. In J. Garber & K. A. Dodge (Eds.), *The development of emotion self-regulation and dysregulation* (pp. 69–88). Cambridge, UK: Cambridge University Press.
- Whybrow, P. C., Akiskal, H. S., & McKinney, W. T. (1984). *Mood disorders: Toward a new psychobiology*. New York: Plenum Press.
- Van der Kolk, B. A. (1994). The complexity of adaptation to trauma: self-regulation, stimulus discrimination, and characterological development. In B. A. van der Kolk, A. C. McFarlane, & L. Weisaeth (Eds.), *Traumatic stress: The effects of overwhelming experience on mind, body, and society* (pp. 182–213). New York: Guilford.
- Weinberger, D. A. & Davidson, M. N. (1994). Styles of inhibiting emotional expression: Distinguishing repressive coping from impression management. *Journal of Personality*, 62, 587–613.
- Weinberger, D. A., Schwartz, G. E., & Davidson, J. R. (1979). Low-anxious and repressive coping styles: Psychometric patterns of behavioral and physiological responses to stress. *Journal of Abnormal Psychology*, 88, 369–380.
- Winnicott, D. W. (1971). *Playing and reality*. London, England: Tavistock.
- Zajonc, R. B., Murphy, S. T., & Inglehart, M. (1989). Feeling and facial efference: Implications of the vascular theory of emotion. *Psychological Review*, 96, 395–416.

CHAPTER 2

Temperament and emotional regulation

Multiple models of early development

Susan D. Calkins

Introduction

Emotional regulation refers to processes that serve to manage emotional arousal and support adaptive social and non-social responses (Calkins 1994; Kopp 1992; Thompson 1994). Over the last several years, the field of developmental psychology has addressed extensively the construct of emotional regulation and its role in normative development and successful adaptation (Calkins & Dedmon 2000; Eisenberg, Murphy, Maszk, Smith, & Karbon 1995; Eisenberg, Fabes, Guthrie, Murphy, Maszk, Holmgren, & Suh 1996; Thompson 1994). This work suggests that these processes begin to develop early in life and that the lack of adequate development of control over emotion (as well as, in some instances, over-control of emotion) may be a precursor to the development of psychopathology (Calkins & Dedmon 2000; Calkins & Fox 2002; Keenan 2000; Shaw, Keenan, Vondra, Delliquadri, & Giovannelli 1997). Given its primary role in early development and developmental psychopathology, much research continues to focus on the construct of emotional regulation, the mechanisms through which it develops, and the implications of different patterns of regulatory functioning for interpersonal functioning.

In this chapter, I focus on the construct of emotional regulation from a developmental perspective, with an emphasis on the temperamental processes or mechanisms that contribute to its development in the early years of life. I begin with a brief overview of recent theorizing about emotional regulation, followed by a description of normative developments in the domain of emotional regulation. Next, I address the specific role of temperament, with consideration of the multiple possible roles that temperament might play in the normative development of emotional regulation skills and in the emergence of individual

differences in emotional regulation. Finally, I address implications of this perspective for the empirical study of emotional regulation and its conceptual integration with other dimensions of interpersonal functioning. I consider a multiple-level approach to the study of emotional regulation as a means of consolidating, from a theoretical perspective, what is known about the behavioral and biological processes involved in emotional regulation.

Theoretical and empirical approaches to emotional regulation

The construct of emotional regulation may be regarded from within a broader literature on self-control. Notions of self-control are discussed in the psychological literature in relation to the development of motor skills, attention and cognition, and with regard to emotion (Calkins 1994; Fox & Calkins 2003; Gross 1999; Posner & Rothbart 2000). Self-control of emotion, or emotional regulation, refers to efforts on the part of the individual to manage, modulate, inhibit and enhance both positive and negative emotions (Cicchetti, Ganiban, & Barnett 1991; Kopp 1982, 1989; Thompson 1994). The capacity for self-control of emotion is a cluster of skills and abilities that develops over the first years of life and has profound effects upon the child's behavioral repertoire (Kopp 1982; Kochanska, Coy, & Murray 2001).

Developmental theories of emotional regulation have addressed normative changes in regulation, the emergence of individual differences in regulation, the predictors of both normative change and individual differences, and the implications of emotional regulation for psychosocial functioning (Calkins 1994; Cassidy 1994; Eisenberg et al. 2001; Kopp 1982, 1989, 2002; Thompson 1994). For example, Claire Kopp's theoretical work on emotional regulation focuses largely on the normative changes that take place in the child's behavioral repertoire as a function of maturation and experience. Her integrated view of the development of emotional regulation identifies physiological, cognitive, and motor components of this developmental process, and suggests that one must examine the learning environment to appreciate fully the emergence of new skills and abilities related to the control of emotions (Kopp 1982, 1992).

The caregiving experience is one learning environment that is the focus of the work of Thompson, Cassidy and others (Cassidy 1994; Thompson 1994). From this perspective, both normative changes and individual differences in emotional regulation are a function of early experiences with caregivers, whose influence may be a function of a range of behaviors across the early years of development. Caregiver's abilities to engage in sensitive and responsive in-

teractions, to establish secure relationships, to model, monitor, and provide feedback about emotions, are just a few of the ways that guide the development of children's emotional repertoire. And, deviations from supportive caregiving may contribute to patterns of emotional regulation that are functional for the child in the short term, but that undermine the development of appropriate skills and abilities needed for later developmental challenges (Cassidy 1994).

Other theories of emotional regulation focus more on the underlying biological components of emotional regulation, and assume that maturation of different biological support systems lays the foundation for increasingly sophisticated emotional regulation. Fox (1989, 1994), for example, has noted that the frontal lobes of the brain are differentially specialized for approach versus avoidance and that these tendencies influence the behaviors that children engage in when emotionally aroused. He further notes that maturation of the frontal cortex provides a mechanism for the more sophisticated and planful regulatory behaviors of older children versus infants. Porges (Porges, Doussard-Roosevelt, & Maita 1994; Porges 1996) also describes an important role for biological maturation, specifically maturation of the parasympathetic nervous system that plays a key role in regulation of state, motor activity, and emotion. Moreover, Porges notes that individual differences in nervous system functioning might mediate the expression and regulation of emotion (Porges 1994). Porges and others have found that parasympathetic nervous system functioning, as reflected in heart rate variability, or vagal tone, is related to the control of attention, emotion, and behavior (Calkins 1997; Calkins & Dedmon 2000; DeGangi, DiPietro, Porges, & Greenspan 1991; Huffman et al. 1998; Porges, Doussard-Roosevelt, Portales, & Greenspan 1996). Specifically, suppression of vagal tone during demanding tasks may reflect physiological processes that allow the child to shift focus from internal homeostatic demands to the generation of coping strategies to control affective or behavioral arousal (Porges 1996).

Eisenberg's theory of emotional regulation focuses less on the source of normative development and individual differences and more on the implications of such functioning for successful social adjustment. In her model (Eisenberg, Fabes, Guthrie, & Rieser 2000) she argues that emotionality and regulation are key components of the emotion system that interact in various ways to produce different social outcomes. She notes that each dimension provides independent information regarding the functioning of the individual in emotion-laden contexts, and that such functioning plays a critical role in subsequent social behavior.

In my work (Calkins 1994; Calkins & Dedmon 2000; Calkins & Johnson 1998; Calkins 2002), I have attempted to integrate these approaches by focusing on both environmental and child characteristics and by hypothesizing that the transactions between the child and her environment may be expected to produce predictable variations in both emotional regulation skills and subsequent psychosocial functioning. In this work, which focuses on the development of social competence versus incompetence characterized by aggression, both behavioral and physiological indices of emotion regulation are examined and are viewed as factors that affect, and are affected by, numerous dimensions of the child's caregiving environment.

Regardless of the theoretical orientation that one adopts in the study of emotional regulation, the construct itself must be operationalized in order to evaluate both its predictors and consequences. In the developmental literature, the construct of emotional regulation has been studied in many ways, including the examination of specific strategies and their effects on affective experience and expression. For example, research reveals that the use of specific emotional regulation strategies such as self-comforting, help-seeking, and distraction may assist the young child in managing early temperament-driven frustration and fear responses in situations where the control of negative emotions may be necessary (Stifter & Braungart 1995). Moreover, emotional regulation skills may be useful in situations that elicit positive affective arousal in that they allow the child to keep such arousal within a manageable and pleasurable range (Grolnick, Cosgrove, & Bridges 1996; Stifter & Moyer 1991). These kinds of affective responses and strategies have been studied using a number of standard paradigms, including the stranger approach task, the still-face paradigm, and frustration tasks (Gianino & Tronick 1988; Mangelsdorf, Shapiro, & Marzolf 1995; Stifter & Braungart 1995). Emotional regulation skills also have been studied by linking them directly with failures of psychosocial adaptation. Thus, for example, lack of emotional control is considered a symptom or indicator of externalizing problems (Campbell 1995; Lewis & Miller 1990).

Because the lack of emotional regulation skills contributes to adjustment difficulties (Calkins 1994; Calkins & Dedmon 2000; Eisenberg et al. 1993, 1994; Cicchetti, Ackerman & Izard 1995; Keenan 2000; Rubin, Coplan, Fox, & Calkins 1995), failure to acquire these skills may lead to difficulties in areas such as social competence and school adjustment. For this reason, the acquisition of emotional regulation skills and strategies is considered a critical achievement of early childhood (Bronson 2000; Sroufe 1996), and the focus on the development of individual differences in emotional regulation has stimulated

much research (Mangelsdorf et al. 1995; Stifter & Braungart 1995; Calkins, Gill, Dedmon, Lomax, & Johnson 2002).

One important assumption of much of the empirical work on the acquisition of emotional regulation is that parental caregiving practices may support or undermine such development and thus contribute to observed individual differences among young children's emotional skills (Thompson 1994). In infancy, there is an almost exclusive reliance on parents for the regulation of emotion. Over time, interactions with parents in emotion-laden contexts teach children that the use of particular strategies may be more useful for the reduction of emotional arousal than other strategies (Sroufe 1996). Although caregiving practices are often attributed a role in the development of emotional regulation, the specific processes by which these practices affect children's development is often left unspecified (Calkins, *in press*; Fox & Calkins 2003).

A second assumption of the research on emotional regulation is that individual differences in emotionality, or temperamental reactivity, play a role in at least the display, if not the development of, emotional regulation skills (Stifter & Braungart 1995; Calkins 1994; Mangelsdorf et al. 1995). From this perspective, it is assumed that the inborn personality characteristics that differentiate infants from one another influence, either directly or indirectly, the kinds of emotional regulatory skills and strategies that children develop.

There are several possible ways that temperament may affect the display and development of emotional regulation (Calkins 1994). First, temperament may exert a direct effect on early emotional regulation by constraining the use and/or practice of such regulation. Second, temperament may be moderated by environmental effects such as caregiving practices. Third, temperament might be mediated by other processes, such as attention or physiological functioning, that then affect the display and development of emotional regulation. Following a description of the normative development of emotional regulation, I examine the empirical evidence for each of these kinds of temperament effects.

Emotional regulation in early childhood: Normative development

Much of the research on the development of emotional regulation skills and abilities has focused on infancy and toddlerhood, primarily because dramatic developments may be observed during this period of development. The developmental process itself may be described broadly as one in which the relatively passive and reactive neonate becomes a child capable of self-initiated behaviors that serve a regulatory function (Calkins 1994; Kopp 1982; Sroufe 1996). In

addition, this process has also been described as one in which the infant progresses from near complete reliance on caregivers for regulation to independent self-regulation. As the infant makes this transition, the use of specific strategies and behaviors become organized into the infant's repertoire of emotional regulation that may be used in a variety of contexts and that lead to independent functioning across a variety of skill domains.

Kopp (1982) provides an excellent overview of the early developments in emotional regulation. This description has been verified by studies of both normative development (Rothbart, Ziaie, & O'Boyle 1992; Buss & Goldsmith 1998; Mangelsdorf, Shapiro, & Marzolf 1995) and studies of individual differences (Braungart & Stifter 1995; Mangelsdorf et al. 1995). These descriptions provide an explanation of how infants develop and utilize a rich behavioral repertoire of strategies in the service of reducing, inhibiting, amplifying, and balancing different affective responses. Moreover, it is also clear from these descriptions that functioning in a variety of non-emotional domains, including motor, language and cognition, and social development, is implicated in these changes as well (Kopp, 1989, 1992).

Early efforts at emotional regulation, specifically those occurring prior to about three months of age, are thought to be controlled largely by physiological mechanisms that are innate (Kopp 1982). By three months of age, primitive mechanisms of self-soothing, such as sucking, simple motor movements such as turning away, and reflexive signaling in response to discomfort, often in the form of crying, are the primary processes operating, independent of caregiver intervention (Kopp 1982; Rothbart et al. 1992).

The period between three and six months of age marks a major transition in infant development. First, biological functioning, specifically sleep-wake cycles and eating and elimination processes, have become more predictable, signaling an important biological transition that is implicated in self-regulation. Second, the ability of the infant to voluntarily control arousal levels begins to emerge. This control depends largely on attentional control mechanisms and simple motor skills (Rothbart et al. 1992; Ruff & Rothbart 1996), and leads to coordinated use of attention engagement and disengagement, particularly in contexts that evoke negative affect. Infants are now capable of engaging in self-initiated distraction, moving attention away from the source of negative arousal to more neutral non-social stimuli. For example, the ability to shift attention away from a negative event (such as something frightening) to a positive distracter may lead to decreases in the experience of negative affect. Importantly, though, there are clear individual differences in the ability to utilize attention to successfully control emotion and behavior. Rothbart (1981,

1986) found increases in positive affect and decreases in distress from 3 to 6 months during episodes of focused attention, suggesting that attentional control is tied to affective experience. Mangelsdorf and colleagues (Mangelsdorf, Sahpiro, & Marzolf 1995) also found that younger infants tended to rely on simple attentional means such as gaze avoidance to control negative affect. These empirical findings are important to understanding the long-term implications of early emotional regulation because the experience of negative affect is believed to interfere with the child's ability to explore and learn about the environment (Ruff & Rothbart 1996). Consequently, there are clear implications of early emotional regulation for development in a range of domains.

By the end of first year of life, infants become much more active and purposeful in their attempts to control affective arousal (Kopp 1982; Mangelsdorf et al. 1995). First, they begin to employ organized sequences of motor behavior that enable them to reach, retreat, redirect, and self-soothe in a flexible manner that suggests they are responsive to environmental cues. Second, their signaling and redirection become explicitly social as they recognize that caregivers and others may behave in a way that will assist them in the regulation of affective states (Rothbart et al. 1992).

During the second year of life, the transition from passive to active methods of emotional regulation is complete (Rothbart et al. 1992). Although infants are not entirely capable of controlling their own affective states by this age, they are capable of using specific strategies to attempt to manage different affective states, albeit sometimes unsuccessfully (Calkins & Dedmon 2000; Calkins, Smith, Gill, & Johnson 1998). Moreover, during this period, infants begin to respond to caregiver directives and, as a consequence of this responsivity, compliance and behavioral self-control begin to emerge (Kopp 1989). This shift is supported by developments in the motor domain, as well as changes in representational ability and the development of language skills. Brain maturation contributes as well, and by the end of toddlerhood, children have executive control abilities that allow control of arousal, regulation of affective expression, and inhibition and activation of behavior (Bronson 2000).

The description of the developmental process of emerging self-regulation, including emotional regulation, has been subject to inquiry regarding the role of the factors that contribute to these developments. Like investigations of other areas of self-control (Sethi, Mischel, Aber, Shoda, & Rodriguez 2000), understanding the development of control of emotions necessitates examination of both intrinsic and extrinsic factors (Calkins 1994). Intrinsic factors include the temperamental disposition of the child, certain cognitive skills, and the underlying neural and physiological systems that support and are engaged in

the processes of control (Calkins 1994; Fox 1994; Fox, Henderson, & Marshall 2001). Extrinsic factors include the manner in which caregivers shape and socialize emotional responses of the child (Thompson 1994). Caregivers may utilize specific strategies to enhance the development of self-control by providing supportive and responsive environments to the child's needs, and by socializing culturally appropriate behavior (Thompson 1994, 1998). In addition, other socializing agents, including siblings and peers, influence the extent to which children utilize self-control strategies and the success of these strategies (Fox & Calkins, in press).

Empirical evidence supports the theoretical notion that multiple dimensions of the caregiving environment play a role in the development of emotional regulation skills (Braungart & Stifter 1991; Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss 1996). Clearly, though, emotional regulatory processes begin to develop as a function of very early behavioral and biological tendencies, or temperament. In the next section, temperament theory is reviewed briefly, with a specific emphasis on the way in which temperament, referring to both behavioral and biological processes, affects developing emotional regulation.

Temperament and early emotional development

Current theorizing about infant and child temperament and its role in emotional functioning and behavioral adjustment has its roots in the work of Thomas and Chess (Thomas, Birch, Chess, Hertzog, & Korn 1964; Thomas & Chess 1977; Thomas, Chess, & Birch 1970). Thomas and Chess conducted a longitudinal study of children's behavioral styles, later termed temperament, in an effort to understand how children's personality emerged and interacted with their environments. On the basis of interviews with parents about children's reactions to a variety of stimuli and situations, Thomas and Chess described nine different behavioral dimensions that clustered into 3 types, labeled easy, difficult, and slow-to-warm-up temperaments. Children displaying these different profiles exhibited characteristic patterns of responding across a variety of situations. Importantly, though, Thomas and Chess viewed the critical predictor of this adaptation to be the "goodness-of-fit" between a child's temperament and his environment. With their work, Thomas and Chess introduced the idea that children bring with them to their development, and to their interactions with others, their own style that plays a role in subsequent behavioral adaptation.

Thomas and Chess's work stimulated a number of researchers interested in early socioemotional development to explore the notion that inborn characteristics of the child contributed substantively to behavior. Subsequent theories of temperament have varied in the numbers of temperament dimensions proposed, the emphasis on emotion versus behavior, and the extent to which the environment influences these initial tendencies (Goldsmith et al. 1987). For example, Seifer (Seifer & Sameroff 1986; Seifer et al. 1996) has extended Thomas and Chess's theory to incorporate the notion that the goodness-of-fit between parent and child is influenced by a number of factors, including infant behavior, parent expectations and parenting practices, and the context in which the interactions occur. And, Rothbart (1981) defines temperament along two broad dimensions of reactivity and self-regulation, which subsume six subscales that place a greater emphasis on emotion. One significant point of agreement among temperament theorists is that temperamental differences reflect biological or physiological differences (Goldsmith et al. 1987; Gunnar 1990; Rothbart & Derryberry 1981). And, some researchers, notably Kagan (1991), Fox (1989, 1991), and Gunnar (1990) place a greater emphasis on exploring the underlying physiological base of temperament that is often referenced only hypothetically in other theories.

Several temperament theories have relevance to the study of the development of emotional regulation. For example, Rothbart's theory views the very young infant as a highly reactive organism whose behavior becomes, with development, increasingly controlled by regulatory processes. A key construct in Rothbart's theory of temperament is effortful control, defined as the ability to inhibit responses to stimuli in the immediate environment while pursuing a cognitively represented goal (Rothbart & Posner 1981; Rothbart & Posner 1985). As a temperamental dimension, effortful control refers to a special class of self-regulatory processes that develop with the maturation of attentional mechanisms, particularly the anterior attention system (Posner & Rothbart 1992). Although it is believed that effortful control begins to emerge at the end of the first year of life, its development continues at least through the preschool years. Thus, the system of regulation that emerges over time, and which began largely as a constitutionally based one, becomes more differentiated, more voluntary and more systematically deployed. Nevertheless, this system is relatively slow to develop and its development, while influenced by temperamental reactivity, is likely a function of other factors as well.

In sum, most theories of temperament focus largely on the dimensions of emotionality, particularly emotional reactivity, which is thought to be biologically based and relatively stable across time. And, although regulatory processes

are often viewed as having an early temperamental basis, and perhaps even may be a dimension of temperament, they are subject to significant maturation and development. Next, I explore how the processes that reflect temperamental reactivity affect the development of emotional regulation and the emergence of individual differences in emotion regulatory style.

Temperament and the development of emotional regulation

The research examining temperament and emotional regulation has focused on multiple dimensions of temperament at different ages and using different indices of responding. So, for example, initial responses of an infant may be characterized by temperament researchers by their physiological and behavioral reactions to sensory stimuli of different qualities and intensities. This reactivity is believed to be present at birth and reflects a relatively stable characteristic of the infant (Rothbart, Derryberry, & Hershey 2000). Moreover, infants will differ initially in their threshold to respond to visual or auditory stimuli as well as in their level of reactivity to stimuli designed to elicit negative affect (e.g., Calkins, Fox, & Marshall 1996). These initial affective responses that are characterized by vocal and facial indices of negativity are presumed to reflect generalized distress. Thus, this initial reactivity has neither the complexity nor the range of later emotional responses. Rather it is a rudimentary form of the more sophisticated and differentiated emotions that will in later infancy be labeled as fear, anger, sadness. These emotions undergo further differentiation with cognitive development and the emergence of self-awareness during early childhood.

There are, then, both behavioral and physiological indices of temperament that may be examined in infancy and early childhood. In addition, in examining the relation between these temperament processes and emotional regulation, one may look at both proximal and distal indices of emotional regulation. Proximal biological indices would include the physiological measures of regulation, as reflected in, for example, heart rate and EEG (Calkins 1997; Fox et al. 1996). Proximal behavioral indices would include the specific strategies that children use in coping with emotional arousal. These behaviors include, for example, self-soothing, distraction, and avoidance. More distal indices of emotional regulation might include, for example, behavior problems with an emotional regulatory component. In fact, within the behavior problem literature, problems of both an internalizing and externalizing nature are often defined by self-regulatory difficulties (Barkley 1997; Calkins & Howse

2003; Keenan 2000). For example, in characterizing the behavior of children with early externalizing behavior problems, there is often reference to a lack of control, undercontrol, or poor regulation (Campbell 1995; Lewis & Miller 1990). In characterizing the behavior of children with internalizing disorders, there is often a discussion of overcontrol (Calkins & Fox 2002). Rarely do these investigations examine specific emotion regulation strategies or processes (though see Calkins & Dedmon 2000 and Gilliom, Shaw, Beck, Schonberg, & Lukon 2002, for exceptions). Rather, it is often assumed that the behavioral symptoms themselves (for example aggression, in the case of externalizing, or withdrawal in the case of internalizing) are either strategies for regulation (Calkins 1994) or behaviors that reflect a lack of adaptive emotional regulation strategies (Keenan 2000). Evidence for the influence of temperament on emotional regulation will be addressed with studies examining both proximal and distal emotional regulation measures, and by examining direct, moderated and mediated effects.

Direct effects of temperament

In understanding how temperament might affect the development of emotional regulation, several hypotheses might be explored. First, temperament might have direct effects on the display and development of specific emotional regulation strategies, particularly during infancy. Characteristics of the infant that are most often explored are temperamental dimensions such as proneness-to-distress (Calkins & Fox 1992; Mangelsdorf, Gunnar, Kestenbaum, Lang, & Andreas 1990; Seifer, Schiller, Sameroff, Resnick, & Riordan 1996). Hypothetically, one might imagine an infant who is highly negatively reactive to novel stimuli. Such an infant might cry easily, intensely, and for a long duration when exposed to new people, objects or environments. Given the level of behavioral disorganization that might accompany such a response, such a child might be unable to display, and therefore practice, the skills of gaze aversion, social referencing, distraction or self-soothing that typically lead to a reduction in the experience and expression of negative affect. Under repeated exposures to novelty, the opportunities for the acquisition of early regulatory behaviors thus become limited. As the child develops, the likelihood of having a rich behavioral repertoire of strategies to draw upon also becomes limited. In this way, temperamental reactivity to novelty may function to limit concurrent strategy use as well as the practice and development of more sophisticated skills that build on early primitive strategies such as gaze aversion or self-soothing, and that are critical to adaptive functioning across a range of more challenging contexts.

Such a scenario has found support in a small number of studies that have investigated the role of temperament in displays of emotional regulation. For example, in a recent series of studies, several researchers have suggested that frustration reactivity, elicited in response to physical restraint or denial, may constrain both the use and the development of appropriate regulatory behaviors (Calkins 1994; Calkins & Fox 1992; Fox & Calkins 1993; Stifter & Fox 1990). First, the concurrent relation between regulation and laboratory elicited negative affect has been examined in a number of these studies. In one study, Stifter and Braungart (1995) examined changes in the types of regulatory behaviors infants use to manage emotional reactivity and observed that there were relations between these behaviors and changes in negative affect. In another study of the efficacy of regulatory behaviors, Rothbart and colleagues (Rothbart, Posner, & Boylan 1990) observed that at least one specific emotional regulation behavior, that of attentional control, is related to decreases in negative emotionality in infancy. And, Buss and Goldsmith (1998) observed that a number of different behaviors that infants display when observed in frustrating or constraining situations appear to reduce negative affect.

In my work, I have demonstrated that there are relations between the level of reactivity to frustration, both behaviorally and physiologically, and emotional and physiological regulation. For example, the degree of baseline of resting heart rate variability, or vagal tone, which is often used as a marker of temperament, predicted the degree of physiological regulation in response to frustration during infancy, toddlerhood and preschool (Calkins et al. 2002; Calkins 1997; Calkins et al. 1998). I have also demonstrated that there are relations between regulatory behaviors and the tendency to be distressed to frustrating situations during toddlerhood (Calkins & Johnson 1998). And, I found that a group of infants characterized by low frustration tolerance differed from less frustrated infants in terms of physiological regulation, emotional regulation strategies, attention, and activity level (Calkins et al. 2002). Similarly, a group of toddlers characterized by disruptive behavior displayed fewer emotional regulation behaviors and poorer physiological regulation (Calkins & Dedmon 2000). The implications of these behavioral and physiological profiles for longer-term emotional regulation are unclear. However, the notion that certain behaviors serve to minimize frustration reactivity has gained clear support, as has the notion that greater levels of frustration are linked to both less regulation and different types of regulation.

A second issue with respect to frustration reactivity is its effect on developing regulatory ability. Braungart-Rieker and Stifter (1996) demonstrated that frustration reactivity at five months of age was related to the use of fewer emo-

tional regulation behaviors at 10 months of age. What is unclear, though, is whether some types of emotional regulation behaviors are more likely to be associated with heightened frustration over time than others. A small number of studies conducted with children of various ages suggest that it might be possible to identify profiles of infants at higher risk for regulatory difficulties. For example, Aksan and colleagues (Aksan et al. 1999) report that a preschool temperament type characterized by noncontrolled-expressive behavior was predicted by infant distress to limitations. Thus, there seems to be evidence that the dimension of frustration, as it is displayed in infancy at least, is linked both concurrently and predictively to the use of less adaptive emotional regulation skills.

To address the more distal effects of temperament on emotional regulation, we may examine the significant body of developmental research that has examined the relations between temperament and behavior problems. This body of work has been conducted across a wide developmental spectrum, in several different countries, with both boys and girls, using different dimensions of temperament and different measurement tools. Despite these differences, however, these studies are consistent in finding modest direct effects of early temperament dimensions such as negativity, resistance to control, and activity level on the display of externalizing, or acting-out behavior problems, at later ages (Bates, Bayles, Bennet, Ridge, & Brown 1991; Caspi, Henry, McGee, Moffitt, & Silva 1995; Prior, Smart, Sanson, Pedlow, & Oberklaid 1992; Shaw, Owens, Giovanelli, & Winslow 2001). Although these studies are suggestive of links between temperament and emotional regulation, there was not any actual measurement of successful versus unsuccessful emotional regulation in these studies. Nevertheless, this work provides support for the direct effects of early temperament on the process of acquiring the kinds of emotional regulation behaviors that underlie successful social functioning and behavioral adaptation.

Moderated effects of temperament

A second way that temperament might affect emotional regulation is that it is moderated by some environmental influences such as parenting behavior (Calkins 1994). Such a hypothesis implies that temperament affects emotional regulation when it is displayed in the context of specific type of parenting. So, for example, an infant who has a tendency to be easily frustrated might have difficulties developing appropriate emotional regulation skills only when the infant's caregiver does not provide sensitive and responsive care. Such an infant

might be forced to rely on immature or ineffective self-regulation skills because the caregiver is not providing the appropriate, indeed necessary, emotional support that will allow the child to become a skilled practitioner of emotional regulation even in the absence of the caregiver.

There is considerable evidence that caregiving behavior is directly related to both proximal and distal measures of emotional regulation, as well as to both behavioral and biological measures of such behavior (Braungart & Stifter 1991; Calkins et al. 1998; Diener, Mangelsdorf, McHale, & Frosch 2002; Donovan & Leavitt 1985; Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss 1996). The question of indirect effects is one that has been examined in studies of both specific emotional regulation strategies (proximal effects) and behavior problems (distal effects).

Empirical investigations of the moderational hypothesis of temperament by caregiving interactions with respect to proximal measures of emotional regulation are quite uncommon. Gilliom and colleagues (Gilliom et al. 2002) conducted a study that examined specific emotional regulation strategy use beyond the infancy period. The focus of this investigation was on preschoolers' use of specific anger control strategies during a waiting paradigm. Specific strategies involving the control of attention were found to predict the anger reaction of the children in this situation. In addition, though, maternal negative control was found to moderate this temperament effect. Children who displayed a difficult temperament and whose mothers used more negative control developed different, potentially less effective, strategies for regulating anger when they were of school age (Gilliom et al. 2002).

A number of other studies have examined the moderational effects of parenting on temperament as a predictor of behavior problems that have an underlying emotional regulation component. In one study conducted in my laboratory, for example, early difficult temperament was moderated by maternal positive behavior such that toddlers who were negative and whose mothers provided more positive guidance were less likely to develop externalizing behavior problems than toddlers with negative temperament and parents who provided little positive guidance (Calkins 2002). Thus, parenting behavior can clearly compensate for the challenges to the emotional regulation system that a difficult temperament poses. Other studies confirm this kind of relation. Bates and colleagues (Bates, Pettit, Dodge, & Ridge 1998) found that children whose early temperament was characterized as resistant to external control were more likely to develop externalizing behavior problems when the parenting to which they were exposed was restrictive. Other studies have found that dimensions of the family environment such as parent psychological functioning interact with

early temperament to predict child behavior problems (Mun, Fitzgerald, Von Eyre, Puttler, & Zucker 2001; Edwards, Leonard, & Eiden 2001). Thus, there is evidence for temperament, moderated by aspects of the caregiving environment, to have indirect effects on both proximal and distal indices of emotional regulation.

Mediational effects of temperament

A third way that temperament may affect the development of emotional regulation is through processes or mechanisms that mediate this relation. As defined by Baron and Kenney (1986), a mediator is a variable that serves as the mechanism through which a predictor affects an outcome. Because mediational analyses allow one to specify possible mechanisms or processes that explain how one variable affects another, they are thus quite useful in understanding developmental process. Although few studies have specified the goal of understanding the mechanism through which temperament affects regulation, it is possible to extrapolate, at least on a theoretical level, some of these processes given the existing literature.

For example, one hypothesized central process in the emergence of emotional regulation is the control of attention (Kopp 2002). The development of attention and its use in the control of emotional reactivity begins to emerge in the first year of life and continues throughout the preschool and school years (Posner & Rothbart 2000; Rothbart 1989; Rothbart & Bates 1998). Individual differences in the ability to voluntarily sustain focus or shift attention are critical components of self-control of attention. Attentional orienting skills, in particular, have been identified as a critical component of the regulatory process, as orienting has the direct effect of amplifying, at a neural level, the stimuli toward which attention is directed, thus changing the affective experience of the individual (Rothbart et al. 1994). Thus, orienting skills assist in the management of both negative and positive emotions, and consequently to the development of adaptive control of emotion. Qualitative shifts in attention skills across the first twelve months of life may be integral to the qualitative shifts in emotional regulation that are observed during this period. However, there are clear individual differences in the ability to utilize attention to successfully control emotion. For example, Rothbart (1981, 1986) found increases in positive affect and decreases in distress from 3 to 6 months during episodes of focused attention, suggesting that control of attention is tied to affective experience. Moreover, negative affectivity is believed to interfere with the child's ability to deploy attention to explore and learn about the environment (Rothbart, Posner, & Rosicky 1994;

Ruff & Rothbart 1996). Clearly, attention must be considered a central process that links temperamental reactivity to emotional regulation very early in development.

A second process that may link temperament to emotional regulation in early development may be physiological in nature. Descriptions of processes underlying the maturation and differentiation of the frontal lobes (Fox 1994), for example, suggest that not only do these brain processes predispose individuals to experience specific types of affects, they also support the initial approach versus withdrawal tendencies of individuals in the face of affective stimuli. With development, these frontal processes play a critical role in the more effortful control of attention, emotions, and behavior. Data from Fox and colleagues suggests, for example, that temperamentally inhibited or fearful infants who display a profile of greater relative right frontal brain electrical activity have difficulty as preschoolers in the regulation of emotion and behavior that are so critical to successful social interaction with peers (Fox et al. 1996).

Similarly, physiological measures of autonomic nervous system activity, such as heart rate variability, have been linked to both temperamental reactivity and emotional regulation. Regulation or suppression of vagal tone during emotional challenge is thought to be a physiological strategy that permits sustained attention and behaviors indicative of active coping that are mediated by the parasympathetic nervous system (Porges 1991 1996; Wilson & Gottman 1996). The degree to which the organism experiences autonomic nervous system arousal, as reflected in heart rate variability, may affect the successful generation of emotional coping skills because of the organism's capacity to regulate physiological arousal.

Although physiological and attentional control mechanisms have been hypothesized to mediate the relation between reactivity and emotional regulation (Calkins 1994; Kopp 2002; Porges 1996), there has been only indirect empirical evidence to support this relation (Calkins et al. 2002). Explicit tests of such a model are needed to verify these relations. Greater emphasis on physiological processes in studies of early emotional development will significantly enhance our ability to understand these relations.

Future directions in the study of temperament and emotional regulation

The theoretical and empirical work reviewed to this point suggests that there are clear implications of temperamental reactivity, particularly distress, for the development of emotional regulation. Based on the work conducted to date,

associations are strongest concurrently and in contexts that have explicit emotional demands. Longer-term relations and relations to emotional functioning in other contexts are more modestly supported. Nevertheless, there is reason to think that future studies of these phenomenon could clarify the nature and extent of these relations.

First, empirical work that is more focused on process, rather than simple associations, might be more informative for elucidating the complex ways that temperament and emotion regulation influence development. I have highlighted here some of these processes, namely, attention and physiology; others are probably involved as well.

Second, it is apparent that the literature has examined extensively the temperament dimension of frustration and its relation to emotional regulation, but there has been less emphasis on other dimensions such as fear, sadness, and positive affectivity. Clearly, the question of whether the direct and indirect effects of temperament described above apply to these other emotional tendencies should be addressed.

Third, it is clear that the direction of effects in development is not always from child to outcome. Transactional influences from the environment to the child and back again are responsible for some pathways in development. Moreover, it must be acknowledged that the child plays an important role in the dyadic interactions with caregivers that lead to the development of relationships that are themselves critical to the development of emotional regulation (Calkins 1994; Thompson 1994). Consequently, these transactional influences may obscure the identification of longer-term effects of temperament on emotional processes, but they are important to understanding developmental pathways (Cicchetti 1984, 1993).

Finally, it may be more useful in studying the development of emotional regulation to adopt an approach that considers multiple levels of analysis of self-regulation (Calkins & Fox 2002), rather than emotional regulatory processes in isolation. It has been noted by a number of researchers that self-regulation occurs on a number of different, and likely interrelated, levels (Calkins & Fox 2002; Eisenberg, Guthrie, Fabes, Shepard, Losoya, Murphy, et al. 2001; Posner & Rothbart 2000). Clearly, there is a rationale provided by the empirical literature reviewed here for examining emotion-related processes across a number of interrelated levels of analysis. Such an analysis may be more fruitful to establishing links from emotional regulation to adaptive functioning in childhood.

From this perspective, we may conceptualize this larger self-regulatory system as adaptive control that may be observed across a number of levels,

including at the levels of physiological, attentional, emotional, behavioral, cognitive, and interpersonal or social processes (Calkins & Fox 2002). Control at these various levels emerges, at least in primitive form, across the prenatal, infancy, toddler and early childhood periods of development. Importantly, though, the mastery of earlier regulatory tasks becomes an important component of later competencies, and by extension, the level of mastery of these early skills may constrain the development of later skills. Thus, understanding the development of specific regulatory processes, such as emotional regulation, becomes integral to understanding how regulatory deficits across multiple levels affect the emergence of childhood behavior and behavior problems (Calkins & Fox 2002; Keenan 2000). Moreover embedding emotional regulation in a larger self-regulatory framework has the advantage of allowing researchers to understand the multiple levels of infants and child functioning that may be influenced by early temperament.

Summary

This review of research examining the effects of temperament on the development of emotional regulation seems to demonstrate that the direct effects of this relationship are quite evident. The level of distress an infant experiences, which is thought to be reflective of a stable, biologically-based trait, influences the tendency to utilize specific emotional regulation strategies and the subsequent level of distress, as well as the likelihood of the future use of particular strategies. In addition, there is evidence from the psychophysiological literature that predictable biological responses can be expected from infants in contexts that activate the emotion system. Beyond these immediate and direct effects, more distal effects of temperament on behavioral and emotional regulation that underlies adaptive functioning in preschool and early childhood have also been observed. There are also clear indirect effects of temperament on emotional regulation. However, because these effects have not been systematically assessed, with respect to the specific types of moderators and mediators, the age at which such processes are observable, and the proximal versus distal emotional regulation outcomes involved, clear interpretation of these data is not possible. However, suggestions for a more integrated analysis of emotional regulation, within the context of a larger self-regulatory system are proposed. Such a multiple levels analysis would have the benefit of integrating the various physiological and psychological processes that have been implicated in the development of emotional regulation.

Acknowledgements

The writing of this chapter was supported by National Institute of Health grants to the author (MH 55584 and MH 58144). Address correspondence to: Susan D. Calkins, Department of Psychology, P. O. Box 26170, University of North Carolina at Greensboro, Greensboro, NC 27402-6170, SDCALKIN@UNCG.EDU.

References

- Aksan, N. Goldsmith, H. H., Smider, N., Essex, M., Clark, R., Klein, M., & Vandell, D. (1999). Derivation and prediction of temperamental types among preschoolers. *Developmental Psychology*, 35, 958–971.
- Barkley (1997). *ADHD and the nature of self-control*. New York: Guilford Press.
- Baron, R. M. & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, 51 (6), 1173–1182.
- Bates, J. E., Bayles, K., Bennett, D. S., Ridge, B., & Brown, M. M. (1991). Origins of externalizing behavior problems at eight years of age. In D. Pepler & K. Rubin (Eds.), *Development and treatment of childhood aggression* (pp. 93–120). Hillsdale, NJ: Erlbaum.
- Bates, J. E., Pettit, G., Dodge, K., & Ridge, B. (1998). Interaction of temperament resistance to control and restrictive parenting in the development of externalizing behavior. *Developmental Psychology*, 34, 982–995.
- Braungart, J. M. & Stifter, C. A. (1991). Regulation of negative reactivity during the Strange Situation: Temperament and attachment in 12-month-old infants. *Infant Behavior and Development*, 14, 349–367.
- Braungart-Rieker, J. & Stifter, C. (1996). Infants' responses to frustrating situations: Continuity and change in reactivity and regulation. *Child Development*, 67, 1767–1769.
- Bronson, Martha B. (2000). *Self-regulation in early childhood: Nature and Nurture*. New York, NY: The Guilford Press.
- Buss & Goldsmith (1998). Fear and anger regulation in infancy: Effects on the temporal dynamics of affective expression. *Child Development*, 69, 359–374.
- Calkins, S. D. (1994). Origins and outcomes of individual differences in emotional regulation. In N. A. Fox (Ed.), *Emotion regulation: Behavioral and biological considerations*. Monographs of the Society for Research in Child Development.
- Calkins, S. D. (1997). Cardiac vagal tone indices of temperamental reactivity and behavioral regulation in young children. *Developmental Psychobiology*, 31, 125–135.
- Calkins, S. D. (2002). Does aversive behavior during toddlerhood matter? The effects of difficult temperament on maternal perceptions and behavior. *Infant Mental Health Journal*, 23, 381–402.

- Calkins, S. D. (in press). Early attachment process and the development of emotional self-regulation. Chapter to appear in Baumeister & Vohs, *Handbook of Self Regulation*, Erlbaum.
- Calkins, S. D. & Dedmon, S. A. (2000). Physiological and behavioral regulation in two-year-old children with aggressive/destructive behavior problems. *Journal of Abnormal Child Psychology*, 2, 103–118.
- Calkins, S. D., Dedmon, S., Gill, K., Lomax, L., & Johnson, L. (2002). Frustration in infancy: Implications for emotion regulation, physiological processes, and temperament. *Infancy*, 3, 175–198.
- Calkins, S. D. & Fox, N. A. (1992). The relations among infant temperament, security of attachment, and behavioral inhibition at 24 months. *Child Development*, 63, 1456–1472.
- Calkins, S. D. & Fox, N. A. (2002). Self-regulatory processes in early personality development: A multilevel approach to the study of childhood social withdrawal and aggression. *Development and Psychopathology*.
- Calkins, S. D., Fox, N. A., & Marshall, T. R. (1996). Behavioral and physiological antecedents of inhibition in infancy. *Child Development*, 67, 523–540.
- Calkins, S. D., Gill, K. A., Johnson, M. C., & Smith, C. (1999). Emotional reactivity and emotion regulation strategies and predictors of social behavior with peers during toddlerhood. *Social Development*, 8, 310–341.
- Calkins, S. D. & Howse, R. (in press). Individual differences in self-regulation: Implications for childhood adjustment. Chapter to appear in P. Philipot & R. Feldman (Eds.), *The Regulation of Emotion*. Mahwah, NJ: Lawrence Erlbaum.
- Calkins, S. D. & Johnson, M. C. (1998). Toddler regulation of distress to frustrating events: Temperamental and maternal correlates. *Infant Behavior and Development*, 21, 379–395.
- Calkins, S. D., Smith, C. L., Gill, K. L., & Johnson, M. C. (1998). Maternal interactive style across contexts: Relations to emotional, behavioral and physiological regulation during toddlerhood. *Social Development*, 7 (3), 350–369.
- Campbell, S. B. (1995). Behavior problems in preschool children: A review of recent research. *Journal of Child Psychology and Psychiatry*, 36, 113–149.
- Caspi, A., Henry, B., McGee, R., Moffitt, T., & Silva, P. (1995). Temperamental origins of child and adolescent behavior problems: From age three to fifteen. *Child Development*, 66, 55–68.
- Cassidy, J. (1994). Emotion regulation: Influences of attachment relationships. In N. A. Fox (Ed.), *Emotion regulation: Behavioral and biological considerations*. Monographs of the Society for Research in Child Development.
- Cicchetti, D. (1984). The emergence of developmental psychopathology, *Child Development*, 55, 1–7.
- Cicchetti, D. (1993). Developmental psychopathology: Reactions, reflections, projections. *Developmental Review*, 13, 471–502.
- Cicchetti, D., Ackerman, B., & Izard, C. (1995). Emotions and emotion regulation in developmental psychopathology. *Development and Psychopathology*, 7, 1–10.

- Cicchetti, D., Ganiban, J., & Barnett, D. (1991). Contributions from the study of high-risk populations to understanding the development of emotional regulation. In J. Garber & K. A. Dodge (Eds.), *The development of emotion regulation and dysregulation* (pp. 69–88). Cambridge: Cambridge University Press.
- Diener, M., Mangelsdorf, S., McHale, J., & Frosch, C. (2002). Infants' behavioral strategies for emotion regulation with fathers and mothers: Associations with emotional expressions and attachment quality. *Infancy*, 3, 153–174.
- Donovan, W. L. & Leavitt, L. A. (1985). Physiologic assessment of mother-infant attachment. *Journal of the American Academy of Child Psychiatry*, 24, 65–70.
- Edwards, E. P., Leonard, K., & Eiden, R. (2001). Temperament and behavior problems among infants in alcoholic families. *Infant Mental Health Journal*, 22, 374–392.
- Eisenberg, N., Cumberland, A., Spinrad, T. L., Fabes, R. A., Shepard, S. A., Reiser, M., Murphy, B. C., Losoya, S. H., & Guthrie, I. K. (2001). The Relations of regulation and emotionality to children's externalizing and internalizing problem behavior. *Child Development*, 72 (4), 1112–1134.
- Eisenberg, N., Fabes, R. A., Bernzweig, J., Karbon, M., Poulin, R., & Hanish, L. (1993). The relations of emotionality and regulation to preschoolers' social skills and sociometric status. *Child Development*, 64, 1418–1438.
- Eisenberg, N., Fabes, R., Guthrie, I., Murphy, B., Maszk, P., Holmgren, R., & Suh, K. (1996). The relations of regulation and emotionality to problem behavior in elementary school. *Development and Psychopathology*, 8, 141–162.
- Eisenberg, N., Fabes, R. A., Nyman, M., Bernzweig, J., Bernzweig, J., & Pinuelas, A. (1994). The relations of emotionality and regulation to children's anger-related reactions. *Child Development*, 65, 109–128.
- Eisenberg, N., Fabes, R. A., Guthrie, & Reiser, M. (2000). Dispositional emotionality and regulation: Their role in predicting quality of social functioning. *Journal of Personality and Social Psychology*, 78, 136–157.
- Eisenberg, N., Fabes, R. A., Shephard, S. A., Murphy, B. C., Guthrie, I. K., Jones, S., Freidman, J., Poulin, R., & Maszk, P. (1997). Contemporaneous and longitudinal prediction of children's social functioning from regulation and emotionality. *Child Development*, 68, 642–664.
- Eisenberg, N., Guthrie, I. K., Fabes, R. A., Shepard, S., Losoya, S., Murphy, B. C., et al. (2000). Prediction of elementary school children's externalizing problem behaviors from attention and behavioral regulation and negative emotionality. *Child Development*, 71 (5), 1367–1382.
- Eisenberg, N., Murphy, B. C., Maszk, P., Smith, M., & Karbon, M. (1995). The role of emotionality and regulation in children's social functioning: A longitudinal study. *Child Development*, 66, 1360–1384.
- Fox, N. A. (1989). Psychophysiological correlates of emotional reactivity during the first year of life. *Developmental Psychology*, 25, 364–372.
- Fox, N. A. (1991). If it's not left, it's right: Electroencephalogram asymmetry and the development of emotion. *American Psychologist*, 46, 863–872.
- Fox, N. A. (1994). Dynamic cerebral process underlying emotion regulation. In N. A. Fox (Ed.), *Emotion regulation: Behavioral and biological considerations*. Monographs of the Society for Research in Child Development.

- Fox, N. A. & Calkins, S. D. (1993). Pathways to aggression and social withdrawal: Interactions among temperament, attachment and regulation. K. Rubin & J. Asendorpf (Eds.), *Social withdrawal, shyness and inhibition in childhood* (pp. 81–100). Hillsdale, NJ: Lawrence Erlbaum.
- Fox, N. & Calkins, S. D. (2003). The development of self-control of emotion: Intrinsic and extrinsic influences. *Motivation and Emotion*, 27, 7–26.
- Fox, N. A., Calkins, S. D., Porges, S. W., Rubin, K., Coplan, R. J., Stewart, S., Marshall, T. R., & Long, J. M. (1995). Frontal activation asymmetry and social competence at four years of age. *Child Development*, 66, 1770–1784.
- Fox, N. A., Henderson, H. A., & Marshall, P. J. (2001). The biology of temperament: An integrative approach. In C. A. Nelson & M. Luciana (Eds.), *The Handbook of Developmental Cognitive Neuroscience*. Cambridge, MA: MIT Press.
- Gianino, A. & Tronick, E. Z. (1988). The mutual regulation model: The infant's self and interactive regulation coping and defense capacities. In T. Field, P. McCabe, & N. Schneiderman (Eds.), *Stress and coping* (pp. 47–68). Hillsdale, NJ: Erlbaum.
- Gilliom, M., Shaw, D. Beck, J., Schonberg, M., & Lukon, J. (2002). Anger regulation in disadvantaged preschool boys: Strategies, antecedents, and the development of self-control. *Developmental Psychology*, 38, 222–235.
- Goldsmith, H. H., Buss, A., Plomin, R., Rothbart, M., Thomas, A., Chess, S., Hinde, R., & McCall, R. (1987). Roundtable: What is temperament? Four approaches. *Child Development*, 58, 505–529.
- Grolnick, W., Cosgrove, T., & Bridges, L. (1996). Age-graded change in the initiation of positive affect. *Infant Behavior and Development*, 19, 153–157.
- Gross, J. (1999). Emotion regulation: Past, present, future. *Cognition and Emotion*, 13, 555–573.
- Gunnar, M. R. (1990). The psychobiology of infant temperament. In J. F. Colombo & J. Fagan (Eds.), *Individual differences in infancy: Reliability, stability, prediction*. Hillsdale, NJ: Lawrence Erlbaum Associates.
- Huffman, L. C., Bryan, Y., del Carmen, R., Pederson, F., Doussard-Roosevelt, J., & Porges, S. (1998). Infant temperament and cardiac vagal tone: Assessments at twelve weeks of age. *Child Development*, 69, 624–635.
- Kagan, J. (1997). Temperament and the reactions to unfamiliarity. *Child Development*, 68, 139–143.
- Keenan, K. (2000). Emotion dysregulation as a risk factor for child psychopathology. *Clinical Psychology: Science and Practice*, 7, 418–434.
- Kochanska, G., Coy, K. C., & Murray, K. Y. (2001). The development of self-regulation in the first four years of life. *Child Development*, 72 (4), 1091–1111.
- Kopp, C. (1982). Antecedents of self-regulation: A developmental perspective. *Developmental Psychology*, 18, 199–214.
- Kopp, C. (1989). Regulation of distress and negative emotions: A developmental view. *Developmental Psychology*, 25, 243–254.
- Kopp, C. (1992). Emotional distress and control in young children. In N. Eisenberg & R. Fabes (Eds.), *Emotion and its regulation in early development. New directions for child development*, No. 55: The Jossey-Bass education series (pp. 7–23). San Francisco, CA: Jossey-Bass/Pfeiffer.

- Kopp, C. (2002). The codevelopment of attention and emotion regulation. *Infancy*, 3, 199–208.
- Lewis, M. & Miller, S. (1990). *Handbook of Developmental Psychopathology*. New York: Plenum Press.
- Mangelsdorf, S., Gunnar, M., Kestenbaum, R., Lang, S., & Andreas (1990). Infant proneness-to-distress temperament, maternal personality, and mother-infant attachment: associations and goodness of fit. *Child Development*, 61, 820–831.
- Mangelsdorf, S., Sahpiro, J., & Marzolf, D. (1995). Developmental and temperamental differences in emotional regulation in infancy. *Child Development*, 66, 1817–1828.
- Mun, E., Fitzgerald, H., Von Eye, A., Puttler, & Zucker, R. (2001). Temperamental characteristics as predictors of externalizing and internalizing child behavior problems in the context of high and low parental psychopathology. *Infant Mental Health Journal*, 22, 393–415.
- Nachmias, M., Gunnar, M., Mangelsdorf, S., Parritz, R., et al. (1996). Behavioral inhibition and stress reactivity: The moderating role of attachment security. *Child Development*, 67, 508–522.
- Porges, S. W. (1991). Vagal tone: An autonomic mediatory of affect. In J. A. Garber & K. A. Dodge (Eds.), *The development of affect regulation and dysregulation* (pp. 11–128). New York: Cambridge University Press.
- Porges, S. W. (1996). Physiological Regulation in high-risk infants: A model for assessment and potential intervention. *Development and Psychopathology*, 8, 43–58.
- Porges, S. W., Doussard-Roosevelt, J. A., & Maiti, A. K. (1994). Vagal tone and the physiological regulation of emotion. In N. A. Fox (Ed.), *Emotion regulation: Behavioral and biological considerations. Monographs of the Society for Research in Child Development*, 59 (2–3, Serial No. 240), 167–186.
- Porges, S. W., Doussard-Roosevelt, J. A., Portales, A. L., & Greenspan, S. I. (1996). Infant regulation of the vagal “brake” predicts child behavior problems: A psychobiological model of social behavior. *Developmental Psychobiology*, 29, 697–712.
- Posner, M. I. & Rothbart, M. K. (1992). Attentional mechanisms and conscious experience. In D. Milner & M. Ruggs (Eds.), *The neuropsychology of consciousness* (pp. 91–111). San Diego, CA: Academic Press.
- Posner, M. I. & Rothbart, M. K. (2000). Developing mechanisms of self-regulation. *Development and Psychopathology*, 12, 427–441.
- Prior, M., Smart, D., Sanson, A., Pedlow, R., & Oberklaid, F. (1992). Transient versus stable behavior problems in a normative sample: Infancy to school age. *Journal of Pediatric Psychology*, 17, 423–443.
- Rothbart, M. K. (1981). Measurement of temperament in infancy. *Child Development*, 52, 569–578.
- Rothbart, M. K. (1986). Longitudinal observation of infant temperament. *Developmental Psychology*, 22, 356–365.
- Rothbart, M. K. (1989). Temperament and development. In G. Kohnstamm, J. Bates, & M. K. Rothbart (Eds.), *Temperament in childhood* (pp. 187–248). Chichester, England: Wiley.
- Rothbart, M. K., Ahadi, S. A., & Hershey, K. L. (1994). Temperament and social behavior in childhood. *Merrill-Palmer-Quarterly*, 40, 21–39.

- Rothbart, M. K. & Bates, J. E. (1998). Temperament. In W. Damon (Series Ed.) & N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (5th ed., pp. 105–176). New York: Wiley.
- Rothbart, M. K. & Derryberry, D. (1981). Development of individual differences in temperament. In M. E. Lamb & A. L. Brown (Eds.), *The neuropsychology of individual differences: A developmental perspective* (pp. 93–123). New York: Plenum.
- Rothbart, M. K., Derryberry, D., & Hershey, K. (2000). Stability of temperament in childhood: Laboratory infant assessment to parent report at seven years. In V. Molfese & D. Molfese (Eds.), *Temperament and personality development across the lifespan* (pp. 85–119). Mahwah, NJ: Erlbaum.
- Rothbart, M. K. & Posner, M. I. (1985). Temperament and the development of self-regulation. In L. C. Hartlage & C. F. Telzrow (Eds.), *The neuropsychology of individual differences: A developmental perspective* (pp. 93–123). New York: Plenum.
- Rothbart, M. K., Posner, M. I., & Boylan, A. (1990). Regulatory mechanisms in infant development. In J. Enns (Ed.), *The development of attention: Research and theory* (pp. 139–160). Amsterdam, Netherlands: Elsevier.
- Rothbart, M., Ziaie, H., & O'Boyle, C. (1992). Self-regulation and emotion in infancy. In N. Eisenberg & R. Fabes (Eds.), *Emotion and its regulation in early development. New directions for child development*, No. 55: The Jossey-Bass education series (pp. 7–23). San Francisco, CA: Jossey-Bass/Pfeiffer.
- Rubin, K. H., Coplan, R. J., Fox, N. A., & Calkins, S. D. (1995). Emotionality, emotion regulation and preschooler's social adaptation. *Development and Psychopathology*, 7, 49–62.
- Ruff, H. & Rothbart, M. K. (1996). *Attention in Early Development*. New York: Oxford University Press.
- Seifer, R., Schiller, M., Sameroff, A., Resnick, S., & Riordan, K. (1996). Attachment, maternal sensitivity, and infant temperament during the first year of life. *Developmental Psychology*, 32, 12–25.
- Sethi, A., Mischel, W., Aber, J. L., Shoda, Y., & Rodriguez, M. L. (2000). The role of strategic attention deployment in development of self-regulation: Predicting preschoolers' delay of gratification from mother-toddler interactions. *Developmental Psychology*, 36 (6), 767–777.
- Shaw, D. S., Keenan, K., & Vondra, J., Delliquadri, & Giovannelli, J. (1997). Antecedents of preschool children's internalizing problems: A longitudinal study of low-income families. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 1760–1767.
- Shaw, D., Owens, E., Giovannelli, J., & Winslow, E. (2001). Infant and toddler pathways leading to early externalizing disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40, 36–43.
- Sroufe, A. L. (1996). *Emotional development: The organization of emotional life in the early years*. New York, NY: Cambridge University Press.
- Stifter, C. A. & Braungart, J. M. (1995). The regulation of negative reactivity in infancy: Function and development. *Developmental Psychology*, 31 (3), 448–455.
- Stifter, C. A. & Fox, N. A. (1990). Infant reactivity: Physiological correlates of newborn and 5-month temperament. *Developmental Psychology*, 26, 582–588.

- Stifter, C. A. & Moyer, D. (1991). The regulation of positive affect: Gaze aversion activity during mother/infant interaction. *Infant Behavior and Development*, 14 (1), 111–123.
- Thomas, A., Chess, S., & Birch, H. G. (1970). The origins of personality. *Scientific American*, 223 (2), 102–109.
- Thomas, A., Birch, H., Chess, S., Hertzog, M., & Korn, S. (1964). *Behavioral Individuality in Early Childhood*. NY: New York University Press.
- Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. In N. A. Fox (Ed.), *The development of emotion regulation: Biological and behavioral considerations. Monographs of the Society for Research in Child Development*, 59 (2–3 Serial No. 240), 25–52.
- Thompson, R. A. (1998). Early sociopersonality development. In W. Damon (Series Ed.) & N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (5th ed., pp. 25–104). New York: Wiley.
- Wilson, B. & Gottman, J. (1996). Attention – the shuttle between emotion and cognition: Risk, Resiliency, and physiological bases. In E. Hetherington & E. Blechman (Eds.), *Stress, coping and resiliency in children and families*. Mahwah, NJ: Lawrence Erlbaum Associates.

CHAPTER 3

Emotion dysregulation and psychopathology

Kimberly Shipman, Renee Schneider, and Amy Brown

Introduction

Interest in emotion and emotion regulation has burgeoned in the past two decades, particularly with regard to the development of emotional competence and the role that emotion may play in facilitating psychosocial adjustment across the lifespan. Recent theory and research in this area highlights the importance of emotion to navigating interpersonal relationships and to organizing an individual's goal-related behavior. This emphasis on the adaptive functions of emotion reflects a shift from traditional emotion theories that viewed emotions as irrational feeling states with little or no influence on external events (Keltner & Gross 1999). The goal of this chapter is to examine the relation between emotion regulation (or dysregulation) and psychopathology within the context of functionalist theory. To do this, we will first present a theoretical discussion of the functional role of emotion. Next we will define the constructs of emotion regulation and dysregulation and discuss different avenues to emotion regulation (e.g., attentional control, expression management, coping). Finally, we will review the empirical literature that examines the links between emotion dysregulation and psychological symptomatology and discuss future directions for research.

Functionalist theory

The functionalist theory of emotion provides a foundation for understanding the importance of emotion regulation to psychosocial adaptation throughout the lifespan (Barrett & Campos 1987; Campos, Mumme, Kermoian, & Campos 1994; Saarni, Mumme, & Campos 1998). Functionalist theory views emotions as "bi-directional processes of establishing, maintain-

ing, and/or disrupting significant relationships between an organism and the external or internal environment” (Barrett & Campos 1987:558). Within this context, emotion is seen as having both intrapersonal and interpersonal regulatory consequences, facilitating individual well-being and adaptation to the social environment. For example, according to the functionalist perspective, joy functions intrapersonally to increase the likelihood that an individual will continue his or her activity while, at the same time, signaling to others to maintain the interaction. Similarly, sadness functions to elicit help in situations in which an individual recognizes he or she will be unable to attain the desired goal without assistance. Embedded within the functionalist definition of emotion is an understanding that emotions are reciprocal in nature. Emotion not only motivates individual behavior toward goal attainment, but emotional expression motivates the behavior of others in goal-relevant ways. Further, emotional development occurs within a relational context such that children learn to manage emotion within significant interpersonal relationships, particularly through parent-child interaction. Finally, emotion-related behavior is characterized by flexibility such that the adaptive value of a given behavior will vary as a function of individual goals, developmental level, and social context. The functional nature of emotion, though emphasized in functionalist theory, is not a unique concept. Rather, it has roots in historical theories of emotion (Darwin 1872) and is central to several current theories of emotional development (see Keltner & Gross 1999 for a review).

Emotion regulation and dysregulation

From a functionalist perspective, emotion regulation is essential given that it allows individuals to respond strategically to emotionally-arousing situations (Saarni 1999). Emotion regulation has been defined in relation to intrinsic or extrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions in order to achieve individual goals and to facilitate adaptation to the social environment (Campos et al. 1994; Thompson 1994). According to this definition, emotion regulation includes a number of inter-related skills (e.g., interpretation of nonverbal emotional expressions of others, clarity of one’s own emotional displays, emotional self-awareness, understanding of causes and consequences of emotion) in addition to the ability to manage emotional arousal and expression. These skills, often de-

scribed under the rubric of emotional understanding, are clearly central to the regulatory process, particularly with regard to monitoring and evaluating emotional reactions. Further, contrary to common conceptions, emotion regulation is associated not only with minimizing emotional arousal and expressivity but also with maintaining and enhancing emotion-related experience for emotions with both positive and negative valences. For example, young children will often intensify their distress after scraping their knee when they see their parents are watching. In addition, emotion regulation involves both emotion self-management and any external influences that serve a regulatory function. Early in development, parents help children to regulate emotion in many ways, including creating a climate with developmentally appropriate emotional demands, providing support and assistance, and teaching strategies for emotion management. Finally, emotion regulation is context dependent and must be regarded functionally in terms of the goals that are motivating the individual's regulatory behavior. Competent emotion regulation skills are central to accomplishing developmental tasks of both childhood and adulthood (e.g., establishing and maintaining interpersonal relationships, achieving academic/occupational success).

So far we have highlighted the regulatory functions that emotions serve with regard to helping to organize and motivate goal-directed behavior essential to adaptation within our social environment. If emotions are fundamentally adaptive and regulated, then we must ask how we should conceptualize emotion dysregulation. Consideration of clinical literature suggest that dysregulated emotion (e.g., blunted affect, emotional lability, high levels of sadness or anxiety) is associated with many, if not most, psychological disorders and often serves as the defining feature of diagnostic categories. Emotion dysregulation is best conceptualized as involving normal regulatory processes that are operating in a dysfunctional manner (Cole, Michel, & Teti 1994). Dysregulated emotion may serve some protective or communicative function for the individual but in a manner that interferes with adjustment by increasing short- or long-term risk for adaptational failures in development (e.g., psychopathology, interpersonal difficulties). Patterns of emotion regulation that impair functioning may become symptoms of psychopathology (e.g., high levels of emotional distress, hypervigilance) or they may interfere with development in other areas in such a way as to create risk for psychosocial difficulties (e.g., creating problematic interpersonal relationships). Further, regulatory behavior may be adaptive in a given context but, at the same time, be dysregulated in that it interferes with current functioning (in this or other central contexts) or jeopardizes the accomplishment of important developmental tasks. When investigating the re-

lation between emotion dysregulation and psychopathology, we must consider an individual's pattern of regulatory behavior, including the potential functions that the dysregulated behavior may be serving as well as the dysfunctions in specific processes through which regulation takes place.

What gets regulated in emotion regulation?

Given that emotion involves physiological, behavioral, and cognitive systems, there are diverse avenues to emotion management (Thompson 1994). Thompson identifies several dimensions as central to the emotion regulation process, including neurophysiological factors, attentional and attributional processes, coping resources, and emotional demands of the environment. Neurophysiological factors include inhibitory and excitatory processes within the nervous system that regulate arousal, including emotional arousal and physiological reactivity. Research suggests that differences in these systems relate to social and personality functioning. For example, children with a lower threshold for reactivity in limbic system mediating fear and defense are more likely to show behavioral inhibition and anxiety (Kagan, Snidman, Zentner, & Peterson 1999; Thompson 1994). Attentional processes serve regulatory functions by managing the intake of emotionally-arousing information, through processes varying in developmental sophistication from visual disengagement (e.g., turning away from stimuli) to behavioral distraction (e.g., engaging in another activity to distract) and cognitive redirection (e.g., thinking positive thoughts). Attributional processes involve altering one's interpretation of a situation or an internal cue in such a way as to facilitate emotion regulation. For example, when left out of a game, a child may interpret this as an oversight instead of an indication that the other children are not interested in his company. Similarly, physiological arousal associated with stage fright may be interpreted as facilitating performance rather than as a sign of impending dysfunction. Access to coping resources also facilitates effective emotion regulation. Children and adults alike elicit interpersonal support and pragmatic assistance from family and friends in order to manage emotional arousal (including maintenance and enhancement of positive emotion as well as amelioration of distress). Finally, individuals regulate the emotional demands of familiar situations. For example, parents create manageable emotional demands for their children by modifying experiences both within the family (e.g., consistency of daily routines, frequency of parent-child separations) and outside the home (e.g., exposure to child care, participation in extra-curricular activities) that fit the child's devel-

opmental level and temperament. These regulatory processes serve important adaptive functions but, in some instances, may become dysregulated such that they do so at the cost of psychological adjustment.

Emotion dysregulation and psychopathology in childhood

Nonclinical samples

Research provides strong evidence for a relation between emotion dysregulation and internalizing and externalizing behavior problems in children. Consistent with both functionalist theory (Barrett & Campos 1987) and Thompson's (1994) definition of emotion regulation, researchers have adopted a broad stance, with particular focus on the modulation of emotional expressivity, attention, coping behavior, and physiological arousal. Overall, differences in emotion regulation across these various domains appear to distinguish children with behavior problems from their peers. With regard to emotional expressivity, research suggests that children with internalizing or externalizing behavior problems differ from nondisordered children in the frequency and intensity of their emotional displays (e.g., Zeman, Shipman, & Suveg 2002). In particular, findings indicate that children with internalizing problems are more likely to express sadness (Eisenberg et al. 2001) but less likely to express anger relative to other children (Zeman et al. 2002). Additionally, children with internalizing problems report a higher incidence of dysregulated emotional expression (e.g., uncontrollable sobbing, slamming doors; Zeman et al. 2002). Similarly, compared with nondisordered children, those with externalizing problems are rated as more emotionally intense by parents and teachers (Eisenberg, Fabes, Guthrie et al. 1996) and are more likely to express anger inappropriately (Cole, Zahn-Waxler, & Smith 1994; Eisenberg, Fabes, Murphy et al. 1999). Though some research indicates that children with externalizing problems express negative emotions, particularly anger, more often than their peers (Cole, Zahn-Waxler, Fox, Usher, & Welsh 1996; Eisenberg et al. 2001), findings also suggest that a subset of children with externalizing problems inhibit emotional expression and that these children may be at risk for the development of comorbid internalizing problems (Cole, Zahn-Waxler et al. 1994, 1996).

Further, research suggests that a low level of attentional control (i.e., the ability to shift or refocus attention as appropriate to the situation) is predictive of behavioral difficulties in early and middle childhood (Derryberry &

Rothbart 1988; Eisenberg, Fabes, Guthrie et al. 1996; Eisenberg et al. 2000, 2001). Attentional control may function to regulate both positive and negative emotions by keeping the child focused on pleasant stimuli or by withdrawing attention away from aversive or threatening stimuli (Rothbart 1989). Eisenberg and colleagues (Eisenberg et al. 2001; Eisenberg, Fabes, Guthrie et al. 1996) have assessed attentional control using both parent and teacher report as well as an observational measure (e.g., degree of gaze aversion in response to a distressing film) in early elementary-school-age children. In general, findings indicate negative relations between attentional control and both externalizing and internalizing behavior problems. Eisenberg et al. (2000) expanded on this research, demonstrating that attentional control predicts behavior problems primarily for children who show high levels of negative emotionality. Related research suggests important relations between attentional control and proneness to anger (Derryberry & Rothbart 1988), aggression (Hart, Keller, Edelstein, & Hofmann 1998), and social difficulties (Calkins, Gill, Johnson, & Smith 1999; Eisenberg et al. 1995). Finally, developmental research has demonstrated that change in the attentional capacity of infants is related to associated reductions in emotional distress (Johnson, Posner, & Rothbart 1991) and has identified attentional mechanisms as central to the development of effortful control (i.e., ability to suppress a dominant response and perform a subdominant response; Rothbart, Derryberry, & Posner 1994).

Similarly, research suggests that problematic coping is related to children's behavior problems. Findings indicate that children who focus on the source of their distress or who use physical or emotional avoidance in an effort to escape distress are more likely to manifest behavior problems relative to their peers (Calkins et al. 1999; Eisenberg, Fabes, Murphy et al. 1999; Gilliom, Shaw, Beck, Schonberg, & Lukon 2002). In contrast, children who rely on distraction to regulate negative emotion are less likely to manifest behavior problems, both concurrently and over time (Gilliom et al. 2002). Additionally, children with behavior problems often rely on venting and aggression (Calkins et al. 1999; Cole, Zahn-Waxler et al. 1994; Zeman et al. 2002) rather than on problem-solving strategies to manage their negative emotions (see Compas, Conner-Smith, Saltzman, Thomsen, & Wadsworth 2001 for a review of the coping literature). Of note, research in this area highlights the importance of attentional control discussed earlier, suggesting that the ability to modulate attention in emotionally-arousing situations is essential to children's use of effective coping strategies such as distraction.

Physiological variables have also been studied in an effort to elucidate the biological underpinnings of emotion regulation and the modulation of

attention and arousal. In particular, research suggests that asymmetrical activation of the right frontal cerebral cortex is associated with behavioral inhibition and negative affect in young children, whereas asymmetrical activation of the left frontal lobe is associated with expressions of positive affect (Fox 1994). Additionally, the ability to problem-solve and to use cognitive coping strategies (e.g., cognitive restructuring) is dependent on effective frontal lobe functioning (e.g., Fox 1994). Other research suggests that the hypothalamic-pituitary-adrenocortical (HPA) axis, responsible for the production of cortisol, is essential to emotion regulation and attentional processes. Cortisol secretion is increased following exposure to a stressful stimulus and is important both in preparing an organism to respond to stress and later in facilitating a return to homeostatic functioning (de Kloet 1991; Stansbury & Gunnar 1994). Some research suggests that prolonged exposure to cortisol may lead to a depressive state (Wolkowitz et al. 1988) and excess cortisol secretion has been linked to internalizing problems in children, though findings in this area have been mixed (see Stansbury & Gunnar 1994 for a review). Finally, recent research on the physiological aspects of emotion regulation has focused on functioning of the vagus nerve. Low resting vagal tone has been correlated with behavioral problems across a number of studies and vagal functioning appears to be important to the regulation of attention and emotion (see Beauchaine 2001 for a review).

Deficits in skills central to emotional understanding (e.g., ability to recognize and label emotions, understanding the causes and consequences of emotion) also relate to children's behavioral problems. Findings suggest children with externalizing behavior problems are less accurate than nondisordered children at interpreting facial expressions of others (Lancelot & Nowicki 1997; Nowicki 1997; Zabel 1979) and have difficulty producing clear and interpretable facial displays of emotion (Burks, Carson, & Parke 1987; Buck 1975). Additionally, they show deficits with regard to recognizing and labeling internal emotional experiences as well as a lack of understanding of the causes and consequences of emotions (Cook, Greenberg, & Kusche 1994). Finally, children with difficulties characterized by aggression process social information differently from their peers, more often attributing angry or hostile motives to others (Crick & Dodge 1994; Schultz, Izard, & Ackerman 2000; Weiss, Dodge, Bates, & Pettit 1992).

The literature from nonclinical populations serves to underscore three important points regarding emotion regulation in children. First, variation in emotional experience and expression is likely reflective of differences in both child temperament and emotion regulation, though the precise nature of the relationship between these variables remains unclear. Research by Calkins

(Calkins et al. 1999) and Eisenberg (Eisenberg, Fabes, Guthrie et al. 1996) suggests that emotion dysregulation may be a more important determinant of behavior problems in children who demonstrate greater emotional reactivity. In particular, findings indicate that emotion regulation may serve as a protective factor in children with more difficult temperaments. Other research by Rubin and colleagues (Rubin, Coplan, Fox, & Calkins 1995) suggests that emotion dysregulation predicts problematic functioning, but that differences in temperament influence how these problems are manifested. In Rubin's study preschoolers were categorized as having an introverted or extroverted temperament based on observations of their play with peers. Maternal ratings of emotion regulation and emotional and behavioral problems were also obtained. Among the introverted preschoolers, those who were more emotionally dysregulated were rated as having more internalizing problems. In contrast, among the extroverted preschoolers, those who were more emotionally dysregulated had more externalizing problems.

Second, consistent with functionalist theory (e.g., Saarni et al. 1998), research has demonstrated that emotion regulation is socialized within the parent-child relationship. Parents who are supportive of their children's emotional expressions have children who are better regulated (see Eisenberg, Cumberland, & Spinard 1998 for a review). Parents also foster the development of healthy emotion regulation by limiting their children's exposure to distressing events and by providing teaching and feedback regarding emotional experience and expression (Halberstadt 1991; Thompson 1991). Conversely, mothers who are more controlling (Calkins, Smith, Gill, & Johnson 1998) or who punish or minimize their children's expressions of negative affect have children who are more likely to be emotionally dysregulated (Eisenberg, Cumberland, & Spinard 1998; Eisenberg, Fabes, & Murphy 1996; Eisenberg, Fabes, Shepard et al. 1999). Additionally, repeated exposure to intense emotional experiences (e.g., maternal depression, parental conflict) may overwhelm children's nascent regulatory capacities (Cole, Michel et al. 1994) and predispose them to experience distress in future emotionally-arousing situations (Cummings, Iannotti, & Zahn-Waxler 1985; Cummings, Zahn-Waxler, & Radke-Yarrow 1981; Saarni et al. 1998). Further, parents who teach their children to recognize and label emotions and who explain the causes and consequences of emotions have children who are better able to regulate their emotions (see Denham 1998 for a review).

Third, emotion dysregulation has been shown to predict problem behaviors over time. Unfortunately, the cross-sectional nature of many of the studies reported here prohibits drawing causal conclusions regarding the direction

of the relationship between emotion dysregulation regulation and behavior problems. Nonetheless, findings from the few longitudinal studies in this area suggests that emotion dysregulation is likely important to the development of child emotional and behavioral problems (e.g., Cole et al. 1996; Eisenberg, Fabes, Murphy et al. 1999; Gilliom et al. 2002).

Clinical samples

Though much less is known about the emotion regulation strategies of children with significant psychopathology, research in this area suggests that emotion dysregulation is a central feature of many childhood disorders. With regard to Oppositional Defiant Disorder (ODD), preliminary research indicates that these children demonstrate a bias for negative emotions, particularly anger and hostility. Casey (1996; Casey & Schlosser 1994) found that compared to nondisordered children, those with ODD were less attentive to positive social cues and expressed more hostility and negativity in interpersonal situations. Children with ODD were also more attentive to a staged conflict, more reactive to the conflict, and more likely to correctly remember details of the conflict after it had been resolved than were nondisordered children (Casey 1996). Additionally, Casey (1996) found that children with ODD exhibited deficits in emotional understanding, specifically the ability to understand the cause of their own emotional experience and to label their own and others' emotional expressions. Interestingly, children with ODD were more likely to mislabel others emotional expressions as negative, providing further evidence for a negative emotion bias in ODD (Casey 1996; Casey & Schlosser 1994).

Children with Attention Deficit/Hyperactivity Disorder (ADHD) also demonstrate regulatory difficulties, with particular deficits in social information processing and in encoding and decoding emotional expressions (Casey 1996; Moore, Hughes, & Robinson 1992). In particular, Moore, Hughes, and Robinson (1992) found that socially rejected hyperactive boys recalled fewer social cues in hypothetical interpersonal situations. Consistent with these findings, Casey (1996) found that children with ADHD observed their partners more than nondisordered children during a laboratory interaction, yet more often failed to respond in a contingent manner to their partner's behavior. Further, children with ADHD were also less accurate in labeling their own and others' emotional expressions (Casey 1996).

Research with depressed children has found, not surprisingly, that these children are more sad and angry relative to their peers (Blumberg & Izard

1985, 1986). Children with depression are also less emotionally expressive than nondisordered children and may more often fail to respond contingently during interpersonal interactions (Casey 1996). Research also suggests that depressed children and adolescents use different strategies for regulating emotion than do their nondepressed peers. One study (Garber, Braafladt, & Zeman 1991) compared the emotion regulation strategies nominated by depressed children and adolescents to those of their medically ill, nondepressed peers. In general, medically ill youths reported using distraction and problem-solving strategies more often than their depressed peers. In contrast, the depressed youths were more likely to report using avoidance and engaging in problematic behaviors to regulate their emotions. A related study (Garber, Braafladt, & Weiss 1995) examined the emotion regulation strategies endorsed by depressed children in both achievement-oriented and interpersonal situations. Findings indicated that depressed children, as compared to their nondepressed peers, reported using less support seeking and cognitive reframing in achievement-oriented situations. Further, in interpersonal situations, depressed girls reported less reliance on problem-solving strategies, and depressed boys endorsed responding with higher levels of anger. Although there has been less research attention devoted to other internalizing disorders, preliminary evidence suggests that children with anxiety disorders may show some emotional understanding deficits (e.g., understanding of inhibited emotional expression, understanding that one can alter one's emotional experience; Southam-Gerow & Kendall 2000). Additionally, children with significant social anxiety may have greater difficulty decoding nonverbal emotion cues (McClure & Nowicki 2001).

At-risk samples

Researchers have also studied the emotion regulation skills of children at-risk for psychological disorders. Populations studied in this research include children exposed to environmental stressors (e.g., parental depression, family violence) as well as children with an intrinsic or constitutional risk (e.g., temperamental vulnerabilities such as behavioral inhibition or attentional difficulties). Research in these areas is important given that children's "at-risk" status provides the opportunity to examine factors in emotional development that may either underlie the development of psychopathology or buffer the child, facilitating psychosocial adjustment. Further, research with at-risk children provides the opportunity to consider how emotion dysregulation may serve important protective or communicative functions for children while, at

the same time, increasing their short- and/or long-term risk for psychopathology. To illustrate these points, we will review research examining the impact of child maltreatment on children's emotional development and subsequent psychological functioning. Child maltreatment provides a good paradigm for examining these issues given that maltreated children are at significant risk for psychopathology (Erikson & Egeland 2002; Kolko 2002) and are developing within a family context characterized by several factors (e.g., high levels of negative emotionality, low levels of empathy and parental support) thought to interfere with healthy emotional development (Milner & Dopke 1997; Pianta, Egeland, & Erickson 1989).

Recent research has examined emotion regulation skills in children who have experienced familial maltreatment (i.e., physical abuse, sexual abuse, and/or neglect). Findings from this research have demonstrated that maltreated children, compared to nonmaltreated children, have more difficulty regulating their emotions throughout childhood. In the Strange Situation Task, maltreated infants display a decreased range of emotional expression as well as less flexibility and sensitivity to environmental events and changing social contexts (Gaensbauer 1982). These patterns continue into middle childhood such that maltreated children display more emotional dysregulation, including higher levels of emotional lability, fewer effective coping strategies, and greater difficulty with attentional control (Shields & Cicchetti 1997; Shields, Cicchetti, & Ryan 1994; Shipman & Zeman 2001; Shipman, Zeman, Penza, & Champion 2000). Further, findings suggest that emotion dysregulation may mediate the relation between child maltreatment and children's psychological difficulties (Shields & Cicchetti 1998).

Maltreated children also report different goals and expectancies with regard to emotion regulation. For example, one study examined children's goals for managing emotional expression (i.e., anger, sadness) with parents in a sample of sexually maltreated and nonmaltreated girls (Shipman, Zeman, Fitzgerald, & Swisher 2003). Findings indicated that maltreated girls reported inhibiting emotional displays to avoid interpersonal conflict as compared to nonmaltreated girls who reported displaying emotion to obtain support or assistance (Shipman et al. 2003). Several related studies have demonstrated that maltreated children expect less support and more conflict from parents in response to their displays of negative emotion and, in turn, they report dissembling emotional expression in the family context (Shipman & Zeman 2001; Shipman et al. 2000; Shipman, Edwards, Brown, Swisher, & Green-Turner 2003).

Maltreated children also show difficulties with regard to several emotional understanding skills that are essential to effective emotion regulation. Considerable research has demonstrated that maltreated children demonstrate difficulties producing clear facial expressions of emotion and accurately identifying the emotions of others (Camras, Sachs-Alter, & Ribordy 1996; Pollak, Cicchetti, Hornung, & Reed 2000). For example, in one study with physically abused children, Camras et al. (1988) found that the posed facial expressions of maltreated children, compared to those posed by nonmaltreated children, were more difficult for raters to interpret. Maltreated children also performed more poorly on a task requiring emotion identification skills in combination with understanding of the situational causes of emotion. Other studies have demonstrated that maltreated children are less likely to talk about emotion (Beeghly & Cicchetti 1994; Shipman & Zeman 1999), show less understanding of causes and consequences of emotion (Rogosch, Cicchetti, & Aber 1995; Shipman & Zeman 1999), and have difficulty taking an individual's prior experience into account when determining their emotional experience (Sachs-Alter 1993). Further, a recent study by Rogosch et al. (1995) highlights the importance of emotional understanding to psychological health by demonstrating that children's emotional understanding mediates the relation between maltreatment and subsequent behavioral problems and peer rejection.

Developing in a maltreating context may influence children's regulatory skills in several ways. In particular, maltreating parents are likely to model dysregulated responses to emotionally-arousing situations and, at the same time, are less likely to provide the scaffolding necessary to help their children learn effective regulatory strategies. Research demonstrates that maltreating parents, compared to nonmaltreating parents, show more frequent and intense negative affect, more emotional lability, and difficulty with impulse control. Further, in response to child distress, maltreating parents experience more physiological arousal and report less sympathy and more aversion toward the child (Frodi & Lamb 1980). Maltreating parents also respond more negatively to children's emotional displays (Shipman et al. 2001), engage in less discussion of emotion with their children (Cicchetti 1990; Shipman & Zeman 1999), and have difficulty identifying children's emotional experience (Camras et al. 1988). These emotion socialization practices may interfere with children's skill development by not providing them with the pragmatic assistance and sense of security important to children's emotional competence. These socialization experiences may also encourage maltreated children to develop 'atypical' strategies for managing emotion that are adaptive only in the maltreating context (Camras et al. 1996; Shipman et al. 2000). For example, when asked how they would

respond to others experiencing anger or sadness, maltreated children indicated that they would ignore the emotional display or remove themselves from the situation (e.g., "I wouldn't do nothing cuz it's their problem and I can't solve it."). In contrast, nonmaltreated children reported providing assistance or support ("I'd go over and help them feel un-mad.") (Shipman et al. 2000). More research is needed in this area, with particular attention to investigating whether (and under what circumstances) regulatory strategies learned to adapt to the maltreating context are generalized to other social contexts. Although research does demonstrate that maltreated children show regulatory difficulties across social contexts, preliminary evidence (Shipman et al. 2000; Smith & Walden 1999) suggests that they also show some flexibility in emotional responding that may help them to adapt within both maltreating and nonmaltreating environments (e.g., seeking support from peers and teachers but not parents).

Emotion regulation and psychopathology in adulthood

Nonclinical samples

Research on emotion dysregulation and psychopathology in adulthood has lagged behind the child literature, and most research in this area has been limited to delineating the relations between coping styles and psychological adjustment. Two broad categories of coping are identified in the adult literature: approach coping and disengagement coping (Herman-Stahl, Stemmler, & Peterson 1995; Soderstrom, Dolbier, Leiferman, & Steinhardt 2000; Wilkinson, Walford, & Espenes 2000). Approach coping is defined as acknowledging and accepting negative thoughts and emotional experiences and often involves the use of problem-solving strategies. In contrast, disengagement coping involves intentional efforts to suppress negative thoughts and emotions (e.g., denying the experience of thinking about a negative event or focusing attention away from the source of distress).

In general, findings demonstrate the utility of approach coping versus disengagement coping in decreasing emotional distress (Herman-Stahl, Stemmler, & Peterson 1995). However, subtypes of disengagement coping, including distraction and avoidance, are associated with differential outcomes. In particular, distraction (i.e., shifting one's attention to positive or neutral thoughts and activities) appears to be a skillful coping strategy that decreases the frequency of intrusive thoughts and is associated with lower levels

of emotional distress (Nolen-Hoeksema 1991; Nolen-Hoeksema, Morrow, & Fredrickson 1993). In fact, the use of distraction differentiates adults with depressed mood from adults without depressed mood (Wegner, Erber, & Zanakos 1993). In contrast, avoidance coping (i.e., efforts to suppress negative thoughts) has been consistently correlated with negative, intrusive thoughts and poorer physical and psychological health. For example, field studies of adult cancer patients demonstrated that higher levels of avoidance were associated with poorer disease status at a 1-year follow-up (Epping-Jordan, Compas, & Howell 1994). Additionally, research suggests that the intrusive thoughts associated with avoidance coping mediate the relation between exposure to traumatic events and emotional distress in adults (Baum 1990; Creamer, Burgess, & Pattison 1992). Thus, findings in this area have led researchers to conclude that efforts to avoid or suppress undesirable thoughts may cause a paradoxical increase in intrusive thoughts and emotional distress (Hayes, Wilson, Gifford, Follette, & Stroshal 1999).

Clinical samples

Though few studies have examined emotion dysregulation in adult clinical populations, indirect evidence for the importance of emotion dysregulation to adult psychopathology comes from research on depression and borderline personality disorder. Depressed adults report both high levels of negative affect, particularly sadness, and low levels of positive affect, suggestive of emotion regulation deficits (Shaver, Schwartz, Kirson, & O'Connor 1987; Watson et al. 1995). Current research is focused on identifying the emotion regulation strategies utilized by depressed adults to understand how these strategies contribute to the maintenance of depressive symptoms (Gross & Levenson 1997; Gross & Munoz 1995; Scott, Winters, & Beevers 2000). In particular, rumination (e.g., dwelling on various aspects of emotional distress) and avoidance (e.g., suppression of negative thoughts) exacerbate depression (Scott, Winters, & Beevers 2000; Nolen-Hoeksema 1991), leading to continued negative thoughts, depressed mood, and overall distress (Gross & Levenson 1997). In contrast, other coping strategies, such as distraction, appear to decrease negative emotion and symptoms of depression (Nolen-Hoeksema 1991; Nolen-Hoeksema, Morrow, & Fredrickson 1993). Further, findings indicate that depressed individuals use fewer effortful coping strategies (i.e., controlled regulation of instinctive responses to stress; Hartlage, Alloy, Vazquez, & Dykman 1993; Wegner 1994), making it difficult for depressed individuals to disrupt negative thought patterns and heighten positive emotional experiences.

Borderline Personality Disorder (BPD), which is characterized by intense, labile emotions and social and behavioral instability, has been conceptualized as a disorder of emotion dysregulation. Adults diagnosed with BPD tend to be “emotionally vulnerable” individuals who demonstrate automatic or involuntary responses to emotional events and have a lower threshold for emotional reactions as compared to non-BPD individuals (Linehan 1993). Linehan (1993) contends that invalidating environments during childhood may serve as catalysts for the development of emotion regulation difficulties. Specifically, an invalidating environment fails to teach tolerance of emotional distress, to offer skills necessary for the volitional control of emotional experiences, or to provide validation of positive and negative emotional expressiveness. As a result, the environment does not foster the internalization of adaptive emotion regulation skills and, instead, teaches the necessity of extreme emotional displays in order to elicit a response from the interpersonal environment. That is, the intense emotional experiences often observed among individuals with BPD may have been functional in their family of origin because they served to elicit help from unresponsive caregivers. Additionally, when a parent serves the role of both nurturer and abuser, as is often the case for children who develop BPD, emotional lability and a combination of approach and avoidance strategies may emerge as defenses for self-protection (Fischer et al. 1997). Although these behaviors may serve adaptive functions, they also create significant risk for psychological difficulties and problematic interpersonal relationships throughout childhood and adulthood.

Summary and future directions

Considerable recent attention has been directed toward understanding the role of emotion in psychosocial adjustment, with an emphasis on the relation between emotion dysregulation and psychopathology. The functionalist theory of emotion (Barrett & Campos 1987; Saarni et al. 1998) provides a foundation for this work by emphasizing the functional nature of emotion with regard to organizing goal-related behavior and facilitating both intrapersonal and interpersonal adjustment. Within this context, emotion regulation has been defined in relation to intrinsic or extrinsic processes for maintaining, evaluating and modifying emotional reactions in order to achieve individual goals and to adapt within the social environment (Thompson 1994). Emotion regulation includes a number of inter-related skills (e.g., management of emotional arousal and expressivity, interpretation of emotional expressions

of others, understanding of causes and consequences of emotion) that allow the individual to respond strategically in emotionally arousing situations. Further, effective emotion regulation requires coordination of several different but inter-related systems (e.g., physiological, behavioral, cognitive). Emotion dysregulation is defined in relation to normal regulatory processes that are operating in a dysfunctional manner (Cole, Michel et al. 1994). Dysregulated emotion may serve important protective or communicative functions but in a manner that interferes with short- or long-term adjustment. Patterns of emotion regulation that impair functioning may result in symptoms of psychopathology (e.g., emotional distress) or in adaptational failures in other areas of development (e.g., interpersonal relationships) that increase risk for psychological difficulties. Emotion dysregulation is associated with many, if not most, psychological disorders and serves as the central feature of several diagnostic categories.

Recent research provides strong support for relations between emotion dysregulation and children's psychological difficulties through work with non-clinical, clinical, and high-risk samples (e.g., Casey 1996; Eisenberg et al. 2001; Zeman et al. 2002; see also Southam-Gerow & Kendall 2002 for a review). Research with nonclinical samples has assessed several different aspects of emotion regulation (e.g., emotional expressivity, attentional control, coping, physiological arousal), with findings consistently indicating that children with behavioral problems demonstrate fewer adaptive emotion regulation skills and more emotion dysregulation. Similarly, children with behavior problems show deficits in emotional understanding skills (e.g., ability to identify other's emotions, understanding of causes and consequences) thought to be central to competent emotion regulation (Cook et al. 1994; Lancelot & Nowicki 1997). Although most of this research has been cross-sectional in nature, available longitudinal research suggests that early deficits in emotion regulation appear to place children at-risk for subsequent social and psychological difficulties (Gilliom et al. 2002). Research with clinical samples has been more limited but provides similar support for relations between emotion dysregulation and psychopathology. For example, findings indicate that depressed children and adolescents show higher levels of anger and sadness and are less effective at coping with emotionally-arousing situations (Blumberg & Izard 1985, 1986; Garber et al. 1991, 1995). Additionally, research with high-risk populations strongly suggests that emotion dysregulation is important to the development of psychopathology. For example, research has shown that the atypical socialization experiences of maltreated children interfere with the development of healthy emotion regulation (Shipman et al. 2000; Shipman & Zeman 1999) and

that difficulties with emotion regulation place these children at subsequent risk for psychopathology (Rogosch et al. 1995; Shields & Cicchetti 1998).

Research on emotion dysregulation in adult populations has generally focused on the relation between differential coping strategies and psychological distress. In particular, research suggests that distraction and approach coping are associated with better outcomes, whereas avoidance coping strategies, which may lead to intrusive thoughts and rumination, are associated with increased distress (Epping-Jordan et al. 1994). Research with clinical populations also suggests that avoidance and rumination strategies may be important to the etiology and maintenance of depression (Gross & Levenson 1997; Nolen-Hoeksema 1991). Further, current theory on the development of BPD indicates that a childhood characterized by parental invalidation may lead to deficits in the ability to manage emotions, resulting in psychological symptomatology and interpersonal difficulties.

Future research with nonclinical, clinical, and high-risk samples is needed to increase our understanding of the ways in which given patterns of emotion dysregulation may increase risk for certain types of psychopathology and how such patterns, once established, may function to maintain psychological symptomatology. Further, research in this area should examine the developmental trajectories of regulatory behavior and their implications for adjustment across the lifespan. More attention needs to be directed toward understanding how children's strategies for regulating emotion influence their ability to achieve central developmental tasks across domains of functioning (i.e., cognitive, social, physiological), elucidating both direct and indirect ways that regulatory behavior may influence psychological adjustment. In addition, given that most research has focused on children, more attention needs to be directed to examining emotion regulation and adjustment in adults. Normative research has demonstrated important changes in regulatory goals and strategies across adulthood. For example, with age, adults become increasingly selective in choosing smaller, more emotionally meaningful, interpersonal networks (Carstensen 1993, 1995). Emotionally satisfying relationships become a priority and serve to buffer the individual from negative emotions by providing access to coping resources and enhanced opportunity for positive emotional experiences (Carstensen, Gross, & Fung 1997; Diehl, Coyle, & Labouvie-Vief 1996). Further, older adults are more expressive (Malatesta-Magai, Jonas, Shepard, & Culver 1992) and are less likely to obey display rules consistent with social convention (Labouvie-Vief, DeVoe, & Bulka 1989). Research in this area will benefit from considering how changes in regulatory behavior across adulthood may impact adult adjustment.

Future research should also explore techniques for teaching clients adaptive emotion regulation strategies given that emotion dysregulation is a core diagnostic and clinical feature of most forms of child and adult psychopathology. Though a number of psychological interventions target clients' emotion regulation deficits (e.g., cognitive-behavioral therapy), available treatments are generally focused on clients' attributions, coping strategies, and problem-solving skills, neglecting more fundamental aspects of emotion regulation, such as attentional regulation and emotional understanding. An exception is Linehan's (1993) Dialectical Behavior Therapy (DBT) for BPD mentioned earlier in this chapter. This therapy teaches clients with BPD to modulate their attention to facilitate effective emotion regulation, to recognize and label their emotions, and to identify effective interpersonal goals. Empirical support for DBT suggests that this therapy is superior to more traditional approaches to the treatment of BPD (Koons et al. 2001; Linehan et al. 1999), and the proliferation and extension of this therapy into a number of diverse arenas (e.g., drug abuse, domestic violence) hints at the utility and clinical appeal of this approach. Further, research with at-risk children suggests that emotion regulation deficits, and related emotional understanding and encoding and decoding deficits, may place children at risk for subsequent psychopathology by hindering their ability to meet developmental milestones and to achieve their intrapersonal and interpersonal goals. Prevention programs designed to target children's emotion regulation deficits would be useful not only in the prevention of psychopathology, but also in increasing our understanding of developmental processes (e.g., parental socialization) that are important to the normative acquisition of these skills.

Acknowledgements

Kimberly Shipman, University of Georgia, Department of Psychology, Athens, GA 30602-3013, email, kshipman@uga.edu; phone, 706-542-1173. Preparation of this chapter was supported in part by a grant from the National Institute of Mental Health (IR03 MH60281-01A1) awarded to the first author.

References

- Barrett, K. & Campos, J. (1987). Perspectives on emotional development: II. A functionalist approach to emotions. In J. Olsofsky (Ed.), *Handbook of infant development* (2nd ed., pp. 555–578). New York: Wiley-Interscience.
- Baum, A. (1990). Stress, intrusive imagery, and chronic distress. *Health Psychology*, 9, 653–675.
- Beauchaine, T. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology*, 13, 183–214.
- Beeghly, M. & Cicchetti, D. (1994). Child maltreatment, attachment, and the self system: Emergence of an internal state lexicon in toddlers at high social risk. *Development and Psychopathology*, 6, 5–30.
- Blumberg, S. & Izard, C. (1985). Affect and cognitive characteristics of depression in 10- and 11-year-old children. *Journal of Personality and Social Psychology*, 49, 194–202.
- Blumberg, S. & Izard, C. (1986). Discriminating patterns of emotion in 10- and 11-year-old children's anxiety and depression. *Journal of Personality and Social Psychology*, 51, 852–857.
- Buck, R. (1975). Nonverbal communication of affect in children. *Journal of Personality and Social Psychology*, 31, 644–653.
- Burks, V. M., Carson, J. L., & Parke, R. D. (1987). Parent-child interactional styles of popular and rejected children Unpublished manuscript. University of Illinois, Urbana.
- Calkins, S. D., Gill, K. L., Johnson, M. C., & Smith, C. L. (1999). Emotional reactivity and emotional regulation strategies as predictors of social behavior with peers during toddlerhood. *Social Development*, 8, 310–334.
- Calkins, S. D., Smith, C. L., Gill, K. L., & Johnson, M. C. (1998). Maternal interactive style across contexts: relations to emotional, behavioral and physiological regulation during toddlerhood. *Social Development*, 7, 350–369.
- Campos, J., Mumme, D., Kermoian, R., & Campos, R. (1994). A functionalist perspective on the nature of emotion. *Monographs of the Society for Research in Child Development*, 59 (2–3, Serial No. 240), 284–304.
- Camras, L. A., Ribordy, S., Hill, J., Martino, S., Spaccarelli, S., & Stefani, R. (1988). Recognition and posing of emotional expressions by abused children and their mothers. *Developmental Psychology*, 24, 776–781.
- Camras, L., Sachs-Alter, E., & Ribordy, S. C. (1996). Emotion understanding in maltreated children: Recognition of facial expressions and integration with other emotion cues. In M. Lewis & M. Sullivan (Eds.), *Emotional development in atypical children* (pp. 203–225). Mahwah, NJ: Lawrence Erlbaum.
- Carstensen, L. L. (1993). Motivation for social contact across the life span: A theory of socioemotional selectivity. In J. Jacobs (Ed.), *Nebraska Symposium on Motivation*, 39 (pp. 209–254). Lincoln, NE: University of Nebraska Press.
- Carstensen, L. L. (1995). Evidence for a life-span theory of socioemotional selectivity. *Current Directions in Psychological Science*, 4, 151–156.

- Carstensen, L. L., Gross, J. J., & Fung, H. H. (1997). The Social Context of Emotional Experience. In K. W. Schaie & M. P. Lawton (Eds.), *Annual Review of Gerontology and Geriatrics*, Vol. 17 (pp. 325–352). New York: Springer Publishing Company.
- Casey, R. (1996). Emotional competence in children with externalizing and internalizing disorders. In M. Lewis & Sullivan, M. W. (Eds.), *Emotional development in atypical children* (pp. 161–183). Mahwah, NJ: Lawrence Erlbaum.
- Casey, R. & Schlosser, S. (1994). Emotional responses to peer praise in children with and without a diagnosed externalizing disorder. *Merrill-Palmer Quarterly*, 40, 60–81.
- Cicchetti, D. (1990). The organization and coherence of socioemotional, cognitive, and representational development: Illustrations through a developmental psychopathology perspective on Downs syndrome and child maltreatment. In R. Thompson (Ed.), *Socioemotional development: Nebraska Symposium on Motivation 1988* (pp. 259–367). Lincoln: University of Nebraska Press.
- Cole, P. M., Michel, M. K., & Teti, L. O. (1994). The development of emotion regulation and dysregulation: A clinical perspective. In N. Fox (Ed.), *The development of emotion regulation. Monographs of the Society of Research in Child Development*, 59 (2–3, Serial No. 240), 73–100.
- Cole, P. M., Zahn-Waxler, C., Fox, N. A., Usher, B. A., & Welsh, J. D. (1996). Individual differences in emotion regulations and behavior problems in preschool children. *Journal of Abnormal Psychology*, 105, 518–529.
- Cole, P. M., Zahn-Waxler, C., & Smith, K. D. (1994). Expressive control during a disappointment: Variations related to preschoolers' behavior problems. *Developmental Psychology*, 30, 835–846.
- Compas, B. E., Conner-Smith, J. K., Saltzman, H., Thomsen, A. H., & Wadsworth, M. E. (2001). Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. *Psychological Bulletin*, 127, 87–127.
- Cook, E. T., Greenberg, M. T., & Kusche, C. A. (1994). The relations between emotional understanding, intellectual functioning, and disruptive behavior problems in elementary-school-aged children. *Journal of Abnormal Child Psychology*, 22, 205–219.
- Creamer, M., Burgess, P., & Pattison, P. (1992). Reaction to trauma: A cognitive processing model. *Journal of Abnormal Psychology*, 101, 452–459.
- Crick, N. R. & Dodge, K. A. (1994). A review and reformulation of social information processing mechanisms in children's social adjustment. *Psychological Bulletin*, 115, 74–101.
- Cummings, E. M., Iannotti, R. J., & Zahn-Waxler, C. (1985). Influence of conflict between adults on the emotions and aggression of young children. *Developmental Psychology*, 21, 495–507.
- Cummings, E. M., Zahn-Waxler, C., & Radke-Yarrow, M. (1981). Young children's responses to expressions of anger and affection by others in the family. *Child Development*, 52, 1274–1282.
- Darwin, C. (1872). *The expression of emotions in man and animals*. London: John Murray.
- De Kloet, E. R. (1991). Brain corticosteroid receptor balance and homeostatic control. *Frontiers in Neuroendocrinology*, 12, 95–164.
- Denham, S. (1998). *Emotional development in young children*. New York: Guilford Press.

- Derryberry, D. & Rothbart, M. K. (1988). Arousal, affect and attention as components of temperament. *Journal of Personality and Social Psychology*, 55, 958–966.
- Diehl, M., Coyle, N., & Labouvie-Vief, G. (1996). Age and sex differences in strategies of coping and defense across the life span. *Psychology and Aging*, 11, 127–130.
- Eisenberg, N., Cumberland, A., & Spinrad, T. L. (1998). Parental socialization of emotion. *Psychological Inquiry*, 9, 241–273.
- Eisenberg, N., Cumberland, A., Spinrad, T. L., Fabes, R. A., Shepard, S. A., Reiser, M., et al. (2001). The relations of regulation and emotionality to children's externalizing and internalizing problem behavior. *Child Development*, 72, 1112–1134.
- Eisenberg, N., Fabes, R. A., Guthrie, I. K., Murphy, B. C., Maszk, P., Holmgren, R., et al. (1996). The relations of regulation and emotionality to problem behavior in elementary school children. *Development and Psychopathology*, 8, 141–162.
- Eisenberg, N., Fabes, R. A., & Murphy, B. C. (1996). Parents' reactions to children's negative emotions: Relations to children's social competence and comforting behavior. *Child Development*, 67, 2227–2247.
- Eisenberg, N., Fabes, R. A., Murphy, B. C., Maszk, P., Smith, M., & Karbon, M. (1995). The role of emotionality and regulation in children's social functioning: A longitudinal study. *Child Development*, 66, 1239–1261.
- Eisenberg, N., Fabes, R. A., Murphy, B. C., Shepard, S., Guthrie, I. K., Maszk, P., et al. (1999). Prediction of elementary school children's socially appropriate and problem behavior from anger reactions at age 4–6 years. *Journal of Applied Developmental Psychology*, 20, 119–142.
- Eisenberg, N., Fabes, R. A., Shepard, S. A., Guthrie, I. K., Murphy, B. C., & Reiser, M. (1999). Parental reactions to children's negative emotions: Longitudinal relations to quality of children's social functioning. *Child Development*, 70, 513–534.
- Eisenberg, N., Guthrie, I. K., Fabes, R. A., Shepard, S., Losoya, S., Murphy, B. C., et al. (2000). Prediction of elementary school children's externalizing problem behaviors from attentional and behavioral regulation and negative emotionality. *Child Development*, 71, 1367–1382.
- Epping-Jordan, J. E., Compas, B. E., & Howell, D. O. (1994). Predictors of cancer progression in young adult men and women: Avoidance, intrusive thoughts, and psychological symptoms. *Health Psychology*, 13, 539–547.
- Erikson, M. F. & Egeland, B. (2002). Child Neglect. In J. E. B. Myers, L. Berliner, J. Briere, C. T. Hendrix, C. Jenny, & T. A. Reid (Eds.), *The APSAC handbook on child maltreatment* (2nd ed., pp. 3–21). London: Sage Publications.
- Fischer, K. W., Ayoub, C., Singh, I., Noam, G., Maraganore, A., & Raya, P. (1997). Psychopathology as adaptive development along distinctive pathways. *Development and Psychopathology*, 9, 749–779.
- Fox, N. A. (1994). Dynamic cerebral processes underlying emotion regulation. In N. Fox (Ed.), *The development of emotion regulation. Monographs of the Society of Research in Child Development*, 59 (2–3, Serial No. 240), 152–166.
- Frodi, A. M. & Lamb, M. E. (1980). Child abusers' responses to infant smiles and cries. *Child Development*, 51, 238–241.

- Gaensbauer, T. (1982). Regulation of emotional expression in infants from two contrasting caretaking environments. *Journal of the American Academy of Child Psychiatry*, 21, 163–171.
- Garber, J., Braadfladt, N., & Weiss, B. (1995). Affect regulation in depressed and non-depressed children and adolescents. *Development and Psychopathology*, 7, 93–115.
- Garber, J., Braadfladt, N., & Zeman, J. (1991). The regulation of sad affect: An information processing perspective. In J. Garber & K. A. Dodge (Eds.), *The development of emotion regulation and dysregulation* (pp. 208–240). New York: Cambridge University Press.
- Gilliom, M., Shaw, D. S., Beck, J. E., Schonberg, M. A., & Lukon, J. L. (2002). Anger regulation in disadvantaged preschool boys: Strategies, antecedents, and the development of self-control. *Developmental Psychology*, 38, 222–235.
- Gross, J. J. & Levenson, R. W. (1997). Hiding Feelings: The acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology*, 106, 95–103.
- Gross, J. J. & Munoz, R. F. (1995). Emotion regulation and mental health. *Clinical Psychology: Science and Practice*, 2, 151–164.
- Halberstadt, A. (1991). Towards an ecology of expressiveness: Family expressiveness in particular and a model in general. In R. S. Feldman & B. Rime (Eds.), *Fundamentals in nonverbal behavior* (pp. 106–160). Cambridge: Cambridge University Press.
- Hart, D., Keller, M., Edelstein, W., & Hofmann, V. (1998). Childhood personality influences on social-cognitive development: A longitudinal study. *Journal of Personality and Social Psychology*, 74, 1278–1289.
- Hartlage, S., Alloy, L. B., Vazquez, C., & Dykman, B. (1993). Automatic and effortful processing in depression. *Psychological Bulletin*, 113, 247–278.
- Hayes, S. C., Wilson, K. G., Gifford, E. V., Follette, V. M., & Strosahl, K. (1996). Experiential avoidance and behavioral disorders: A functional dimensional approach to diagnosis and treatment. *Journal of Consulting and Clinical Psychology*, 64, 1152–1168.
- Herman-Stahl, M. A., Stemmler, M., & Petersen, A. C. (1995). Approach and avoidant coping: Implications for adolescent mental health. *Journal of Youth & Adolescent*, 24, 649–665.
- Johnson, M., Posner, M., & Rothbart, M. (1991). Components of visual orienting in early infancy: Contingent learning, anticipatory looking, and disengaging. *Journal of Cognitive Neuroscience*, 4, 335–344.
- Kagan, J., Snidman, N., Zenter, M., & Peterson, E. (1999). Infant temperament and anxious symptoms in school age children. *Development and Psychopathology*, 11, 209–224.
- * Keltner, D. & Gross, J. J. (1999). Functional accounts of emotion. *Cognition and Emotion*, 13, 467–480.
- Kolko, D. (2002). Child Physical Abuse. In J. E. B. Myers, L. Berliner, J. Briere, C. T. Hendrix, C. Jenny, & T. A. Reid (Eds.), *The APSAC handbook on child maltreatment* (2nd ed., pp. 21–54). London: Sage Publications.
- Koons, C., Robins, C. J., Tweed, J. L., Lynch, T., Gonzalez, A., Morse, J. Q., et al. (2001). Efficacy of dialectical behavior therapy in women veterans with borderline personality disorder. *Behavior Therapy*, 32, 371–390.
- Labouvie-Vief, G., DeVoe, M., & Bulka, D. (1989). Speaking about feelings: Conceptions of emotion across the life span. *Psychology & Aging*, 4, 425–437.

- Lancelot, C. & Nowicki S., Jr. (1997). The association between receptive nonverbal processing abilities and internalizing/externalizing problems in girls and boys. *Journal of Genetic Psychology*, 158, 297–302.
- Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. Guilford: New York.
- Linehan, M. M., Schmidt, H., Dimeff, L., Craft, J. C., Kanter, J., & Comtois, K. A. (1999). Dialectical behavior therapy for patients with borderline personality disorder and drug-dependence. *American Journal on Addictions*, 8, 279–292.
- Malatesta-Magai, C. Z., Jonas, R., Shepard, B., & Culver, C. (1992). Type A personality and emotional expressivity in younger and older adults. *Psychology and Aging*, 7, 551–561.
- McClure, E. B. & Nowicki, S., Jr. (2001). Associations between social anxiety and nonverbal processing skill in preadolescent boys and girls. *Journal of Nonverbal Behavior*, 25, 3–19.
- Moore, L. A., Hughes, J. N., & Robinson, M. (1992). A comparison of the social information-processing abilities of rejected and accepted hyperactive children. *Journal of Clinical Child Psychology*, 21, 123–131.
- Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology*, 100, 569–582.
- Nolen-Hoeksema, S., Morrow, J., & Fredrickson, B. L. (1993). Response styles and the duration of episodes of depressed mood. *Journal of Abnormal Psychology*, 102, 20–28.
- Nowicki, S., Jr. (1997). The relation of nonverbal processing ability of faces and voices and children's feelings of depression and competence. *Journal of Genetic Psychology*, 158, 357–363.
- Pianta, R., Egeland, B., & Erickson, M. (1989). The antecedents of maltreatment: Results of the Mother Child Interaction Project. In D. Cicchetti & V. Carlson (Eds.), *Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect* (pp. 203–253). Cambridge: Cambridge University Press.
- Pollak, S., Cicchetti, D., Hornung, K., & Reed, A. (2000). Recognizing emotion in faces: Developmental effects of child abuse and neglect. *Developmental Psychology*, 36, 679–688.
- Rogosch, F., Cicchetti, D., & Aber, J. L. (1995). The role of child maltreatment in early deviations in cognitive and affective processing abilities and later peer relationship problems. *Development and Psychopathology*, 7, 591–609.
- Rothbart, M. (1989). Temperament and development. In G. Kohnstamm, J. Bates, & M. Rothbart (Eds.), *Temperament in childhood* (pp. 187–247). New York: Wiley.
- Rothbart, M., Derryberry, D., & Posner, M. (1994). A psychobiological approach to the development of temperament. In J. E. Bates & T. D. Wachs (Eds.), *Temperament: Individual differences in biology and behavior* (pp. 83–116). Washington, DC: American Psychological Association.
- Rubin, K. H., Coplan, R. J., Fox, N. A., & Calkins, S. D. (1995). Emotionality, emotion regulation, and preschoolers' social adaptation. *Development and Psychopathology*, 7, 49–62.
- Saarni, C. (1999). *The development of emotional competence*. New York: Guilford Press.

- Saarni, C., Mumme, D., & Campos, J. (1998). Emotional development: Action, communication and understanding. In N. Eisenberg (Ed.), *Handbook of child psychology, vol. 3: Social, emotion, and personality development* (5th ed., pp. 237–309). New York: Wiley.
- Sachs-Alter, E. (1993). Maltreated and nonmaltreated children's use of cues in understanding the emotion of others. Unpublished doctoral dissertation, DePaul University Chicago, IL.
- Scott, W. D., Winters, R. W., & Beevers, C. G. (2000). Affective distress as a central and organizing symptom in depression: Psychological mechanisms. In S. L. Johnson, A. M. Hayes, T. M. Field, N. Schneiderman, & P. M. McCabe (Eds.), *Stress, coping, and depression* (pp. 145–175). New Jersey: Lawrence Erlbaum Associates.
- Shaver, P., Schwartz, J., Kirson, D., & O'Connor, C. (1987). Emotion knowledge: Further explorations of a prototype approach. *Journal of Personality & Social Psychology*, 52, 1061–1086.
- Shields, A. & Cicchetti, D. (1997). Emotion regulation among school-age children: The development and validation of a new criterion Q-sort scale. *Developmental Psychology*, 33, 906–916.
- Shields, A. & Cicchetti, D. (1998). Reactive aggression among maltreated children: The contributions of attention and emotion dysregulation. *Journal of Child Clinical Psychology*, 27, 381–395.
- Shields, A. M., Cicchetti, D., & Ryan, R. M. (1994). The development of emotional and behavioral self-regulation and social competence among maltreated school-age children. *Development and Psychopathology*, 6, 57–75.
- Shipman, K., Edwards, A., Brown, A., Swisher, L., & Green-Turner, E. (2003). *Development of children's emotion management skills: Implications of child neglect*. Manuscript submitted for publication.
- Shipman, K. & Zeman, J. (1999). Emotional understanding: A comparison of physically maltreating and nonmaltreating mother-child dyads. *Journal of Clinical Child Psychology*, 28, 407–417.
- Shipman, K. & Zeman, J. (2001). Socialization of children's emotion regulation in mother-child dyads: A developmental psychopathology perspective. *Development and Psychopathology*, 13, 317–336.
- Shipman, K., Zeman, J., Fitzgerald, M., & Swisher, L. (2003). Learning to manage emotion: A comparison of sexually maltreated and nonmaltreated girls. *Child Maltreatment*, 8, 163–172.
- Shipman, K., Zeman, J., Penza, S., & Champion, K. (2000). Emotion management skills in sexually maltreated and nonmaltreated girls: A developmental psychopathology perspective. *Development and Psychopathology*, 12, 47–62.
- Shultz, D., Izard, C. E., & Ackerman, B. P. (2000). Children's anger attribution bias: Relations to family environment and social adjustment. *Social Development*, 9, 284–301.
- Smith, M. & Walden, T. (1999). Understanding feelings and coping with emotional situations: A comparison of maltreated and nonmaltreated preschoolers. *Social Development*, 8, 93–116.

- Soderstrom, M., Dolbier, C., Leiferman, J., & Steinhardt, M. (2000). The relationship of hardiness, coping strategies, and perceived stress to symptoms of illness. *Journal of Behavioral Medicine*, 23, 311–328.
- Southam-Gerow, M. A. & Kendall, P. C. (2000). A preliminary study of the emotion understanding of youths referred for treatment of anxiety disorders. *Journal of Clinical Child Psychology*, 29, 319–327.
- Southam-Gerow, M. A. & Kendall, P. C. (2002). Emotion regulation and understanding: Implications for child psychopathology and therapy. *Clinical Psychology Review*, 22, 189–222.
- Stansbury, K. & Gunnar, M. R. (1994). Adrenocortical activity and emotion regulation. In N. Fox (Ed.), The development of emotion regulation. *Monographs of the Society of Research in Child Development*, 59 (2–3, Serial No. 240), 108–134.
- Thompson, R. (1994). Emotion regulation: A theme in search of a definition. In N. Fox (Ed.), The development of emotion regulation. *Monographs of the Society of Research in Child Development*, 59 (2–3, Serial No. 240), 25–52.
- Thompson, R. (1991). Emotional regulation and emotional development. *Educational Psychology Review*, 3, 269–307.
- Watson, D., Clark, L. A., Weber K., Assenheimer, J. S., Strauss, M. E., & McCormick, R. A. (1995). Testing a Tripartite Model: II. Exploring the Symptom Structure of Anxiety and Depression in Student, Adult, and Patient Samples. *Journal of Abnormal Psychology*, 104, 15–25.
- Wegner, D. M. (1994). Ironic processes of mental control. *Psychological Review*, 101, 34–52.
- Wegner, D. M., Erber, R., & Zanakos, S. (1993). Ironic processes in the mental control of mood and mood-related thought. *Journal of Personality and Social Psychology*, 65, 1093–1104.
- Weiss, B., Dodge, K. A., Bates, J. E., & Pettit, G. S. (1992). Some consequences of early harsh discipline: Child aggression and a maladaptive social information processing style. *Child Development*, 63, 1321–1335.
- Wilkinson, R. B., Walford, W. A., & Espenes, G. A. (2000). Coping styles and psychological health in adolescents and young adults: A comparison of moderator and main effects models. *Australian Journal of Psychology*, 52, 155–162.
- Wolfe & R. McMahon (Eds.), *Child abuse: New directions in prevention and treatment across the lifespan* (pp. 27–54). Thousand Oaks, CA: Sage.
- Wolkowitz, O. M., Breier, A., Doran, A., Rubinow, D., Berrettini, W., Coppola, R., et al. (1988). Prednisone-induced behavioral and biological changes in medically healthy volunteers. *Psychopharmacology Bulletin*, 24, 492–494.
- Zabel, R. (1979). Recognition of emotions in facial expressions by emotionally disturbed and nondisturbed children. *Psychology in the Schools*, 16, 119–126.
- Zeman, J., Shipman, K., & Suveg, C. (2002). Anger and sadness regulation: Predictions to internalizing and externalizing symptoms in children. *Journal of Clinical Child and Adolescent Psychology*, 31, 393–398.

CHAPTER 4

Neural substrates of conscious emotional experience

A cognitive-neuroscientific perspective

Richard D. Lane and Kateri McRae

Overview

This chapter addresses the neural substrates of emotion from the perspective of cognitive neuroscience. The guiding theme of this chapter, the distinction between implicit and explicit processes, is fundamental to cognitive neuroscience because these processes are mediated by distinct neural substrates. In the first section individual differences in the conscious experience of emotion are considered. To the extent that the course of cognitive development can be understood as the transformation from implicit to explicit representations, a cognitive-developmental framework can be used to understand individual differences in the conscious (explicit) processing of emotional information. The theory and empirical support for a psychological model based on cognitive-developmental principles called “levels of emotional awareness” is reviewed. In the second section, the neural substrates of emotional experience are considered. The neural substrates of attention to feelings, reflection upon feelings, background feelings and implicit emotion are considered. A hierarchical model is then presented that is parallel to the psychological model and that highlights the distinction between implicit and explicit processes. This model holds that the neural substrates of implicit emotional processes are unique to emotion whereas the neural substrates of explicit emotional processes are domain-general. An implication of the latter is that the conscious experience of emotion competes with other mental contents for conscious processing. In the third section, the implications of this model for self-regulation are reviewed. The conscious experience of emotion appears to involve participation of cortical structures that help to regulate vagal tone. As such, the ability to experience

one's own emotional responses in a complex and differentiated way may contribute to the ability to re-equilibrate following an emotional response in a way that is health-promoting. In the final section, the implications of this model for research on the neural substrates of conscious experience in healthy individuals and for clinical research on emotion dysregulation will be discussed.

A cognitive neuroscientific approach to emotional experience

Within the field of cognitive neuroscience a critical distinction is made between implicit and explicit processes. Implicit cognition includes procedures or habits that are expressed in the body or behavior in the absence of conscious awareness. Implicit processes have been demonstrated in a variety of cognitive domains, including memory, attention, perception, problem solving, etc. This body of research has led to a growing recognition that consciousness is the tip of the cognitive iceberg in the sense that most cognitive processing occurs outside of conscious awareness.

With the discovery of implicit cognitive processes the boundary between emotion and cognition has become blurred. Cognitive scientists have demonstrated that humans can make use of unconsciously processed stimuli, as in the case of masked priming effects, or can exhibit an appropriate skin conductance response to a previously presented object, even in the absence of conscious recognition of the object. These unconscious cognitive abilities extend into emotional processing, as evidenced by classical conditioning to emotional stimuli presented outside of explicit awareness of the stimuli.

A particularly poignant example is that of an amnesic patient in the care of French physician Edouard Claparede. This patient had severe anterograde amnesia, such that she was unable to recognize her physician if he stepped out of the room for a few minutes. On one such occasion when he reached out to shake her hand, she received a painful surprise from a pin in the palm of his hand. When he left the room and returned within minutes, she exchanged greetings with him as if it was their first meeting, but refused to shake his hand (LeDoux 1996). Such examples raise questions about whether one can make a meaningful distinction between implicit cognition and implicit emotion. A group of investigators including one of us (RDL) concluded recently that there is no way of clearly specifying how cognition differs from emotion (Lane, Nadel, Allen, & Kaszniak 2000a). To the extent that this is true, a cognitive neuroscientific approach to emotion is justified.

For those not steeped in the scientific study of emotion it may be difficult to understand the concept of implicit emotion if one equates emotion with feelings (conscious experiences). Yet, the initiation and implementation of an emotional response can clearly occur without the participation of conscious processing. Emotions involve evaluations of the extent to which goals are being met in interaction with the environment. As such, this evaluation process involves complex cognition that is typically not conscious. Implicit emotional responses consist of automatic visceral and somatic sensory and motor responses that do not involve conscious awareness. Autonomic responses are not unique to emotions – we know that they occur with cognitive processes such as attention, and, as noted above, skin conductance responses are used to demonstrate recognition in the absence of conscious awareness that it has occurred. Action tendencies, such as an inclination to approach or avoid, may occur outside of conscious awareness and can be thought of as enactive (procedural) or implicit representations of an emotional state. The conscious experience of emotion, or feeling, is a mental or cognitive state. Thus, when considering the full array of elements that contribute to and constitute an emotional response, it is reasonable to conclude that *there is nothing about emotion that is not cognitive*.

It should be added that one can differentiate emotion from other types of cognition, just as one can differentiate between different established areas of cognition, such as memory, language, attention and perception. The unique content of emotion may involve judgments of value as the individual interacts with the environment. Our position is that emotion should be considered one of these domains. This viewpoint is consistent with the fact that major chapters on emotion are included in the latest edition of textbooks in cognitive neuroscience (e.g. Gazzaniga, Ivry, & Mangun 2002).

If one accepts that it is impossible to distinguish between emotion and cognition in any formal sense, then it follows that *what applies to cognition in the traditional sense also applies to emotion*. To the extent that this is true, then the implicit vs. explicit distinction also applies to emotion. There are a number of implications of this conclusion. Let's turn to the implications for the study of conscious experience of emotion.

A cognitive-developmental approach to emotional experience

We have established that the distinction between implicit and explicit processes is important for emotion. A critical question in a discussion of emotional

experience is how implicit and explicit emotional processes are related to one another. Are they separate, distinct processes or are they related to one another in a more fundamental, integrated sense?

The concept of emotional intelligence (Salovey & Mayer 1990) may be useful in this context. People who are high in emotional intelligence are able to consciously use emotional information for successful social adaptation. They can, for example, anticipate how they or another person would feel if a particular course of action were taken. By contrast, lower emotional intelligence is characterized by impulsive behavior, an inability to delay gratification, a tendency for action rather than contemplation, and in general a behavioral style that involves less conscious processing of emotional information. Higher and lower emotional intelligence may be conceptualized as involving a relative emphasis on explicit and implicit processes, respectively. Furthermore, higher and lower emotional intelligence may be understood in a developmental sense, with the latter reflecting less advanced development relative to the former.

It is noteworthy, then, that the process of cognitive development has been conceptualized as consisting of the transformation of knowledge from implicit to explicit representations through a process called “representational redescription” (Karmiloff-Smith 1992). From this perspective, implicit emotion involves sensori-motor representations (e.g., action tendencies) whereas explicit emotion involves mental representations (e.g., feelings). The way we know something is a function of how we represent it. If one accepts the premise that emotion and cognition are indistinguishable, it therefore follows that the relationship between implicit and explicit emotion can be understood from a cognitive-developmental perspective, and that the way emotion is represented drives this developmental process.

Lane and Schwartz (1987) proposed that an individual’s ability to recognize and describe emotion in oneself and others, called emotional awareness, is a cognitive skill that undergoes a developmental process similar to that which Piaget described for cognition in general. A fundamental tenet of this model is that individual differences in emotional awareness reflect variations in the degree of differentiation and integration of the schemata (implicit programs or sets of rules) used to process emotional information, whether that information comes from the external world or the internal world through introspection.

The model posits five “levels of emotional awareness” that share the structural characteristics of Piaget’s stages of cognitive development (Piaget 1937). The five levels of emotional awareness in ascending order are awareness of physical sensations, action tendencies, single emotions, blends of emotion, and

blends of blends of emotional experience (the capacity to appreciate complexity in the experiences of self and other).

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

The five levels therefore describe the cognitive organization of emotional experience. They describe traits, although they may also be used to describe states. The levels are hierarchically related in that functioning at each level adds to and modifies the function of previous levels but does not eliminate them. For example, blends of emotion (Level 4 experiences) should be associated with more differentiated representations of somatic sensations (Level 1) than action tendencies (Level 2 experiences). The feelings associated with a given emotional response can be thought of as a construction consisting of each of the levels of awareness up to and including the highest level attained.

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

The Levels of Emotional Awareness Scale (LEAS): Psychometric findings

The LEAS is a written performance measure that asks a person to describe his or her anticipated feelings and those of another person in each of twenty scenes described in two to four sentences (Lane, Quinlan, Schwartz, Walker, & Zeitlin 1990). Scoring is based on specific structural criteria aimed at determining the

degree of differentiation in the use of emotion words (the degree of specificity in the terms used and the range of emotions described) and the differentiation of self from other. The scoring involves essentially no inference by raters. Because the scoring system evaluates the structure of experience and not its content, individuals cannot modify their responses to enhance their score, as is the case with some self-report instruments. A glossary of words at each level was created to guide scoring.

Each the 20 scenes receives a score of 0 to 5 corresponding to the cognitive-developmental theory of emotional awareness that underlies the LEAS (Lane & Schwartz 1987). A score of 0 is assigned when non-affective words are used, or when the word "feel" is used to describe a thought rather than a feeling. A score of 1 is assigned when words indicating physiological cues are used in the description of feelings (e.g. "I'd feel tired"). A score of 2 is assigned when words are used that convey relatively undifferentiated emotion (e.g. "I'd feel bad"), or when the word "feel" is used to convey an action tendency (e.g., "I'd feel like punching the wall"). A score of 3 is assigned when one word conveying a typical, differentiated emotion is used (e.g. happy, sad, angry, etc.). A score of 4 is assigned when two or more Level 3 words are used in a way that conveys greater emotional differentiation than would either word alone. Respondents receive a separate score for the "self" response and for the "other" response ranging from 0 to 4. In addition, a total LEAS score is given to each scene equal to the higher of the self and other scores. A score of 5 is assigned to the total when self and other each receive a score of 4 and are differentiated from one another; thus, a maximum total LEAS score of 100 is possible.

To date eight separate psychometric studies have been conducted with the LEAS. The first study in Yale undergraduates ($n = 94$) enabled us to examine the reliability of the LEAS and its correlation with other psychological tests (Lane et al. 1990). The second study involved students at Chicago Medical School (CMS) ($n = 57$) and focused on the correlation with the Levy Chimeric Faces Test (Lane, Kevley, DuBois, Shamasundara, & Schwartz 1995). The third study in Arizona and Minnesota ($n = 385$) established norms for the scale (Lane, Sechrest, Reidel, Weldon, Kaszniak, & Schwartz 1996). A fourth study with University of Arizona undergraduates ($n = 215$) involved additional psychometric and psychophysiologic assessments. The fifth and sixth studies have been conducted in collaboration with Dr. Lisa Feldman Barrett at Boston College. In addition, two international studies have been conducted: a study of 331 German students (Wrana, Thomas, Heindichs, Huber, Obliers, Koerfer, & Köhle 2000) and a Canadian study of 30 subjects with borderline personality

disorder and 40 control subjects (Levine, Marziali, & Hood 1997). The findings from these studies are selectively reviewed below.

The LEAS has consistently been shown to have high inter-rater reliability and internal consistency (Lane, Reiman, Axelrod, Yun, Holmes, & Schwartz 1998). The test-retest reliability at 2 weeks has been shown to be quite good ($n = 135$, Spearman Brown $r = .67$, $p < 0.001$). Norms for age, sex and socioeconomic status have been established based on the study completed in Arizona and Minnesota.

In the Yale study we administered two instruments which, like the LEAS, are cognitive-developmental measures based on Piaget's model: the Sentence Completion Test of Ego Development by Loevinger (Loevinger & Wessler 1970; Loevinger, Wessler, & Redmore 1970) and the cognitive complexity of the description of parents by Blatt and colleagues (Blatt, Wein, Chevron, & Quinlan 1979). The LEAS correlated moderately ($r = .37$ and $r = .36$, respectively) and significantly ($p < 0.01$) in the predicted direction in both cases. These results support the claim that the LEAS is measuring a cognitive-developmental continuum and that the LEAS is not identical to these other measures.

A key question is whether the LEAS is simply another measure of verbal ability. In the Yale sample the LEAS correlated $r = .38$ ($p < 0.001$) with the vocabulary subtest of the WAIS. In the CMS study the LEAS correlated $r = .17$ (NS) with the Shipley Institute of Living Scale (Shipley 1940), a multiple choice measure of verbal ability. These data suggest that verbal ability may contribute to LEAS performance. However, several studies have now been conducted demonstrating that when verbal ability is controlled significant effects are still observed. For example, LEAS scores in men and women could be compared in all eight studies. In three of these studies measures of verbal ability, including the WAIS vocabulary subtest and the Shipley Institute of Living Scale, were also obtained. In each study women scored higher than men on the LEAS ($p < 0.01$), even when controlling for verbal ability ($p < 0.05$) (Barrett, Lane, Sechrest, & Schwartz 2000). Thus, the finding that women score higher than men on the LEAS is a highly stable and generalizable finding.

Lisa Feldman Barrett administered the LEAS and the Weinberger Adjustment Inventory to 63 subjects at Penn State and 55 subjects at Boston College. In both samples the LEAS correlated significantly ($p < 0.05$, two-tailed) with self-restraint, one of three superordinate dimensions of the scale. The LEAS also correlated significantly with impulse control, ($r = .35$, $p < 0.01$, two-tailed and $r = .30$, $p < 0.05$, two-tailed), a component of self-restraint that involves the tendency to think before acting. Self-restraint refers directly to suppression of egoistic desires in the interest of long-term goals and relations with oth-

ers. This replication in independent samples indicates that greater emotional awareness is associated with greater self-reported impulse control, and is consistent with the theory that functioning at higher levels of emotional awareness (Levels 3–5) modulates function at lower levels (actions and action tendencies at Level 2).

A key assumption in this work on emotional awareness is that language promotes the development of schemata for the processing of emotional information, whether that information comes from the internal or external world. Once the schemata are established, they should affect the processing of emotional information whether the information is verbal or nonverbal. Thus, the LEAS should correlate with the ability to recognize and categorize external emotional stimuli. Furthermore, this correlation should hold whether the external stimulus and the response are purely verbal or purely non-verbal.

These hypotheses were tested in the Norms study by use of the Perception of Affect Task (PAT), a set of four emotion recognition tasks (35 items each) developed by Jim Rau and Alfred Kaszniak at the University of Arizona (Rau 1993). The first subtask consists of stimuli describing an emotional situation without the use of emotion words. For example, “The man looked at the photograph of his recently departed wife.” The response involves choosing one from an array of seven terms (happy, sad, angry, afraid, disgust, neutral, surprise) to identify how the person in question was feeling. The fourth subtask is purely nonverbal. The stimuli consist of photographs of faces developed by Ekman (1982), each of which depicts an individual emotion. The response consists of selecting one from an array of seven photographs depicting emotional scenes without faces (e.g. two people standing arm-in-arm by a grave with their backs to the camera). The other two subtasks involved a verbal stimulus (sentence) and a non-verbal response (from an array of seven faces) and a nonverbal stimulus (face) and a verbal response (from an array of seven words).

Across the entire scale, the correlation between the LEAS and the PAT was high ($r = .43$, $n = 385$, $p < 0.001$), accounting for about 18% of the variance. Furthermore, significant correlations were observed between the LEAS and each of the PAT subtasks. When dividing the sample into upper (high), middle and lower thirds on the LEAS, the high LEAS subjects scored higher on each of the PAT subtasks than the low LEAS subjects. Thus, high LEAS scores were associated with better emotion recognition no matter whether the task was purely verbal or purely nonverbal (Lane, Sechrest, Riedel, Weldon, Kaszniak, & Schwartz 1996). Furthermore, when combining results for each of the seven emotion categories across the four subtasks (there were five stimuli of each emotion type in each subtask), the same findings for

high, moderate and low LEAS subjects were observed (Lane, Sechrest, Riedel, Shapiro, & Kaszniak 2000). These findings support the claim that the LEAS is: (a) a measure of the cognitive-developmental schemata used to process emotional information, whether the information is verbal or nonverbal; (b) a measure of the complexity of experience and; (c) not merely a measure of verbal ability.

Evidence for the discriminant validity of the LEAS is provided by data from the Norms study and the Arizona undergraduate study. In both studies ($n = 385$ and $n = 215$, respectively) the Affect Intensity Measure (Larsen & Diener 1987), a trait measure of the tendency to experience emotions intensely, did not correlate significantly with the LEAS despite the large sample sizes. Thus, inadequate statistical power cannot explain the lack of correlation. The LEAS also does not correlate significantly with measures of negative affect, such as the Taylor Manifest Anxiety Scale and the Beck Depression Inventory. These results are consistent with the view that the LEAS measures the structure or complexity and not the intensity or valence of affective experience.

Neural correlates of emotional awareness

A systematic survey of the neural correlates of each of the levels of emotional awareness has not yet been undertaken, either in between-subjects or within-subjects designs. Nevertheless, it is still possible to consider certain aspects of the conscious experience of emotion. In this section four different aspects of the conscious experience of emotion will be considered: focal attention to feelings, reflective awareness, background feelings and implicit emotion. This sequence corresponds to the chronological order of findings in our laboratory for the first three topics and our attempts to integrate these findings with those of other investigators, particularly with regard to the latter two topics.

A. Focal attention to feelings

To explore the underlying functional neuroanatomy of emotional awareness, we administered the LEAS to participants in a positron emission tomography (PET) study of emotion (Lane, Reiman, Axelrod, Yun, Holmes, & Schwartz 1998). They included twelve right-handed female volunteers who were free of medical, neurological, or psychiatric abnormalities. The LEAS and other psychometric instruments were completed prior to PET imaging. Happiness, sadness, disgust, and three neutral control conditions were induced by film

and recall of personal experiences (12 conditions). Twelve PET images of blood flow were obtained in each person using the ECAT 951/31 scanner (Siemens, Knoxville, TN), 40 mCi intravenous bolus injections of ^{15}O -water, a 15 second uptake period, 60 second scans, and an interscan interval of 10 minutes.

The recall paradigm imposed constraints that emphasized the attention component of the stimulus conditions. Specifically, recalled experiences of happiness, sadness and disgust were prepared in advance by asking subjects to select a memory of each type and to focus on an aspect of the memory in which the target emotion was particularly intense. At the time of imaging, we asked subjects to focus on feeling the target emotion as they recalled the scene in question. In order to make the film and recall conditions similar, prior to each film clip we told the subjects what emotion was being targeted by the film clip and asked them to concentrate on feeling that emotion. As a result, the subjects were intentionally focusing their attention on the experience of each emotion in both the recall and film conditions.

To examine neural activity attributable to emotion generally, rather than to specific emotions, one can subtract the three neutral conditions from the three emotion conditions in a given stimulus modality (film or recall). This difference, which can be calculated separately for the six film and six recall conditions, identifies regions of the brain where blood flow changes specifically attributable to emotion occur. These blood flow changes, which indicate neural activity in that region, can then be correlated with the LEAS scores to identify regions of the brain that are associated with emotional awareness during emotional arousal.

Findings from this covariate analysis revealed one cluster for film-induced emotion with a maximum located in the right mid-cingulate cortex (Brodmann area – BA – 23; coordinates of maximum = $[16, -18, 32]$; $z = 3.40$; $p < 0.001$ uncorrected). For recall-induced emotion, the most statistically significant cluster was located in the right anterior cingulate cortex (BA 24; coordinates of maximum = $[16, 6, 30]$; $z = 2.82$; $p < 0.005$ uncorrected). An analysis was then performed to identify areas of significant overlap between the two covariance analyses. With a height threshold of $z = 3.09$, $p < 0.001$, and an extent threshold of five voxels, a single cluster was observed in the right anterior cingulate cortex (BA 24) maximal at $[14, 6, 30]$.

Traditionally the anterior cingulate cortex has been thought to have a primarily affective function (Papez 1937; Vogt, Finch, & Olson 1992). However, in addition to emotion, it is now recognized to play important roles in attention, pain, response selection, maternal behavior, vocalization, skeletomotor function, and autonomic control (Vogt & Gabriel 1993). The multiple functions of

the anterior cingulate cortex no doubt contribute to the significant changes in activation that have been observed in a variety of studies. How can these different functions be reconciled with the present findings involving emotional awareness?

One answer might be that these various functions of the anterior cingulate cortex may reflect its superordinate role in executive control of attention and motor responses (Lane et al. 1998). According to this view, emotion, pain or other salient exteroceptive or interoceptive stimuli provide moment-to-moment guidance regarding the most suitable allocation of attentional resources for the purpose of optimizing motor responses in interaction with the environment. The conscious experience of emotion could occur concomitantly and automatically as attention gets redirected by emotion. As such, a role for the anterior cingulate cortex in the conscious experience of emotion fits well with its other functions, but suggests that this role is not exclusive to emotion. To the extent that people who are more emotionally aware attend more to internal and external emotion cues, the cognitive processing of this information can contribute to ongoing emotional development. This is so because attention to an emotional experience necessarily precedes and facilitates the description of that experience using language or other representation mode, which in turn will contribute to the further elaboration and refinement of the schemata used for processing emotional information in the future. The dorsal ACC may have been activated because attention was intentionally focused on the experience of the emotion in question. An almost identical area of ACC was implicated in emotional experience in a study by Rainville and colleagues (1997). The study involved manipulating the unpleasantness of a painful stimulus by hypnotic suggestion. As a result of the hypnotic suggestion the subjects focused conscious attention on the unpleasantness of the painful stimulus. As such, these studies together indicate that this area of ACC may be involved in that aspect of phenomenal awareness involved in focused attention on emotional experience.

B. Reflective awareness

Within the field of consciousness research a distinction is made between phenomenal and reflective awareness. Phenomenal awareness refers to the actual content of consciousness (focal attention to feelings and background feelings) whereas reflective awareness involves attending to or performing a cognitive operation upon the contents of conscious experience (Farthing 1992). Reflective awareness, or metacognition, requires the creation of a representation of

experience, and this representation will affect how future emotional information is interpreted and experienced.

Subsequent to the study described above we examined the pattern of neural activation associated with attending to one's own emotional experience (Lane, Fink, Chua, & Dolan 1997). To confirm that subjects were allocating their attention as we instructed, we had them indicate on a keypad how each emotion-evoking picture made them feel. In essence, we were examining an aspect of conscious experience involving commentary on that experience (Weiskrantz 2000). By having subjects attend to and label their experience in this next study, we were studying reflective awareness.

We studied ten healthy men as they viewed twelve picture sets, each consisting of pleasant, unpleasant and neutral pictures from the International Affective Picture System (Lang, Bradley, & Cuthbert 1995). Pictures were presented for 500 msec every 3.0 seconds. Twelve PET-derived measures of cerebral blood flow were obtained in each subject, one for each picture set. During half the scans subjects attended to their emotional experience (indicating on a keypad whether the picture evoked a pleasant, unpleasant or neutral feeling); during the other half they attended to spatial location (indicating whether the scene depicted was indoors, outdoors, or indeterminate). Across subjects, picture sets were counterbalanced across the two attention conditions.

During attention to subjective emotional responses increased neural activity was elicited in rostral anterior cingulate cortex (BA 32) and medial prefrontal cortex (coordinates: 0,50,16; $Z = 6.74$, $p < 0.001$, corrected), right temporal pole, insula and ventral cingulate cortex (all $p < 0.001$, corrected). Under the same stimulus conditions when subjects attended to spatial aspects of the picture sets activation was observed in parieto-occipital cortex bilaterally ($Z = 5.71$, $p < 0.001$, corrected), a region known to participate in the evaluation of spatial relationships.

In that PET study all of the scan conditions included pleasant, unpleasant and neutral pictures ("mixed" picture sets). We sought to replicate those findings in the functional magnetic resonance imaging (fMRI) environment and determine the relative influence of pleasant, unpleasant and neutral stimuli on the observed activations. Subjects included 10 healthy men and women. Relative to the mixed external attention condition, dorsal medial prefrontal cortex activation was observed during the mixed, pleasant, and neutral internal attention conditions; attention to unpleasant pictures revealed activity that was slightly more posterior in anterior cingulate cortex. In addition, posterior parieto-occipital cortex was activated bilaterally during the external relative to the internal focus conditions. Findings from our previous PET study were

therefore replicated and extended in this fMRI paradigm. Indeed these same fMRI findings, activation of dorsomedial prefrontal cortex during attention to and labeling of emotional experience and activation of parieto-occipital areas during the evaluation of spatial relationships, have been replicated in another independent laboratory (Gusnard, Akbudak, Shulman, & Raichle 2001).

Frith and Frith (1999) hypothesize that the ability to mentalize evolved from the action system for the purpose of identifying the intentions of conspecifics and anticipating their future actions. We know that emotional states may fundamentally consist of action tendencies (Frijda 1986), which can be construed as equivalent to the intentions of the self. This dovetails beautifully with the Piagetian perspective on cognitive development in that all mental representations at the conceptual level are fundamentally derived from action schemes. It is therefore reasonable to consider, as a first approximation, that the paracingulate sulcus, which includes the dorsomedial prefrontal cortex, is a substructure within the prefrontal cortex that participates in establishing the representations of mental states (including emotional states) of both self and other. *As such, another aspect of conscious emotional experience, the ability to reflect upon one's own emotional state, requires participation of a structure that serves a more general function that is not specific to emotion.*

This conclusion is supported by several recent findings. Sterling Johnson and colleagues (Johnson, Baxter, Wilder, Pipe, Heiserman, & Prigatano 2002) demonstrated activation of this region during self-reflective thought (considering whether statements or attributes pertain to the self). Vogeley and associates (Vogeley, Bussfeld, Newen, Herrmann, Falkai, Maier, Shah, Fink, & Zilles 2001) have observed activation of this region during the process of evaluating what someone else is thinking (theory of mind), as have others previously (e.g. Happe et al. 1996).

Recently, investigators have attempted to isolate the effects of attention to one's own emotional state that is necessary to rate one's emotional reaction to a stimulus. Passive viewing of emotional stimuli has been found to activate the amygdala, hippocampus, putamen, cerebellum and lingual gyrus (Lange et al. 2003) while an emotional rating task did not activate the amygdala or hippocampus and, in addition, activated the left ventral frontal gyrus. Another study found activation of anterior cingulate, dorsomedial prefrontal cortex and cerebellum when subjects were asked to rate emotional images but found greater activity in the right amygdala, bilateral insula and other areas during passive viewing (Taylor et al. 2003). These findings are consistent with findings that amygdala activity is modulated by cognitive tasks such as labeling stimuli (Hariri et al. 2003).

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

C. Background feelings

The fMRI study from our laboratory that was just described generated another important set of findings. In an event-related analysis based on keypad responses (independent of condition), the pleasant-minus-neutral and the unpleasant-minus-neutral comparisons yielded activation of a region that included ventromedial prefrontal cortex and ventral anterior cingulate cortex but not dorsomedial prefrontal cortex.

For both the emotion and neutral conditions, the subjects were attending to how they felt and labeling the feeling (or its absence) with a keypad label. As such these elements canceled one another out in the subtraction analysis. What remains from this subtraction is the conscious experience of emotion that is not attended to or labeled. This aspect of consciousness that is not in focal awareness may be akin to what Damasio refers to as “background feelings” (Damasio 2003) or what Farthing (1992) refers to as “peripheral awareness.” Background feelings are bodily states that color conscious experience but are not noticed as such unless attended to. They are generated by internal regulatory functions as well as external stimuli and provide information about one’s current state of well-being. Farthing refers to peripheral awareness as those mental contents that are on the periphery of focal awareness. They include stimuli that are being processed automatically while focal attention is directed elsewhere, as well as events that have been in focal awareness recently. It is our

intention to include in this concept both undifferentiated (e.g. feeling lousy) and differentiated (e.g. feeling sad) emotional states that are situated on the periphery of focal awareness, are part of conscious experience, and are easily attended to if necessary.

We speculate that a number of paralimbic and neocortical areas participate in background feelings of this type. The ventral anterior cingulate cortex is an area known to participate in visceral regulation. Bechara and colleagues (1997) have shown that the nearby ventromedial prefrontal cortex biases behavior and decision-making without conscious awareness of an emotional state necessarily occurring, although the somatic marker hypothesis (Damasio 1994) clearly implicates it in some type of conscious bodily experience. The so-called "affective division" of the anterior cingulate cortex (ACC) has dense inputs from amygdala and other subcortical structures and may well participate in background feelings. This interpretation is consistent with the findings of Bush and colleagues (1998) involving the Emotional Counting Stroop, in which the rostral anterior cingulate cortex was activated during a foreground cognitive task while background emotional stimuli served as distractors. Similarly, Blakemore and colleagues (1998) showed that this region was activated by a ticklish sensation induced by someone else compared to that induced by oneself – again the pleasant feeling of being tickled is revealed by a subtraction that removes attention to the feeling. In a study by Lévesque and colleagues (2003) in which subjects viewed sad film clips, self-reported sadness correlated with activity in bilateral ventrolateral prefrontal cortex, the affective division of the anterior cingulate cortex and the left insula. Since the subjects in that study retrospectively rated their sadness after viewing of the film clips was completed, whereas imaging was performed during the actual viewing of the film clips, the sadness that subjects reported may have been in the background of their conscious awareness during imaging.

The insula is another cortical area that is thought to contribute to the representation of one's own bodily state (Augustine 1996). The insula contains a topographic representation of inputs from visceral, olfactory, gustatory, visual, auditory and somatosensory areas and is proposed to integrate representations of external sensory experience and internal somatic state (Mesulam & Mufson 1982). The insula has been found to be activated in a number of studies of emotional processing (Phillips et al. 1997), especially those involving emotion that is induced by imagery or recall (Reiman et al. 1997), and emotional tasks that have a concurrent cognitive demand (Phan, Wager, Taylor, & Liberzon 2002). Consistent with a role in background emotion, the insula was recently found to be more engaged when emotional stimuli were passively viewed than when

attention was directed at one's own emotional state (Hariri, Mattay, Tessitore, Fera, & Weinberger 2003).

The right parietal cortex has also been implicated broadly while subjects engage in a task that involves emotional stimuli that are not necessarily the focus of attention. For example, the right parietal cortex has been found to be activated preferentially to emotional stimuli during a gender decision task (Iidaka et al. 2001). Somatosensory cortex plays a major role in the mapping of bodily sensations and thus contributes to background feelings as well (Damasio 1994; Damasio 2003).

D. Implicit emotion

Up to this point we have considered the neural substrates of different aspects of conscious emotional experience. In order to complete our neural model of emotional awareness, we must consider the neural substrates of implicit emotional processes. In this context, it is important to recall that the distinction between implicit and explicit processes is well established in cognitive neuroscience because *there are distinct neural substrates of implicit and explicit processes*. For example, explicit memory for facts and events requires participation of medial temporal lobe structures (such as the hippocampus) and diencephalon, whereas implicit memory requires structures such as the striatum (skills and habits), neocortex (priming), amygdala and cerebellum (classical conditioning) and reflex pathways (non-associative learning). Given the evidence presented above that the implicit-explicit distinction applies to emotion, it follows from this general principle that *there are distinct neural substrates of implicit and explicit emotional processes*. What might these neural substrates be?

One clue may be derived from the concept of modular processing. Ladavas and colleagues (Ladavas Cimatti, del Pesce, & Tuoizzi 1993) have shown that the presence or absence of emotional content can be accurately detected in subliminally presented pictures. Drawing on Fodor's concept of modularity (Fodor 1983), these researchers suggest that these and other findings support a critical distinction between modular and non-modular emotion information processing. Modules are domain-specific computational devices that are cognitively impenetrable and generate shallow output. Cognitive penetrability (or informational encapsulation) refers to the degree to which information processing can be influenced by prior knowledge, expectations, beliefs or other cognitive input. Ladavas et al. (1993) argue, based on their own work and that of others, that the automatic, unconscious process of emotion-generation is cognitively impenetrable. The output of this module is crude and diffuse,

but becomes the substrate for further cognitive elaboration, which is highly cognitively penetrable.

Such a module seems logical when considering the evolutionary significance of emotional responses – when faced with an unpredictable, dangerous environment, our evolutionary ancestors relied on an automatic and accurate system of identifying maleficent and beneficial elements of the environment and quickly forming the appropriate actions in response. In a recent review by Öhman and Mineka (2001), an evolutionarily defined fear module is proposed, citing evidence that the fear response is elicited by stimuli of evolutionary significance, these responses are elicited automatically, they are free from the influence of cognitive expectancy or instruction, and they find a neuroanatomical substrate in the amygdala (Öhman & Mineka 2001).

There is evidence from brain imaging studies in recent years that amygdala activation can occur in response to emotional stimuli in the absence of conscious awareness of the stimulus. This is consistent with the notion that the amygdala performs its functions implicitly and plays a key role in establishing whether exteroceptive stimuli have an emotional meaning. The concept of cognitive penetrability fits well here also. Certain autonomic responses that are known to be modulated by the amygdala, such as the skin conductance response (Mangina & Mangina 1996), are unaffected by information that brings the possibility of shock associated with negative stimuli to zero. In other words, conditioned responses to negative stimuli persist despite full access to the knowledge that the contingency has been dissolved (Hugdahl & Öhman 1977).

LeDoux has written widely about the distinction between the thalamo-amygdala pathway for processing exteroceptive stimuli rapidly and crudely in the absence of conscious awareness (LeDoux 1996). In contrast, the neocortical-amygdala pathway provides more precise and differentiated identification of the stimulus requiring an additional 12 msec of processing time. Evidence for similar subcortical processing in humans comes from neuroimaging studies utilizing backward masking of emotional stimuli that disrupt cortical processing and obscure the explicit knowledge of the stimuli. The subjective experience of these stimuli is simply that the masking stimulus (usually neutral) has been displayed, but the amygdala responds to emotional stimuli such as fearful (Whalen, Rauch, Etcoff, McInerney, Lee, & Jenike 1998) and angry faces (Morris, Öhman, & Dolan 1998).

In addition, Morris and colleagues found a dissociation of left and right amygdalae, finding increased right amygdala activation during masked stimuli presentation and left amygdala activation during unmasked presentation. Although the literature on the determinants of left vs. right amygdala acti-

vation are complex (Zald 2003), the findings by Morris and colleagues are consistent with other evidence that the left amygdala is more readily activated during states of high arousal and habituates less readily than the right amygdala (Wright et al. 2001; Phillips et al. 2001).

The amygdala may indeed be a domain-specific computational device specific to the domain of emotion that operates in a modular fashion. If so, the output of the amygdala should be crude and diffuse but can be the substrate for further processing. LeDoux (1996) has hypothesized that the output of the amygdala attains consciousness when it is relayed to working memory areas in the prefrontal cortex. This idea can be traced back to Walter Cannon who, in 1928, hypothesized that the conscious experience of emotion occurs when subcortical emotion information is relayed to the cortex (Cannon 1928). This idea was further elaborated by MacLean (1949) in his writings on the visceral brain. In that paper he hypothesized that impairments in communication between limbic and neocortical structures were expressed as an arrest in emotional development characterized by limited emotional awareness which in turn was associated with a predisposition for physical illness.

Several general principles can be derived from these considerations that are consistent with the psychological model presented above: (1) *The structures that are uniquely responsible for emotion generation operate implicitly, i.e. outside of conscious awareness*; (2) *The brain structures needed for conscious emotional experiences emerged more recently during phylogenesis compared to those required for implicit processes.*

A neural model of implicit and explicit emotional processing

A rudimentary neuroanatomical model of emotional awareness that distinguishes between implicit and explicit processes can now be formulated. Parallels between the corresponding neuroanatomical and psychological models are schematically depicted in Figure 1.

Both models are hierarchical and show a similar architecture of concentric shells. A remarkably similar conception has been independently developed by Fellous (1999). The concentric architecture means that each new level subsumes and modulates that of previous levels. Although both the psychological and neuroanatomical models designate five levels, we do not intend to suggest a direct correspondence between a given level in one model and that of the other model. In general, implicit functions at levels 1 and 2 in the psychological model correspond to Levels 1–3 in the neuroanatomical model, and explicit

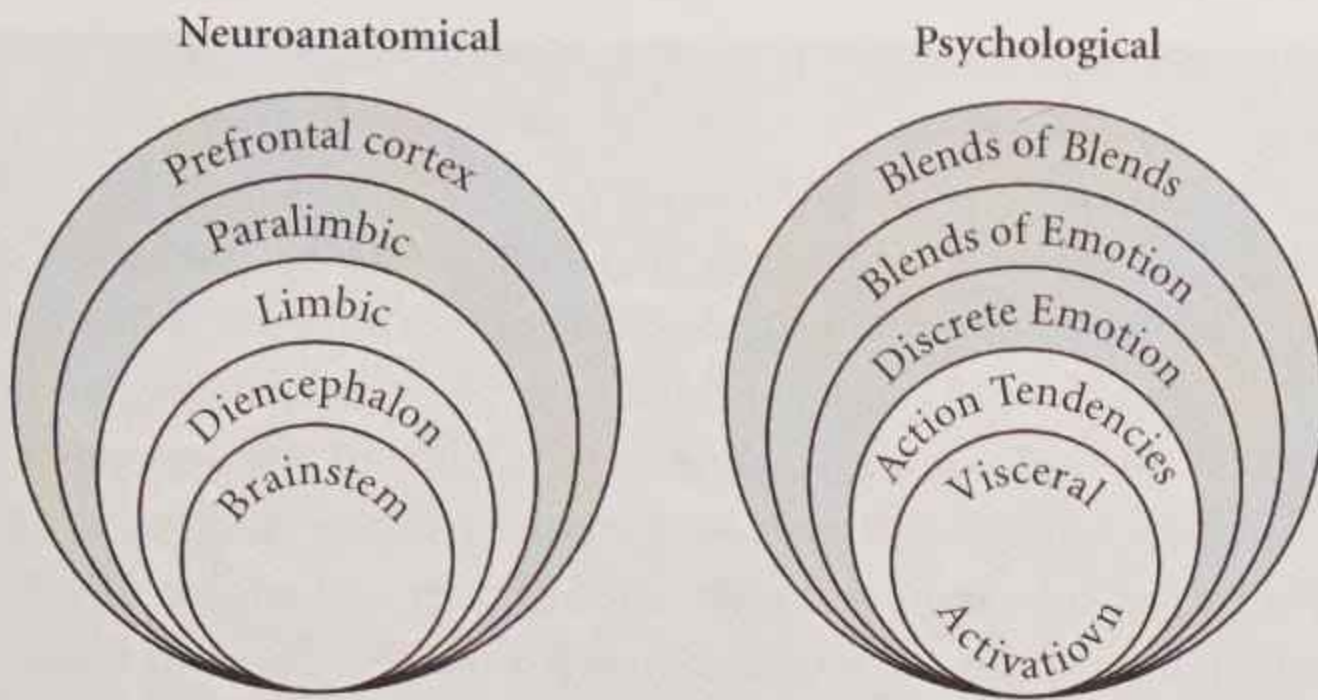


Figure 1. Parallels in the hierarchical organization of emotional experience and its neural substrates. The shell structure is intended to convey that each succeeding level adds to and modulates lower levels but does not replace them. Although each model contains five levels, a one-to-one correspondence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

functions at Levels 3–5 in the psychological model correspond to Levels 4 and 5 in the neuroanatomical model.

Following Ladavas et al. (1993), Levels 1 and 2 in the psychological model involve implicit processes that are automatic, modular and cognitively impenetrable. It would appear that subcortical structures participate in the automatic generation of emotional responses associated with absent or diffusely undifferentiated awareness. It may be speculated that the neural substrates of Level 1 include the thalamus and hypothalamus (diencephalon) and brainstem. At Level 2, the sensorimotor enactive level, crude distinctions between globally positive and globally negative states can be made. Given that decorticate cats can demonstrate fear and pleasure reactions (Bard & Rioch 1937), it is likely that the thalamus participates at this level. The amygdala appears to be preferentially activated in association with aversive stimuli (Zald 2003), and the ventral striatum, including the nucleus accumbens, is preferentially activated by appetitive or reward stimuli (Koob & Goeders 1989). The outputs from this stage of processing are widespread. Emotions at this level are represented in actions such as gestures and other movements that have an either/or quality. Much evidence suggests that the basal ganglia participate in the automatic behavioral displays of emotional gestures and expression (Gray 1995; Rolls 1990). A key tenet of this model is that structures at this level, such as the amygdala

(LeDoux 1996; Cahill & McGaugh 1998), are essential for implicit processing, and contribute to but are not sufficient for the explicit experience of discrete emotions or combinations thereof (i.e. Levels 3–5).

Levels 3–5 in the psychological model involve explicit processes that are influenced by higher cognitive processes, including prior explicit knowledge. The on-line experience of Levels 3–5 constitutes phenomenal awareness, which is mediated by the addition of paralimbic structures to the network of structures that mediate implicit emotion. Paralimbic structures include the anterior cingulate cortex, insula, temporal pole and orbitofrontal cortex. Orbitofrontal cortex activity appears to be associated with the perception of somatic sensations in context, biasing behavior either toward or away from a stimulus (Damasio 1994), overriding automatic processes in the amygdala and participating in extinction, among other functions (Emery & Amaral 2000).

Phenomenal awareness can be differentiated into background feelings and feelings that are the focus of attention. When focal attention is directed to the experience of emotion the dorsal anterior cingulate is activated and interacts with the other structures at this level and below. In the absence of focal attention participation of paralimbic structures, somatosensory cortex and right parietal cortex are associated with background emotions. These structures may well be sufficient to mediate experience at Levels 3–5.

The highest level of the neuroanatomical model is prefrontal cortex. The paracingulate region of medial prefrontal cortex is necessary for reflective awareness. The paracingulate region includes rostral anterior cingulate cortex and medial prefrontal cortex *per se*. The critical importance of representations in creating knowledge and their pivotal role in emotional intelligence was discussed above.

The anatomical relationship between key structures in the model is depicted in Figure 2. As noted, there are three types of conscious emotional experience that are addressed: (1) background feelings; (2) focal attention to feelings; and, (3) reflection upon feelings. Background feelings are subserved by a variety of structures in the ventral medial frontal lobe (labeled “1”) including orbitofrontal cortex, ventromedial prefrontal cortex, and ventral and pregenual ACC, insula; temporal pole; and right parietal lobe structures involved in mapping of somatic states (the structures not in the frontal lobe are not visible on the medial surface of the brain). Focal attention to feelings is subserved by dorsal ACC (labeled “2”). The establishment of a representation of mental state that can be used for purposes of reflection and future planning is subserved by the paracingulate region of the dorsomedial prefrontal cortex (labeled “3”). The function of all of these structures in mediating different as-

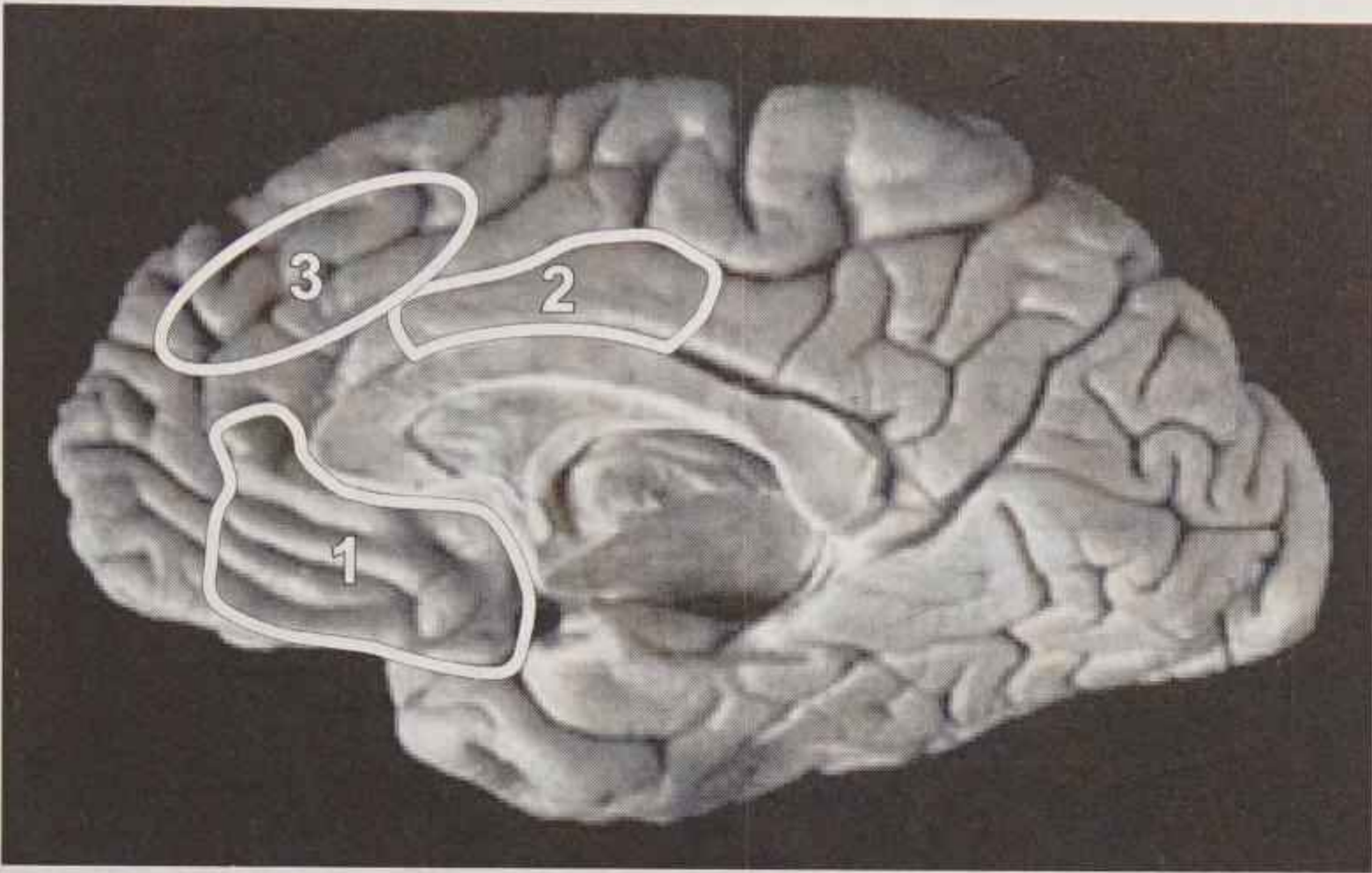


Figure 2. Structures on the medial surface of the frontal lobe that participate in (1) background feelings, (2) attention to feelings and (3) reflective awareness of feelings.

pects of conscious experience is dependent upon the generation of emotional responses in limbic, diencephalic and brainstem structures.

Self-regulation

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

One approach is to consider cortical influences on autonomic regulation. The dorsal ACC and dorsomedial prefrontal cortex have autonomic regulatory functions that are mediated by direct neural connections with subcortical visceromotor centers (Price, Carmichael, & Drevets 1996). Drevets (1999) has observed in PET research that blood flow in medial prefrontal cortex is inversely related to heart rate (Drevets 1999). Stimulation of the medial prefrontal cor-

tex is also known to produce decreases in heart rate and blood pressure in rabbits (Buchanan, Valentine, & Powell 1985). Critchley and colleagues observed in an fMRI study of physical and mental stress that heart rate variability (HRV) correlated positively with activity in the dorsal ACC (Critchley, Josephs, O'Doherty, Mathias, & Dolan 2002). We have similarly observed a positive correlation between the high frequency, vagal, component of HRV and activity in the medial prefrontal cortex (BA10) (Lane et al. 1999). Thus, these centers, when activated in conjunction with conscious emotional experience, have a tonic inhibitory effect mediated through the vagus nerve.

The positive correlation between medial prefrontal activity and vagal tone is interesting in two respects. First, one aspect of the correlation is that higher levels of vagal tone are associated with greater activity in medial prefrontal cortex. Based on the findings reviewed above, it would appear that a greater degree of conscious processing of emotion is associated with greater vagal tone. This can be understood in terms of parallel levels of complexity in the experience of emotion and the associated autonomic substrate for this experience. Greater HRV constitutes greater variation and complexity in the patterning of autonomic responses. Greater emotional awareness is associated with greater differentiation and complexity of experience. Support for the association between the two comes from studies of anxiety disorders, in which less differentiated emotional experience is associated with lower levels of vagal tone (Thayer & Lane 2000).

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

This observation may reflect a broader pattern involving an inverted U pattern for cortical processing. Arnstein (1998) has shown in rats that spatial memory performance is poor with very low levels of dopamine input to the dorsolateral prefrontal cortex, and is also poor with very high levels of dopamine input, whereas performance is maximal with moderate levels of dopamine input. This may be a general pattern for various subsectors of the frontal lobe in association with arousal, and is consistent with the Yerkes-

Dodson Law. Thus, at very high states of arousal and at very low states of arousal, reflection upon or attention to one's own emotional state may be minimal or non-existent. One might hypothesize that a hierarchy of "dissolution" may exist whereby reflection, focal attention and background feelings are successively eliminated from conscious awareness as arousal gets progressively more or progressively less intense.

It therefore appears that when emotional experience is consciously attended to, a feedback process occurs whereby vagal tone is facilitated and emotional arousal is modulated. In contrast to a generalized state of arousal, in which a diffuse network of neural structures is mobilized to process sensory information, attention is selective for certain inputs and not others, and involves selecting certain actions as others are inhibited (Thayer & Lane 2000; Paus 2000).

Recent neuroimaging evidence has shown that when individuals are instructed to regulate their emotional response, a conscious effort to do so can modulate amygdala activation. Decreased amygdala activation to emotional stimuli can result from the use of a cognitive reappraisal strategy (Ochsner, Bunge, Gross, & Gabrieli 2002), and amygdala activation can be enhanced by an instruction to maintain one's emotional state (Schaefer 2002). The anterior cingulate is often activated to a greater extent during the emotion regulation conditions than during the unregulated processing of emotional stimuli. This is consistent with the ACC's role in allocating attention as described above. For example, Beauregard and colleagues (2001) observed that free viewing of erotic film excerpts was associated with activation of right amygdala, right anterior temporal pole and hypothalamus, whereas attempted inhibition of sexual arousal by these male subjects was associated with activation of the right anterior cingulate cortex and right dorsolateral prefrontal cortex and the absence of subcortical activation. The ACC's contribution to successful relaxation using biofeedback (Critchley, Melmed, Featherstone, Mathias, & Dolan 2001) implicates it in accurate monitoring and modulation of somatic state. As noted above (Lange et al. 2003; Taylor et al. 2003) rating one's emotional responses is associated with decreased activity of the amygdala compared to passive viewing of emotional stimuli.

Thus, through a process of equilibration of bottom-up and top-down interactions, bodily state is altered as diffuse arousal mechanisms become modulated and differentiated. One implication of this model is that becoming consciously aware of one's own emotional responses in itself has a physiological effect that is self-regulatory. It is entirely consistent with the clinical notion that to get through an episode of emotional distress one must first allow one-

self to experience the distress fully, as this will then lead to a process of recovery (Greenberg 2002).

Discussion

In this chapter we have presented a comprehensive model of the neural substrates of emotional experience. This chapter extends a previous model published in 2000 (Lane et al. 2000b) by incorporating the concept of background emotion as a component of phenomenal awareness. This chapter also elaborates upon the previous model by selectively incorporating neuroimaging findings over the past four years. In this final section, we wish to consider the usefulness of the model. These implications can be divided into two broad categories: neuroimaging research on normative emotion and clinical applications.

Normative emotional experience

Our model has attempted to broadly parse the landscape of the conscious experience of emotion. Using the levels of emotional awareness model as a starting point, the functional neuroimaging literature has led us to focus on the neural substrates of background, phenomenal and reflective awareness. We thank Damasio and colleagues for introducing the concept of background emotions and find that it captures an important aspect of phenomenal experience not previously incorporated in our model.

Our model shares many similarities with the Damasio model of emotion (Damasio 2003) that are too numerous to delineate here. One area of overlap in the two models is the recognition that emotion generation per se is mediated by subcortical structures and that conscious feelings require the participation of higher cortical centers. One difference in the two models is Damasio's focus on the experience of somatic states, with particular emphasis placed on the insula. Damasio's focus is therefore on the afferent limb of emotional experience. Damasio's model assumes that the conscious experience of bodily states and feelings are the same, whereas somatic sensations constitute one of five levels in our model. Our model also highlights additional distinctions between phenomenal awareness (including background feelings) and reflective awareness that Damasio does not make. Relatedly, our model places more emphasis on the ACC than on the insula. The insula and ACC are densely interconnected and function together as predominantly sensory and motor paralimbic

structures, respectively. Following from the levels of emotional awareness perspective of emotional experience as a construction, our focus is on the efferent, effortful limb of experience involving the self-organizing aspects of attention and representation as they influence the contents of conscious experience. At the current state of knowledge these models should be considered complementary rather than contradictory.

With regard to the ACC, our model differs in important ways from a popular model that distinguishes between the cognitive (dorsal ACC) and affective (pregenual and ventral anterior cingulate) divisions of the ACC (Bush et al. 1998; Drevets & Raichle 1998). The latter model is supported by evidence that focusing attention on certain cognitive tasks leads to inhibition of the pregenual ACC and that affective tasks tend to produce the opposite pattern. One problem with this categorization is that it does not account for evidence that the dorsal ACC may have functions related to emotion. Specifically, there are affective tasks that activate the "cognitive division" including our study on emotional awareness, Rainville's study of the unpleasantness of pain, as well as a study of sadness by Drevets and Raichle in which the cognitive/affective distinction was made. Another problem is that it presents a stark contrast between cognition and affect that we believe is over-stated. A key question is whether the "affective division" may participate in processing both afferent and efferent visceral sensory and motor information that may accompany both "cognitive" and "emotional" tasks. For the purpose of parsing how the various sectors of the frontal lobe participate in emotional processing, we would hold that a distinction between background vs. focal attention to somatic sensations and feelings may more accurately characterize the distinction between ventromedial and dorsal ACC.

Further research is needed in a variety of areas to confirm the model that we have presented. This chapter has pointed out the complexity of the experiential dimension of emotion. As such, our model has implications for the design of neuroimaging studies of emotion. The paradigms that currently dominate the literature do not fully address the distinctions between background emotions, focal attention to emotional experience or reflection upon that experience. Recognition of these distinctions could lead to changes in how such imaging research is conducted.

For example, tasks that instruct people to attend to their own emotional experience typically allow subjects to either maintain attention to those feelings that spontaneously capture one's attention or bring their background emotions into focal awareness. Subjects may also reflect upon their emotional state and focus their attention on the products of this meta-cognitive effort. Few at-

tempts have been made to control these aspects in an experiment or to query subjects after completion of the experiment about which of these emotional tasks they performed.

With regard to reflective awareness, instructions that subjects have been given tend not to differentiate between an evaluation of the emotional stimulus itself and an evaluation of that subject's emotional response to the stimulus. When asked to rate their emotional response to stimuli such as emotion-evoking pictures, for example, it is typically unknown whether subjects are surveying their own immediate emotional response (phenomenal awareness), reporting the elaborated emotional processes evoked by the images (reflective awareness), or rating the images according to the emotional response that was targeted (a normative judgement about how others might rate the image).

Studies that address background emotions have attempted to explore the neural processing of emotional information by explicitly asking subjects to attend to non-emotional aspects of the stimuli. Examples of such tasks include gender decision tasks for emotional faces (Morris, Frith, Perrett, Rowland, Young, Calder, & Dolan 1996), indoor/outdoor decision tasks for emotional scenes (Lane et al. 1997), and counting the number of emotional stimuli as in the Emotional Stroop Task (Whalen et al. 1998). Passive viewing tasks (Lange et al. 2003) may also target background emotions. Although reaction time for cognitive tasks can be used to index the degree to which emotional salience interferes with the assigned cognitive operations, the tasks themselves do not address the degree to which the emotional stimuli are processed as such. Results from studies implementing passive viewing can be particularly difficult to interpret because without instructions guiding how the subject's attention is allocated, individuals are free to focus on the global gestalt of the stimulus, the perceptual details of the stimulus, their own emotional state, or the vast array of emotional and non-emotional topics triggered by the stimulus.

We supported the current model of background feelings using a subtraction paradigm in our own neuroimaging research as well as findings from other laboratories. One approach to substantiating the proposed neural basis of background feelings more directly would be to adapt the paradigm of Shergill and colleagues (2000) in their study of hallucinations. Because attention and reflection have neural substrates of their own, Shergill and colleagues ask subjects to perform an unrelated task and intermittently signal the subject to indicate whether s/he is currently experiencing an hallucination. Such an approach could be adapted to tasks such as the Emotional Counting Stroop or gender identity determination during processing of pictures of facial affect. This paradigm may be adapted further by varying exposure duration in

conjunction with backward masking in order to examine the transition from implicit to explicit processing. To our knowledge no studies of this kind have yet appeared in the literature.

To the extent that these different aspects of conscious emotional experience are not controlled they will introduce variability in the findings. Indeed, we have found that many of the neuroimaging studies of normative emotion in recent years do not permit a clear differentiation between background feelings, focal attention to feelings and reflective awareness. It is our hope that this model will lead to adjustments in experimental conditions that will permit a rigorous test of this model with the ultimate goal of generating replicable findings on the neural substrates of emotional experience.

Clinical implications

The section on self-regulation focused on the role of anterior cingulate and medial prefrontal structures in the regulation of vagal tone. It is notable that various clinical conditions such as major depression (Stein, Carney, Freedland, Skala, Jaffe, Kleiger, & Rottman 2000; Krittanyaphong, Cascio, Light, Sheffield, Golden, Finkel, Glekas, Koch, & Sheps 1997; Carney, Blumenthal, Stein, Watkins, Catellier, Berkman, Czajkowski, O'Connor, Stone, & Freedland 2001) and posttraumatic stress disorder (PTSD) (Cohen, Benjamin, Geva, Matar, Kaplan, & Kotler 2000) are associated both with deficits in vagal tone and dysfunction of ACC (Mayberg, Liotti, Brannan, McGinnis, Mahurin, Jerabek, Silva, Tekell, Martin, Lancaster, & Fox 1999; Liotti & Mayberg 2001; Rauch, Whalen, Shin, McInerney, Macklin, Lasko, Orr, & Pitman 2000; Bremner, Staib, Kaloupek, Southwick, Soufer, & Charney 1999; Pitman, Shin, & Rauch 2001). Given the fact that these disorders involve emotional responses that are too intense and prolonged, a physiologic substrate may be the deficits in vagal tone that keep arousal mechanisms in check. As such, a given change in the functional activity of the ACC may simultaneously account for deficits in vagal tone, a loss of complexity in emotional experience and perhaps changes in certain cognitive functions in these disorders.

In the case of both major depression and PTSD, current evidence suggests that abnormalities exist in ACC and amygdala that are reciprocally related. A decrease in activity in the dorsal ACC has been observed during the clinical state of depression, which returns to baseline when depression is successfully treated (Mayberg et al. 1999). Consistent with the modulatory affect of the ACC on the amygdala, Sheline and colleagues found that the left amygdala

was over-responsive to masked emotional faces in depressed patients, but that this response resolved following treatment with a selective serotonin reuptake inhibitor (SSRI) (Sheline, Barch et al. 2001). Studies in patients with PTSD reveal activation of ACC to a lesser extent than in healthy controls (Lanius, Williamson, Hopper, Densmore, Boksman, Gupta, Neufeld, Gati, & Menon 2003) or combat controls (Bremner et al. 1999) during induction of traumatic symptoms. Consistent with prediction, Shin and colleagues (2001) observed that the pregenual ACC was not activated during performance of the Emotional Counting Stroop task, whereas it was in healthy controls, and PTSD patients exhibit an enhanced amygdala response to masked negative stimuli compared to healthy controls (Rauch et al. 2000). Although not all studies in PTSD have found relative deactivation of ACC (Hull 2002), this may be related to some of the variability in experimental design discussed above.

Indirect support for the relationship between conscious experience, activity of medial prefrontal cortex and HRV can be derived from research on cognitive-behavioral therapy (CBT) of depression. CBT involves recognizing when dysphoric emotional states occur, identifying thoughts that induce these states and then modifying the thoughts for the purpose of reducing affective symptoms (Beck 1979). As such, CBT promotes awareness and attention to one's own emotions. Mayberg and colleagues have shown that successful CBT treatment for depression increases activity in the medial prefrontal cortex (Goldapple, Segal, Garson, Bieling, Lau, & Mayberg 2002). Carney and colleagues have shown that successful CBT treatment of depression is associated with increased HRV (Carney et al. 2000). To date, however, the neural substrates of HRV regulation in the context of depression or PTSD have not been studied. These findings are sufficiently suggestive that further investigation into the similarities and differences in the neural regulation of vagal tone in these two disorders appears warranted.

The findings from these clinical studies may appear to raise a paradox. We have taken the position that those brain structures that operate implicitly do so in a modular manner. As such they are impenetrable to preexisting expectations, ideas and attitudes. Yet, we know clinically that emotional responses can change. How does this occur? Consistent with the findings just reported, we would contend that such change occurs as a result of top-down influences being superimposed upon subcortical processing. Recent evidence (Milad & Quirk 2002) showed that the process of extinction of conditioned fear in rats does not involve simply unlearning the conditioned response. Rather, extinction involves new learning mediated by ventromedial prefrontal cortex. Changing old patterns does not eliminate the patterns – it involves adding to those old pat-

terns and modulating them. This is consistent with the orbitofrontal cortex, a paralimbic structure, having a modulating influence on a limbic structure, the amygdala, consistent with the concentric shell architecture. Although subjects are able to successfully modulate their amygdala activation to negative stimuli, amygdala activation is still much greater compared to that when subjects are viewing neutral images (Ochsner, Bunge, Gross, & Gabrieli 2002). Relatedly, our hierarchical model may help to explain the phenomenon of "regression" to old patterns of behavior in the face of severe stress as prefrontal structures go off-line.

Summary

We have explored the neural substrates of conscious emotional experience using a cognitive neuroscientific approach. This approach highlighted the distinction between implicit and explicit processes, which can be viewed on a continuum of progressive differentiation and integration. These structural characteristics of the psychological model led to a hierarchical model of the neural substrates of emotional experience. Although a direct mapping between the psychological and neuroanatomical models is not yet possible, an exploration of the functional properties of the neural structures identified by the model supported the distinction between implicit emotion and three aspects of conscious emotional experience: background feelings, attention to feelings and reflective awareness. When phenomenal awareness (background feelings and attention to feelings) or reflective awareness occur, subcortical generators of emotion are modulated. Conversely, pathological states of emotion dysregulation such as major depression and PTSD appear to be associated with brain changes that may simultaneously account for alterations in emotional experience and autonomic indices of arousal. This model may therefore be a useful guide to research on normative emotion and on the dysregulation of emotion that occurs in certain psychiatric disorders. To the extent that anxiety and depression are associated with adverse medical outcome, this model may also provide insight into the physiological mechanisms by which emotion dysregulation affects the longitudinal course of systemic medical disorders.

References

- Adleman, N., Menon, V., Blasey, C., White, C., Warsofsky, I., Glover, G., & Reiss A. L. (2002). A Developmental fMRI Study of the Stroop Color-Word Task. *Neuroimage*, 16 (1), 61–75.
- Arnstein, A. F. (1998). Catecholamine modulation of prefrontal cortical cognitive function. *Trends Cognitive Science*, 2 (11), 436–447.
- Augustine, J. R. (1996). Circuitry and functional aspects of the insular lobe in primates including humans. *Brain Research Review*, 22, 229–244.
- Bard, P. & Rioch, D. M. (1937). A study of four cats deprived of neocortex and additional parts of the forebrain. *Bulletin of the Johns Hopkins Hospital*, 60, 73–147.
- Barrett, L. E., Lane, R. D., Sechrest, L., & Schwartz, G. E. (2000). Sex differences in emotional awareness. *Personality and Social Psychology Bulletin*, 26, 1027–1035.
- Beauregard, M., Lévesque, J., & Bourgouin, P. (2001). Neural correlates of the conscious self-regulation of emotion. *J. Neuroscience*, 21, RC165 (1–6).
- Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1997). Deciding advantageously before knowing the advantageous strategy. *Science*, 275, 1293–1295.
- Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive Therapy of Depression*. New York: Guilford Press.
- Blakemore, S. J., Wolpert, D. M., & Frith, C. D. (1998). Central cancellation of self-produced tickle sensation. *Nature Neuroscience*, 1 (7), 635–640.
- Blatt, S. J., Wein, S. J., Chevron, E., & Quinlan, D. M. (1979). Parental representations and depression in normal young adults. *Journal of Abnormal Psychology*, 88, 388–397.
- Bremner, J. D., Staib, L. H., Kaloupek, D., Southwick, S. M., Soufer, R., & Charney, D. S. (1999). Neural correlates of exposure to traumatic pictures and sound in combat veterans with and without posttraumatic stress disorder: A positron emission tomography study. *Biological Psychiatry*, 45 (7), 806–816.
- Buchanan, S. L., Valentine, J., & Powell, D. A. (1985). Autonomic responses are elicited by electrical stimulation of the medial but not lateral frontal cortex in rabbits. *Behavioral Brain Research*, 18, 51–62.
- Bush, G., Whalen, P. J., Rosen, B. R., Jenike, M. A., McInerney, S. C., & Rauch, S. L. (1998). The Counting Stroop: An interference task specialized for functional neuroimaging: Validation study with functional MRI. *Human Brain Mapping*, 6 (4), 270–282.
- Cahill, L., & McGaugh, J. L. (1998). Mechanisms of emotional arousal and lasting declarative memory. *Trends in Neurosciences*, 21 (7), 294–299.
- Cannon, W. B. (1928). The mechanism of emotional disturbance of bodily functions. *NEJM*, 198, 877–884.
- Carney, R. M., Freedland, K. E., Stein, P. K., Skala, J. A., Hoffman, P., & Jaffe, A. S. (2000). Change in heart rate and heart rate variability during treatment for depression in patients with coronary heart disease. *Psychosomatic Medicine*, 62, 639–647.
- Carney, R. M., Blumenthal, J. A., Stein, P. K., Watkins, L., Catellier, D., Berkman, L. F., Czajkowski, S. M., O'Connor, C., Stone, P. H., & Freedland, K. E. (2001.) Depression, heart rate variability, and acute myocardial infarction. *Circulation*, 104, 2024–2028.

- Cohen, H., Benjamin, J., Geva, A. B., Matar, M. A., Kaplan, Z., & Kotler, M. (2000). Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: Application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. *Psychiatry Research*, 96 (1), 1–13.
- Critchley, H., Josephs, O., O'Doherty, J., Mathias, C. J., & Dolan, R. J. fMRI correlates of heart rate variability during cognitive and motor behaviour. Abstract #283. 8th International Conference on Functional Mapping of Human Brain, Sendai, Japan, June 2002.
- Critchley, H. D., Melmed, R. N., Featherstone, E., Mathias, C. J., & Dolan, R. J. (2001). Brain activity during biofeedback relaxation: A functional neuroimaging investigation. *Brain*, 124 (5), 1003–1012.
- Damasio, A. R. (1994). *Descartes' error: Emotion, reason, and the human brain*. New York: G.P. Putnam's Sons.
- Damasio, A. R., Grabowski, T. J., et al. (2000). Subcortical and cortical brain activity during the feeling of self-generated emotions. *Nature Neuroscience*, 3 (10), 1049–1056.
- Damasio, A. R. (2003). *Looking for Spinoza: Joy, sorrow and the feeling brain*. New York, Harcourt.
- Drevets, W. C. (1999). Prefrontal cortical-amygdalar metabolism in major depression. *Annals of the New York Academy of Sciences*, 877, 614–637.
- Drevets, W. C. & Raichle, M. E. (1998). Reciprocal suppression of regional cerebral blood flow during emotional versus higher cognitive processes: implications for interactions between emotion and cognition. *Cognition and Emotion*, 12 (3), 353–385.
- Ekman, P. (1982). *Emotion in the human face* (2nd ed.). New York: Cambridge University Press.
- Emery, N. J. & Amaral, D. G. (2000). The role of the amygdala in social cognition. In R. Lane, L. Nadel, G. Ahern, J. Allen, A. Kaszniak, S. Rapsack, & G. E. Schwartz (Eds.), *The cognitive neuroscience of emotion*. New York: Oxford University Press.
- Farthing, G. W. (1992). *The psychology of consciousness*. Englewood Cliffs, NJ, Prentice Hall.
- Fellous, J. M. (1999). The neuromodulatory basis of emotion. *Neuroscientist*, Summer, 1–15.
- Fodor, J. A. (1983). *The modularity of the mind*. Cambridge, MA: MIT Press.
- Frijda, N. (1986). *The emotions*. Cambridge, UK: Cambridge University Press.
- Frith, C. D. & Frith, U. (1999). Interacting minds – a biological basis. *Science*, 286 (5445), 1692–1695.
- Gazzaniga, M. S., Ivry, R. B., & Mangun, G. R. (2002). *Cognitive neuroscience – the biology of the mind* (2nd ed.). New York: Norton.
- Goldapple, K., Segal, Z., Garson, C., Bieling, P., Lau, M., & Mayberg, M. Effects of cognitive behavioral therapy on brain glucose metabolism in patients with major depression. Presented at the 2002 Annual Meeting of the Society of Biological Psychiatry.
- Gray, J. A. (1995). A model of the limbic system and basal ganglia: Applications to anxiety and schizophrenia. In M. S. Gazzaniga (Ed.), *The Cognitive Neurosciences* (pp. 1165–1176). Cambridge, MA: MIT Press.
- Greenberg, L. S. (2002). *Emotion-focused therapy: Coaching clients to work through their feelings*. American Psychological Association: Washington, DC.

- Gusnard, D. A., Akbudak, E., Shulman, G. L., & Raichle, M. E. (2001). Medial prefrontal cortex and self-referential mental activity: Relation to a default mode of brain function. *Proceedings of the National Academy of Sciences of the United States of America*, 8 (7), 4259–4264.
- Happé, F., Ehlers, S., Fletcher, P., Frith, U., Johansson, M., Gillberg, C., Dolan, R., Frackowiak, R., & Frith, C. (1996). 'Theory of mind' in the brain. Evidence from a PET scan study of Asperger syndrome. *Neuroreport*, 8 (1), 197–201.
- Hariri, A. R., Mattay, V. S., Tessitore, A., Fera, F., & Weinberger, D. R. (2003). Neocortical modulation of the amygdala response to fearful stimuli. *Biological Psychiatry*, 56 (6), 494–501.
- Hugdahl, K. & Oehman, A. (1977). Effects of instruction on acquisition and extinction of electrodermal responses to fear-relevant stimuli. *Journal of Experimental Psychology: Human Learning & Memory*, 3 (5), 608–618.
- Hull, A. M. (2002). Neuroimaging findings in post-traumatic stress disorder. Systematic review. *British Journal of Psychiatry*, 181 (2), 102–110.
- Iidaka, T., Omori, M., Murata, T., Kosaka, H., Yonekura, Y., Tomohisa, O., et al. (2001). Neural interaction of the amygdala with the prefrontal and temporal cortices in the processing of facial expressions as revealed by fMRI. *Journal of Cognitive Neuroscience*, 13 (8), 1035–1047.
- Johnson, S. C., Baxter, L. C., Wilder, L. S., Pipe, J. G., Heiserman, J. E., & Prigatano, G. P. (2002). Neural correlates of self-reflection. *Brain*, 125 (Pt 8), 1808–1814.
- Karmiloff-Smith, A. (1992). *Beyond modularity: A developmental perspective on cognitive science*. Cambridge, MA: MIT Press.
- Koob, G. F. & Goeders, N. E. (1989). Neuroanatomical substrates of drug self-administration. In J. M. Liebman & S. J. Cooper (Eds.), *Neuropharmacological Basis of Reward* (pp. 214–263). New York: Oxford University Press.
- Krittayaphong, R., Cascio, W. E., Light, K. C., Sheffield, D., Golden, R. N., Finkel, J. B., Glekas, G., Koch, G. G., & Sheps, D. S. (1997). Heart rate variability in patients with coronary artery disease: differences in patients with higher and lower depression scores. *Psychosomatic Medicine*, 59, 231–235.
- Ladavas, E., Cimatti, D., del Pesce, M., & Tuoizzi, G. (1993). Emotional evaluation with and without conscious stimulus identification: Evidence from a split-brain patient. *Cognition and Emotion*, 7 (1), 95–114.
- Lane, R. D. & Schwartz, G. E. (1987). Levels of emotional awareness: A cognitive-developmental theory and its application to psychopathology. *American Journal of Psychiatry*, 144, 133–143.
- Lane, R., Quinlan, D., Schwartz, G., Walker, P., & Zeitlin, S. (1990). The levels of emotional awareness scale: A cognitive-developmental measure of emotion. *Journal of Personality Assessment*, 55, 124–134.
- Lane, R. D., Kevley, L. S., DuBois, M. A., Shamasundara, P., & Schwartz, G. E. (1995). Levels of emotional awareness and the degree of right hemispheric dominance in the perception of facial emotion. *Neuropsychologia*, 33, 525–528.
- Lane, R. D., Sechrest, B., Riedel, R., Weldon, V., Kaszniak, A., & Schwartz, G. (1996). Impaired verbal and nonverbal emotion recognition in alexithymia. *Psychosomatic Medicine*, 58, 203–210.

- Lane, R. D., Fink, G. R., Chua, P. M. L., & Dolan, R. J. (1997). Neural activation during selective attention to subjective emotional responses. *Neuroreport*, 8 (18), 3969–3972.
- Lane, R. D., Reiman, E. M., Axelrod, B., Yun, L.-S., Holmes, A., & Schwartz, G. E. (1998). Neural correlates of levels of emotional awareness: evidence of an interaction between emotion and attention in the anterior cingulate cortex. *Journal of Cognitive Neuroscience*, 10 (4), 525–535.
- Lane, R., Reiman, E., Ahern, G., & Thayer, J. (1999). Activity in medial prefrontal cortex correlates with vagal component of heart rate variability during emotion. Presented at the Annual Meeting of the Society for Neuroscience, Miami, FL, October 1999.
- Lane, R., Nadel, L., Allen, J., & Kaszniak, A. (2000a). The study of emotion from the perspective of cognitive neuroscience. In R. Lane, L. Nadel, G. Ahern, J. Allen, A. Kaszniak, S. Rapcsak, & G. Schwartz (Eds.), *Cognitive neuroscience of emotion* (pp. 3–1). New York: Oxford University Press.
- Lane, R. (2000). Neural correlates of conscious emotional experience. In R. Lane, L. Nadel, G. Ahern, J. Allen, A. Kaszniak, S. Rapcsak, & G. Schwartz (Eds.), *Cognitive neuroscience of emotion* (pp. 345–370). New York: Oxford University Press.
- Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1995). *The International Affective Picture System (IAPS): Photographic slides*. University of Florida: The Center for Research in Psychophysiology.
- Lange, C. G. & James, W. (1922). *The Emotions*. Oxford, England: Williams & Wilkins Co.
- Lange, K., Williams, L. M., Young, A. W., Bullmore, E. T., Brammer, M. J., Williams, S. C. R., et al. (2003). Task instructions modulate neural responses to fearful facial expressions. *Biological Psychiatry*, 53 (3), 226–232.
- Lanius, R. A., Williamson, P. C., Hopper, J., Densmore, M., Boksman, K., Gupta, M. A., Neufeld, R. W. J., Gati, J. S., & Menon, R. S. (2003). Recall of emotional states in posttraumatic stress disorder: An fMRI investigation. *Biological Psychiatry*, 53 (3), 204–210.
- Larsen, R. J. & Diener, E. (1987). Affect intensity as an individual difference characteristic: A review. *Journal of Research in Personality*, 21, 1–39.
- LeDoux, J. E. (1996). *The emotional brain: The mysterious underpinnings of emotional life*. New York, NY: Simon & Schuster.
- Lévesque, J., Joanette, Y., Paquette, V., Mensour, B., Beaudoin, G., Leroux, J.-M., Bourgouin, P., & Beauregard, M. (2003). Neural circuitry underlying voluntary self-regulation of sadness. *Biological Psychiatry*, 53, 502–510.
- Levine, D., Marziali, E., & Hood, J. (1997). Emotion processing in borderline personality disorders. *Journal of Nervous and Mental Disease*, 185 (4), 240–246.
- Liotti, M. & Mayberg, H. S. (2001). The role of functional neuroimaging in the neuropsychology of depression. *Journal of Clinical & Experimental Neuropsychology*, 23 (1), 121–136.
- Loevinger, J., & Wessler, R. (1970). *Measuring ego development, Vol. I. Construction and use of a sentence completion test*. San Francisco: Jossey-Bass.
- Loevinger, J., Wessler, R., & Redmore, C. (1970). *Measuring ego development, Vol. II. Scoring manual for women and girls*. San Francisco: Jossey-Bass.
- MacLean, P. D. (1949). Psychosomatic disease and the “visceral brain”: Recent developments bearing on the Papez theory of emotion. *Psychosomatic Medicine*, 11, 338–353.

- Mangina, C. A. & Beuzeron-Mangina, J. H. (1996). Direct electrical stimulation of specific human brain structures and bilateral electrodermal activity. *Int. J. Psychophysiology*, 22 (1–2), 1–8.
- Mayberg, H. S., Liotti, M., Brannan, S. K., McGinnis, S., Mahurin, R. K., Jerabek, P. A., Silva, J. A., Tekell, J. L., Martin, C. C., Lancaster, J. L., & Fox, P. T. (1999). Reciprocal limbic-cortical function and negative mood: converging PET findings in depression and normal sadness. *American Journal of Psychiatry*, 156 (5), 675–682.
- Mesulam, M. M. & Mufson, E. J. (1982). Insula of the old world monkey. III: Efferent cortical output and comments on function. *Journal of Computational Neurology*, 212 (1), 38–52.
- Milad, M. R. & Quirk, G. J. (2002). Neurons in medial prefrontal cortex signal memory for fear extinction. *Nature*, 420, 70–74.
- Morris, J. S., Frith, C. D., Perrett, D. I., Rowland, D., Young, A. W., Calder, A. J., & Dolan, R. J. (1996). A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature*, 383, 812–815.
- Morris, J. S., Öhman, A., & Dolan, R. J. (1998). Conscious and unconscious emotional learning in the human amygdala. *Nature*, 393, 467–470.
- Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. E. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience*, 14 (8), 1215–1229.
- Öhman, A. & Mineka, S. (2001). Fears, phobias, and preparedness: Toward an evolved module of fear and fear learning. *Psychological Review*, 108 (3), 483–522.
- Papez, J. W. (1937). A proposed mechanism of emotion. *Archives of Neurology and Psychiatry*, 38, 725–734.
- Paus, T. (2000). Functional anatomy of arousal and attention systems in the human brain. In *Cognition, emotion and autonomic responses: the integrative role of the prefrontal cortex and limbic structures*. In H. B. M. Uylings, C. G. Van Eden, J. P. C. DeBruin, M. G. P. Feenstra, & C. M. A. Pennartz (Eds.), *Proceedings of the 21st international summer school of brain research*, Amsterdam, August 1999. Reprinted from *Progress in Brain Research* 126, 65–77.
- Phan, K. L., Wager, T., Taylor, S. F., & Liberzon, I. (2002). Functional neuroanatomy of emotion: a meta-analysis of emotion activation studies in PET and fMRI. *Neuroimage*, 16 (12), 331–348.
- Phillips, M. L., Young, A. W., Senior, C., Brammer, M., Andrew, C., Calder, A. J., Bullmore, E. T., Perrett, D. I., Rowland, D., Williams, S. C. R., Gray, J. A., & David, A. S. (1997). A specific neural substrate for perceiving facial expressions of disgust. *Nature*, 389, 495–498.
- Phillips, M. L., Medford, N., Young, A. W., Williams, L., Williams, S. C., Bullmore, E. T., Gray, J. A., & Brammer, M. J. (2001). Time courses of left and right amygdalar responses to fearful facial expressions. *Hum Brain Mapp*, 12 (4), 193–202.
- Piaget, J. (1937). *La construction du réel*. Neuchâtel: Delachaux et Niestlé.
- Pitman, R. K., Shin, L. M., & Rauch, S. L. (2001). Investigating the pathogenesis of posttraumatic stress disorder with Neuroimaging. *Journal of Clinical Psychiatry*, 62 (17), 47–54.

- Price, J. L., Carmichael, S. T., & Drevets, W. C. (1996). Networks related to the orbital and medial prefrontal cortex; a substrate for emotional behavior? *Progress in Brain Research*, 107, 523–536.
- Rainville, P., Duncan, G. H., Price, D. D., Carrier, B., & Bushnell, M. C. (1997). Pain affect encoded in human anterior cingulate but not somatosensory cortex. *Science*, 277, 968–971.
- Rau, J. C. (1993). Perception of verbal and nonverbal affective stimuli in complex partial seizure disorder. *Dissertation Abstracts International [B]*, 54, 506–B.
- Rauch, S. L., Whalen, P. J., Shin, L. M., McInerney, S. C., Macklin, M. L., Lasko, N. B., Orr, S. P., & Pitman, R. K. (2000). Exaggerated amygdala response to masked facial stimuli in posttraumatic stress disorder: A functional MRI study. *Biological Psychiatry*, 47 (9), 769–776.
- Reiman, E., Lane, R., Ahern, G., Schwartz, G., Davidson, R., Yun, L., & Chen, K. (1997). Neuroanatomical correlates of externally and internally generated emotion. *American Journal of Psychiatry*, 154, 918–925.
- Rolls, E. T. (1990). A theory of emotion, and its application to understanding the neural basis of emotion. *Cognition and Emotion*, 4 (3), 161–190.
- Salovey, P. & Mayer, J. D. (1990). Emotional intelligence. *Imagination, Cognition and Personality*, 9, 185–211.
- Schaefer, S. M. J., Daren C., Davidson, R. J., Aguirre, G. K., Kimberg, D. Y., & Thompson-Schill, S. L. (2002). Modulation of amygdalar activity by the conscious regulation of negative emotion. *Journal of Cognitive Neuroscience*, 14 (6), 913–921.
- Shin L. M., Whalen P. J., Pitman R. K., Bush, G., Macklin, M. L., Lasko, N. B., Orr, S. P., McInerney, S. C., & Rauch, S. L. (2001). An fMRI study of anterior cingulate function in posttraumatic stress disorder. *Biological Psychiatry*, 50 (12), 932–942.
- Shipley, W. (1940). A self-administering scale for measuring intellectual impairment and deterioration. *Journal of Psychology*, 9, 371–377.
- Sheline, Y. I., Barch, D. M., et al. (2001). Increased amygdala response to masked emotional faces in depressed subjects resolves with antidepressant treatment: An fMRI study. *Biological Psychiatry*, 50 (9), 651–658.
- Shergill S. S., Brammer M. J., Williams, S. C., Murray, R. M., & McGuire, P. K. (2000). Mapping auditory hallucinations in schizophrenia using functional magnetic resonance imaging. *Archives of General Psychiatry*, 57 (11), 1033–1038.
- Stein, P. K., Carney, R. M., Freedland, K. E., Skala, J. A., Jaffe, A. S., Kleiger, R. E., & Rottman, J. N. (2000). Severe depression is associated with markedly reduced heart rate variability in patients with stable coronary heart disease. *Journal of Psychosomatic Research*, 48, 493–500.
- Taylor, S. F., Phan, K. L., Decker, L. R., & Liberzon, I. (2003). Subjective rating of emotionally salient stimuli modulates neural activity. *Neuroimage*, 18 (3), 650–659.
- Thayer, J. F. & Lane, R. (2000). A model of neurovisceral integration in emotional regulation and dysregulation. *Journal of Affective Disorders*, 61, 201–216.
- Vogeley, K., Bussfeld, P., Newen, A., Herrmann, S., Happe, E., Falkai, P., Maier, W., Shah, N. J., Fink, G. R., & Zilles, K. (2001). Mind reading: neural mechanisms of theory of mind and self-perspective. *Neuroimage*, 14 (1 Pt 1), 170–181.

- Vogt, B. A., Finch, D. M., & Olson, C. R. (1992). Functional heterogeneity in cingulate cortex: The anterior executive and posterior evaluative regions. *Cerebral Cortex*, 2 (6), 435–443.
- Vogt, B. A. & Gabriel, M. (1993). *Neurobiology of cingulate cortex and limbic thalamus*. Boston: Birkhauser.
- Weiskrantz, L. (2000). Blindsight: Implications for the conscious experience emotion. In R. Lane, L. Nadel, G. Ahern, J. Allen, A. Kaszniak, S. Rapsack, & G. E. Schwartz (Eds.), *Cognitive Neuroscience of Emotion* (pp. 277–295). New York: Oxford University Press.
- Whalen, P. J., Bush, G., McNally, R. J., Wilhelm, S., McInerney, S. C., Jenike, M. A., & Rauch, S. L. (1998). The emotional counting Stroop paradigm: A functional magnetic resonance imaging probe of the anterior cingulate affective division. *Biological Psychiatry*, 44 (12), 1219–1228.
- Whalen, P. J., Rauch, S. L., Etcoff, N. L., McInerney, S. C., Lee, M. B., & Jenike, M. A. (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *Journal of Neuroscience*, 18 (1), 411–418.
- Wrana, C., Thomas, W., Heindichs, G., Huber, M., Obliers, R., Koerfer, A., & Köhle, K. (Leipzig, March 1998). Levels of Emotional Awareness Scale (LEAS): A contribution to an empirical examination of the validity and reliability of the German version. Poster presentation at the 47th working conference of the German College of Psychosomatic Medicine.
- Wright, C. I., Fischer, H., Whalen, P. J., McInerney, S. C., Shin, L. M., & Rauch, S. L. (2001). Differential prefrontal cortex and amygdala habituation to repeatedly presented emotional stimuli. *Neuroreport*, 12 (2), 379–383.
- Yamasaki, H. L. K., & McCarthy, G. (2002). Dissociable prefrontal brain systems for attention and emotion. *Proceedings of the National Academy of Science USA*, 99 (17), 11447–11451.
- Zald, D. H. (2003). The human amygdala and the emotional evaluation of sensory stimuli. *Brain Research – Brain Research Reviews*, 41 (1), 88–123.

CHAPTER 5

Self-regulation by the medial frontal cortex

Limbic representation of motive set-points

Phan Luu and Don M. Tucker

Introduction

In researching the neural mechanisms of psychological function, a key question is the adaptive, motivational control of cognition. Although studies of emotion and self-regulation are now popular topics in cognitive neuroscience research, the theoretical framework of cognitive neuroscience provides only limited concepts of motivational control. This limitation may have resulted from the common shadow of behaviorism, cast on the historical development of both predecessor fields of cognitive psychology and neuroscience. As cognitive psychologists struggled against the prevailing dogma of behaviorism in the 1940s and 1950s, they focused on describing cognitive processes of perception, attention, and memory as objectively definable information processing tasks, ignoring the domains of reward, motivation, and learning that were the purview of behaviorism. Biologists studying neuroscience, on the other hand, were trained only with simplest behavioristic notions of classical and operant conditioning, the only framework accepted as scientifically reputable for explaining behavior, as they studied neural mechanisms of behavior control. The result of these historical trends is that the current paradigm of cognitive neuroscience approaches questions of emotional self-regulation with relatively naïve experimental designs and theoretical interpretations, often as if these questions were being asked for the first time.

Future historians of science may find this to be an ironic turn of events. It was, in fact, the scientific analysis of physiological self-regulation that led to concepts of control theory that allowed the development of modern electronic information processing. From Barnard's studies of the "internal milieu" in the 1900s came the realization that the brain has some circuits specialized for

monitoring internal states, such as temperature and thirst, and other circuits for adjusting physiological controls relevant to those states, such as metabolic rate and water-seeking behavior. Recognizing that these circuits are mechanistically and dynamically coupled, Canon (1915), formulated the principle of homeostasis, in which deviations from physiological set-points lead to adaptive mechanisms. To describe influences that create these deviations, Canon introduced the concept of *stress*. Although nineteenth century mechanics and inventors had implemented feedback systems in applications such as heating thermostats, it took until the mid twentieth century for electrical engineers to realize the general principle of feedback control. This principle was the central idea of Wiener's (1948) *cybernetics*. With this principle, engineers could then apply dynamic feedback control to modern transistor electronics and data-processing machines, leading to the profound informatics revolution that allowed today's science, industry, and daily life (Heims 1991).

Wiener's idea of cybernetics was taken directly from Canon's study of physiological homeostasis, and it in turn influenced scientific thinking in fields from anthropology to psychopathology (Heims 1991). This idea also led to a popular appreciation of complex forms of causality, as exemplified by the integration of the term "feedback" in daily life. Drawing from cybernetic principles, Pribram (1960) proposed a feedback model of human planning and self-regulation. In this model, the organism set goals, monitors actions in relation to them, and corrects deviations from desired goal trajectories. In reviewing the neural mechanisms relevant to these processes, Pribram emphasized that brain systems operate as cybernetic servomechanisms, with the posterior brain responding to discrepancies from the expected perceptual context, and the anterior brain responding to discrepancies from the expected motivational context. Following his anatomical analysis of the dense connectivity between frontal neocortex and limbic cortex, Nauta (1971) drew from Teuber's (1964) feedforward cybernetic model of "corollary discharge." Teuber speculated that, as the frontal lobe develops action plans, it sends an efferent copy of the action plan to sensory networks, creating an adaptive expectancy of the changing perceptual input to be caused by the action. In a similar fashion, Nauta proposed that the frontal lobe sends an efferent copy of the action plan to limbic networks, which, with their rich interconnections with hypothalamic and brainstem homeostatic centers, can evaluate the likely motivational significance of the action plan. Prepared by feedforward expectancy in this way, limbic networks can create motive expectancies that provide evaluative set-points for dynamic feedback regulation of the ongoing action sequence (Nauta 1971).

Recent studies of brain activity in relation to executive processes such as evaluation and planning have again raised the question of the neural mechanisms of self-regulation. We propose that the classical cybernetic approaches to self-regulation should be revisited in the light of new evidence. In the present paper, we review findings with both hemodynamic and electrophysiological measures showing activity in the limbic networks of the frontal lobe when subjects detect an error, when they receive feedback on their actions, or when they discriminate good from bad targets. Because this limbic activity varies with motivation and with the subject's emotional state, research with these measures promises new insights into neural mechanisms of adaptive self-regulation.

Concepts of self-regulation

Self-regulation describes how an organism adjusts the parameters of its own functions to meet the challenges of existence. This concept requires a cybernetic or feedback explanation, in that the causality of adjustment is internal or recursive, without the intervention of an external agent or controller. At a neurophysiological level, the self-regulatory mechanisms of basic physiological processes, such as body temperature, are understood relatively well (Satinoff 1978). Homeostasis, in Canon's original formulation, means "same state," describing the stabilization of internal states according to preset internal cues. In a strict sense, homeostasis is a concept that is appropriately applied to a limited number of systems, such as pH, body temperature, and oxygen tension (McEwen 2000).

Homeostatic systems are essential to life because small deviations from their set-points can lead to death. Homeostatic mechanisms respond to deviations from a set-point by initiating restorative responses to correct the deviations and return the system to its homeostatic set-point. Researchers studying self-regulatory mechanisms at this level have found that homeostatic mechanisms by themselves cannot explain the complexity of the observed phenomenon of self-regulation (Winn 1995).

Classically, the concept of "biased homeostasis" was used to explain the shifting of homeostatic set-points under varying motivational conditions (Pribram 1960). More recently, systems that show varying set-points with varying motivational states, such as the hypothalamic-pituitary-adrenal system, have been referred to as allostatic systems (McEwen 2000). Allostasis can be defined as the regulation of many variables (including behavioral as well as physiological variables) over time to meet motive set points that are estab-

lished dynamically. Allostasis can create an anticipatory form of self-regulation. Rather than just responding to deviations from fixed set-points, allostasis allows the organism to anticipate needs and to adjust configurations of regulatory goals in advance to meet those needs (Schulkin, McEwen, & Gold 1994; Winn 1995). In essence, allostasis incorporates learning and adaption into the process of homeostatic self-regulation.

Cognitive psychology describes the control of behavior with the construct of executive functions (sometimes also referred to as cognitive control processes). Executive functions include setting goals, planning, correcting errors, carrying out novel actions, inhibiting routinized behavior, and alerting to danger (Posner & DiGirolamo 1998; Posner & Rothbart 1998). Cognitive theories conceive of executive functions as higher-level processes that exert control over elementary mental operations (Norman & Shallice 1986). These executive functions include supervisory processes that must be engaged in situations in which well-learned behavior is inadequate.

When framed in this way, the construct of executive functions risks being disembodied, separated as an external cause rather than an integral, bodily, self-regulatory function. The problem of resorting to an external supervisor is well known in attention theory (Posner 1978), tending to imply a homunculus as the executive controller, rather than explaining the intrinsic self-regulation provided by basic neurophysiological and motivational mechanisms. Disembodied concepts of executive functions may be particularly inappropriate when dysfunctions of executive control are invoked to explain pathologies of mood and motivation. Rather than a disorder of an external supervisory control, these pathologies suggest fundamental alterations in the internal goals and set-points guiding self-regulation.

In this chapter we review electrophysiological and hemodynamic studies of an elementary component of the executive functions, action monitoring. We place action monitoring in the larger framework of action regulation, which involves correction and adjustment as well as monitoring. Within this framework, it becomes apparent that certain executive functions can be described as allostatic processes, cybernetic mechanisms that create expectancies for the outcomes of actions, evaluate the outcomes against the expectancies, and adjust coping efforts to deal with discrepancies that arise. Motivation, learning, and memory are integral to the process of action regulation. We will review evidence that implicate the anterior cingulate cortex (ACC) in those aspects of action regulation in which behavior is evaluated against expectations (represented as contexts for action). Electrophysiological responses are often observed when events (perceptual or motor) violate expectancies, consistent

with the view that brain networks operate through cybernetic servomechanisms, responding to the discrepancy between a target goal and the actual operation (Pribram 1960). In human action monitoring, one electrophysiological signal, the error-related negativity (ERN), is seen regularly when actions are discrepant from an intended goal representation. Because they appear to reflect the representation of motive set points, the ERN and related medial frontal negativities (MFNs) may provide important clues to the corticolimbic mechanisms of allostatic cybernetics. Although the ERN can be localized to the ACC, we will argue that this signal indexes multiple components of a limbic network. By understanding the function of this larger network, we can theorize how the executive psychological operations of conscious attention might emerge from more elementary, and largely preconscious, processes of corticolimbic self-regulation.

The anterior cingulate cortex in self-regulation

Of the brain structures involved in the executive functions, it was recognized early on that the frontal lobes played a critical role. Patients with frontal lobe lesions were often characterized as being changed and unable to manage their daily lives, often being uncharacteristically unconcerned about their mistakes (Rylander 1947). At first glance frontal lobe patients appear unimpaired because lesions to the frontal lobe do not usually alter performance on traditional exams of intellectual functions (Eslinger & Damasio 1985). However, in the context of their daily lives, when patients have to make their own choices and learn from their mistakes, they show striking failures. The inability to incorporate error information into ongoing planning operations has long been recognized to be a key deficit of the executive functions following frontal lesions (Konow & Pribram 1970; Luria & Pribram 1964).

More recent research has focused on the cingulate gyrus, which lies along the medial aspect of each hemisphere, in the control of executive processes. Early neuroimaging studies identified ACC activity in a variety of tasks, such as generating verbs for a given noun, or naming the color of a word (in which the word is also a name of a color) in the Stroop interference task (George et al. 1994; Pardo, Pardo, Janer, & Raichle 1990; Posner, Petersen, Fox, & Raichle 1988). These results were interpreted to indicate that the ACC is involved in executive attention, sometimes described as attention for action (Posner & Dehaene 1994). These neuroimaging findings are complemented by studies that show patients with ACC lesions perform poorly on tasks requiring execu-

tive control (Cohen, Kaplan, Moser, Jenkins, & Wilkinson 1999; Janer & Pardo 1991; Ochsner, Kosslyn, Cosgrove, Cassem, Price, Nierenberg et al. 2001).

Some recent theories of the ACC have shifted away from the view that the ACC is involved in strategic control. Rather, these theories posit that the ACC plays an evaluative role, that of detecting when control is required (Carter, Botvinick, & Cohen 1999; MacDonald, Cohen, Stenger, & Carter 2000; Ochsner et al. 2001). In a fMRI study using the Stroop interference task, MacDonald and colleagues (MacDonald, Cohen, Stenger, & Carter 2000) found that when subjects were instructed, prior to the presentation of the target word, to either name the word or name the color of the word, activity in the dorsolateral prefrontal cortex (DLPFC) differentiated between the two types of instructions. These authors found greater activity in the DLPFC in response to the instruction requiring subjects to name the color of the word, which would require more control because reading the word is considered more automatic. Moreover, activation of the DLPFC was related to subjects' reaction time. Activity in the ACC, in contrast, differentiated between the congruent and incongruent conditions (i.e., when the word was or was not the same color as the color it named). MacDonald et al. (2000) interpreted these findings to indicate that the ACC is involved in the evaluation of when cognitive control is required, whereas the DLPFC is involved in the strategic control of task performance.

The shift from conceptualizing the ACC's role as strategic control to conceptualizing it as evaluation is an important one. However, the nature of the evaluation is still a topic of intense debate. For several researchers, the issue has become whether the ACC is involved in evaluating when conflict occurs or evaluating when an error is committed. In the next sections we review the evidence on each position in turn, and we will suggest that neither is fully explanatory. Rather, we will propose that the ACC is part of a system that is involved in the representation of adaptive goals, i.e., motivational set-points. Activity in the ACC is strongly engaged when events are discrepant with these adaptive, emotionally-significant, goal representations.

Animal studies of action monitoring

In one of the earliest reports of error responsive neurons in primate cortex, Niki and Watanabe (Niki & Watanabe 1979) found cells in the ACC that appeared to code the consequences of action. They found cells that increased their firing when the animal made an error but not when the animal makes a correct re-

sponse. Moreover, these same error responsive cells increased their firing when an expected reward was omitted. Niki and Watanabe noted the existence of cells from other areas, such as the orbitofrontal cortex, that also respond to both errors and the omission of expected rewards.

Later studies by Rosenkilde et al. (Rosenkilde, Bauer, & Fuster 1981) and Thorpe et al. (Thorpe, Rolls, & Maddison 1983) also confirmed the existence of error-responsive cells in the orbitofrontal cortex. Rosenkilde and colleagues (1981) reported the existence of a class of cells whose activity was found to be related to the consequence of behavior. These authors noted that the crucial factor determining the response of these cells was reinforcement. Within this class of cells there are cells whose activity was specifically related to errors and non-reinforced but correctly responded to (i.e., extinction) trials. These latter cells showed an inhibitory response to trials in which an unexpected reward was given. Thorpe et al. (1983) reported very similar results for error responsive cells in the orbitofrontal cortex.

More recent studies have found dopaminergic cells in the ventral tegmental area (VTA) that are responsive to errors and unexpected rewards (Schultz, Dayan, & Montague 1997; Schultz & Dickinson 2000; Schultz, Tremblay, & Hollerman 1998). Schultz and associates found that VTA cells initially respond to primary rewards. After learning, the response to the primary reward is lost. Instead, these cells now respond to the reward-predicting stimuli. When errors are made, these cells show a phasic depression in activity at the time that the reward would be delivered. The fact that the cells show these opposite responses suggest that they receive two convergent but different inputs (Schultz, Apicella, & Ljungberg 1993).

Using a task in which performance parameters can be used to index learning, Gemba et al. (Gemba, Sasaki, & Brooks 1986) found that at the point of gaining insight into the nature of the task, error-related potentials can be recorded from the ACC when an error is committed, and the trial is thus not rewarded. This error potential is not apparent prior to this stage, and it is not apparent after this stage when animals become proficient at the task. This finding led Gemba and colleagues (1986) to propose that the ACC is involved in learning what to do in a motor task.

Human studies of action monitoring

The work of Falkenstein et al. (Falkenstein, Hohnsbein, Hoormann, & Blanke 1991) and Gehring et al. (Gehring, Goss, Coles, Meyer, & Donchin 1993) iden-

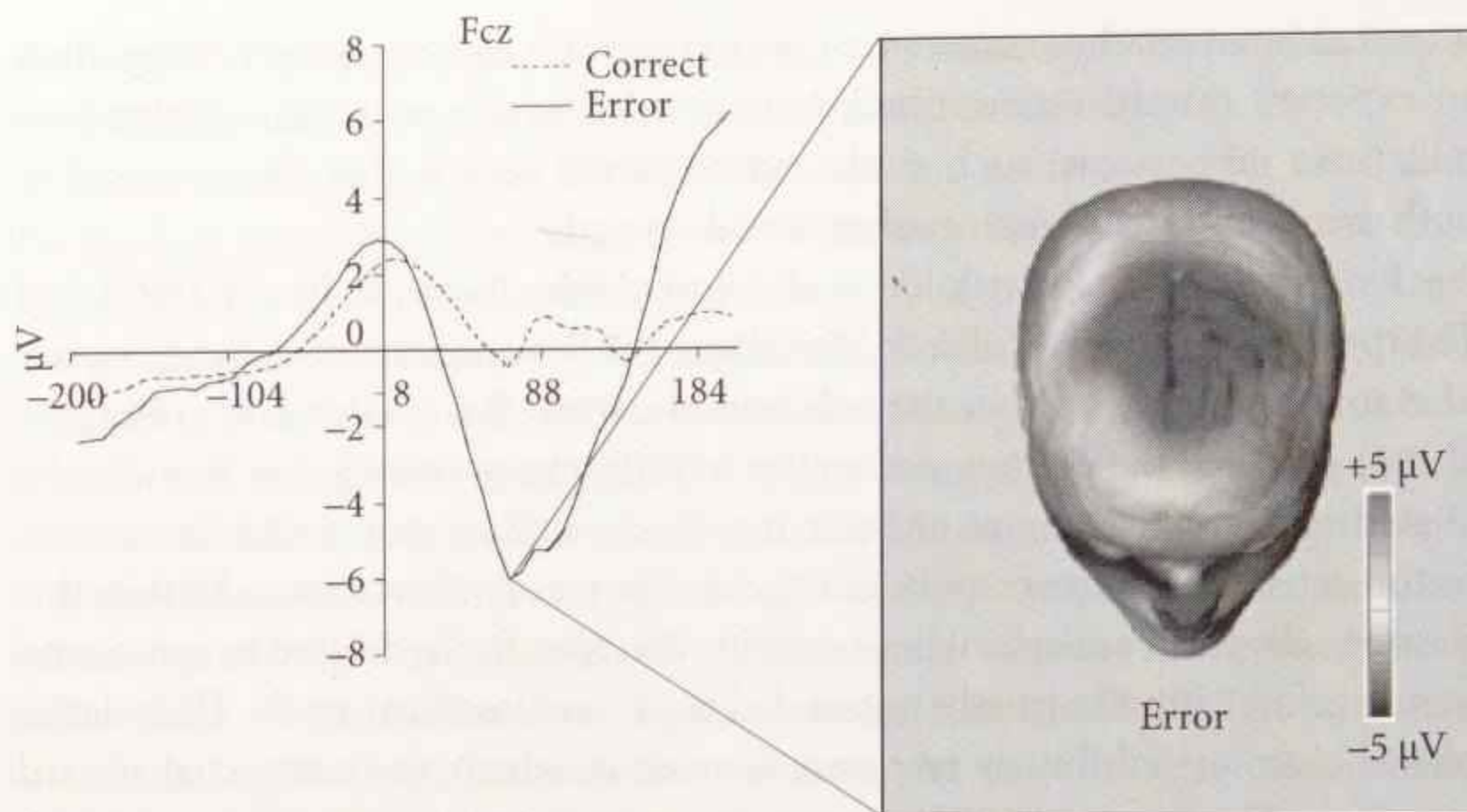


Figure 1. Left. The waveforms are for correct and error responses at electrode position FCz. The peak of the difference, approximately 80 ms after button press, is the ERN. Right. The scalp distribution of the ERN mapped on to a realistic head.

tified a response-locked component of the EEG that differentiated between correct and error responses in speeded response tasks. This component is referred to as the error negativity (Ne) or the error-related negativity (ERN). This component has a midline distribution (see Figure 1) and peaks approximately between 50 and 100 ms after a button press.

When first described, the ERN was thought to result from the automatic detection of a mismatch between the overt response and the outcome of the response selection process (Falkenstein et al. 1991). This detection of an error does not seem to depend upon sensory or proprioceptive information, because its onset is coincident with the movement. Rather, the detection may occur through some corollary discharge mechanism related to execution of the movement (Gehring et al. 1993; Kopp, Rist, & Mattler 1996). Later studies have identified the generator of the ERN to be located in the vicinity of the ACC (Dehaene, Posner, & Tucker 1994).

Because of the importance of such a signal to the understanding of human behavior and control, the discovery of ERN initiated many other studies that seek to further understand the nature of the ERN and the underlying neurophysiological system.

The ERN and action monitoring

The ERN is believed to index an output-independent, action monitoring system. The ERN is seen after hand (Falkenstein et al. 1991), foot (Holroyd, Dien, & Coles 1998), vocal (Masaki, Tanaka, Takasawa, & Yamazaki 2001), and saccadic eye movement errors (Van't Ent & Apkarian 1999). Execution of a wrong response is not necessary because slow responses, executed with the correct hand, also generate an ERN response (Luu, Flaisch, & Tucker 2000). The ERN appears to be unrelated to other error-related processes, such as inhibiting or correcting the erroneous response. For example, the ERN still occurs in situations in which errors cannot be corrected, such as pushing a button when a response is not required (Scheffers, Coles, Bernstein, Gehring, & Donchin 1996) or when the response is erroneous by a speed criterion (Luu et al. 2000).

One interpretation from these studies is that the important factor is discrepancy between what is expected in the context of the action and what is actually executed. This interpretation is consistent with a study by Stemmer et al. (Stemmer, Witzke, & Schönle 2001) who asked subjects to make purposeful mistakes. In this study, subjects were instructed to intentionally make mistakes in order to fool the experimenters. When these intentional mistakes were compared with unintended mistakes, the ERN was not apparent. According to the expectancy-violation interpretation, there was no ERN because the error was both expected and intended.

There are task situations in which discrepancies between action and correct outcome states are not so easily detected. In tasks in which subjects are required to estimate the passage of time (Miltner, Braun, & Coles 1997), to generate a response within a pre-specified interval (Badgaiyan & Posner 1998), or to guess the correct response (Ruchow, Grothe, Spitzer, & Kiefer 2002), the criterion for a correct response can not be readily determined. In these cases, erroneous responses do not generate the ERN. Rather, an ERN-like deflection can be observed in the stimulus-locked event-related potential (ERP) when an error feedback signal is presented. This negative deflection has a mediofrontal distribution and peaks between 250 and 350 ms after feedback onset. Like the response-locked ERN, the response is not modality specific: visual, auditory, or tactile feedback will elicit similar responses. Source analysis identified an ACC generator for this potential (Miltner et al. 1997). Therefore, it was believed that the response-locked ERN and the stimulus-locked error feedback potential are isomorphic (see Kopp et al. 1996). When error feedback is delayed from the mistaken action itself, discrepancy with the representation of the goal is not apparent until feedback is presented, consistent with the finding

that the ERN is then seen in response to the feedback signal, not the action (also see Brooks 1986).

We have observed midline negativities in several tasks requiring either monitoring of actions or the monitoring of feedback. Tucker et al. (Tucker, Hartry-Speiser, McDougal, Luu, & deGrandpre 1999) found that when subjects were given either a good or bad feedback signal, activity over mediofrontal sites, at approximately 350 ms, differentiated the two types of feedback. In addition, a similar medial frontal negativity (MFN) response discriminated between good and bad targets when they were initially presented (i.e., before the subject's response), suggesting the MFN reflected the initial target evaluation as well as the evaluation of feedback on the subject's performance. In a study in which subjects had to indicate whether a trait descriptor applied to them or not, activity at mediofrontal sites differentiated between good and bad trait-descriptive words approximately 300 ms after stimulus onset (Tucker, Luu, Desmond, Hartry-Speiser, Davey, & Flaisch 2003), suggesting that an evaluative discrimination formed the initial basis for semantic processing of the trait word. Gehring and Willoughby (Gehring & Willoughby 2002) used a choice task to determine the effects of decision outcomes on the ACC. Subjects were given a choice of either 5 or 25 points. After making their choice, they were provided with information that indicated whether subjects won or lost the amount of points they had just chosen. Gehring and Willoughby noted that a medial frontal negativity was sensitive to losses but not errors. Source analysis of the MFN placed its generator within the ACC.

In another study, we looked at the MFN to evaluative feedback (which was differentiated from feedback indicating correctness of response) and compared it to the ERN in the same subjects (Luu, Tucker, Derryberry, Reed, & Poulsen 2003). In this task, subjects were presented with a feedback signal (a letter grade) about their performance from a trial that was completed five trials previously. The feedback gave subjects information about the speed of their response and whether it was correct or incorrect. The delay of five trials was used to separate immediate control value of the feedback from its affective value. We found that a MFN with a peak around 250–350 ms post-stimulus differentiated between A, C, and F feedback (error feedback was not analyzed). The MFN was more negative for C and F feedback than for A. We then compared the source(s) of the ERN with the MFN in the same group of subjects. We found that the ERN was actually made up of two sources: one located in the rostral ACC and the other in the dorsal ACC or supplementary motor area (SMA). The MFN, in contrast, was made up of only the dorsal ACC/SMA source. Therefore, it appears that the stimulus-locked ERN (or MFN) and the response-locked

ERN are not the same, but rather they share an overlapping dorsal ACC/SMA generator. van Veen et al. (van Veen & Carter 2002) also found a dorsal ACC generator for the ERN and the stimulus-locked N2. However, these authors did not find a rostral ACC source unique to the ERN.

The source model of the ERN obtained in the Luu et al. (2003) study is consistent with source models of midline theta oscillations (Asada, Fukuda, Tsunoda, Yamaguchi, & Tonoike 1999). This is an important finding because an earlier study by Gevins et al. (A. S. Gevins et al. 1989) found that presentation of error feedback produced correlated EEG activity between mediofrontal and left-lateral frontal sites at the theta frequency, and this was absent when correct feedback was presented. In addition to the topographic similarity of this effect with the MFN, the time course is also very similar (occurring approximately 300 ms after stimulus presentation).

The ERN and conflict monitoring

Carter and colleagues (Carter, Braver, Barch, Botvinick, Noll, & Cohen 1998) put forth an alternative interpretation of the ERN. Carter et al. argued that rather than detection of an error per se, the ERN may reflect detection of conflict between competing responses. These authors propose that a condition of high response conflict will lead to errors, and that it is the conflict that is indexed by the ERN. Carter et al. conducted a fMRI study in which they found that both errors and correct responses in which response conflict was high were associated with increased activation in the same ACC region.

The proposal put forth by Carter et al. (Carter et al. 1998) has been tested extensively in the past several years, using both EEG and fMRI methodologies. Falkenstein et al. (Falkenstein, Hoorman, Christ, & Hohnbein 2000) found the ERN amplitude to be of equivalent size for tasks that have strong response conflict and those that have no response conflict. Similarly, when responses are sorted according to degree of response conflict, as measured by activation of motor cortices, the ERN was largest for errors with the wrong response hand, which had little or no response conflict (Luu, Flaisch et al. 2000). More recently, Swick and Turken (Swick & Turken 2002) tested this hypothesis by examining the effects of response conflict and errors on the EEG of a patient with a rostral ACC lesion. In this patient, the ERN was reduced when an error was committed. The ERN was found not to be responsive to the response conflict manipulation. Rather, Swick and Turken found a negativity at approximately 450 ms post-stimulus that varied with the conflict manipulation and was unaffected by the ACC lesion.

More direct examinations of the response conflict theory have come from fMRI studies. These are important studies because they can be compared directly to the report by Carter and colleagues (Carter et al. 1998). To date there have been at least five studies showing that regions within the medial frontal cortex are differentially activated by conflict and errors. Kiehl et al. (Kiehl, Liddle, & Hopfinger 2000) was the first to show, using a go/nogo paradigm, that conflict activated dorsal aspects of the ACC and that error uniquely activated more rostral aspects of the ACC. Menon et al. (Menon, Adelman, White, Glover, & Reiss 2001) also found that rostro-ventral ACC activation was specific to errors whereas caudal ACC activation was specific to conditions of high response conflict. These findings suggest that the rostral or ventral ACC region is uniquely active in response to errors, and that this activity is not attributable to increased response conflict on error trials, as originally found by Carter et al. (Carter et al. 1998). More recent findings help to clarify this apparent disparity.

Ullsperger et al. (Ullsperger & von Cramon 2001) found response conflict activated regions of the medial frontal cortex that extended from the pre-SMA all the way to the banks of the ACC (also see results of Menon et al. 2001). However, the rostral aspect of the ACC specifically activated in response to errors was located in the cingulate motor area (CMA). Two other studies have confirmed the finding by Ullsperger et al. Braver and colleagues (Braver, Barch, Gray, Molfese, & Snyder 2001) essentially replicated the results reported by Carter et al. (Carter et al. 1998) and found an additional source of activation for errors in the rostral ACC. Garavan et al. (Garavan, Ross, Kaufman, & Stein, submitted) found that the pre-SMA was active under conditions of high response conflict and more rostral aspects of the ACC were responsive to errors. Between these two regions was an area that was responsive to both conflict and errors.

How can the findings of Carter et al. (Carter et al. 1998) be resolved with these later findings? The first discrepancy is the responsiveness of pre-SMA cortex versus caudal ACC to response conflict. According to Garavan et al. (Garavan et al., submitted), the pre-SMA region identified in the later studies and the caudal ACC activation identified by Carter et al. are less than four mm apart, and it is likely that the activations of these two areas exhibit substantial overlap. The second discrepancy is the finding that both errors and conflict activate the same ACC region. This discrepancy is only apparent, given the finding that there is a rostral to caudal (and dorsal) axis in which conflict and error related processes overlap substantially.

As pointed out by Braver et al. (Braver et al. 2001), the region of the ACC identified in their study and the Carter et al. study was defined a priori, and

that this region happened to be located in a region that is responsive to both conflicts and errors. Braver et al. suggest that along the rostral-to-caudal axis of the ACC the subregions are not completely selective in their response to the experimental manipulation. Rather, these subregions seem to demonstrate degrees of functional specialization. That is, the rostral ACC was still somewhat responsive to conflict, but it was more strongly responsive to errors, and vice versa for the more caudal aspect of the ACC.

Disorders of self-regulation

Both schizophrenia and obsessive compulsive disorder (OCD) are characterized by alterations in the monitoring of personal actions. Certain symptoms in schizophrenia suggest the inability to accurately assign action to oneself. In schizophrenic patients with control delusions, the patient gets the feeling that his or her actions are somehow under the control of others (see C. D. Frith, Blakemore, & Wolpert 2000). In contrast, in OCD the patient has an accurate sense of ownership of personal thoughts and actions, but there is a monitoring deficit suggested by the symptom of over-checking (i.e., checking and rechecking) of actions. The checking behavior is compulsive, and the patient experiences anxiety if prevented from carrying out the compulsive behavior.

Monitoring deficits and schizophrenia

The first study to examine error monitoring deficits indexed by the ERN in schizophrenic patients was reported by Kopp and Rist (Kopp & Rist 1999). Kopp and Rist found that paranoid schizophrenic patients showed smaller ERN amplitudes after an error was committed, in comparison to controls or non-paranoid schizophrenic patients. Importantly, they found that paranoid patients do not make more errors than non-schizophrenic patients or controls, and paranoid patients also showed normal error correction behaviors (i.e., making a correct response after the incorrect response). This pattern of results suggested that the ERN was not related to behavioral compensation or awareness of errors.

A second study of the ERN in schizophrenic patients was reported by Mathalon et al. (Mathalon et al. 2002). Like Kopp and Rist (1999), these authors found that schizophrenic patients exhibited a smaller ERN to errors when compared to controls. They also found that RTs on trials after error trials do not differ between patients and controls, which also suggests that schizophrenic pa-

tients were equally aware of making an error. However, Mathalon et al. found that, in addition to the reduced ERN amplitude, schizophrenic patients also displayed an ERN like negativity after a correct response. Mathalon et al. referred to this potential as the correct-related negativity (CRN). The topography of the CRN and ERN were similar for the schizophrenic patients but not for the control subjects. However, without source analysis, it is difficult to determine whether the CRN and ERN are generated by the same neural source(s).

Two more studies followed the report by Mathalon et al. Bates and colleagues (Bates, Kiehl, Laurens, & Liddle 2002) found that the ERN is indeed smaller in schizophrenic patients, but they did not find a difference in the CRN for correct trials. These authors also found no behavioral differences (slower RTs after error responses) between patients and controls. These authors found that the amplitude of the ERN was significantly correlated with scores on a psychomotor poverty scale. Alain et al. (Alain, McNeely, He, Christensen, & West 2002) also found smaller ERN amplitudes in patients and a larger CRN for patients when compared to controls.

In summary, the finding of smaller ERN amplitudes in schizophrenia appears to be quite robust. Also quite robust is the finding that error correction and the Pe response (positivity after the ERN) is not faulty in schizophrenia. What is less certain are differences in CRN amplitudes. These findings appear to indicate that monitoring processes, but not error awareness or error compensating behaviors, are indeed impaired in schizophrenic patients. The results of these studies may be consistent with the hypothesis put forth by Frith and colleagues (C. D. Frith et al. 2000) that schizophrenia is characterized by a deficit in awareness of the predicted state. Schizophrenic patients do not lack the ability to represent the desired state of an action, nor are they deficient in the ability to determine whether a desired state has been achieved, but the reduced ERN suggests they may be impaired in some aspect of the prediction or monitoring of the action process.

Monitoring deficits and OCD

When compared to normal controls, obsessive-compulsive disorder (OCD) patients exhibit larger ERN amplitude (Gehring, Himle, & Nisenson 2000). Moreover, Gehring et al. found that OCD severity correlated with the amplitude of the ERN. The finding of an association between larger ERN amplitudes and OCD has been replicated twice with adult subjects and have been extended to children. The first replication was by Johannes and colleagues (Johannes, Wieringa, Nager, Rada et al. 2001), who found larger ERN amplitudes in OCD

patients. Additionally, these authors also found latency and topographic differences, with the ERN being later and distributed at more posterior sites in OCD patients than in controls. Hajcak and Simons (Hajcak & Simons 2002) studied university students who scored high on OCD measures. Like OCD patients, normal university students who scored high on the OCD measure exhibited larger ERN amplitude compared to university students with low OCD scores. In addition, the amplitude of the ERPs on correct trials was also larger for students with high OCD scores. A more recent study of OCD with 10-year old children have shown that ERN amplitude correlates with parents reports of OCD symptoms (Santesso, Segalowitz, & Schmidt, submitted).

Integral to OCD is the dysregulation of affect. OCD is not only a disorder of action monitoring but also of the affective evaluation of whether further monitoring is required. Therefore, an important question is whether the ERN is related to action monitoring or the affective consequences of action monitoring. Similarly, the smaller ERN amplitude found in schizophrenic patients may be related in some way to affective evaluation as well as to action monitoring directly. Bates et al. (Bates et al. 2002) found that within schizophrenic patients, psychomotor poverty was the only symptom that was significantly correlated with ERN amplitude. Bates et al. suggest the ERN findings may be specific to the emotional response associated with errors, because blunted affect is a characteristic of psychomotor poverty.

Thus, several findings from research on the ERN suggest that a theory of executive control must account for affective and motivational influences as well as specific processes of action monitoring and regulation. The evidence from neuroanatomy also demands such an integrative explanation, pointing to the integral connections of frontal networks with visceral and motivational circuits (see Allman, Hakeem, Erwin, Nimchinsky, & Hof 2001; Paus, Petrides, Evans, & Meyer 1993). Several lines of evidence point to activity in medial frontal networks in relation to affective evaluation specifically.

Affective control of action monitoring

Clear links to affective control are provided by evidence that the ACC is involved in the processing and representation of painful stimuli. The processing of painful stimuli can be separated into two distinct paths (Vogt, Sikes, & Vogt 1993). One leads to the somatosensory cortex, where information about the temporal and spatial extent of the stimulus is represented. The second leads to the ACC, where the aversiveness of the stimulus is represented. The ACC is

not involved in representing the specific location (either spatial or temporal) of the stimulus.

It is not obvious how the representation of pain can be integrated into the cognitive functions identified with the ACC. One explanation is that the ACC is involved in pain processing because painful stimuli require attention, which is known to activate the ACC. However, ACC activity appears to vary with the affective evaluation of painful stimuli specifically, increasing in subjects who appear to have a hyperemotional response, but not increased attention, to the stimuli (see Derbyshire, Vogt, & Jones 1998). On the other hand, the link between affect and attention may be inherently close.

These findings are consistent with several results suggesting that the ERN is influenced by the affective response to an error (Luu, Collins, & Tucker 2000; Luu, Flaisch et al. 2000; Pailing & Segalowitz, submitted; Vidal, Hasbroucq, Grapperon, & Bonnet 2000). The ACC is a limbic structure with rich connections to diencephalic structures (Pandya, Van Hoesen, & Mesulam 1981; Vogt, Pandya, & Rosene 1987), providing connectivity that is directly related to the role of the ACC in pain evaluation. It may be that affective evaluation of response outcomes occurs along the dimension of negative affect, similar to the evaluation of subjective distress induced by painful stimuli (Luu, Collins et al. 2000), and similar to other individual difference findings, such as the relation between ERN amplitude and OCD described above. In addition, Dikman and Allen (Dikman & Allen 2000) found that subjects who score low on a socialization scale exhibited small ERN amplitudes when their error responses were punished. In typical psychometric studies, low socialization subjects report low levels of negative affect (see Tellegen & Waller 1996).

The importance of negative affect in the evaluation of action outcomes has been supported by research on the effects of anxiolytic medication on ERN amplitude. Johannes and colleagues (Johannes, Wieringa, Nager, Dengler, & Münte 2001) compared the amplitude of the ERN in subjects who received an anxiolytic drug with those subjects who received a placebo. The anxiolytic drug decreased ERN amplitude but did not alter reaction time or increase errors. Johannes et al. concluded that their findings are consistent with the proposal by Luu et al. (2000). These findings of affective influences on the amplitude of the ERN are consistent with fMRI findings that show activation of the rostral ACC, which is engaged in emotionally-related tasks (Bush, Luu, & Posner 2000), when errors are committed (Garavan et al., submitted; Kiehl et al. 2000; Menon et al. 2001), and when subjects have to inhibit feelings associated with sexually explicit materials (Beauregard, Levesque, & Bourgouin 2001).

The MFN, like the ERN, is also responsive to affective manipulations. As reviewed previously, we found that when subjects are presented with evaluative (i.e., graded) feedback about their performance, negative feedback (e.g., Cs and Fs) elicited larger MFN amplitudes than positive feedback (Luu et al. 2003). More recently, we extended this work to examine self-monitoring in community subjects meeting diagnostic criteria for clinical depression (Tucker, Luu, Frishkoff, Quiring, & Poulsen, *in press*). The depressed subjects had larger MFN responses than controls, and their MFNs to the F grades were particularly striking.

Thus, a subject's emotional self-regulation appears to be indexed by the medial frontal electrophysiology of action monitoring, such that greater depression and anxiety are associated with greater MFN and ERN responses. From the perspective of neurocybernetic mechanisms, it may not be surprising that negative affect is integral to the detection of a discrepancy between a behavioral goal and an event outcome. In fact, we could conceptualize negative affect as the emotional self-regulatory state engaged when there is a significant deviance between events and the allostatic reference frame. The fact that subjects with higher anxiety and depression as their characteristic state have particularly strong MFNs in task monitoring suggests that negative affect may itself create a "homeostatic bias," such that deviations from represented goal states are particularly productive of a discrepancy response. This would be a form of allostatic regulation in which, rather than setting the reference goal at a certain level, the emotional state sets the magnitude of the response to the discrepancy.

In a similar fashion, negative affect may be important to the corticolimbic response that is indexed by the ERN. Detecting a discrepancy between a current event and the expected goal state may have implications not only for coping efforts but also for the immediate affective state. As they make errors in speeded tasks, subjects are often aware of the error as soon as it is initiated, and they often make vocalizations of irritation and frustration. The ERN may reflect the elicitation of negative affect in response to detecting a significant goal-action discrepancy.

Although the affective evaluation hypothesis of the ERN and MFN is novel, and it requires additional theoretical and empirical analysis, it may offer an important alternative to the error detection and conflict monitoring hypotheses. In the following sections, we present a model of ACC function in which affective control is central to self-regulation. Rather than separating the executive functions from affect and motivation, this model will argue that motivational

process is integral both to the control of simple actions and to the most complex forms of executive self-regulation.

A model of action-regulation

Systems for discriminative learning

A model of the ACC based solely on monitoring cannot account for the important findings that have implicated the ACC and related limbic structures in learning and memory. Although the medial temporal lobe, particularly the hippocampus, is widely known to be involved in memory operations (Squire & Zola-Morgan 1991), Mishkin (1982) recognized that the hippocampus is integral to only one of two memory circuits. Mishkin pointed out the existence of a second memory system centered on the amygdala. Gabriel and his colleagues (Gabriel 1990, 1993; Gabriel, Sparenborg, & Stolar 1986) have studied the contribution of both these memory circuits, and their thalamostriatal targets, to the learning process.

Previously, we have outlined a system for action regulation (Luu & Tucker 2002) based upon the work of Gabriel et al. (Gabriel, Burhans, Talk, & Scalf 2002). This model explains how affect and the executive functions are integral to the functioning of a medial frontal network. Rather than emphasizing only monitoring functions, the theory of action regulation emphasizes the role of the ACC in learning which action is relevant in a given motivational context, monitoring the outcome of that action, and switching to a different set of actions when outcomes are violated.

Gabriel et al. (Gabriel 1990, 1993; Gabriel et al. 1986) have demonstrated the role of the ACC circuit in discriminative avoidant learning (DAVL, see Figure 2). This circuit is made up of the ACC, amygdala, and the medial dorsal nucleus of the thalamus. The ACC is important to the early stages of discrimination learning, when rapid encoding of novel reinforcement contingencies is critical to adaptive action. Once the animal can reliably differentiate between a stimulus that predicts the delivery of an aversive unconditioned stimulus and one that does not, further learning is not dependent on the ACC circuit. Critical to the overall functioning of this circuit is the integrity of the amygdala. Lesion to or neurochemical inactivation of the amygdala will impair DAVL, but only in the early stages of learning (Poremba & Gabriel 1997, 1999, 2001). A theoretically important observation is that although this circuit is also involved in discriminative approach learning (DAPL), there appears to be a bias for

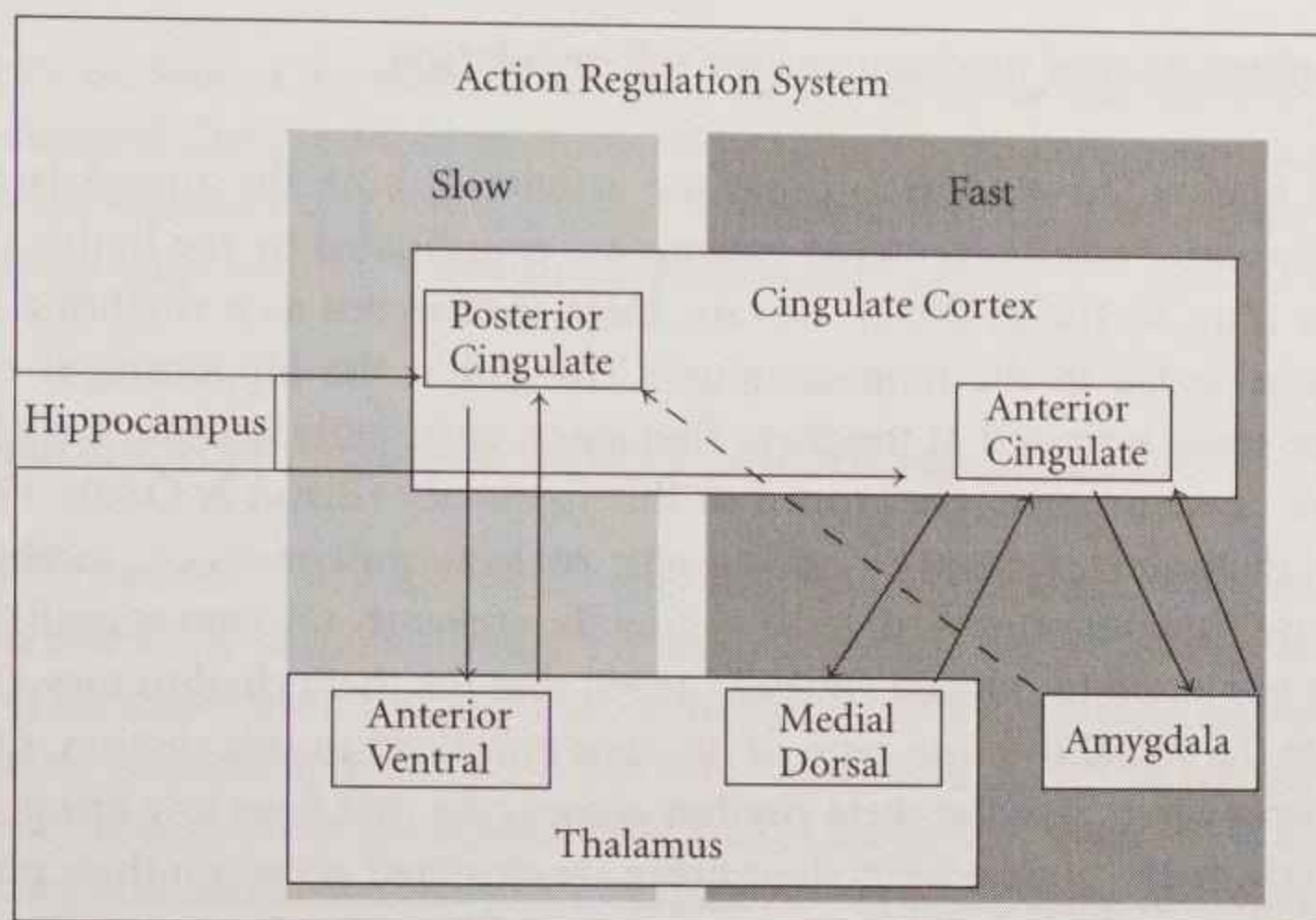


Figure 2. A model of the action regulation system based upon Gabriel's model of discriminant learning (see text for details).

learning to avoid aversive stimuli (Freeman, Cuppernell, Flannery, & Gabriel 1996; Gabriel et al. 2002). The neuronal responses that discriminate between predictive and non-predictive conditioned stimuli are weaker for DAPL compared to DAVL. Furthermore the learning rate for DAPL in the ACC circuit is much slower than that for DAVL.

In contrast to the ACC, the posterior cingulate cortex (PCC) is involved in later stages of discrimination learning. PCC cells are responsive to the novelty-familiarity dimension of the predictive conditioned stimulus. This suggested to Gabriel et al. (Gabriel et al. 2002) that the PCC is involved in selecting action based upon the environmental context, and that it does so with hippocampal input. The role of the hippocampal complex in action regulation is to compare inputs against an expected context for a certain action. In humans, neuroimaging studies have specifically identified hippocampal activation when expectations, particularly of an affective nature, are violated (Ploghaus, Tracey, Clare, Gati, Rawlins, & Matthews 2000).

Neurophysiological mechanisms for self-regulation

Several findings have suggested that the activity of both the amygdala/ACC and hippocampus/PCC centered systems are coordinated by the limbic theta rhythm (Luu & Tucker 2002). In rats, theta is observed as a rhythmic slow activity recorded in the hippocampus. Cells within the hippocampal complex are easily activated at the theta frequency, apparently because they have intrinsic resonant properties tuned to this frequency (Bland & Oddie 1998). During theta activity, most pyramidal cells are hyperpolarized (i.e., inhibited) and those that are active seem to be coding the currently relevant stimuli. This finding suggested to Buzsáki (Buzsáki 1996) that the theta rhythm may act to enhance the signal to noise ratio of neurons entrained to this rhythm. Other evidence suggests that the theta rhythm allows cells that have low firing rates to be activated in unison when their firing are clustered at certain theta phases (Paré, Collins, & Pelletier 2002).

These properties suggested to Buzsáki that theta is dominant in the “throughput mode” of neuronal transmission, when the animal is taking in new information (also see Miller 1991). In this mode, processing is centered on the input stages of the hippocampal circuit, such as the entorhinal complex and dentate gyrus. Consistent with this notion, Gabriel and colleagues (Gabriel et al. 1986) observed that, during the acquisition stages of discriminative learning, cells in the hippocampus display bursting at the theta frequency. In contrast, Gabriel et al. observed that, during the inter-trial period when the animal was quiescent, hippocampal cells show brief bursts of activity. This appears to reflect the consolidation stage of hippocampal function, in which irregular sharp waves are prominent in the hippocampus (Buzsáki 1989).

There is now evidence that theta activity can also be recorded in the amygdala and that this activity is phase locked to hippocampal theta activity (Paré et al. 2002). Paré et al. suggest that the amygdalar theta rhythm, like the hippocampal theta rhythm, is critical for the establishment of synaptic plasticity, a hypothesis that may provide an important link between the literature on hippocampal theta and the substantial evidence that important aspects of emotional learning and memory are mediated by the amygdala (Mishkin 1982; Packard & Cahill 2001).

If the action regulation system functions as an integrated system, in which the other structures are critically dependent on information from the hippocampus, one would expect that a major mode of transmission of neural information between the structures of the system would be at the theta frequency. An important finding in support of this is that bilateral lesions to the

anterior nucleus of the thalamus or the cingulate gyrus reduce the duration of hippocampal theta induced by electrical stimulation. Similarly, stimulation to either the cingulate cortex or the anterior thalamus induces theta activity in the ipsilateral hippocampus (Azzaroni & Parmeggiani 1967).

Human electrophysiology of corticolimbic self-regulation

In ERP research, it is a common belief that the components of the averaged ERP is due solely to transient EEG events that are locked to either the presentation of the stimulus or the response. Although this may be a valid assumption, it ignores the neurophysiological fact that endogenous rhythms (recorded either on the scalp or in the extracellular space) are inextricably tied to sensory, memory, and other cognitive processes (see Paré et al. 2002). Accordingly, there is increasing evidence for the view that components of the average ERP reflects phase-alignment of ongoing EEG rhythms (see Basar 1998; Basar-Eroglu, Basar, Demiralp, & Schürmann 1992; Demiralp, Ademoglu, Comerchero, & Polich 2001; Makeig et al. 2002).

We have found that, like certain other ERP components of the EEG, the ERN reflects, to a large degree, phase alignment of ongoing, midline, theta oscillations that are interleaved with motor potentials (Luu & Tucker 2001). Figure 3 illustrates that the ERN theta rhythm can be observed in single trials in several subjects. Independent components analysis also reveal similar results (Makeig et al., in preparation). As discussed earlier, source analysis of the ERN revealed solutions that are identical, in terms of location and phase relation of the different sources, to source solutions for midline theta (Luu et al. 2003).

In rats, hippocampal theta is seen when the animal is exploring its environment (Miller 1991). However, in primates the most reliable method for eliciting hippocampal theta is withholding an expected reward (Crowne & Radcliffe 1975). The amygdalar theta rhythm is observed clearly under conditions of intense arousal, such as when the animal is anticipating the delivery of a noxious stimulus. In humans, increased theta activity in the region of the ACC and temporal lobe is associated with increases in working memory demands (A. S. Gevins, Smith, McEvoy, & Yu 1997; Raghavachari et al. 2001; Slobounov, Fukada, Simon, Rearick, & Ray 2000). Superficially, these findings appear to be disparate, but recently Kahana et al. (Kahana, Seelig, & Madsen 2001) concluded that the most parsimonious description regarding the functional significance of theta across species is that it is related to cognitive control.

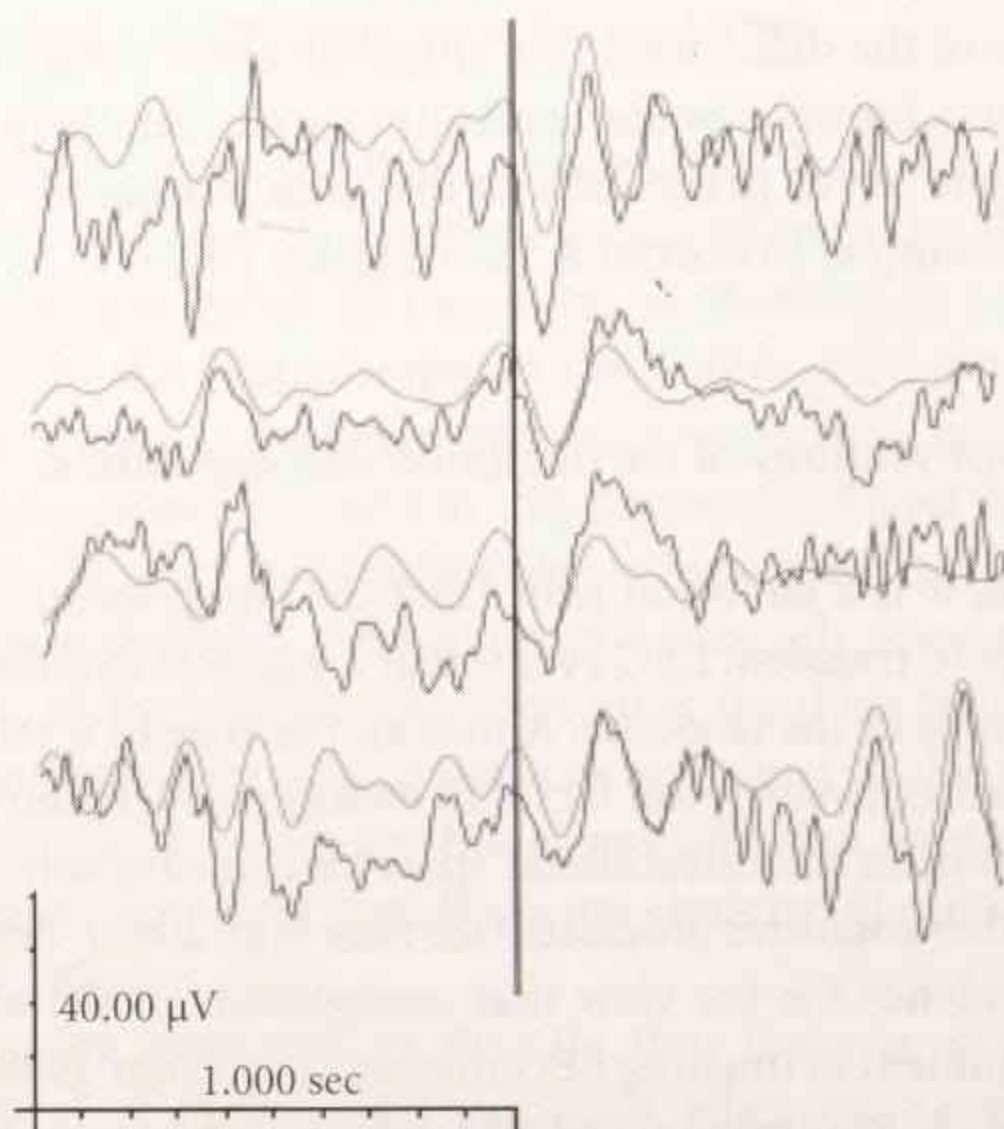


Figure 3. Single trial EEG from four different subjects. The data is shown for error trials only. The dark lines represent data filtered with a 30 Hz low pass and the light line represent the same data filtered with a 4–7 Hz (theta) band pass. The vertical line represent the time of the button press. Note the prominent negativity (ERN) immediately after the button press.

More specifically, we hypothesize that the theta rhythm reflects the control of information-gathering by the hippocampal and amygdalar/ACC systems. This information-gathering requires communication between multiple brain regions, but particularly within these two working memory systems engaged in the task of action regulation. The theta rhythm allows the incoming information to be integrated into permanent memory traces (Buzsáki 1996), according to its evaluated adaptive significance (Luu & Tucker 2002; Luu et al. 2003). The finding that the ERN is part of the theta rhythm is consistent with this idea; when errors occur, action must be adjusted and the information about the error must be integrated within the current contextual model to achieve effective learning. Given the localization of the ERN to the ACC, we hypothesize that the ERN indexes the coordinated activity of the amygdalar/ACC network that is involved in regulating action in the early stages of discriminative avoidance learning (Luu & Tucker 2002; Luu et al. 2003).

Phase resetting of the theta rhythm by dopaminergic signals

The phase alignment of the theta rhythm to the erroneous response, which gives rise to the appearance of the ERN in the average ERP, is likely signaled by a dopaminergic response from the VTA (Luu & Tucker 2002). Gabriel and Taylor (1998) found that dopamine played a specific role in discriminative learning. When the conditioned stimulus is made difficult to detect by reducing its duration, ACC cells show an enhancement in their firing. Importantly, prior to this enhancement there is a brief pause (40–80 ms) in the activity of these cells. In animals exposed to cocaine in utero, resulting in altered DA function, this pause of firing is absent, and the animals show DAVL deficits in the early phases of learning. The firing pause observed by Gabriel and Taylor (1998) may act as a resetting mechanism that allows for the recruitment of neurons to fire together, perhaps at certain phases of theta. This pause appears to be similar of the pause function identified by Brazhnik et al. (Brazhnik, Vinogradova, & Karanov 1985), who demonstrated that one mechanism of theta phase resetting is a 40–90 ms pause in neuronal activity after an electrical stimulus delivery.

Integrating the Schultz evidence of dopamine coding of prediction errors with the human evidence of error-related negativities in the anterior cingulate cortex, Holroyd and Coles (2002) have recently proposed that the ERN reflects the transmission of an error signal from brainstem dopaminergic cells to the ACC. The finding of ERN amplitude differences in patients with Parkinson's disease, which is a disease brought on by damage to dopaminergic cells, would provide support for this proposal. There have been two published studies that have examined how ERN amplitudes are altered in patients with Parkinson's disease. Falkenstein et al. (Falkenstein et al. 2001) found reduced ERN amplitudes in patients with Parkinson's disease whereas Holroyd et al. (Holroyd, Praamstra, Plat, & Coles 2002) did not. The negative findings from the later study were interpreted as being due to the subject population; the patients studied were in the early stages of Parkinson's disease where dopaminergic systems are not as compromised.

Direct evidence for the role of dopaminergic involvement in error processing in animals has recently been reported by Morgan et al. (Morgan, Garavan, Mactutus, Levitsky, Booze, & Strupp 2002). Similar to the findings by Gabriel and Taylor (1998), Morgan et al. found that animals with prenatal exposure to cocaine demonstrated poor performance when cues were presented for a very brief period. In particular, cocaine-exposed rats made significantly more errors, as predicted by the findings of Gabriel and Taylor. Paralleling the findings in humans of slower responses on those trials following correct tri-

als, Morgan et al. found that when rats make an error, they are likely to make omission errors on the trials following the error trials. In the cocaine-exposed rats, this effect was blunted. Moreover, when these rats do make a response, it is likely to be an error. These several findings suggest that integrity of the dopaminergic system is critical to correct performance under conditions in which discrimination of environmental cues is difficult. Very likely, the salience-compensation mechanism that is engaged under difficult conditions contributes to action adjustments (e.g., error compensating behaviors) when errors are made.

Opiates and the theta rhythm: Modification of the signal-to-noise ratio

If the limbic mechanisms of action regulation are adjusted in an allostatic fashion, with motivational states such as negative affect increasing the gain for discrepancy detection, we might expect that modulation of the organism's affective state would tune both the limbic mechanisms and the response to negative feedback. The response to pain provides an important example of allostatic stress, and the modulation of this response by endogenous opiates may provide a model of self-regulation of allostatic mechanisms.

It has been observed that cells in the medial septum are especially responsive to noxious stimulation, whether mechanical or thermal (Dutar, Lamour, & Jobert 1985). The receptive fields of these cells are broad, a characteristic feature of cells found in the medial pain system that encode affective significance of painful stimuli (Vogt et al. 1993). The pain-responsive cells of the medial septum project to the hippocampus to produce changes in the activity of pyramidal neurons. Lesions of the medial septum, or administration of a cholinergic antagonist, abolish this pain-induced effect (Khanna 1997).

In the hippocampus, painful stimuli produce a depression of the population spikes with an accompanying increase in theta activity (Khanna 1997). This effect results specifically from medial septal projections, which act on the GABAergic hippocampal interneurons. The GABAergic interneurons inhibit most of the pyramidal cells, resulting in broad suppression of population spikes accompanied by increases in theta activity. In the midst of this inhibitory influence, the activity of a small population of pyramidal neurons is enhanced, giving rise to a signal-to-noise increase that may facilitate specific processes of associative learning.

Examining the effects of several neuromodulators on the theta rhythm, Vanderwolf et al. (Vanderwolf, Kramis, & Robinson 1978) discovered that only

opiates were effective at attenuating hippocampal theta activity (but see results by Miura, Ito, & Kadokawa 1987). Recently, Khanna and colleagues (Khanna 1997; Khanna & Zheng 1998; Zheng & Khanna 1999) showed that intraperitoneal injections of opiates attenuate the hippocampal nociceptive response (i.e., the characteristic population suppression and increase in theta activity in response to a painful stimulus). Furthermore, this effect was naloxone-reversible, suggesting that it reflects a specific opiate mechanism. Opiates appear to reduce hippocampal nociceptive response either by inhibiting the GABAergic hippocampal interneurons (Zieglagänsberger, French, Siggins, & Bloom 1979) or by inhibiting the function of the medial septum itself.

Injection of β -endorphine into the medial septal area impairs performance on a spatial task that requires alternating positions of reward location (Wan, Givens, & Olton 1995). This task situation may be similar to manipulations requiring new learning and salience compensation studied by Gabriel et al, in that the animal must shift to the previously unrewarded location in order to obtain a reward. In well-learned tasks that do not require shifting of contingencies, opiates do not impair performance unless administered in the early stages of learning. Wan et al. (1995) found that the performance decrement to contingency shifts was directly correlated with decreases of hippocampal theta produced by opiate injection. In contrast, theta activity within the amygdala appears to be important for avoidance learning, but not for the types of spatial learning mediated by the hippocampal system.

It is well known that drugs such as cocaine and heroin have powerful mood altering properties. Their effects on the theta rhythm illustrate how affect and cognition are inextricable. For example, a recognized analgesic effect of the opiates is that it does not affect the localization of the painful stimulus. Rather, when opiates are given, it acts to blunt the emotional response to the pain (Kupers, Konings, Adriaensen, & Gybels 1991). This blunted affect can have the behavioral expression of apathy or indifference to the pain. The neurophysiological equivalent of this behavioral (or cognitive) expression is the reduction of the signal-to-noise ratio in the amygdalar/ACC and septohippocampal circuits.

Cybernetics of motivational congruence

We propose that a cybernetic approach can provide an embodied model of self-regulation. This approach can integrate both cognitive and emotional influences, and it can integrate both neurophysiological and neuroanatomical

evidence. This model proposes that the amygdalar/ACC network, as outlined in Figure 2, is involved in action regulation (i.e., is responsible for discriminative learning), and not just action evaluation. The ACC, with substantial input from the amygdala and associated ventral limbic networks, forms a limbic representation of a motivational expectancy, the goal state, that serves as the evaluative reference, the set-point, for planning and monitoring action (Nauta 1971; Pribram 1960). The negativities seen in medial frontal cortex reflect the detection of an incongruence between events (actions or perceptions) and the motive set-point and the resulting coordination of the entire network.

Although the ACC appears to have a central role in both monitoring for prediction discrepancies and motivating corrective actions, it is one element of the telencephalic circuits for action regulation. The dopaminergic control of the ACC and frontolimbic cortices, for example, is closely related to regulation of the basal ganglia (Redgrave, Prescott, & Gurney 1999). Within the limbic system, the amygdala is also important to modulating functions of the basal ganglia in both motivational and motor control. Although human EEG studies have focused on the ACC, after reviewing the Gabriel findings we have been impressed with the importance of the PCC, with its strong hippocampal influence and its interconnectivity with the Papez circuit, in the maintenance of the expectancy for the current context, representing both motivational and perceptual qualities. Although both Pribram (1960) and Nauta (1971) emphasized that frontal cortex (and thus the ACC) have the major input from limbic cortex, it is clear that posterior cortex (and thus the PCC) has limbic territories as its major projection zones, and that the memory operations of the posterior brain are regulated by corticolimbic interactions just as those of the anterior brain. Therefore monitoring of motive discrepancies by the ACC must be considered in relation to the qualitatively different monitoring and updating of the context-expectancy model of the PCC and associated posterior cortical networks.

By representing expectancies for the motivational outcome of events and actions, both PCC and ACC contribute to action regulation as an allostatic process. As reviewed above, allostasis describes how physiological set-points vary to meet anticipated demands (McEwen 2000; Schulkin et al. 1994). Rather than passively responding to changes in set-points, allostatic systems anticipate and recruit both physiological and behavioral control to anticipate and meet the challenges posed by incongruence of events with motive set-points. Although feedback controls are integral to detection and correction of incongruence, an allostatic system operates with feedforward control, in that anticipated challenges to set-points can motivate behavior in advance of the challenges.

Furthermore, the motive set-points can themselves be changed not only in response to internal needs, but to allow strategic processes of self-regulation. By adjusting the cybernetics (feedforward and feedback) dynamically, the allostatic system achieves greater flexibility than a homeostatic system in which set-points are fixed and behavior is rigidly motivated in response to set-point deviations.

To develop an adequate model of motivational cybernetics, it will be important to integrate the full range of allostatic physiological responses, including those of the hypothalamic-pituitary-adrenal (HPA) axis, which regulate multiple physiological states including glucocorticoid responses in stressful situations. As the cortical representation of the visceromotor control system (Neafsey, Terreberry, Hurley, Ruit, & Fryszak 1993), the ACC is involved in the inhibitory control of the HPA stress response. The glucocorticoids are central to this control function (Diorio, Viau, & Meaney 1993). Moreover, the interactions between the opiates and glucocorticoids are fundamental to the function of the HPA axis.

We propose that the amygdalar/ACC action regulation system reflects the encephalization of homeostatic motivational systems of the mesencephalic brainstem and the diencephalic hypothalamus. The encephalization of self-regulatory mechanisms, taking over of function by more recently evolved systems, allows for complexity in behavior not seen in the more primitive self-regulatory systems (Jackson 1931). As the visceromotor and viscerosensory functions were encephalized by the ACC and insular cortices respectively, they provided not only elementary physiological self-regulation, but more complex representations of motivational goals to guide memory consolidation and action planning (Tucker 2001). Even in complex decision processes, detection of motive incongruence by the ACC may be integral to the evaluative control of the executive functions.

The error and conflict monitoring theories of ACC function may be too narrow because they cannot consider how motivation, goals, and context contribute to adaptive adjustments. Responses by cells in ACC and orbitofrontal cortex are not limited to errors; these cells are also responsive to the omission of reward. Similarly, ACC cells that are responsive to noxious stimuli also anticipate the delivery of a noxious stimulus (Hutchinson, Davis, Lozano, Tasker, & Dostrovsky 1999; Koyama, Tanaka, & Mikami 1998), consistent with the idea that representations of expectancies are central to the function of the amygdalar/ACC network. When developed in encephalized form in cortical networks, allostatic set-points may become motivational expectancies. The brains of higher vertebrates are not passive and reflexive, but continually

generate predictions with the goal of controlling the interaction with the environment. Within the hierarchy of corticolimbic networks (Tucker 2001), the limbic cortices are unique in receiving projections from hypothalamic homeostatic centers (Swanson 2000) and are thus the only regions of cortex directly informed on the animal's internal state. In the representation of motive set-points in conceptual form in human self-regulation, we can expect limbic networks to be essential. What is the nature of these representations?

Although it may be natural in a psychological analysis to assume that motives are separate from actions, this assumption may prove difficult in a neurophysiological analysis. The motive set-point representations arise in visceromotor cortex, and this cortex (the ACC) sits at the base of the somatomotor cortex (SMA, CMA) of the frontal lobe. In the representation of set-points, behavior and motive representations may be inseparable. Illustrative of this inseparability are findings in studies in which reward reductions serve as cues for behavioral change. In one paradigm, when a reward for a particular behavior is reduced, the animal must change its behavior in order to obtain reward at the previous level. Shima and Tanji (Shima & Tanji 1998) found cells in the cingulate motor area (CMA) that respond to changes in reward level only when the change in reward was accompanied by a corresponding behavior change. If the reward reduction was not accompanied by a behavior change, the cells were not responsive. Deactivation of the CMA prevented the expected behavioral shift after a reward reduction. Bush et al. (Bush et al. 2002) used the same paradigm to study ACC activation in humans. Similar to the results described by Shima and Tanji, Bush et al. (2002) observed CMA activation associated with reward reduction and behavioral change. This activation was above the activation level obtained for a non-reward related behavioral switch cue.

The behavior that is represented integral to motive set-points is likely to be amodal, broad, and abstract. Clues to the nature of representations of the behavior in set-points come from findings that have shown that the ERN can be elicited by hand, foot, eye, and vocal errors and that the MFN can be elicited by visual, auditory, and tactile feedback. Tasks that require subjects to attribute intentions to the behaviors of others, or that require subjects to infer the affective and motivational states of others, activate the same ACC regions (primarily the rostral ACC) as those tasks that require subjects to monitor their own actions (U. Frith & Frith 2001), such as error monitoring. Therefore, the representation of behavior integral to the motive set-point may not be a concrete personal action, but a more general representation of the concept of an action (i.e., the goal act).

Another clue comes from the suggestion that there may be a distribution of discrepancy or incongruence representations across multiple divisions of the ACC. Studies have shown that along the rostral-to-caudal axis of the ACC, there is a transition between regions responsive to errors and those regions that are responsive to conflict, with substantial overlapping responses by those cortical regions in intermediate positions (Braver et al. 2001; Garavan et al., submitted). These findings tell us that there exists the possibility of many set-points involved in action regulation. Elliot and Dolan (1998) conducted a study in which subjects had to generate a hypothesis about stimulus properties that would predict a correct choice. They found that generation of a hypothesis resulted in activation of the dorsal ACC. In contrast, making a choice resulted in activation of the ventral ACC. Similarly, we find that there is a response-locked component of the ERN located in the rostral ACC and another component located in the dorsal ACC (Luu et al. 2003). The activity of this latter source appears to be stimulus-locked, and is uniquely responsive to reward feedback.

We take these findings to indicate that the representation of motive-action set-points occurs along the entire axis of the cingulate cortex. These representations may progress from action represented as intention and goals all the way up to action as represented in context. When represented in context, and thus integrated with the perceptual context model of the PCC, the motive-action-idea is then prepared to lead to motor outflow (Brown 1987). At the level of the ventral ACC, behavior representation is amodal, and as one moves along the ACC axis towards the more dorsal region, the set-points seem to incorporate the representation of the context of action. This later representation is still behavior-based in that responses are inseparable from the context. For example, the representation of response conflict is a representation of the context of both the task demands and the conflicting responses engendered by the task. Similarly, the dorsal ACC activation that we found for the MFN in response to performance feedback can be thought of as a response to incongruence of the remembered response (motive set-point) from the now-updated task context.

This line of reasoning can be used to interpret the ERN findings, the findings of MFN responses to loss and negatively valenced feedback stimuli (Gehring & Willoughby 2002; Luu et al. 2003; Tucker et al. 1999; Tucker et al., in press), and the results on ACC contributions to discriminant avoidance learning in animals (Gabriel et al. 2002). The ERN response indexes a deviation from the motive set-point (i.e., a reduction in the reward). In the case of negative feedback, the set-point represents performance at a level that emphasizes good performance. Therefore, negative feedback (such as a C or an F) is perceived as incongruous with this motive expectation. Under dynamic

task conditions, the set-points represented in the amygdalar/ACC network can be constantly changing. By definition, allostatic set-points are not rigid; they can vary with the state of the individual. In discriminant avoidance learning, associating the appropriate response with the predictive conditioned stimulus integrates the new perceptual context cue as an element of the motive set-point for behavior.

Further research may show how discrepancies from set-points trigger the theta response, and how this response coordinates the function of the entire amygdalar/ACC network to adjust behavior to meet the set-point, or to update the set-point to a new expectancy, thus establishing dynamic adjustments to memory circuits according to motivational realities. Although we have emphasized the influence of motivational factors in establishing set-points, the response of the amygdalar/ACC network to detection of incongruence may itself be aversive. Therefore, it may not be a coincidence that the amygdalar/ACC network is integral to the evaluation of pain. Very likely the pain-evaluation mechanisms of visceromotor cortex have been elaborated in vertebrate evolution to evaluate incongruence of outcomes and expectancies generally.

Summary

We started the chapter with a brief discussion of self-regulation and how it may be conceptualized in relation to homeostasis, allostasis, and executive control. Executive control is the dominant construct in cognitive psychology used to study self-regulation and its disorders. Of the executive control functions, action monitoring has been extensively studied during the past decade, particularly after the discovery of an electrophysiological signal that appears to be a specific response to an error. An important task for this line of research, as for cognitive neuroscience generally, is to integrate concepts of motivational control. There are several findings in the error-monitoring literature suggesting that emotional variables alter the process of action monitoring. We have made suggestions about how these findings can be interpreted in light of the abundant findings from animal studies showing motivational control of learning mechanisms.

By emphasizing the integral relation between representations of allostatic set-points and the regulation of actions, we have attempted to apply classical concepts of cybernetics to interpret the recent findings of limbic contributions to self-regulation. Over thirty years have passed since Pribram (1960) and Nauta (1971) emphasized the limbic base of the frontal executive func-

tions. We now have powerful new tools for measuring the component brain mechanisms, and for clarifying how these mechanisms contribute to the process of self-regulation. It may be time to revisit classical theorizing on the control theory of brain systems, so that cognitive neuroscience can proceed beyond mapping isolated mental operations to address the self-regulation of the whole organism.

Acknowledgements

This work was supported by National Institutes of Mental Health grants #MH42129 and #MH42669 and an Augmented Cognition project grant funded by DARPA to Don M. Tucker.

Address correspondence to: Phan Luu, Electrical Geodesics, 1600 Millrace Dr. Suite 307, Eugene, OR 97401, Ph: 541-687-7962, Fax: 541-687-7963, email: pluu@oregon.uoregon.edu

References

- Alain, C., McNeely, H. E., He, Y., Christensen, B. K., & West, R. (2002). Neurophysiological evidence of error-monitoring deficits in patients with schizophrenia. *Cerebral Cortex*, 12, 840-846.
- Allman, J. M., Hakeem, A., Erwin, J. M., Nimchinsky, E., & Hof, P. (2001). The anterior cingulate cortex: the evolution of an interface between emotion and cognition. *Annals of the New York Academy of Sciences*, 935, 107-117.
- Asada, H., Fukuda, Y., Tsunoda, S., Yamaguchi, M., & Tonoike, M. (1999). Frontal midline theta rhythms reflect alternative activation of prefrontal cortex and anterior cingulate cortex in humans. *Neuroscience Letters*, 274, 29-32.
- Azzaroni, A. & Parmeggiani, P. L. (1967). Feedback regulation of the hippocampal theta-rhythm. *Helvetica Physiologica et Pharmacologica Acta*, 25, 309-321.
- Badgaiyan, R. D. & Posner, M. I. (1998). Mapping the cingulate cortex in response selection and monitoring. *Neuroimage*, 7, 255-260.
- Basar, E. (1998). *Brain function and oscillations*. Berlin: Springer.
- Basar-Eroglu, C., Basar, E., Demiralp, T., & Schürmann, M. (1992). P300-response: possible psychophysiological correlations in delta and theta frequency channels. A review. *International Journal of Psychophysiology*, 13, 161-179.
- Bates, A. T., Kiehl, K. A., Laurens, K. R., & Liddle, P. F. (2002). Error-related negativity and correct response negativity in schizophrenia. *Clinical Neurophysiology*, 113, 1454-1463.
- Beauregard, M., Levesque, J., & Bourgouin, P. (2001). Neural correlates of conscious self-regulation of emotion. *Journal of Neuroscience*, 21 (RC165), 1-6.

- Bland, B. H. & Oddie, S. D. (1998). Anatomical, electrophysiological and pharmacological studies of ascending brainstem hippocampal synchronizing pathways. *Neuroscience and Biobehavioral Reviews*, 22, 259–273.
- Braver, T. S., Barch, D. M., Gray, J. R., Molfese, D. L., & Snyder, A. (2001). Anterior cingulate cortex and response conflict: effects of frequency, inhibition and errors. *Cerebral cortex*, 11, 825–836.
- Brazhnik, E. S., Vinogradova, O. S., & Karanov, A. M. (1985). Frequency modulation of neuronal theta-bursts in rabbit's septum by low-frequency repetitive stimulation of the afferent pathways. *Neuroscience*, 14, 501–508.
- Brooks, V. B. (1986). How does the limbic system assist motor learning? A limbic comparator hypothesis. *Brain, Behavior and Evolution*, 29, 29–53.
- Brown, J. W. (1987). The microstructure of action. In E. Perceman (Ed.), *The frontal lobes revisited* (pp. 251–272). New York: The IRBN press.
- Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences*, 4, 215–222.
- Bush, G., Vogt, B. A., Holmes, J., Dale, A. M., Greve, D., Jenike, M. A., et al. (2002). Dorsal anterior cingulate cortex: a role in reward-based decision making. *Proceedings of the National Academy of Sciences*, 99, 523–528.
- Buzsáki, G. (1989). Two-stage model of memory trace formation: A role for "noisy" brain states. *Neuroscience*, 31, 551–570.
- Buzsáki, G. (1996). The hippocampal-neocortical dialogue. *Cerebral Cortex*, 6, 81–92.
- Canon, W. B. (1915). *Bodily changes in pain, hunger, fear, and rage*. New York: Appleton.
- Carter, C. S., Botvinick, M. M., & Cohen, J. D. (1999). The contribution of the anterior cingulate cortex to executive processes in cognition. *Reviews in Neuroscience*, 10, 49–57.
- Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, 280, 747–749.
- Cohen, R. A., Kaplan, R. F., Moser, D. J., Jenkins, M. A., & Wilkinson, H. (1999). Impairments of attention after cingulotomy. *Neurology*, 53, 819–824.
- Crowne, D. P. & Radcliffe, D. D. (1975). Some characteristics and functional relations of the electrical activity of the primate hippocampus and a hypothesis of hippocampal function. In R. L. Isaacson & K. H. Pribram (Eds.), *The hippocampus: neurophysiology and behavior*, Vol. 2 (pp. 185–206). New York: Plenum.
- Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a neural system for error detection and compensation. *Psychological Science*, 5, 303–305.
- Demiralp, T., Ademoglu, A., Comerchero, M., & Polich, J. (2001). Wavelet analysis of P3a and P3b. *Brain Topography*, 13, 251–267.
- Derbyshire, S. W. G., Vogt, B. A., & Jones, A. K. P. (1998). Pain and stroop interference tasks activate separate processing modules in anterior cingulate cortex. *Experimental Brain Research*, 118, 52–60.
- Dikman, Z. V. & Allen, J. J. B. (2000). Error monitoring during reward and avoidance learning in high and low-socialized individuals. *Psychophysiology*, 37, 43–54.
- Diorio, D., Viau, V., & Meaney, M. J. (1993). The role of the medial prefrontal cortex (cingulate gyrus) in the regulation of hypothalamic-pituitary-adrenal responses to stress. *Journal of Neuroscience*, 13, 3839–3847.

- Dutar, P., Lamour, Y., & Jobert, A. (1985). Activation of identified septo-hippocampal neurons by noxious peripheral stimulation. *Brain Research*, 328, 15–21.
- Elliot, R. & Dolan, R. J. (1998). Activation of different anterior cingulate foci in association with hypothesis testing and response selection. *Neuroimage*, 8, 17–29.
- Eslinger, P. J. & Damasio, A. R. (1985). Severe disturbance of higher cognition after bilateral frontal lobe ablation. *Neurology*, 35, 1731–1741.
- Falkenstein, M., Hielscher, H., Dziobek, I., Schwarzenau, P., Hoormann, J., Sundermann, B., et al. (2001). Action monitoring, error detection, and the basal ganglia: an ERP study. *Neuroreport*, 12, 157–161.
- Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. *Electroencephalography and clinical Neurophysiology*, 78, 447–455.
- Falkenstein, M., Hoorman, J., Christ, S., & Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: a tutorial. *Biological Psychology*, 51, 87–107.
- Freeman, J. H., Jr., Cuppernell, C., Flannery, K., & Gabriel, M. (1996). Limbic thalamic, cingulate cortical and hippocampal neuronal correlates of discriminative approach learning in rabbits. *Behavioral Brain Research*, 80 (1–2), 123–136.
- Frith, C. D., Blakemore, S.-J., & Wolpert, D. M. (2000). Explaining the symptoms of schizophrenia: abnormalities in the awareness of action. *Brain Research Reviews*, 31, 357–363.
- Frith, U. & Frith, C. D. (2001). The biological basis of social interaction. *Current Direction in Psychological Science*, 10, 151–155.
- Gabriel, M. (1990). Functions of anterior and posterior cingulate cortex during avoidance learning in rabbits. *Progress in Brain Research*, 85, 467–483.
- Gabriel, M. (1993). Discriminative avoidance learning: a model system. In B. A. Vogt & M. Gabriel (Eds.), *Neurobiology of the cingulate cortex and limbic thalamus* (pp. 478–523). Boston: Birkhäuser.
- Gabriel, M., Burhans, L., Talk, A., & Scalf, P. (2002). Cingulate cortex. In V. S. Ramachandran (Ed.), *Encyclopedia of the human brain* (pp. 775–791). Elsevier Science.
- Gabriel, M., Sparenborg, S. P., & Stolar, N. (1986). An executive function of the hippocampus: pathway selection for thalamic neuronal significance code. In R. L. Isaacson & K. H. Pribram (Eds.), *The hippocampus* (pp. 1–39). New York: Plenum.
- Gabriel, M. & Taylor, C. (1998). Prenatal exposure to cocaine impairs neuronal coding of attention and discriminative learning. *Annals of the New York Academy of Sciences*, 846, 194–212.
- Garavan, H., Ross, T. J., Kaufman, J., & Stein, E. A. (submitted). A midline rostral-caudal axis for error processing and response conflict monitoring.
- Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science*, 4, 385–390.
- Gehring, W. J., Himle, J., & Nisenson, L. G. (2000). Action monitoring dysfunction in obsessive-compulsive disorder. *Psychological Science*, 11, 1–6.
- Gehring, W. J. & Willoughby, A. R. (2002). The medial frontal cortex and the rapid processing of monetary gains and losses. *Science*, 295, 2279–2282.

- Gemba, H., Sasaki, K., & Brooks, V. B. (1986). "Error" potentials in limbic cortex (anterior cingulate area 24) of monkeys during motor learning. *Neuroscience Letters*, 70, 223–227.
- George, M. S., Ketter, P. I., Rosinsky, N., Ring, H., Casey, B. J., Trimble, M. R., et al. (1994). Regional brain activity when selecting a response despite interference: an H215O Pet study of the stroop and emotional stroop. *Human Brain Mapping*, 1, 194–209.
- Gevins, A., Smith, M. E., McEvoy, L., & Yu, D. (1997). High-resolution EEG mapping of cortical activation related to working memory: effects of task difficulty, type of processing, and practice. *Cerebral Cortex*, 7, 374–385.
- Gevins, A. S., Cutillo, B. A., Bressler, S. L., Morgan, N. H., White, R. M., Illes, J., et al. (1989). Event-related covariances during a bimanual visuomotor task. II. Preparation and feedback. *Electroencephalography and clinical Neurophysiology*, 74, 147–160.
- Hajcak, G. & Simons, R. F. (2002). Error-related brain activity in obsessive-compulsive undergraduates. *Psychiatry Research*, 110, 63–72.
- Heims, S. J. (1991). *The cybernetics group*. Cambridge: MIT Press.
- Holroyd, C. B. & Coles, M. G. H. (2002). The basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, 109, 679–709.
- Holroyd, C. B., Dien, J., & Coles, M. G. H. (1998). Error-related scalp potentials elicited by hand and foot movements: evidence for an output-independent error-processing system in humans. *Neuroscience Letters*, 242, 65–68.
- Holroyd, C. B., Praamstra, P., Plat, E., & Coles, M. G. H. (2002). Spared error-related potentials in mild to moderate parkinson's disease. *Neuropsychologia*, 40, 2116–2124.
- Hutchinson, W. D., Davis, K. D., Lozano, A. M., Tasker, R. R., & Dostrovsky, J. O. (1999). Pain-related neurons in the human cingulate cortex. *Nature Neuroscience*, 2 (5), 403–405.
- Jackson, J. H. (1931). The evolution and dissolution of the nervous system. In *Selected writings of John Hughlings Jackson, Vol II* (pp. 45–75). London: Hodder and Stoughton.
- Janer, K. W. & Pardo, J. V. (1991). Deficits in selective attention following bilateral anterior cingulotomy. *Journal of Cognitive Neuroscience*, 3, 231–241.
- Johannes, S., Wieringa, B. M., Nager, W., Dengler, R., & Münte, T. F. (2001). Oxazepam alters action monitoring. *Psychopharmacology*, 155, 100–106.
- Johannes, S., Wieringa, B. M., Nager, W., Rada, D., Dengler, R., Emrich, H. M., et al. (2001). Discrepant target detection and action monitoring in obsessive-compulsive disorder. *Psychiatry Research*, 108, 101–110.
- Kahana, M. J., Seelig, D., & Madsen, J. R. (2001). Theta returns. *Current Opinion in Neurobiology*, 11, 739–744.
- Khanna, S. (1997). Dorsal hippocampus field CA1 pyramidal cell responses to a persistent versus an acute nociceptive stimulus and their septal modulation. *Neuroscience*, 77, 713–721.
- Khanna, S. & Zheng, F. (1998). Morphine reversed formalin-induced CA1 pyramidal cell suppression via an effect on septohippocampal neural processing. *Neuroscience*, 89, 61–71.
- Kiehl, K. A., Liddle, P. F., & Hopfinger, J. B. (2000). Error processing and the rostral anterior cingulate: an event-related fMRI study. *Psychophysiology*, 37, 216–223.

- Konow, A. & Pribram, K. H. (1970). Error recognition and utilization produced by injury to the frontal cortex in man. *Neuropsychologia*, 8, 489–491.
- Kopp, B. & Rist, F. (1999). An event-related brain potential substrate of disturbed response monitoring in paranoid schizophrenic patients. *Journal of Abnormal Psychology*, 108, 337–346.
- Kopp, B., Rist, F., & Mattler, U. (1996). N200 in the flanker task as a neurobehavioral tool for investigating executive control. *Psychophysiology*, 33, 282–294.
- Koyama, T., Tanaka, Y. Z., & Mikami, A. (1998). Nociceptive neurons in the macaque anterior cingulate activate during the anticipation of pain. *Neuroreport*, 9 (11), 2663–2667.
- Kupers, R. C., Konings, H., Adriaensen, H., & Gybels, J. M. (1991). Morphine differentially affects the sensory and affective pain ratings in neurogenic and idiopathic forms of pain. *Pain*, 47, 5–12.
- Luria, A. R. & Pribram, K. H. (1964). An experimental analysis of the behavioral disturbance produced by a left frontal arachnoidal endothelioma (meningioma). *Neuropsychologia*, 2, 257–281.
- Luu, P., Collins, P., & Tucker, D. M. (2000). Mood, personality, and self-monitoring: negative affect and emotionality in relation to frontal lobe mechanisms of error monitoring. *Journal of Experimental Psychology: General*, 129, 43–60.
- Luu, P., Flaisch, T., & Tucker, D. M. (2000). Medial frontal cortex in action monitoring. *Journal of Neuroscience*, 20, 464–469.
- Luu, P. & Tucker, D. M. (2001). Regulating action: alternating activation of human prefrontal and motor cortical networks. *Clinical Neurophysiology*, 112, 1295–1306.
- Luu, P. & Tucker, D. M. (2002). Self-regulation and the executive functions: electrophysiological clues. In A. Zani & A. M. Preverbio (Eds.), *The cognitive electrophysiology of mind and brain* (pp. 199–223). San Diego: Academic Press.
- Luu, P., Tucker, D. M., Derryberry, D., Reed, M., & Poulsen, C. (2003). Activity in human medial frontal cortex in emotional evaluation and error monitoring. *Psychological Science*, 14, 47–53.
- MacDonald, A. W., Cohen, J. D., Stenger, V. A., & Carter, C. S. (2000). Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science*, 288, 1835–1838.
- Makeig, S., Luu, P., Briggman, K., Visser, E., Sejnowski, T. J., & Tucker, D. M. (in preparation). Oscillatory sources of human error-related brain activity.
- Makeig, S., Westerfield, M., Jung, T.-P., Enghoff, S., Townsend, J., Courchesne, E., et al. (2002). Dynamic Brain Sources of Visual Evoked Responses. *Science*, 295, 690–694.
- Masaki, H., Tanaka, H., Takasawa, N., & Yamazaki, K. (2001). Error-related brain potentials elicited by vocal errors. *Neuroreport*, 12, 1851–1855.
- Mathalon, D. H., Fedor, M., Faustman, W. O., Gray, M., Asakari, N., & Ford, M. J. (2002). Response-monitoring dysfunction in schizophrenia: an event-related brain potential study. *Journal of Abnormal Psychology*, 111, 22–41.
- McEwen, B. S. (2000). The neurobiology of stress: from serendipity to clinical relevance. *Brain Research*, 886, 172–189.

- Menon, V., Adelman, N. E., White, C. D., Glover, G. H., & Reiss, A. L. (2001). Error-related brain activation during a go/nogo response inhibition task. *Human Brain Mapping*, 12, 131–143.
- Miller, R. (1991). *Cortico-hippocampal interplay and the representation of contexts in the brain*. New York: Springer-Verlag.
- Miltner, W. H. R., Braun, C. H., & Coles, M. G. H. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: Evidence for a “generic” neural system for error detection. *Journal of Cognitive Neuroscience*, 9, 787–797.
- Mishkin, M. (1982). A memory system in the monkey. *Philosophical Transactions of the Royal Society of London: Series B*, 298, 85–95.
- Miura, Y., Ito, T., & Kadokawa, T. (1987). Effects of intraseptally injected dopamine and noradrenaline on hippocampal synchronized theta wave activity in rats. *Japanese Journal of Pharmacology*, 44, 471–479.
- Morgan, R. E., Garavan, H. P., Mactutus, C. F., Levitsky, D. A., Booze, R. M., & Strupp, B. J. (2002). Enduring effects of prenatal cocaine exposure on attention and reaction to errors. *Behavioral Neuroscience*, 116, 624–633.
- Nauta, W. J. H. (1971). The problem of the frontal lobe: A reinterpretation. *Journal of Psychiatric Research*, 8, 167–187.
- Neafsey, E. J., Terreberry, R. R., Hurley, K. M., Ruit, K. G., & Fryszak, R. J. (1993). Anterior cingulate cortex in rodents: Connections, visceral control functions, and implications for emotion. In B. A. Vogt & M. Gabriel (Eds.), *Neurobiology of the cingulate cortex and limbic thalamus* (pp. 206–223). Boston: Birkhauser.
- Niki, H. & Watanabe, M. (1979). Prefrontal and cingulate unit activity during timing behavior in the monkey. *Brain Research*, 171, 213–224.
- Norman, D. A. & Shallice, T. (1986). Attention to action: willed and automatic control of behavior. In R. J. Davidson, G. E. Schwartz, & D. Shapiro (Eds.), *Consciousness and self-regulation* (pp. 1–18). New York: Plenum.
- Ochsner, K. N., Kosslyn, S. M., Cosgrove, G. R., Cassem, E. H., Price, B. H., Nierenberg, A. A., et al. (2001). Deficits in visual cognition and attention following bilateral anterior cingulotomy. *Neuropsychologia*, 39, 219–230.
- Packard, M. G. & Cahill, L. (2001). Affective modulation of multiple memory systems. *Current Opinion in Neurobiology*, 11, 752–756.
- Pailing, P. E. & Segalowitz, S. J. (submitted). The error-related negativity (ERN/Ne) as a state and trait measure: motivation, personality and ERPs in response to errors.
- Pandya, D. N., Van Hoesen, G. W., & Mesulam, M.-M. (1981). Efferent connections of the cingulate gyrus in the rhesus monkey. *Experimental Brain Research*, 42, 319–330.
- Pardo, J. V., Pardo, P. J., Janer, K. W., & Raichle, M. E. (1990). The anterior cingulate cortex mediates processing selection in the stroop attentional conflict paradigm. *Proceedings of the National Academy of Sciences*, 87, 256–259.
- Paré, D., Collins, D. R., & Pelletier, J. G. (2002). Amygdala oscillations and the consolidation of emotional memories. *Trends in Cognitive Sciences*, 6, 306–314.
- Paus, T., Petrides, M., Evans, A. C., & Meyer, E. (1993). Role of the human anterior cingulate cortex in the control of oculomotor, manual, and speech responses: A positron emission tomography study. *Journal of Neurophysiology*, 70, 453–469.

- Ploghaus, A., Tracey, I., Clare, S., Gati, J. S., Rawlins, J. N. P., & Matthews, P. M. (2000). Learning about pain: the neural substrate of the prediction error for aversive events. *Proceedings of the National Academy of Sciences*, 97, 9281–9286.
- Poremba, A. & Gabriel, M. (1997). Amygdalar lesions block discriminative avoidance learning and cingulothalamic training-induced neuronal plasticity in rabbits. *Journal of Neuroscience*, 17, 5237–5244.
- Poremba, A. & Gabriel, M. (1999). Amygdala neurons mediate acquisition but not maintenance of instrumental avoidance behavior in rabbits. *Journal of Neuroscience*, 19, 9635–9641.
- Poremba, A. & Gabriel, M. (2001). Amygdalar efferents initiate auditory thalamic discriminative training-induced neuronal activity. *Journal of Neuroscience*, 21, 270–278.
- Posner, M. I. & Dehaene, S. (1994). Attentional networks. *Trends in Neuroscience*, 17, 75–79.
- Posner, M. I. & DiGirolamo, G. (1998). Executive attention: conflict, target detection and cognitive control. In R. Parasuraman (Ed.), *The attentive brain* (pp. 401–423). Cambridge: MIT Press.
- Posner, M. I., Petersen, S. E., Fox, P. T., & Raichle, M. E. (1988). Localization of cognitive operations in the human brain. *Science*, 240, 1627–1631.
- Posner, M. I. & Rothbart, M. K. (1998). Attention, self regulation and consciousness. *Philosophical Transactions of the Royal Society of London: Series B*, 353, 1–13.
- Pribram, K. H. (1960). A review of theory in physiological psychology. *Annual Review of Psychology*, 11, 1–40.
- Raghavachari, S., Kahana, M. J., Rizzuto, D. S., Caplan, J. B., Kirschen, M., Bourgeois, D., et al. (2001). Gating of human theta oscillations by a working memory task. *Journal of Neuroscience*, 21, 3175–3183.
- Redgrave, P., Prescott, T. J., & Gurney, K. (1999). Is the short-latency dopamine response too short to signal reward error? *Trends in Neurosciences*, 22, 146–151.
- Rosenkilde, C. E., Bauer, R. H., & Fuster, J. M. (1981). Single cell activity in ventral prefrontal cortex of behaving monkeys. *Brain Research*, 209, 375–394.
- Ruchsow, M., Grothe, J., Spitzer, M., & Kiefer, M. (2002). Human anterior cingulate cortex is activated by negative feedback: evidence from event-related potentials in a guessing task. *Neuroscience Letters*, 325, 203–206.
- Rylander, G. (1947). Personality analysis before and after frontal lobotomy. In J. F. Fulton, C. D. Aring, & B. S. Wortis (Eds.), *Research publications association for research in nervous and mental disease: The frontal lobes* (pp. 691–705). Baltimore: Williams & Wilkins.
- Santesso, D. L., Segalowitz, S. J., & Schmidt, L. A. (submitted). Enhanced error-related ERPs in 10-year-old children with high obsessive-compulsive behavior.
- Satinoff, E. (1978). Neural organization and evolution of thermal regulation in mammals. *Science*, 201, 16–22.
- Scheffers, M. K., Coles, M. G. H., Bernstein, P., Gehring, W. R., & Donchin, E. (1996). Event-related brain potentials and error-related processing: An analysis of incorrect responses to go and no-go stimuli. *Psychophysiology*, 33, 42–53.
- Schulkin, J., McEwen, B. S., & Gold, P. W. (1994). Allostasis, amygdala, and anticipatory angst. *Neuroscience and Biobehavioral Reviews*, 18, 385–396.

- Schultz, W., Apicella, P., & Ljungberg, T. (1993). Responses of monkey dopamine neurons to reward and conditioned stimuli during successive steps of learning a delayed response task. *Journal of Neuroscience*, 13, 900–913.
- Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science*, 275, 1593–1599.
- Schultz, W. & Dickinson, A. (2000). Neuronal coding of prediction errors. *Annual Review of Neuroscience*, 23, 473–500.
- Schultz, W., Tremblay, L., & Hollerman, J. F. (1998). Reward prediction in primate basal ganglia and frontal cortex. *Neuropharmacology*, 37, 421–429.
- Shima, K. & Tanji, J. (1998). Role for cingulate motor area cells in voluntary movement selection based on reward. *Science*, 282, 1335–1338.
- Slobounov, S. M., Fukada, K., Simon, R., Rearick, M., & Ray, W. (2000). Neurophysiological and behavioral indices of time pressure effects on visuomotor task performance. *Cognitive Brain Research*, 9, 287–298.
- Squire, L. R. & Zola-Morgan, S. (1991). The medial temporal lobe memory system. *Science*, 253, 1380–1386.
- Stemmer, B., Witzke, W., & Schönle, P. W. (2001). Losing the error related negativity in the EEG of human subjects: An indicator for willed action. *Neuroscience Letters*, 308, 60–62.
- Swanson, L. W. (2000). Cerebral hemisphere regulation of motivated behavior. *Brain Research*, 886, 113–164.
- Swick, D. & Turken, A. U. (2002). Dissociation between conflict detection and error monitoring in the human anterior cingulate cortex. *Proceedings of the National Academy of Science*, 99, 16354–16359.
- Tellegen, A. & Waller, N. G. (1996). Exploring personality through test construction: Development of the multidimensional personality questionnaire. In S. R. Briggs & J. M. Cheek (Eds.), *Personality measures: Development and evaluation* (pp. 133–161). Greenwich: JAI Press.
- Teuber, H. L. (1964). The riddle of the frontal lobe function in man. In J. M. Warren & K. Akert (Eds.), *The frontal granular cortex and behavior* (pp. 410–444). New York: McGraw Hill.
- Thorpe, S. J., Rolls, E. T., & Maddison, S. (1983). The orbitofrontal cortex: Neuronal activity in the behaving monkey. *Experimental Brain Research*, 49, 93–115.
- Tucker, D. M. (2001). Motivated Anatomy: A core-and-shell model of corticolimbic architecture. In G. Gainotti (Ed.), *Handbook of Neuropsychology*. Amsterdam: Elsevier.
- Tucker, D. M., Hartry-Speiser, A., McDougal, L., Luu, P., & deGrandpre, D. (1999). Mood and spatial memory: Emotion and the right hemisphere contribution to spatial cognition. *Biological Psychology*, 50, 103–125.
- Tucker, D. M., Luu, P., Desmond, R. E., Hartry-Speiser, A. L., Davey, C., & Flaisch, T. (2003). Corticolimbic Mechanisms in Emotional Decisions. *Emotion*, 3, 127–149.
- Tucker, D. M., Luu, P., Frishkoff, G., Quiring, J., & Poulsen, C. (in press). Frontolimbic Response to Negative Feedback in Clinical Depression.
- Ullsperger, M. & von Cramon, D. Y. (2001). Subprocesses of performance monitoring: a dissociation of error processing and response competition revealed by event-related fMRI and ERPs. *Neuroimage*, 14, 1387–1401.

- van Veen, V. & Carter, C. S. (2002). The timing of action monitoring processes in anterior cingulate cortex. *Journal of Cognitive Neuroscience*, 14, 593–602.
- Van't Ent, D. & Apkarian, P. (1999). Motoric response inhibition in finger movement and saccadic eye movement: a comparative study. *Clinical Neurophysiology*, 110, 1058–1072.
- Vanderwolf, C. H., Kramis, R., & Robinson, T. E. (1978). Hippocampal electrical activity during waking behaviour and and sleep: Analyses using centrally acting drugs. In *Functions of the septo-hippocampal system.*, Vol. 58 (pp. 199–221). Amsterdam: Elsevier.
- Vidal, F., Hasbroucq, T., Grapperon, J., & Bonnet, M. (2000). Is the 'error negativity' specific to errors? *Biological Psychology*, 51, 109–128.
- Vogt, B. A., Pandya, D. N., & Rosene, D. L. (1987). Cingulate cortex of the rhesus monkey: I. Cyto architecture and thalamic afferents. *Journal of Comparative Neurology*, 262, 256–270.
- Vogt, B. A., Sikes, R. W., & Vogt, L. J. (1993). Anterior cingulate cortex and the medial pain system. In B. A. Vogt & M. Gabriel (Eds.), *Neurobiology of the cingulate cortex and limbic thalamus* (pp. 314–344). Boston: Birkhäuser.
- Wan, R.-Q., Givens, B. S., & Olton, D. S. (1995). Opioid modulation of working memory: intraseptal, but not intraamygdaloid, infusion of β -endorphin impair performance in spatial alternation. *Neurobiology of Learning and Memory*, 63, 74–86.
- Wiener, N. (1948). *Cybernetics, or control and communication in the animal and the machine*. New York: MIT Press.
- Winn, P. (1995). The lateral hypothalamus and motivated behavior: An old syndrome reassessed and a new perspective gained. *Current Directions in Psychological Science*, 4, 182–187.
- Zheng, F., & Khanna, S. (1999). Hippocampal field CA1 interneuronal nociceptive responses: Modulation by medial septal region and morphine. *Neuroscience*, 93, 45–55.
- Zieglagänsberger, W., French, E. D., Siggins, G. R., & Bloom, F. E. (1979). Opioid peptides may excite hippocampal pyramidal neurons by inhibiting adjacent inhibitory neurons. *Science*, 205, 415–417.

Neural basis of conscious and voluntary self-regulation of emotion

Mario Beauregard, Johanne Lévesque, and Vincent Paquette

Introduction

Until recently, few things were known regarding the neural basis of emotional self-regulation. With respect to this issue, various evidence from experimental lesion studies in animals, and clinical neuropsychology in humans, have led Nauta (1971) and, two decades later, Tucker, Luu, and Pribram (1995) to postulate that the prefrontal cortex (PFC) and limbic/paralimbic structures are key components of a frontolimbic circuit involved in emotional self-regulation. Lately, Davidson and associates (Davidson, Putnam, & Larson 2000) have hypothesized, along the same lines, that emotional self-regulation may be normally implemented by a neural circuit encompassing several regions of the PFC (e.g., the orbitofrontal – OFC, the dorsolateral – DLPFC, the anterior cingulate cortex – ACC), as well as subcortical limbic structures, such as the amygdala and the hypothalamus.

A few years ago, we undertook a research program that sought to test the validity of the views proposed by Nauta (1971), Tucker et al. (1995), and Davidson et al. (2000) regarding the neural substrate of emotional self-regulation in healthy individuals. Within this program, we used functional magnetic resonance imaging (fMRI) to investigate the neural circuitry underlying conscious and voluntary self-regulation of sexual arousal in male subjects (although sexual arousal is not an emotion *per se*, it certainly can be considered as a powerful alteration of the emotional state) (Beauregard, Lévesque, & Bourgouin 2001). In the following two studies, we examined the neural basis of voluntary regulation of sadness in adults (Lévesque, Eugène, Joannette, Paquette, Mensour, Beaudoin, Leroux, Bourgouin, & Beauregard 2003) as well as in children (Beauregard, Lévesque, Eugène, Joannette, Mensour, Beaudoin,

Leroux, & Bourgouin 2002). At about the same time, three other groups, respectively led by Schaefer (Schaefer, Jackson, Davidson, Aguirre, Kimberg, & Thompson-Schill 2002), Ochsner (Ochsner, Bunge, Gross, & Gabrieli 2002), and Hariri (Hariri, Bookheimer, & Mazziotta 2000; Hariri, Mattay, Tessitore, Fera, & Weinberger 2003) also used fMRI to investigate various aspects pertaining to the neural basis of emotional self-regulation in healthy people.

In the first section of this chapter, we will introduce a few key theoretical concepts related to the emotional self-regulation construct. In the following section, we will present the findings of the functional neuroimaging studies aforementioned. Finally, in the last section of the chapter, we will discuss the implications of these findings, with special reference to the interaction between mind and brain.

Definition of emotional self-regulation

According to evolutionary psychology, biologically based emotions represent efficient modes of adaptation to changing environmental demands (Tooby & Cosmides 1990; Levenson 1994). From this theoretical perspective, emotions have emerged in the course of evolution on account of their capacity to adequately coordinate the various response systems (e.g., cognitive, subjective or experiential, physiological, and behavioral) that characterize emotion's multidimensional nature. In so doing, emotions have helped the human species to survive, facing very challenging events (Levenson 1994). However, as noted by Gross (1999), emotions are not always the most appropriate response to the diverse situations encountered in daily life. Gross further contends that, despite the many possible benefits of emotion, it is very important to correctly evaluate the dangers that arise from blindly following emotional impulses. Indeed, negative emotions are undoubtedly one of the main causes of suffering and dysfunction on our planet. For instance, it becomes increasingly evident that, collectively, the emotional/impulsive use of certain weapons could have dreadful consequences for the political, economical, and ecological stability of our world. This is why, at both an individual and a collective level, it is peremptory for human beings to learn how to properly control and modulate their emotional reactions using their cognitive capacities. Fortunately, individuals can change the way they feel by changing the way they think, thereby diminishing the emotional impact of otherwise negative experiences (Ochsner et al. 2002). This point leads us directly to the construct of emotional self-regulation.

Emotional self-regulation refers to the heterogeneous set of cognitive processes by which emotions are regulated, i.e., the ways individuals influence which emotions they have, when they have them, and how they experience and express these emotions (Gross 1999). This instance of self-regulation involves changes in one or more of the various components (or response systems) of emotion (Gross 1999). These changes implicate modifications in what Thompson (1990) has termed "emotion dynamics", i.e., the magnitude, rise time, duration, and offset of responses in the various components of emotion (Gross 1999). Emotional self-regulation involves either generating, maintaining, decreasing, or increasing either positive or negative emotions (Masters 1991; Parrott 1993; Cole, Michel, & Teti 1994; Langston 1994). This capacity implicates several types of adjustments that favour the adaptation of the individual to life circumstances (Cole et al. 1994) and allow to accomplish one's goals (Thompson 1990). The cognitive strategies used to self-regulate emotion can be more or less conscious, effortful, and controlled (Gross 1999). These cognitive strategies are numerous and include, among others, rationalization, reappraisal, and suppression (Gross 1999).

Emotional self-regulation certainly constitutes one of the cornerstones of socialization and moral development (Kochanska, Murray, & Coy 1997). Consistent with this, several lines of evidence indicate that a chronic inability to self-regulate negative emotions, such as sadness and fear, may play a pivotal role in the genesis of clinical depression and anxiety disorders (Davidson et al. 2000; Jackson, Malmstadt, Larson, & Davidson 2000). There is also mounting evidence that impulsive aggression and violence arise as a consequence of defective regulation of anger (Davidson et al. 2000). The fact that the ability to self-regulate negative emotion is essential to a healthy psyche explains why, in several modern psychotherapeutic approaches, the client is taught by the therapist how to use efficient emotion regulatory strategies.

Because the cognitive processes underlying emotional self-regulation are closely meshed with those implicated in emotion generation, it has been theorized that emotional self-regulation is an integral part of emotion (Frijda 1986). With regard to the relationship between emotion regulation and generation, Gross (2001) has postulated, in his process model, that emotion may be regulated at five different points in the emotion-generative process: (1) selection of the situation, (2) modification of the situation, (3) deployment of attention, (4) change of cognitions, and (5) modulation of experiential, behavioral, or physiological responses. In Gross' terminology, the first four of these cognitive processes are antecedent-focused while the fifth is response-focused.

Neural basis of emotional self-regulation: Findings from functional neuroimaging studies

Neural correlates of conscious and voluntary regulation of sexual arousal

In the first fMRI study we conducted regarding the neural basis of emotional self-regulation (Beauregard et al. 2001), ten healthy male volunteers (right-handed Caucasian university students) (aged 20–42) were scanned during two experimental conditions, i.e., a Sexual Arousal condition and a Suppression condition. In the Sexual Arousal condition, subjects viewed series of erotic film excerpts and emotionally neutral film excerpts. They were instructed to react normally to both categories of stimuli (i.e., they had to allow themselves to become sexually aroused during the viewing of the erotic film excerpts). In the Suppression condition, subjects were instructed to suppress any feelings elicited by the erotic film excerpts. In order to do so, subjects were encouraged to become a detached observer of the erotic film excerpts and the sexual arousal induced by these stimuli (subjects were not trained beforehand). Such a metacognitive strategy shares some similarity with mindfulness, a central mental state in Buddhist meditation.

The Sexual Arousal condition consisted in the viewing of, first, four blocks of emotionally neutral film excerpts and then, four blocks of erotic film excerpts. Such an approach was adopted to avoid contamination of the neutral stimuli by the erotic stimuli (unpublished data). Each block lasted 39 sec. Blocks were separated by resting periods of 15 sec during which subjects saw a blank cyan screen. A similar design was used in the Suppression condition. To assess the emotional reactions of the subjects to the film excerpts, at the outset of each condition, they were asked to rate verbally - on a numerical (analogue) rating scale ranging from 0 (absence of any emotional reaction) to 8 (strongest emotion ever felt in one's lifetime) - the intensity of primary emotions (e.g., sadness, happiness, disgust, fear, anger, surprise) felt during the viewing of both categories of film excerpts.

From an experiential perspective, the viewing of the erotic film excerpts during both experimental conditions induced a state of sexual arousal in all subjects. In the Suppression condition, most subjects reported having been successful at distancing themselves from the sexual arousal induced by the erotic film excerpts. Consistent with this, the mean level of sexual arousal was significantly higher in the Sexual Arousal condition (mean = 5; range: 3–8) than in the Suppression condition (mean = 2; range: 1–4) ($p < 0.005$). In agreement with the results of a previous study by our group (Karama, Lecours,

Leroux, Joubert, Bourgouin, Beaudoin, & Beaugregard 2002), the viewing of erotic film excerpts, in the Sexual Arousal condition, produced a significant activation of the right amygdala, right anterior temporal pole (Brodmann area – BA – 38), and hypothalamus. These findings support the view that the amygdala plays a key role in the evaluation of the emotional significance of stimuli (for a review, see Lane & Nadel 2000), that the hypothalamus is a pivotal brain structure implicated in the endocrine and autonomic expression of emotion (Henry 1986), and that the anterior temporal pole is involved in imparting emotional colour to subjective experience (Mesulam 1985). The results of the Suppression condition revealed that the volitional inhibitory action exerted by the subjects, in order to decrease the intensity of the sexual arousal produced by the erotic film excerpts, was associated with activation peaks in BA10 of the right lateral prefrontal cortex (LPFC) and BA 32 of the right ACC. Contrary to the pattern of brain activity seen in the Sexual Arousal condition, in the Suppression condition, no significant loci of activation were noted in the amygdala, the anterior temporal polar region, and the hypothalamus (even for a low height threshold value of $p < 0.10$, uncorrected).

These findings accord well with those of the fMRI study conducted by Hariri and colleagues (Hariri, Bookheimer, & Mazziotta 2000). In this investigation, subjects either matched the emotion (e.g., anger or fear) displayed by one of two faces to that of a simultaneously presented target face (a perceptual task) or identified the emotion (e.g., anger or fear) showed by a target face by selecting one of two simultaneously presented linguistic labels (a cognitive task). Matching angry or frightened facial expressions was associated with increased regional cerebral blood flow (rCBF) in the amygdala, bilaterally. Labeling these same facial expressions was associated with decreased rCBF response in the amygdalae. This rCBF decrease correlated with a rCBF increase in the right prefrontal cortex. Our results (Beaugregard et al. 2001) and those of Hariri et al. (2000) provide strong evidence for the view previously proposed by various researchers (Nauta 1971; Tucker et al. 1995; Davidson et al. 2000) that emotional self-regulation depends on a neural circuit in which prefrontal cortical areas can cognitively modulate emotional responses generated at a subcortical level.

In other respects, these results are consistent with findings indicating that the LPFC is implicated in metacognitive/executive top-down processes, which refer to the ability to monitor and control the information processing necessary to produce voluntary action (Flavell 1979). This prefrontal cortical region has been implicated in the selection and control of behavioral strategies and action (Fuster 1999), especially in the inhibition of inherent response tendency

(Goldman-Rakic 1987; Damasio 1995; Frith & Dolan 1996; Fuster 1997). These results also concur with the view that, by virtue of its anatomic connections with brain regions implicated in the modulation of autonomic and endocrine functions, such as the amygdala, hypothalamus, and OFC, the rostral-ventral subdivision of the ACC plays a key role in the regulation of the autonomic aspect of emotional responses (Vogt, Finch, & Olson 1992; Devinsky, Morrell, & Vogt 1995; Bush, Luu, & Posner 2000). The fact that this portion of ACC has both afferent and efferent connections with neural structures mediating autonomic functions such as the periaqueductal gray, the dorsal motor nucleus of the vagus, and preganglionic sympathetic neurons in the intermediolateral cell column of the spinal cord (for a review, see Benes 1997) is supportive of such a view.

Neural correlates of conscious and voluntary regulation of sadness in adults

In our next study (Lévesque et al. 2003) regarding the neural basis of emotional self-regulation, we used whole-brain fMRI to delineate the neural circuitry associated with the voluntary suppression of sadness, a primary emotion with a negative valence (Plutchik 1994). Twenty healthy female volunteers (right-handed Caucasian university students) (age range: 20–30) took part in the study. The experimental design was similar to that used in the Beaudregard et al. (2001) study, except that the blocks of film excerpts (sad and emotionally neutral) were presented for 48 sec (instead of 39 sec). Also, a ‘random-effects model’ was implemented to process the fMRI data (instead of a ‘fixed-effects model’).

Phenomenologically, the viewing of the sad film excerpts, in both the Sad and the Suppression conditions, induced a transient state of sadness in all subjects. As expected, the mean level of reported sadness was significantly higher in the Sad condition (mean = 5.15; SD = 1.30; range: 2–7) than in the Suppression condition (mean = 1.85; SD = 1.42; range: 0–4) ($p < 0.0001$). During both experimental conditions, the viewing of the sad film excerpts did not produce other marked changes in the emotional state than sadness and viewing the emotionally neutral film excerpts did not generate any primary emotion. In addition, the strategy questionnaire completed at the end of the scanning session revealed that, in the Suppression condition, all subjects reported having succeeded in distancing themselves from the sad film excerpts, i.e., in becoming a detached observer. The mean suppression percentage reflecting the degree

to which subjects thought having succeeded in suppressing sad feelings was eighty-four percent (range: 50–100; SD: ± 11).

In the Sad condition, significant bilateral loci of activation were measured in the anterior temporal pole (BA 21 and BA 38) and the midbrain. Significant loci of activations were also seen in the right ventrolateral prefrontal cortex (VLPFC) (BA 47), left amygdala, and left insula. In the Suppression condition, significant loci of activation were noted in the right OFC (BA 11) and right LPFC (BA 9). Additionally, when using a liberal threshold of $p < 0.05$, uncorrected for multiple comparisons, BOLD signal increases were found in the right VLPFC (BA 47). *A posteriori* correlational analyses were also carried out between self-report ratings of sadness and BOLD signal changes found in the regions of interest. In the Sad condition, positive correlations were found in the right VLPFC (BA 47; coordinates of maximum = [27, 20, -11]; $z = 2.80$; $p < 0.05$, uncorrected), the rostral division of the left ACC (BA 24/32; coordinates of maximum = [-3, 38, 1]; $z = 3.69$; $p < 0.05$, uncorrected), and the left insula (coordinates of maximum = [-42, 9, -2]; $z = 2.01$; $p < 0.05$, uncorrected) whereas in the Suppression condition, positive correlations were noted in the right OFC (BA 11; coordinates of maximum = [27, 34, -19]; $z = 2.50$; $p < 0.05$, uncorrected) and right LPFC (BA 9; coordinates of maximum = [36, 34, 28]; $z = 3.13$; $p < 0.05$, uncorrected).

We have proposed (Lévesque et al. 2003) that the amygdala activation seen in the Sad condition reflected the appraisal process through which significance was attributed to the sad film excerpts while the anterior temporal activation was related to the attribution of the emotional tone to the subjects' experience. With respect to the midbrain, it has been demonstrated that this brain structure is involved in the mediation of autonomic responses such as skin conductance responses (Sequeira & Roy 1993) and body temperature changes (Nagashima, Nakai, Tanaka, & Kanosue 2000). Therefore, the midbrain activations noted during sadness likely reflected the autonomic responses accompanying subjects' sad feelings. Regarding the physiological dimension of emotion, we posited that the insular activation measured in the Sad condition may be a neural correlate of the autonomic changes associated with the subjective experience of sadness given the rich interconnection of the insula with regions involved in autonomic regulation (Cechetto 1994). The positive correlation found between average ratings of sadness and BOLD signal increases in the rostral division of the left ACC fits rather nicely with the results of a recent PET study by Lane and colleagues (Lane, Fink, Chau, & Dolan 1997) suggesting that the rostral portion of the ACC plays an important role in the interoceptive or exteroceptive detection of emotional signals, and, therefore, in

emotional awareness. The sensory inputs (olfactory, gustatory, visceral afferent, somatic sensory, and visual) as well as the “limbic” inputs that the VLPFC receives from the amygdala, entorhinal and perirhinal cortex, and subiculum suggests that this cortical region may be implicated in the integration of viscerosensory information with affective signals (Price 1999). In addition, the positive correlation found between self-report ratings of sadness and BOLD signals increases in the right VLPFC indicates that this paralimbic area may also be involved in the subjective aspect of sadness. Regarding this issue, increased VLPFC activity has been reported in association with sad thoughts/sadness in subjects with major depressive disorder (Brody, Barsom, Bota, & Saxena 2001). It thus appears that this brain region is associated with the processing of normal as well as pathological aspects of sadness.

In the Suppression condition, the right LPFC activation demonstrated that, in addition to being involved in the voluntary suppression of a positive emotional reaction such as sexual arousal (Beauregard et al. 2001), this prefrontal cortical region is also associated with the voluntary suppression of a negative emotion such as sadness. These results, and the positive correlation found between the self-report ratings of sadness and the significant loci of activation in the right LPFC, are consistent with a variety of evidence indicating that the LPFC plays a crucial role in willed actions (Frith & Dolan 1996) and with the holding in mind of information on which an action is to be based (Goldman-Rakic 1987; Fuster 1999; Roberts & Wallis 2000). In our previous study, BA 10 of the right LPFC was associated with the suppression of sexual arousal. In this study (Lévesque et al. 2003), the group analysis revealed activation only in BA 9 of the LPFC. However, careful examination of the individual statistical parametric maps revealed that 5 subjects were also showing activation in BA 10 of the LPFC ($p < 0.001$, uncorrected). At this point, few things are known about the respective roles of the LPFC’s BA 9 and BA 10 in emotional self-regulation. With respect to this question, Dias and colleagues (Dias, Robbins, & Roberts 1996) have shown that, in monkeys, BA 9 is involved in inhibitory control, namely, in attentional set-shifting capacity. In humans, further studies are needed to delineate the functional specificity of these two PF regions regarding volitional regulation of emotion.

Robust activation of the right OFC (BA 11) was also noted during suppression of sad feelings. From a neural systemic point of view, this prefrontal region is located at the junction of the prefrontal associative cortex and the limbic system. Given the fact that the OFC sends projections to the amygdala, diencephalon, brain stem and spinal cord, this prefrontal region has been associated with the integration of visceral-autonomic processes with cogni-

tive and behavioral processes (Öngür, An, & Price 1998; Rempel-Clower & Barbas 1998; Eslinger 1999a). It has been further proposed that the OFC plays a central role in the adaptation to complex changing environments (Eslinger 1999b) and the regulation of socio-emotional behavior in settings involving social affiliation and social judgment, self-awareness, inhibition, and the self-guidance of behavior through judgments and decisions about one's actions (Mesulam 1986; Cummings 1993; Damasio 1995; Zald & Kim 1996; Dolan 1999; Bechara, Damasio, Damasio, & Anderson 1994; Fuster 1997; Damasio, Tranel, & Damasio 1990; Elliott 1990; Giancola & Zeichner 1994; Grafman, Schwab, Warden, Pridgen, Brown, & Salazar 1996; Eslinger 1999b; Grafman & Litvan 1999; Lapierre, Braun, & Hodgins 1995). The OFC is the only PFC region richly connected to the amygdala. The central nucleus of the amygdala is the main source of efferent connections to brain stem and hypothalamic structures modulating a wide range of endocrine and autonomic responses (Davis 1992). The caudal OFC receives information from the central nucleus of the amygdala and, in return, projects directly to this nucleus, procuring thereby a circuit through which the OFC may directly modulate the activity of the amygdala. The medial and lateral OFC send extensive connections to the lateral hypothalamus, suggesting that these OFC areas are particularly involved in the regulation of visceral responses to stimuli and events. Furthermore, the OFC has also strong links with the anterior temporal pole, the insular cortex, and the LPFC (Cavada, Company, Tejedor, Cruz-Rizzolo, & Reinoso-Suarez 2000; Morecraft, Geula, & Mesulam 1992).

Clinical neuropsychological studies indicate that the OFC exerts an inhibitory control to protect goal-directed behavior from interference (Casey, Cohen, Jezard, Turner, Noll, Trainor, Giedd, Kaysen, Hertz-Pannier, & Rapoport 1997; Fuster 1999; Roberts & Wallis 2000). Damage to the OFC leads to a *frontal lobe syndrome* (Silver & Yudofsky 1987) or *pseudopsychopathic syndrome* (Stuss & Benson 1984) that is characterized by distractibility, impulsivity, emotional outbursts, shallowness, argumentativeness, verbal and physical aggressiveness, hypersexuality, hyperphagia, lack of concern of consequences of behavior, failure to observe social and moral rules, and risky decision-making behavior. Individuals with OFC lesions tend to be unpredictable, their humor is labile, and they often display inappropriate and childish humour. Interestingly, these individuals show abnormal autonomic responses to emotional elicitors, difficulty to experience emotion related to situations that would normally evoke emotion, and impaired understanding of the adverse consequences of detrimental social behaviors (Damasio et al. 1990). Early-acquired OFC damage has been associated with additional disruption to

social-emotional processes such as social self-awareness, social sensitivity (or empathy), and social self-regulation (Eslinger, Biddle, & Grattan 1997). Individuals with early-acquired OFC lesions bear resemblance to psychopaths in the sense that they manifest high levels of aggression and antisocial behavior without guilt or empathy for their victims (Anderson, Bechara, Damasio, Tranel, & Damasio 1999). The most important difference from patients injured in adulthood is found on tasks measuring moral reasoning and verbal responses to social situations. In contrast to adult-onset patients, the performance of early-damaged patients on such tasks is greatly impaired. According to Anderson and colleagues (1999), such deficits reflect a failure to acquire social and moral knowledge, i.e., an abnormal development of social and moral behavior. These clinical findings strongly suggest that the integrity of the OFC is a prerequisite for the acquisition of specific forms of knowledge essential for self-guidance of social and moral behavior. This is consistent with functional neuroimaging studies having shown that the OFC is involved in decision making related to reward and punishment (O'Doherty, Kringelbach, Rolls, Hornak, & Andrews 2001), as well as in empathic and moral judgments (Farrow, Zheng, Wilkinson, Spence, Deakin, Tarrier, Griffiths, & Woodruff 2001; Greene, Sommerville, Nystrom, Darley, & Cohen 2001; Moll, Eslinger, & de Oliveira-Souza 2001; Moll, de Oliveira-Souza, Eslinger, Bramati, Mourao-Miranda, Andreiuolo, & Pessoa 2002).

Contrary to our *a priori* hypothesis, as well as to the results of our fMRI study regarding the neural substrate of volitional suppression of sexual arousal (Beauregard et al. 2001), no significant activation was noted in the ACC during voluntary suppression of sad feelings. In order to interpret this negative result, we decided to examine the individual statistical parametric maps produced in the Suppression condition. These maps revealed that the ACC was activated in seventy-five percent of the subjects while they attempted to voluntarily suppress sadness. These maps also showed an important degree of interindividual variability. That is, the activated voxels in the ACC were not consistently localized, across subjects, in the same sub-areas of this cortical region. Thus, it appears that the apparent absence of ACC activation, during the Suppression condition, was probably ascribable to interindividual variability from a functional neuroanatomical standpoint.

Clinically, it has been postulated that a permanent inability to suppress a negative emotion, such as sadness, may be crucially related to the etiology of major depression (Davidson et al. 2000). We submit that such inability may result from a dysfunction in the neural circuit identified here. In keeping with this, preliminary fMRI data gathered in our lab indicate that the difficulty to

suppress sadness (externally induced using sad film excerpts), in subjects suffering from major (unipolar) depression, is correlated with an hypoactivation (in terms of spatial extent) of the LPFC (BA 9) and OFC (BA 11) (relative to normal control subjects). Interestingly, a recent review by Brody and colleagues (2001) revealed that depressed patients often show decreased activity in LPFC (BA 9). Brody et al. (2001) also reported that, in several studies, a negative correlation has been found between the scores of depressed patients on the Hamilton Depression Rating Scale (HAM-D) and either metabolism or rCBF in the PFC. In view of these findings, Brody and colleagues (2001) have proposed that, in major depression, ventral prefrontal and limbic circuits (originating in the VLPFC and ventral ACC) override the relatively hypoactive dorsal circuit (originating in the DLPFC and dorsal ACC) and may allow the content of depressive/sad thoughts to be prominent. Regarding the OFC, a recent study conducted with elderly depressed patients has shown that, compared to normal control subjects, these patients had an increased density of medial OFC white matter lesions (MacFall, Payne, Provenzale, & Krishnan 2001). Furthermore, a correlation analysis revealed that this prefrontal cortical region was significantly correlated with the severity of depression of these patients (MacFall et al. 2001). These results contribute to the growing evidence that the OFC is critically involved in affective disorders.

Neural correlates of conscious and voluntary regulation of sadness in children

Children begin to regulate very early (even as infants and toddlers) the stress generated by emotions and emotional situations. For instance, 18-month-olds self-soothe when their mothers are absent (Mangelsdorf, Shapiro, & Marzolf 1996). To be able to consciously and voluntarily self-regulate emotion, a child must be first aware of having an emotional experience, that is, this child must interpret and evaluate his/her perceived emotional state (Lewis 1998). To have an emotional experience – a set of internal events – this child must be cognitively able to make reference to the fact that it is “I” to whom these internal events are happening. This child must also interpret these events in the context of the meaning systems that he/she has acquired through interactions with others. Indeed, children learn strategies for controlling their emotional responses in agreement with the social rules of their culture. These capacities involve the development of self-consciousness, a critical meta-cognition involving the idea of a referencing self (“idea of me”) (Lewis 1995). There is some evidence that self-consciousness emerges at around 15–18 months. It is the emergence of this

referencing self which allows the child to monitor and react to his/her emotional state, i.e., to self-reflect on his/her thoughts, emotions, and behavior. Cognitive development and social development thus play a pivotal role in the evolution of the capacity to regulate emotion (Cole et al. 1994).

Emotional self-regulation begins with the control of distress. Infants aged 8 to 18 months respond with distress and negative affect to brief separation with the mother. Between 19 and 24 months there is a significant reduction of negative emotional responses to brief maternal separation. Fox and colleagues (Fox, Henderson, & Marshall 2001) have speculated that changes in response to this event during the second year of life may be the result of an infant's increasing capacity to recognize, define, and represent the cause of distress; to appraise the contextual features of the emotion elicitor; to maintain a cognitive representation of his/her mother during her absence; and, to form a plan for a sequence of actions in order to modify the situation so that distress is diminished. Additionally, the infant must suppress the negative affect and distress that were elicited earlier in life by brief separation from the mother. Results of EEG studies conducted by Fox and colleagues (2001) have evidenced an important role for the PFC – “the repository of the most advanced and civilized functions of the brain” (Filley 1995:5) – in the regulation of negative affect and emotional distress. Fox and Bell (1990) have hypothesized that a combination of neurobiological maturation in the PFC and social learning experiences underlie the emergence of emotional self-regulation. Regarding this issue, during the first 6 months, the young infant's emotional regulatory capacity advances from a primarily reflexive response to physiological stimuli (e.g., thirst, hunger, pain, fatigue, etc.) to a nascent awareness of internal state and the ability to temporally associate emotional states with specific external stimuli (Kopp 1989). By 6 to 8 months of age, emotional regulation begins to involve typical PFC functions, such as selective attention to stimuli and perception of temporal contingent sequences implicating the infant's own actions and external stimuli, that allow the infant to regulate states of emotional arousal, for instance, by signalling to caregivers to act in response to his/her emotional states. With the emergence of self-consciousness and intentionality (or goal-directedness), emotional regulatory capacities augment markedly during the second half of the first year of life. Emotion regulation then becomes truly emotional *self*-regulation, i.e., more conscious and self-directed. At this stage, the infant acquires new cognitive executive capacities (e.g., representational thought, self-monitoring, working memory, goal formulation, flexibility of responding internalization of rules to guide behaviour, response inhibition, attentional control, planning) that critically depend on

PFC development (Dawson, Panagiotides, Grofer Klinger, & Hill 1992) and allow him/her to engage in intentional self-regulatory behaviours.

Phylogenetically as well as ontogenetically, the PFC is one of the last neocortical regions to develop. Evolutionarily, the PFC achieves maximal relative growth in the human brain, where it represents approximately one-third of the total volume of the neocortex. In the normal human individual, full PFC maturation is not reached until late adolescence/early adulthood (Fuster 1999). Indeed, myelination and dendritic development occur later in the human PFC than in other cortical regions. Synaptogenesis reaches a plateau between the ages of 1 and 7 and declines through adolescence to the adult level (Bourgeois, Goldman-Rakic, & Rakic 1994; Huttenlocher 1994; Rakic, Bourgeois, & Goldman-Rakic 1994). This decrease in synaptic number coincides with the continued development of cognitive capacities (Caviness, Kennedy, Richelme, Rademacher, & Filipek 1996). Furthermore, cortical gray matter volume achieves its peak at around 5 years of age and decreases from that time forward, while the white matter volume increases constantly until about the age of 20 years (Pfefferbaum, Mathalon, Sullivan, Rawles, Zipursky, & Lim 1994). With respect to PFC myelination, Yakovlev and Lecours (1967) have postulated that it might continue well into the third decade of life. Of note, Fox et al. (2001) have emphasized the obvious similarities between the disinhibited behaviors of adults following damage to the PFC and the emotional reactions and behaviors of normal infants and young children. The cognitive and emotional changes following PFC damage have been attributed to a disruption in the inhibitory control normally exerted by the PFC on subcortical limbic structures such as the hypothalamus and the amygdala. In line with this, Posner and Rothbart (1998) have postulated that emotional self-regulation likely involves the interaction of the midfrontal ACC region with the amygdala.

Based on the corpus of knowledge discussed above regarding the development of emotional self-regulation, this fMRI study (Beauregard et al. 2002) was carried out to identify the neural circuitry underlying voluntary self-regulation of sadness in healthy children. The experimental protocol used was identical to that of the study presented in the preceding section. This time, however, fourteen girls (age range: 8–10, mean age: 9) were scanned in the Sad condition and the Suppression condition. From an experiential perspective, the mean level of reported sadness was significantly higher in the Sad condition (mean = 4.9; range: 2–8) than in the Suppression condition (mean = 3.2; range: 1–8) ($p < 0.01$). As in adults, the viewing of the sad film excerpts did specifically produce the expected emotional reaction (i.e., sadness). With respect to functional brain activity, in the Sad condition, significant loci of activation were

seen in the left VLPFC (BA 47) and, bilaterally, in the midbrain and anterior temporal pole (BA 21). In the Suppression condition, significant loci of activation were noted, bilaterally, in the LPFC (BA 9 and 10), OFC (BA 11), MPFC (BA 10), and rostral ACC (BA 24).

When looking at those results, it is noteworthy that a certain portion of the rostral ACC is connected to the OFC (Devinsky et al. 1995) and has outflow to autonomic, visceromotor, and endocrine systems. The co-activation of the rostral ACC and the OFC, while subjects were attempting to suppress sadness, strongly suggest that these PF regions are part of a neural circuit involved in the regulation of primary emotions. It is also worth mentioning that a region of the MPFC close to that identified in this study, during the Suppression condition, has previously been postulated to be implicated in theory-of-mind tasks (inferences about the mental states of others) (Fletcher, Happe, Frith, Dolan, Frackowiak, & Frith 1995) and metacognitive representation of one's own emotional state (Reiman, Lane, Ahern, Schwartz, Davidson, Friston, Yun, & Chen 1997; Lane 2000). This cortical region receives sensory information from the body and the external environment via the OFC (Barbas 1993; Carmichael & Price 1995) and is heavily interconnected with limbic structures such as the amygdala, ventral striatum, hypothalamus, midbrain periaqueductal gray region, and brainstem autonomic nuclei (Barbas 1995; Carmichael & Price 1995). Such anatomical relationships suggest a role for this area of the MPFC in the integration of the visceromotor aspects of emotional processing with information gathered from the internal and external environments. It thus seems plausible that the MPFC activation noted in children, during the Suppression task, reflected "reflective conscious awareness" (awareness of awareness) (Farthing 1992).

Taken together, our findings indicate that the neural substrate underlying the subjective experience of sadness is comparable in children and adults. Such a similitude provides empirical support to the view that the neural substrate of primary emotions is biologically based and functional from birth or nearly after (Izard 1992). Relative to the differential between the subjective scores noted in the Sad and Suppression conditions (Sad minus Suppression), it is clear that voluntary suppression of sadness was more difficult for children (Sad: 4.9, Suppression: 3.2, $p < 0.01$) than adults (Sad: 5.15, Suppression: 1.85, $p < 0.0001$). With regard to brain regions activated in both studies, the Suppression task recruited more prefrontal areas in children than in adults. In addition, the spatial extent of the LPFC activation was five-fold greater in children than in adults. These differences suggest that voluntary suppression of a primary emotion, such as sadness, requires more prefrontal work in children

than in adults. In agreement with this, it has been demonstrated that children show greater volume of PFC activity than adults when performing tasks requiring active maintenance and/or suppression of different types of information (e.g., go no-go paradigm) (Casey et al. 1995; Cohen, Forman, Braver, Casey, Servan-Schreiber, & Noll 1994). Casey and colleagues have proposed that such differences may reflect maturational differences with respect to the PFC. Along the same lines, it appears reasonably fair to assume that conscious and voluntary self-regulation of emotion is more challenging (cognitively and affectively) in children than in adults because the maturation of the prefronto-subcortical (limbic) connections is not yet completed.

fMRI studies of emotional self-regulation conducted by other groups

Using event-related fMRI, Schaefer and her colleagues (2002) tested the hypothesis that voluntary regulation of negative emotion is associated with modifications in neural activity within the amygdala, a cardinal component of emotion perception. Negative and neutral pictures selected from the International Affective Picture System (IAPS) (Lang, Bradley, & Cuthbert 1998) were presented to seven healthy, right-handed women (aged 19–37 years). Subjects were instructed to either Maintain the initial emotional response induced by the picture throughout its presentation or passively View the picture without regulating the emotion. Each picture presentation lasted 6 sec and was followed by a delay of 8 sec during which the screen was blank. After this delay, subjects indicated how they currently felt using a Likert-type scale and a four-button response keypad (“1” being effectively neutral and “4” being the most negative). Trials were separated by a 10-sec interval. The experiment consisted of 15 runs of 12 trials each.

As expected, subjects reported feeling more negative during negative picture trials than neutral picture trials ($p < 0.0001$). Subjects also reported feeling more negative on Maintain trials than View trials ($p < 0.0005$). In agreement with previous functional neuroimaging studies having shown increased activation in the amygdala in response to emotionally negative stimuli, greater amygdalar signal change was noted during the presentation of negative compared to neutral pictures ($p < 0.01$). Increased activation in the amygdala was associated with maintenance of negative emotion compared to the passive viewing condition ($p < 0.05$). Moreover, a prolonged BOLD signal increase was found in the amygdala when subjects maintained the negative emotional state during the presentation of negative pictures. This amygdalar signal increase was significantly correlated with subjects’ self-reported levels of negative emotion.

These findings suggest that the conscious cognitive processes that modulated the emotional responses of the subjects to the negative pictures were associated with an alteration of the degree of neural activity within the amygdala, i.e., the degree of amygdalar activity can be consciously and voluntarily regulated.

Along the same lines, Hariri et al. (Hariri, Mattay, Tessitore, Fera, & Weinberger 2003) have recently demonstrated, using fMRI, that whereas perceptual processing of threatening and fearful non-face (IAPS) stimuli was associated with a bilateral amygdala response, cognitive appraisal of the same stimuli was associated with a reduction of this amygdala response and a concomitant increase in response of the right prefrontal and anterior cingulate cortices.

Ochsner and co-workers (2002) have conducted another event-related fMRI study about the neural basis of emotional self-regulation. In this study, "reappraisal" was used as a cognitive strategy to modulate emotional experience. Reappraising a negative event in non-emotional terms allows to reduce unpleasant emotion with few of the subjective, cognitive, and physiological negative consequences associated with other emotion-regulatory strategies such as suppression (Jackson, Malmstadt, Larson, & Davidson 2000; Richards & Gross 2000; Gross 1998, 2002). Ochsner et al. (2002) hypothesized that the neural circuits and processing dynamics implicated in the cognitive control of emotion would be similar to those involved in other forms of cognitive control. More specifically, these researchers predicted that reappraisal would involve three processes implemented by the LPFC, MPFC, and dorsal ACC. The first process is the self-generation of a cognitive strategy for reframing an emotional event in non-emotional terms. This strategy has to be kept in mind as long as the eliciting conditions of the emotional experience are present. Clinical neuropsychological (Barcelo & Knight 2002; Stuss, Alexander, Palumbo, Buckle, Sayer, & Pogue 1994) and functional neuroimaging (Cabeza & Nyberg 2000; Smith & Jonides 1999; Barch, Braver, Nystrom, Forman, Noll, & Cohen 1997) studies have associated these functions with working memory processes that depend on the LPFC. The second process allows to monitor interference between top-down reappraisals that counteract emotion and bottom-up evaluations that keep generating an emotional response, indicating the need for further reappraisal. This function is associated with the dorsal ACC (for reviews, see Botvinick, Braver, Barch, Carter, & Cohen 2001; Bush et al. 2000), which has been shown to be involved in various tasks such as monitoring ongoing processing, evaluating the need for cognitive control, response conflict (van Veen, Cohen, Botvinick, Stenger, & Carter 2001), or overriding prepotent response tendencies (Carter, Macdonald, Botvinick, Ross, Stenger, Noll, & Cohen 2000). The third process implicates the re-evaluation of the relationship

between internal (subjective or physiological) states and external emotion-generation stimuli. Such a re-evaluation would be used to monitor changes in one's emotional state during reappraisal. Ochsner et al. (2002) predicted that the dorsal regions of the MPFC would be involved in this process, given that they are implicated in emotional awareness (Lane 2000; Lane et al. 1997), self-related processing (Gusnard, Akbudak, Shulman, & Raichle 2001), and attributions about one's own (Paradiso, Johnson, Andreasen, O'Leary, Watkins, Ponto, & Hichwa 1999; Lane et al. 1997) or another person's emotional state (Happé, Ehlers, Fletcher, Frith, Johansson, Gillberg, Dolan, Frackowiak, & Frith 1996). Ochsner and his colleagues (2002) further hypothesized that reappraisal would influence the cognitive processes involved in the evaluation of the emotional significance of a stimulus. With regard to this issue, it has been postulated that there are at least two types of evaluative processes implicated in the generation of emotion (Lazarus 1991). The first type, relatively unconscious and unintentional, is recruited to determine whether a stimulus is emotionally significant. The second type is recruited consciously and intentionally to evaluate contextual meaning and the adequacy of possible behavioural responses (Lazarus 1991). Various lines of evidence indicate that the amygdala plays a pivotal role in the pre-attentive detection and recognition of emotionally significant stimuli (Anderson & Phelps 2001; Whalen, Rauch, Etcoff, McInerney, Lee, & Jenike 1998), whereas the MOFC is a key structure involved in the representation of the emotional valence of a stimulus (Kawasaki, Kaufman, Damasio, Damasio, Granner, Bakken, Hori, Howard, & Adolphs 2001; O'Doherty, Kringelbach, Rolls, Hornak, & Andrews 2001; Davidson & Irwin 1999; Elliott, Frith, & Dolan 1997).

At the beginning of each trial, a picture appeared in the center of a screen for 4 sec. Subjects (15 healthy, right-handed women, aged 18–30) were instructed to view the picture and allow themselves to experience/feel any emotional response it might elicit. The photo remained on the screen for an additional 4 sec with an instruction either to Attend or Reappraise. On Attend trials, either a negative or a neutral picture (stimuli were selected from the IAPS) was shown and subjects were requested to attend to and be aware of, but not to try to modify, any feelings induced by it. On Reappraise trials, a negative picture was shown and subjects were instructed to reinterpret the picture so that it no longer generated a negative emotional response. The photo then disappeared and, for 3.1 sec, subjects could continue to attend, or reappraise, any feelings that persisted after its presentation. A four-point scale (1 = "weak" to 4 = "strong") rating the strength of current negative emotion was then presented for 3 sec and subjects indicated their current emotional state. Finally,

subjects were instructed to relax for 5 sec. A 900-msec interval separated each trial. Overall, subjects completed one hundred and fourteen 20-sec trials over six separate runs.

Behaviorally, reappraisal of negative pictures successfully lessened negative emotion. Indeed, the average ratings of the strength of negative emotion were significantly lower on Reappraise trials than on Attend trials ($p < 0.01$). Neurally, reappraisal was associated with a significant activation of the dorsal and ventral regions of the left LPFC, as well as the dorsal MPFC. In addition, the right amygdala was significantly more activated on Attend than Reappraise trials. Interestingly, the MOFC displayed greater activation to most negative pictures on Attend than on Reappraise trials, whereas activated areas of the LPFC showed the opposite trend. For one of these areas – the ventral LPFC (VLPFC) – increased activation during reappraisal was correlated across subjects with decreased activation in amygdala.

For Ochsner et al. (2002), these results provide good evidence that reappraisal may modulate the emotion processes implemented in the amygdala and MOFC and involved in the evaluation of the emotional significance and contextual relevance of a stimulus (or event). The specific LPFC and MPFC areas found associated with reappraisal are comparable to the prefrontal areas frequently seen activated in diverse working memory and response-selection tasks that entail resisting to interference from competing stimuli and maintaining information in awareness (Cabeza & Nyberg 1999; Smith & Jonides 1999; Courtney, Petit, Maisog, Ungerleider, & Haxby 1998; Petit, Courtney, Ungerleider, & Haxby 1998). This similitude strongly suggests that a common ensemble of LPFC and MPFC areas underlie the cognitive regulation of both feelings and thoughts (Miller & Cohen 2001; Ochsner & Feldman Barrett 2001; Knight, Staines, Swick, & Chao 1999; Smith & Jonides 1999). The fact that the ventral LPFC activation was inversely correlated with the activation of the amygdala and MOFC indicates that the VLPFC may play an important role in conscious and voluntary regulation of emotion processes. Furthermore, the modulation of the neural activity within the amygdala and the MOFC supports the view that reappraisal can influence processes involved in the evaluation of the emotional significance of a stimulus (Anderson & Phelps 2001; Whalen et al. 1998), as well as those implicated in the evaluation of the significance of that stimulus with reference to current contextual meaning (which is determined by situational or personal goals) (Kawasaki et al. 2001; Ochsner & Feldman Barrett 2001; O'Doherty et al. 2001; Bechara et al. 2000; Rolls 1999; Elliott et al. 1997). Ochsner et al. (2002) postulated that the reappraisal processes implemented by lateral and medial prefrontal regions may play an important role in

the regulation of evaluation processes associated with the OFC. This cortical region may be responsible for the selection of appropriate, and the transient suppression of inappropriate, emotional responses. Because the LPFC and the amygdala have few direct anatomical connections, Ochsner and his colleagues (2002) further contended that the LPFC could influence the amygdala via the MOFC, which has reciprocal connections with both structures (Cavada et al. 2000). By directly modulating the representations of the emotional salience of a stimulus in the MOFC, activation in the LPFC could indirectly down-regulate processing in the amygdala.

Neural circuitry underlying emotional self-regulation

In agreement with the results of the studies conducted by Schaefer et al. (2002) and Ochsner et al. (2002), our findings clearly indicate that conscious and voluntary cognitive processes used to alter the impact of an externally-induced emotion (e.g., sadness) or emotional state (e.g., sexual arousal) can significantly modulate neural activity within cortical (e.g., MPFC, anterior temporal pole, insula, etc.) and subcortical (e.g., amygdala, hypothalamus, midbrain, etc.) structures involved in various aspects of emotion processing (e.g., evaluation of the emotional significance, endocrine and autonomic expression of emotion, etc.).

Based on the results of our fMRI studies and on findings from clinical neuropsychology, we submit that the following neural circuit implements the cognitive strategy used by participants in these studies to self-regulate emotion: first, the LPFC (BA 9, 10) holds in mind the instruction given to the subject, selects the appropriate cognitive operations in order to produce the desired outcome (i.e., suppression of the emotional response induced by the emotional film excerpts), and sends an executive command to the OFC (BA 11), which is charged to suppress the various dimensions associated with emotion. The OFC then sends a message to the amygdala, which is led to change its interpretation of the emotional significance of the stimuli presented. The OFC is then informed about this cognitive "reframing" by virtue of the anatomic projections that the amygdala sends back to this cortical region. Thereafter the OFC orders the rostral ACC (BA 24, 32) to modulate activity in brain structures involved in autonomic, visceral, and endocrine functioning (e.g., hypothalamus, insula, midbrain, brainstem nuclei). Following this, the rostral ACC conveys information back to the OFC with regard to the emotional state of the organism from a physiological perspective. Afterwards, the OFC informs the anterior temporal pole (BA 21, 38) to modify accordingly its attribution of the emotional

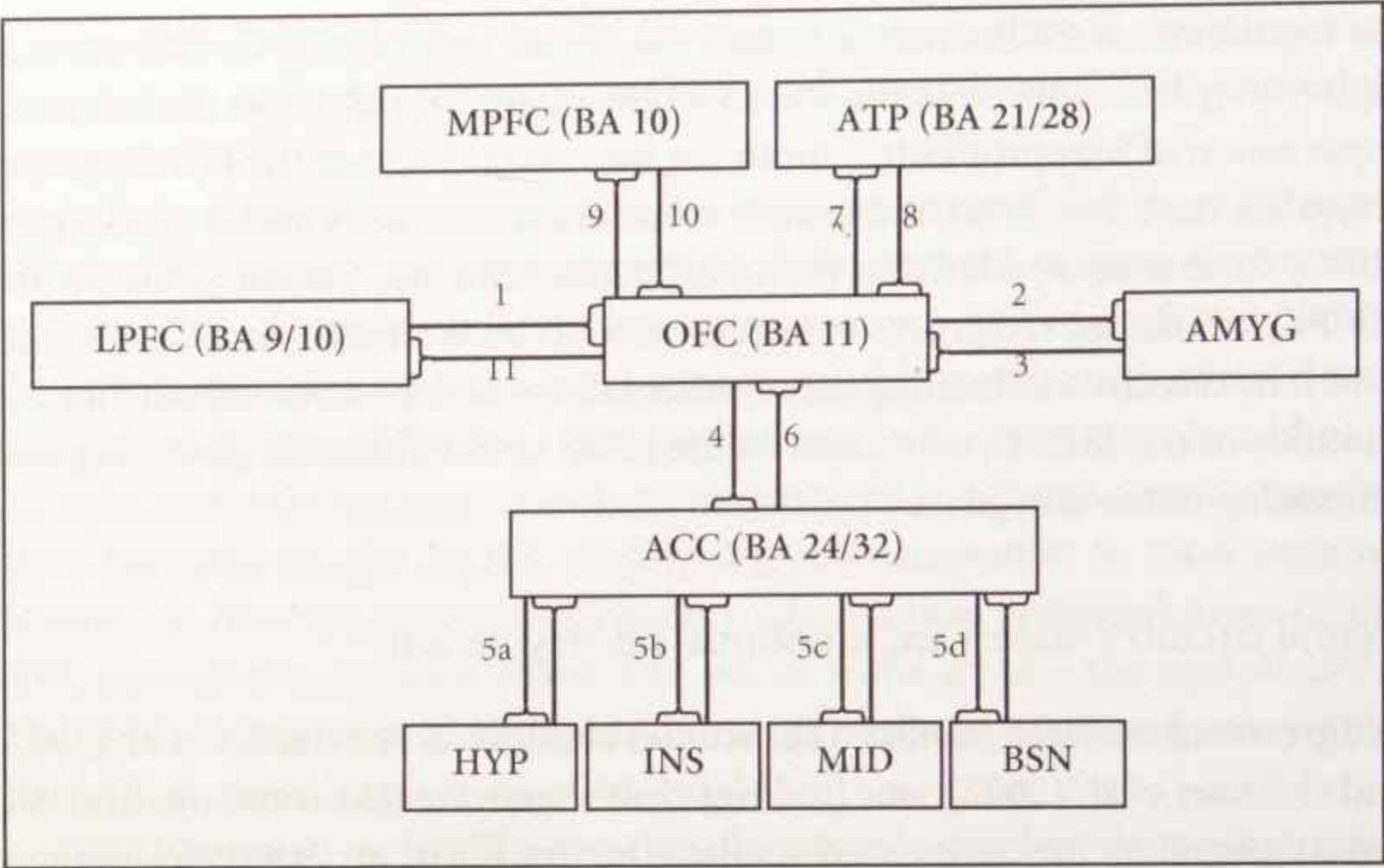


Figure 1. Schematic diagram of a putative neural circuit underlying emotional self-regulation in our fMRI studies. The numbers refer to the temporal sequence of connection between the various components of the circuit. ACC: anterior cingulate cortex; AMYG: amygdala; ATP: anterior temporal pole; BA: Brodmann area; BSN: brainstem nuclei; HYP: hypothalamus; INS: insula; LPFC: lateral prefrontal cortex; MID: mid-brain; MPFC: medial prefrontal cortex; OFC: orbitofrontal cortex.

colour to the subject’s experience. Then the OFC informs the MPFC (BA 10) of the diverse changes related to the individual’s emotional state. This allows the MPFC to carry out a metacognitive analysis about the subjective experience of the individual from an emotional standpoint. Finally, the feedback from the MPFC authorizes the OFC to inform the LPFC regarding whether the current emotional state of the individual (cognitively, physiologically, experientially, behaviorally) has to be further modulated (see Figure 1).

A theoretical note about emotional self-regulation and mind-brain interaction

To a large extent, cognitive psychology and cognitive neuroscience still rest on computationalism to explain the functioning of the mind and brain. According to this view, human behavior is determined by the activity of unconscious sub-personal executive processors (modules) and their neural counterparts. This

view dismisses essential human attributes such as subjectivity, intentional self-guidance, and self-reactiveness by merely reducing human beings to biological computers. As noted by Bandura (2001), a major problem with this view is that the explanatory and predictive value of agentic factors such as beliefs, goals, aspirations, desires, and expectations is very high. Humans are conscious, intentional beings, creative and proactive agents of experiences rather than strictly undergoers of experiences. Self-consciousness (i.e., being conscious of oneself as an immediate subject of experience), self-agency (i.e., experiencing oneself as being the cause of an action), and self-regulatory metacognitive capacities (e.g., self-reflectiveness; self-regulation of motivation, emotion, and action; deliberate processing of information for the evaluation, selection, or regulation of courses of action, etc.) enable human beings to achieve tasks and attain goals that provide direction and meaning to their lives. In contradistinction to eliminative materialists, we posit that self-consciousness and agentic mind processes, although closely related to brain processes, cannot be strictly reduced to neuroelectrical or neurochemical events. Indeed, agentic factors are not eliminatable and the mentalistic terminology used to refer to these variables remains absolutely essential. Regarding this issue, Bandura (2001) rightly points out that mapping the activity of the neural circuit underlying Martin Luther King's "I Have a Dream" speech would reveal little with respect to the socially powerful significance of this speech and the agentic effort that invigorated its creation.

It follows from this that the distinction between mind processes – which belong to the Mind domain (this domain includes self-consciousness and agentic capacities) – and brain processes – which belong to the Brain domain – is necessary and critical to reach a satisfying understanding of human functioning and behavior. Yet, we contend that these two domains (or dimensional levels) are not radically separated, since they originate from a unified field underlying the web of relationships uniting energy and information in our universe. These domains and their processes can be conceived as complex dynamic systems (or informational structures) in a constant state of flux (the "*enchanted loom*" of Sherrington) and transforming through microstates lasting for very brief periods of time (i.e., ms). The components within both systems process and exchange information (from the Latin word *Informare*, which means: to form, to shape, and to organize) so that these systems can function and evolve. For instance, in the Brain domain, information processed by neurons in the cerebellum may be conveyed to neurons in the motor cortex to adjust the posture of a given man. In the Mind domain, the cognitive evaluation made by a woman of an event occurring within a social context may reactivate old

memories and lead to a specific type of emotional reaction. Importantly, the components of one system can also influence the activity of the components found in the other system. In line with this, cognitive neuroscience and cognitive neuropsychology have gathered an impressive amount of data showing how specific types of mind processes are correlated with neural activity in selective regions of the brain. Conversely, conscious and agentic mind processes (for example, a desire or a belief) are capable to significantly influence the nature of the neuroelectrical and neurochemical processes occurring in the Brain domain. Evolution has brought a natural transduction between conscious/agentic mind processes and electrical/chemical activity in selective regions of the brain (regarding this transduction mechanism, see Chapter seven). Indeed, the language of the mind is naturally translated into the language of the brain (at different levels of organization – biophysical, molecular, cellular, neural circuit, etc.). This coupling mechanism accounts for the fact that, for instance, sad thoughts maintained by an individual for a given period of time may affect the rate of synthesis of serotonin in brain regions implicated in emotion processing. In turn, this neurochemical change may affect the immune and endocrine systems via the psychosomatic network (see Chapter 9).

The capacity of self-consciousness and self-agentic abilities to influence brain activity is clearly supported by the results of the functional neuroimaging studies reported in this chapter. The causal efficaciousness of self-consciousness and self-agency is further demonstrated in neuroimaging studies that have measured the impact of cognitive-behavioral therapy (CBT) on brain activity. In the first of these studies, Schwartz and colleagues (Schwartz, Stoessel, Baxter, Martin, & Phelps 1996) have shown, using positron emission tomography (PET), that CBT can lead to significant changes in glucose metabolic rates in the caudate nucleus of patients with obsessive-compulsive disorder. Along the same lines, we recently (Paquette, Lévesque, Mensour, Leroux, Beaudoin, Bourgouin, & Beaugregard 2003) used fMRI to probe the effects of CBT on the neural correlates of spider phobia. Twelve subjects suffering from spider phobia were scanned, before and after CBT, during the viewing of film excerpts depicting living spiders in motion. Results showed that, in phobic subjects before CBT, the transient state of fear triggered, during the viewing of the phobogenic stimuli, was correlated with significant activation of the right LPFC (BA 10), right parahippocampal gyrus, and visual associative cortical areas (bilaterally). We hypothesized that the LPFC activation reflected the use of metacognitive strategies aimed at self-regulating the fear triggered by the spider film excerpts, whereas the parahippocampal activation related to an automatic reactivation of the contextual fear memory that led to the de-

velopment of avoidance behavior and the maintenance of spider phobia. After successful completion of CBT, no significant activation was found in the LPFC or the parahippocampal gyrus. It is noteworthy that a crucial element of CBT consisted in the correction of the erroneous cognitive schemas entertained by phobic subjects about spiders. These findings strongly suggest that CBT has the potential to modify the dysfunctional neural circuitry associated with anxiety disorders. They further indicate that changes made at the mind level, within a psychotherapeutic context, are capable of functionally “rewire” the brain. In other words, “change the mind and you change the brain”.

With the emergence of self-consciousness and agentic capacities, biological evolution has enabled human beings to consciously and voluntarily shape the functioning of their brains. These advanced capacities prevent humans from being totally enslaved by genetic determinism and allow them to deliberately create new social and cultural environments. Through *Homo sapiens*, evolution becomes conscious, that is, much more than simply survival and reproduction, and human action begins to be driven more by complex sets of insights, goals, desires and beliefs than by “selfish” genes. This explains why the moral values associated with a given spiritual world view will help certain individuals to keep in check their emotional impulses and to behave in an altruistic fashion. In such cases, moral conscience replace innate programming as behavioral regulator, and ethical behaviors constitute an emancipation from genetic determinism and the primitive dictates of the mammalian brain. Such freedom is responsible for the fact that, despite the homogeneity of the human genome across human societies, we find fighting cultures that value and foster aggression whereas in pacific cultures, aggression is negatively perceived and almost non-existent (Alland 1972; Sanday 1981). Given the current state of affairs on our planet, it is comforting to realize that more and more people are now awakening to the notion that they can be at the helm of their own future, individually as well as collectively. A future that will hopefully favour the development of the whole human species.

Summary

In this chapter, we have presented the functional neuroimaging studies that have been conducted to date regarding emotional self-regulation. Results of fMRI studies carried out by our group (Beauregard et al. 2001; Lévesque et al. 2003) have revealed that, in adults, the neural substrates associated with conscious and voluntary regulation of an emotional impulse (such as sexual

arousal) or of a primary emotion (such as sadness) show an important overlap. Moreover, the fMRI study we have conducted in children, with respect to the regulation of sadness, showed that the same sub-divisions of the PFC underlie self-regulation of sad feelings in childhood and adulthood (at the exception of the MPFC – BA 10 – which was not seen in adults). Both in adults and children, these neural substrates involved the LPFC (BA 9 and 10), the OFC (BA 11), and the rostral portion of the ACC (BA 24 and 32). These findings fit rather nicely with those of clinical neuropsychology showing an involvement of these brain regions in emotional self-regulation. A few differences were noted, however, between children and adults in regard to regulation of sadness. Actually, in adults, the loci of activation were essentially localized in the right hemisphere whereas they were distributed bilaterally in children. In addition, the spatial extent of the loci of activation in the LPFC was five-fold greater in children than in adults. We have interpreted these differences as suggesting that the voluntary suppression of a primary emotion requires more prefrontal work in children than in adults. It is noteworthy that Ochsner et al. (2002) found also activation in areas of the LPFC and the MPFC located close to the loci of activation found in our studies. Such a similitude may indicate that comparable regions of the PFC may underlie the use of different cognitive strategies to self-regulate emotions.

Taken together, these findings have led us to propose the existence of a putative neural circuit that may have underlied emotional self-regulation in our studies. Finally, in the last section of the chapter, we have argued in favour of a new view in cognitive psychology and cognitive neuroscience that recognize the human agentic capacities and take into account these capacities and self-consciousness to explain the interaction between mind processes and brain processes.

Acknowledgements

The writing of this chapter was supported by grants from National Sciences and Engineering Research Council of Canada (NSERC) and National Alliance for Research in Schizophrenia and Depression (NARSAD) to M.B.

Address correspondence to: Mario Beauregard, Department of Psychology, Université de Montréal, C.P. 6128, succursale Centre-Ville, Montréal (Québec) Canada, H3C 3J7, mario.beauregard@umontreal.ca

References

- Alland Jr., A. (1972). *The human imperative*. New York: Columbia University Press.
- Anderson, S. W., Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1999). Impairment of social and moral behavior related to early damage in human prefrontal cortex. *Nature Neuroscience*, 11, 1032–1037.
- Anderson, A. K. & Phelps, E. A. (2001). Lesions of the human amygdala impair enhanced perception of emotionally salient events. *Nature*, 17, 305–309.
- Bandura, A. (2001). Social cognitive theory: an agentic perspective. *Annual Review of Psychology*, 52, 1–26.
- Barbas, H. (1993). Organization of cortical afferent input to the orbitofrontal area in the rhesus monkey. *Neuroscience*, 56, 841–864.
- Barcelo, F. & Knight, R. T. (2002). Both random and perseverative errors underlie WCST deficits in prefrontal patients. *Neuropsychologia*, 40, 349–56.
- Barch, D. M., Braver, T. S., Nystrom, L. E., Forman, S. D., Noll, D. C., & Cohen, J. D. (1997). Dissociating working memory from task difficulty in human prefrontal cortex. *Neuropsychologia*, 35, 1373–80.
- Beauregard, M., Lévesque, J., & Bourgouin, P. (2001). Neural correlates of the conscious self-regulation of emotion. *Journal of Neuroscience*, 21, RC165 (1–6).
- Beauregard, M., Lévesque, J., Eugène, F., Joannette, Y., Mensour, B., Beaudoin, G., Leroux, J.-M., & Bourgouin, P. (2002). Functional neuroanatomy underlying voluntary suppression of sadness in children. *Society for Neuroscience 32nd Annual meeting, Orlando. Abstract: 10434*.
- Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994) Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*, 50, 7–12.
- Bechara, A., Tranel, D., & Damasio, H. (2000). Characterization of the decision-making deficit of patients with ventromedial prefrontal cortex lesions. *Brain*, 123, 2189–202.
- Benes, F. (1997). Corticolimbic circuitry and the development of psychopathology during childhood and adolescence. In N. A. Krasnegor, Reid Lyon, G., & Goldman-Rakic, P. S. (Eds.), *Development of the prefrontal cortex* (pp. 211–240). Baltimore, MD: Paul H. Brookes Publishing Co., Inc.
- Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, 108, 624–52.
- Bourgeois J.-P., Goldman-Rakic, P. S., & Rakic P. (1994). Synaptogenesis in the prefrontal cortex of rhesus monkeys. *Progress in Brain Research*, 102, 227–43.
- Brody, A. L., Barsom, M. W., Bota, R. G., & Saxena, S. (2001). Prefrontal-subcortical and limbic circuit mediation of major depressive disorder. *Seminars in Clinical Neuropsychiatry*, 6, 102–12.
- Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Science*, 4, 215–222.
- Cabeza, R. & Nyberg, L. (2000). Neural bases of learning and memory: functional neuroimaging evidence. *Current Opinion in Neurology*, 13, 415–421.
- Carmichael, S. T. & Price, J. L. (1995). Limbic connections of the orbital and medial prefrontal cortex in macaque monkeys. *Journal of Comparative Neurology*, 25, 615–641.

- Carter, C. S., Macdonald, A. M., Botvinick, M., Ross, L. L., Stenger, V. A., Noll, D., & Cohen, J. D. (2000). Parsing executive processes: Strategic vs. evaluative functions of the anterior cingulate cortex. *Proceedings of the National Academy of Sciences of the USA*, 97, 1944–1948.
- Casey, B. J., Cohen, J. D., Jezzard, P., Turner, R., Noll, D. C., Trainor, R. J., Giedd, J., Kaysen, D., Hertz-Pannier, L., & Rapoport, J. L. (1995). Activation of prefrontal cortex in children during a nonspatial working memory task with functional MRI. *Neuroimage*, 2, 221–229.
- Cavada, C., Company, T., Tejedor, J., Cruz-Rizzolo, R. J., & Reinoso-Suarez, F. (2000). The anatomical connections of the macaque monkey orbitofrontal cortex. A review. *Cerebral Cortex*, 10, 220–242.
- Caviness, V. S. Jr., Kennedy, D. N., Richelme, C., Rademacher, J., & Filipek, P. A. (1996). The human brain age 7–11 years: a volumetric analysis based on magnetic resonance images. *Cerebral Cortex*, 6, 726–736.
- Cechetto, D. F. (1994). Identification of a cortical site for stress-induced cardiovascular dysfunction. *Integration of Physiological and Behavioral Sciences*, 29, 362–373.
- Cohen, J. D., Forman, S. D., Braver, T. S., Casey, B. J., Servan-Schreiber, D., & Noll, D. C. (1994). Activation of prefrontal cortex in a nonspatial working memory task with functional MRI. *Human Brain Mapping*, 1, 293–304.
- Cole, P. M., Michel, M. K., & Teti, L. O. (1994). The development of emotion regulation and dysregulation: A clinical perspective. *Monograph of the Society for Research on Child Development*, 59, 73–100.
- Courtney, S. M., Petit, L., Maisog, J. M., Ungerleider, L. G., & Haxby, J. V. (1998). An area specialized for spatial working memory in human frontal cortex. *Science*, 279, 1347–1351.
- Cummings, J. L. (1993). Frontal-subcortical circuits and human behavior. *Archives of Neurology*, 50, 873–880.
- Damasio, A. R. (1995). On some functions of the human prefrontal cortex. *Annals of the New York Academy of Sciences*, 769, 241–251.
- Damasio, A. R., Tranel, D., & Damasio, H. (1990). Individuals with sociopathic behavior caused by frontal damage fail to respond autonomically to social stimuli. *Behavioural Brain Research*, 41, 81–94.
- Davidson, R. J. & Irwin, W. (1999). The functional neuroanatomy of emotion and affective style. *Trends in Cognitive Science*, 3, 11–21.
- Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation – A possible prelude to violence. *Science*, 289, 591–594.
- Davis, M. (1992). The role of the amygdala in fear and anxiety. *Annual Review of Neuroscience*, 15, 353–75.
- Dawson, G., Panagiotides, H., Grofer Klinger, L., & Hill, D. (1992). The role of frontal lobe functioning in the development of infant self-regulatory behavior. *Brain and Cognition*, 20, 152–175.
- Devinsky, O., Morrell, M. J., & Vogt, B. A. (1995). Contributions of anterior cingulate cortex to behaviour. *Brain*, 118, 279–306.
- Dias, R., Robbins, T. W., & Roberts, A. C. (1996). Dissociation in prefrontal cortex of affective and attentional shifts. *Nature*, 380, 69–72.

- Dolan, R. J. (1999). On the neurology of morals. *Nature Neuroscience*, 11, 927–929.
- Elliott, F. A. (1990). Neurology of aggression and episodic dyscontrol. *Seminars in Neurology*, 10, 303–312.
- Elliott, R., Frith, C. D., & Dolan, R. J. (1997). Differential neural response to positive and negative feedback in planning and guessing tasks. *Neuropsychologia*, 35, 1395–1404.
- Eslinger, P. J., Biddle, K. R., & Grattan, L. M. (1997). Cognitive and social development in children with prefrontal cortex lesions. In N. A. Krasnegor, G. Reid Lyon, & P. S. Goldman-Rakic (Eds.), *Development of the prefrontal cortex* (pp. 295–335). Baltimore, MD: Paul H. Brookes Publishing Co., Inc.
- Eslinger, P. J. (1999a). Orbital frontal cortex: historical and contemporary views about its behavioral and physiological significance (Part I). *Neurocase*, 5, 225–229.
- Eslinger, P. J. (1999b). Orbital frontal cortex: behavioral and physiological significance (Part II). *Neurocase*, 5, 299–300.
- Farrow, T. F. D., Zheng, Y., Wilkinson, I. D., Spence, S. A., Deakin, J. F., Tarrier, N., Griffiths, P. D., & Woodruff, P. W. (2001). Investigating the functional anatomy of empathy and forgiveness. *NeuroReport*, 12, 2433–2438.
- Farthing, W. G. (1992). *The Psychology of consciousness*. Englewood Cliffs, NJ: Prentice Hall.
- Filley, C. M. (1995). *Neurobehavioral anatomy*. Niwot, CO: University Press of Colorado.
- Flavell, J. H. (1979). Metacognition and cognitive monitoring: A new area of cognitive development inquiry. *American Psychologist*, 34, 906–911.
- Fletcher, P. C., Happe, E., Frith, U., Dolan, R. J., Frackowiak, R. S., & Frith, C. D. (1995). Other minds in the brain: A functional imaging study of “theory of mind” in story comprehension. *Cognition*, 57, 109–128.
- Fox, N. A. & Bell, M. A. (1990). Electrophysiological indices of frontal lobe development. *Annals of the New York Academy of Sciences*, 608, 677–704.
- Fox, N. A., Henderson, H. A., & Marshall, P. J. (2001). The biology of temperament: An integrative approach. In C. A. Nelson & M. Luciana (Eds.), *Handbook of developmental cognitive neuroscience* (pp. 631–645). Cambridge, MA: MIT Press.
- Frijda, N. H. (1986). *The Emotions*. Cambridge University Press.
- Frith, C. & Dolan, R. (1996). The prefrontal cortex in higher cognitive functions. *Cognitive Brain Research*, 5, 175–181.
- Fuster, J. M. (1997). *The prefrontal cortex: Anatomy, physiology and neuropsychology of the frontal lobe* (3rd edition). Philadelphia, PA: Lippincott-Raven.
- Fuster, J. M. (1999). Synopsis of function and dysfunction of the frontal lobe. *Acta Psychiatrica Scandinavica*, 99, 51–57.
- Giancola, P. R. & Zeichner, A. (1994). Neuropsychological performance on tests of frontal-lobe functioning and aggressive behavior in men. *Journal of Abnormal Psychology*, 103, 832–835.
- Goldman-Rakic, P. S. (1987). Circuitry of primate prefrontal cortex and regulation of behavior by representational memory. In V. B. Mountcastle, F. Plum, & S. R. Geiger (Eds.), *Handbook of Physiology*, Vol. 5 (pp. 373–417). Bethesda, MD: American Physiological Society.
- Grafman, J., Schwab, K., Warden, D., Pridgen, A., Brown, H. R., & Salazar, A. M. (1996). Frontal lobe injuries, violence, and aggression: a report of the Vietnam Head Injury Study. *Neurology*, 46, 1231–1238.

- Grafman, J. & Litvan, I. (1999). Importance of deficits in executive functions. *Lancet*, 354, 1921–1923.
- Greene, J. D., Sommerville, R. B., Nystrom, L. E., Darley, J. M., & Cohen, J. D. (2001). An fMRI investigation of emotional engagement in moral judgment. *Science*, 293, 2105–2108.
- Gross, J. J. (1999). Emotion regulation: past, present, future. *Cognition and Emotion*, 13, 551–573.
- Gross, J. J. (2001). Emotion regulation in adulthood: timing is everything. *Current Directions in Psychological Science*, 10, 214–219.
- Gross, J. J. (2002). Emotion regulation: affective, cognitive, and social consequences. *Psychophysiology*, 39, 281–291.
- Gusnard, D. A., Akbudak, E., Shulman, G. L., & Raichle, M. E. (2001). Medial prefrontal cortex and self-referential mental activity: Relation to a default mode of brain function. *Proceedings of the National Academy of Sciences of the USA*, 98, 4259–4264.
- Happe, E., Ehlers, S., Fletcher, P., Frith, U., Johansson, M., Gillberg, C., Dolan, R., Frackowiak, R., & Frith, C. (1996). 'Theory of mind' in the brain. Evidence from a PET scan study of Asperger syndrome. *Neuroreport*, 8, 197–201.
- Hariri, A., Bookheimer, S. Y., & Mazziotta, J. C. (2000). Modulating emotional responses: effects of a neocortical network on the limbic system. *NeuroReport*, 11, 43–48.
- Hariri, A. R., Mattay, V. S., Tessitore, A., Fera, F., & Weinberger, D. R. (2003). Neocortical modulation of the amygdala response to fearful stimuli. *Biological Psychiatry*, 53, 494–501.
- Henry, J. P. (1986). Neuroendocrine patterns of emotional response. In R. Plutchik & H. Kellerman (Eds.), *Biological foundations of emotion*. New York: Academic Press.
- Huttenlocher, P. R. (1994). Synaptogenesis, synapse elimination, and neural plasticity in human cerebral cortex. In C. A. Nelson (Ed.), *Threats to optimal development: Integrating, biological, psychological, and social risk factors* (Minnesota Symposia on Child Psychology), Vol. 27 (pp. 35–54). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Izard, C. E. (1992). Basic emotions, relations among emotions, and emotion-cognition relations. *Psychological Review*, 99, 561–565.
- Jackson, D. C., Malmstadt, J. R., Larson, C. L., & Davidson, R. J. (2000). Suppression and enhancement of emotional responses to unpleasant pictures. *Psychophysiology*, 37, 515–522.
- Karama, S., Lecours, A. R., Leroux, J.-M., Joubert, S., Bourgouin, P., Beaudoin, G., & Beaugregard, M. (2002). Areas of brain activation in males and females during viewing of erotic film excerpts. *Human Brain Mapping*, 16, 1–13.
- Kawasaki, H., Kaufman, O., Damasio, H., Damasio, A. R., Granner, M., Bakken, H., Hori, T., Howard, M. A. 3rd, & Adolphs, R. (2001). Single-neuron responses to emotional visual stimuli recorded in human ventral prefrontal cortex. *Nature Neuroscience*, 4, 15–16.
- Knight, R.T., Staines, W. R., Swick, D., & Chao, L. L. (1999). Prefrontal cortex regulates inhibition and excitation in distributed neural networks. *Acta Psychologica (Amst.)*, 101, 159–78.
- Kochanska, G., Murray, K., & Coy, K. C. (1997). Inhibitory control as a contributor to conscience in childhood: from toddler to early school age. *Child Development*, 68, 263–277.

- Kopp, C. B. (1989). Regulation of distress and negative emotions: A developmental view. *Developmental Psychology*, 25, 343–354.
- Lane, R. D., Fink, G. R., Chau, P. M. L., & Dolan, R. J. (1997). Neural activation during selective attention to subjective emotional responses. *NeuroReport*, 8, 3969–3972.
- Lane, R. D. (2000). Neural correlates of conscious emotional experience. In R. D. Lane & L. Nadel (Eds.), *Cognitive neuroscience of emotion* (pp. 345–370). New York: Oxford University Press.
- Lane, R. D. & Nadel, L. (2000). *Cognitive neuroscience of emotion*. New York: Oxford University Press.
- Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1998). Emotion and motivation: Measuring affective perception. *Journal of Clinical Neurophysiology*, 15, 397–408.
- Langston, C. A. (1994). Capitalizing on and coping with daily-life events: Expressive responses to positive events. *Journal of Personality and Social Psychology*, 67, 1112–1125.
- Lapierre, D., Braun, C. M. J., & Hodgings, S. (1995). Ventral frontal deficits in psychopathy: neuropsychological test findings. *Neuropsychologia*, 33, 139–151.
- Lazarus, R. S. (1991) *Emotion and adaptation*. Oxford, UK: Oxford University Press.
- Levenson, C. A. (1994). Human emotion: A functional view. In P. Ekman & R. J. Davidson (Eds.), *Fundamental questions about the nature of emotion* (pp. 123–126). New York: Oxford University Press.
- Lévesque, J., Eugène, F., Joannette, Y., Paquette, V., Mensour, B., Beaudoin, G., Leroux, J.-M., Bourgouin, P., & Beauregard, M. (2003). Neural circuitry underlying voluntary self-regulation of sadness. *Biological Psychiatry*, 53, 502–510.
- Lewis, M. (1995). Aspects of self: From systems to ideas. In P. Rochat (Ed.), *The self in infancy: Theory and research*. Advances in psychology (pp. 95–115). North Holland: Elsevier Science Publishers.
- Lewis, M. (1998) The development and structure of emotions. In M. F. Mascolo & S. Griffin (Eds.), *What develops in emotional development?* (pp. 29–50). New York: Plenum Press.
- Mangelsdorf, S. C., Shapiro, J. R., & Marzolf, D. (1996). Developmental and temperamental differences in emotional regulation in infancy. *Child Development*, 66, 1817–1828.
- Masters, J. C. (1991). Strategies and mechanisms for the personal and social control of emotion. In J. Garber & K. A. Dodge (Eds.), *The development of emotion regulation and dysregulation* (pp. 182–207). Cambridge, UK: University Press.
- MacFall, J. R., Payne, M. E., Provenza, J. E., Krishnan, K. R. R. (2001). Medial orbital frontal lesions in late-onset depression. *Biological Psychiatry*, 49, 803–806.
- Mesulam, M.-M. (1985). Patterns in behavioural neuroanatomy: Association areas, the limbic system, and hemispheric specialization. In M.-M. Mesulam (Ed.), *Principles of Behavioral Neurology* (pp. 1–70). Philadelphia: F.A. Davis Co.
- Mesulam, M.-M. (1986). Frontal cortex and behavior. *Annals of Neurology*, 19, 320–325.
- Miller, E. K. & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, 24, 167–2202.
- Moll, J., Eslinger, P. J., & de Oliveira-Souza, R. (2001). Frontopolar and anterior temporal cortex activation in a moral judgment task: Preliminary functional MRI results in normal subjects. *Arquiva de Neuropsiquiatria*, 59, 657–664.

- Moll, J., de Oliveira-Souza, R., Eslinger, P. J., Bramati, I. E., Mourao-Miranda, J., Andreiuolo, P. A., & Pessoa, L. (2002). The neural correlates of moral sensitivity: A functional magnetic resonance imaging investigation of basic and moral emotions. *Journal of Neuroscience*, 22, 2730–2736.
- Morecraft, R. J., Geula, C., & Mesulam, M.-M. (1992). Cytoarchitecture and neural afferents of orbitofrontal cortex in the brain of the monkey. *Journal of Comparative Neurology*, 323, 341–358.
- Nagashima, K., Nakai, S., Tanaka, M., & Kanosue, K. (2000). Neuronal circuitries involved in thermoregulation. *Autonomic Neuroscience*, 85, 18–25.
- Nauta, W. J. H. (1971). The problem of the frontal lobe: A reinterpretation. *Journal of Psychiatry Research*, 8, 167–187.
- Ochsner, K. N. & Feldman Barrett, L. (2001). A multiprocess perspective on the neuroscience of emotion. In *Emotions: Current Issues and Future Directions* (pp. 38–81). New York: The Guilford Press.
- Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. (2002). Rethinking feelings: an fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience*, 14, 1215–1229.
- O'Doherty, J., Kringelbach, M. L., Rolls, E. T., Hornak, J., & Andrews, C. (2001). Abstract reward and punishment representations in the human orbitofrontal cortex. *Nature Neuroscience*, 4, 95–102.
- Ongür, D., An X., & Price, J. L. (1998). Prefrontal cortical projections to the hypothalamus in macaque monkeys. *Journal of Comparative Neurology*, 401, 480–505.
- Paquette, V., Lévesque, J., Mensour, B., Leroux, J.-M., Beaudoin, G., Bourgouin, P., & Beaugue, M. (2003) "Change the mind and you change the brain": Effects of cognitive-behavioral therapy on the neural correlates of spider phobia. *NeuroImage*, 18, 401–409.
- Paradiso, S., Johnson, D. L., Andreasen, N. C., O'Leary, D. S., Watkins, G. L., Ponto, L. L., & Hichwa, R. D. (1999). Cerebral blood flow changes associated with attribution of emotional valence to pleasant, unpleasant, and neutral visual stimuli in a PET study of normal subjects. *American Journal of Psychiatry*, 156, 1618–1629.
- Parrott, W. G. (1993). Beyond hedonism: Motives for inhibiting good moods and for maintaining bad moods. In: D. M. Wegner & Pennebaker J. W. (Eds.), *Handbook of mental control* (pp. 278–308). Englewood Cliffs: Prentice-Hall.
- Petit, L., Courtney, S. M., Ungerleider, L. G., & Haxby, J. V. (1998). Sustained activity in the medial wall during working memory delays. *Journal of Neuroscience*, 18, 9429–9437.
- Pfefferbaum, A., Mathalon, D. H., Sullivan, E. V., Rawles, J. M., Zipursky, R. B., & Lim, K. O. (1994). A quantitative magnetic resonance imaging study of changes in brain morphology from infancy to late adulthood. *Archives of Neurology*, 51, 874–887.
- Plutchik, R. (1994). *The psychology and biology of emotion*. New York: Harper Collins College Publishers.
- Posner, M. I. & Rothbart, M. K. (1998). Attention, self-regulation and consciousness. *Philosophical Transactions of the Royal Society of London B Biological Sciences*, 353, 1915–1927.
- Price, J. L. (1999). Prefrontal cortical networks related to visceral function and mood. *Annals of New York Academy of Sciences*, 877, 383–396.

- Rakic, P., Bourgeois, J. P., & Goldman-Rakic, P. S. (1994). Synaptic development of the cerebral cortex: implications for learning, memory, and mental illness. *Progress in Brain Research*, 102, 227–243.
- Reiman, E. M., Lane, R. D., Ahern, G. L., Schwartz, G. E., Davidson, R. J., Friston, K. J., Yun, L.-S., & Chen, K. (1997). Neuroanatomical correlates of externally and internally generated human emotion. *American Journal of Psychiatry*, 154, 918–925.
- Rempel-Clower, N. L. & Barbas, H. (1998). Topographic organization of connections between the hypothalamus and prefrontal cortex in the rhesus monkey. *Journal of Comparative Neurology*, 398, 393–419.
- Richards, J. M. & Gross, J. J. (2000). Emotion regulation and memory: the cognitive costs of keeping one's cool. *Journal of Personality and Social Psychology*, 79, 410–24.
- Roberts, A. C. & Wallis, J. D. (2000). Inhibitory Control and Affective Processing in the prefrontal Cortex: Neuropsychological Studies in the common Marmoset. *Cerebral Cortex*, 10, 252–262.
- Sanday, P. R. (1981). The socio-cultural context of rape: A cross-cultural study. *Journal of Societal Issues*, 37, 5–27.
- Schaefer, S. M., Jackson, D. C., Davidson, R. J., Aguirre, G. K., Kimberg, D. Y., & Thompson-Schill, S. L. (2002). Modulation of amygdalar activity by the conscious regulation of negative emotion. *Journal of Cognitive Neuroscience*, 14, 913–921.
- Schwartz, J. M., Stoessel, P. W., Baxter, L. R. Jr., Martin, K. M., & Phelps, M. E. (1996). Systematic changes in cerebral glucose metabolic rate after successful behavior modification treatment of obsessive-compulsive disorder. *Archives of General Psychiatry*, 53, 109–113.
- Sequeira, H. & Roy, J.-C. (1993). Cortical and hypothalamo-limbic control of electrodermal responses. In J.-C. Roy, W. Boucsein, D. Fowles, & J. Gruzelier (Eds.), *Progress in electrodermal research* (pp. 93–114). New York: Plenum.
- Silver, J. M. & Yudofsky, S. C. (1987). Aggressive behavior in patients with neuropsychiatric disorders. *Psychiatric Annals*, 17, 367–370.
- Smith, E. E. & Jonides, J. (1999). Storage and executive processes in the frontal lobes. *Science*, 283, 1657–1661.
- Stuss, D. T. & Benson, D. F. (1984). Neuropsychological studies of the frontal lobes. *Psychological Bulletin*, 95, 3–28.
- Stuss, D. T., Alexander, M. P., Palumbo, C. L., Buckle, L., Sayer, L., & Pogue, J. (1994). Organizational strategies of patients with unilateral or bilateral frontal lobe injury in word list learning tasks. *Neuropsychology*, 8, 355–373.
- Thompson, R. A. (1990). Emotion and self-regulation. In R. A. Thompson (Ed.), *Socioemotional development: Nebraska symposium on motivation* (pp. 367–467). Lincoln, NE: University of Nebraska Press.
- Tooby, J. & Cosmides, L. (1990). On the universality of human nature and the uniqueness of the individual: the role of genetics and adaptation. *Journal of Personality*, 58, 17–67.
- Tucker, D. M., Luu, P., & Pribram, K. H. (1995). Social and emotional self-regulation. *Annals of New York Academy of Sciences*, 769, 213–239.
- van Veen, V., Cohen, J. D., Botvinick, M. M., Stenger, V. A., & Carter, C. S. (2001). Anterior cingulate cortex, conflict monitoring, and levels of processing. *Neuroimage*, 14, 1302–1308.

- Vogt, B. A., Finch, D. M., & Olson, C. R. (1992). Functional heterogeneity in cingulate cortex: the anterior executive and posterior evaluative regions. *Cerebral Cortex*, 2, 435–443.
- Whalen, P. J., Rauch, S. L., Etcoff, N. L., McInerney, S. C., Lee, M. B., & Jenike M. A. (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *Journal of Neuroscience*, 18, 411–418.
- Yakovlev, P. I. & Lecours, A. R. (1967). The myelogenetic cycles of regional maturation of the brain. In A. Mankowski (Ed.), *Regional development of the brain in early life* (pp. 3–69). Philadelphia: Davis.
- Zald, D. H. & Kim, S. W. (1996). The anatomy and function of the orbito-frontal cortex I: Anatomy, neurocircuitry, and obsessive-compulsive disorder. *Journal of Neuropsychiatry and Clinical Neuroscience*, 8, 125–138.

CHAPTER 7

The volitional influence of the mind on the brain, with special reference to emotional self-regulation

Jeffrey M. Schwartz, Henry P. Stapp, and Mario Beauregard

Suppose I say of a friend: "He isn't an automaton". – What information is conveyed by this, and to whom would it be information? To a *human being* who meets him in ordinary circumstances? What information *could* it give him? (At the very most that this man always behaves like a human being, and not occasionally like a machine.)

"I believe that he is not an automaton", just like that, so far makes no sense.

Ludwig Wittgenstein, *Philosophical Investigations*, Part II, Section IV

Think of the expression "I heard a plaintive melody". And now the question is: "Does he *hear* the plaint?"

And if I reply: "No, he doesn't hear it, he merely has a sense of it" – where does that get us? One cannot mention a sense-organ for this 'sense'... – No, what we have here is a modified concept of *sensation*.

Ludwig Wittgenstein, *Philosophical Investigations*, Part II, Section XI

Introduction

There has now appeared in the neuroimaging literature a robust number of studies which significantly support the thesis that, with appropriate training and effort, people are capable of systematically altering neural circuitry associated with a variety of mental and physical states, some of which are frankly pathological (Schwartz, Stoessel, Baxter, Martin, & Phelps 1996; Schwartz 1998; Musso, Weiller, Kiebel, Muller, Bulau, & Rijntjes 1999; Paquette, Lévesque, Mensour, Leroux, Beaudoin, Bourgouin, & Beauregard 2003). Many of these findings have been reviewed in this book. Further, and

quite importantly, an accelerating number of studies are being performed applying an experimental paradigm that utilizes directed mental effort in order to produce, via training in the cognitive reattribution and attentional recontextualization of conscious experience, systematic and predictable changes in central nervous system (CNS) function (Beauregard, Levesque, & Bourgouin 2001; Ochsner, Bunge, Gross, & Gabrieli 2002). A recent review of this, and the related neurological literature has coined the term “self-directed neuroplasticity” to serve as a general description of the principle that focused training and effort can systematically alter cerebral function in a predictable and potentially therapeutic manner (Schwartz & Begley 2002).

In addition, these findings have potentially profound implications for our understanding of the physics of the mind-brain interface. This is especially so for two reasons we will discuss in detail in this chapter. First, current approaches to understanding the functional activity of the nervous system are entirely derived from the principles of classical physics, which only recognize direct interactions between material factors to be causally efficacious. Intrinsically mentalistic terms such as “feeling,” “knowing” and “effort” cannot be described solely in terms of such material interactions, yet such mentalistic terminology is absolutely and critically necessary to the design and execution of the experiments in which the data demonstrating the principle of self-directed neuroplasticity is acquired and described. Thus mechanisms based solely on a classical physics perspective do not allow for the meaningful discussion of the neuropsychological aspects of this category of neuroplastic phenomena. Second, and crucially, current approaches to physics as currently practiced utilize the principles of quantum mechanics which, as we will demonstrate in detail, fully allow for the scientific integration of mentalistic and neurophysiological terminology. They are therefore entirely capable of explaining the mechanisms necessary to understand the rapidly emerging field of self-directed neuroplasticity and the neuropsychology of emotional self-regulation.

Practical and theoretical aspects of self-directed neuroplasticity

The clinical implications of recent work investigating self-directed neuroplasticity, and its possible application to the design and discovery of new cognitive-behavioral approaches to the treatment of neuropsychological and neuropsychiatric disease states, is obviously of great practical importance. Specifically, the expanding data base demonstrating that self-directed neuroplasticity can be directly applied to disease states with frank neuropathological changes, such

as stroke and dystonia, can have a potentially profound impact on issues concerning the culture of public health (Musso et al. 1999; Byl & McKenzie 2000).

Beyond the obvious clinical significance of scientifically demonstrating the fact that humans can, with appropriate effort and training, functionally reorganize their own brain circuitry, perhaps the most important aspect of this rapidly developing field of research is its implications for our understanding of the role of willfully directed attention in shaping brain activity. The cognitive frame in which neuroscience research, including research on cerebral aspects of behavior, is generally conducted contains within it the unstated assumption that brain mechanisms *per se*, once discovered, are fully sufficient to explain whatever phenomenon it is that's being investigated. In the fields of neuroimaging this has led to experimental paradigms that primarily focus on changes in brain tissue activation as primary dependent variables used to explain whatever behavioral changes are observed – including ones understood as involving primarily cognitive and emotional responses. As long as one is investigating phenomena that are primarily passive in nature this may well be fully justified. A person is shown a picture depicting an emotionally or perhaps a sexually arousing scene. The relevant limbic and/or diencephalic structures are activated. The investigator generally concludes that the observed brain activation has some intrinsic causal role in the emotional changes reported (or perhaps, the hormonal correlates of those changes). All well and good, as far as it goes. And all quite passive from the experimental subject's perspective – all that's really required on his or her part is to remain reasonably awake and alert, or, more precisely, at least somewhat responsive to sensory inputs. But when, as happens in a growing number of studies reported in this volume and elsewhere, the subject makes an active response aimed at systematically *altering* the nature of the emotional response – for example by actively performing a cognitive reattribution – understanding the data solely from the perspective of brain-based causal mechanism can be severely limiting and counterproductive. This is especially so when one is investigating how to develop improved methods for altering the emotional and cerebral responses to significantly stressful external or internally generated stimuli.

Simply stated, confusion concerning the relative importance of neurophysiological and mentalistic variables can lead to serious misunderstandings about causes and effects in the study of human behavior. While one may immediately grant that that these two types of variables are quite intimately related, and that complete clarity concerning their respective role in any given human action can be difficult (and sometimes even impossible), the fact remains that the serious investigator of human neuropsychology must make a concerted effort

to sort out the differences. This is especially so when the phenomena under investigation are value-laden, i.e., involve the possibility of making choices and decisions about how to respond to sensory phenomena.

In the case of studying clinical phenomena such as psychological treatments and their biological effects the distinction between mind and brain (or, if one prefers, mentalistic and neurophysiological variables) becomes absolutely critical. That's because if one simply assumes the most common generic belief of our era of neuroscience research, namely that all aspects of emotional response are passively determined by neurobiological mechanisms, then the possibility of developing genuinely effective self-directed psychological strategies that cause real neurobiological changes becomes, in principle, impossible. The clinician thus becomes locked, as it were, into at least the implicit view that the psychological treatment of ailments caused by neurobiological impairments is not a realistic goal.

There is already a wealth of data arguing against this view. For instance, work in the 1990's on patients with obsessive compulsive disorder demonstrated significant changes in caudate nucleus metabolism and the functional relationships of the orbitofrontal cortex-striatum-thalamus circuitry in patients who responded to a psychological treatment utilizing cognitive reframing and attentional refocusing as key aspects of the therapeutic intervention (for review see Schwartz & Begley 2002). More recently work by Beauregard and colleagues (Paquette et al. 2003) have demonstrated systematic changes in the dorsolateral prefrontal cortex and parahippocampal gyrus after cognitive-behavioral therapy for spider phobia, with brain changes significantly related to both objective measurements and subjective reports of fear and aversion. There are now numerous reports, many reviewed in this volume, on the effects of self-directed regulation of emotional response, via cognitive reframing and attentional recontextualization mechanisms, on cerebral function (e.g., Beauregard et al. 2001; Lévesque, Joanette, Paquette, Mensour, Beaudoin, Leroux, Bourgouin, & Beauregard 2003; Ochsner et al. 2002; Paquette et al. 2003; Schwartz et al. 1996).

One recent set of quite different yet interestingly related studies in normal subjects makes the point particularly clearly – those of Ochsner et al. (2002) on cognitive reattribution and the Beauregard group's work (2001) on sexual arousal. The Ochsner study demonstrates that using basic techniques of cognitive reappraisal, which they define as “the cognitive transformation of emotional experience” (e.g., “reappraisal was commonly accomplished by generating an interpretation of...[a stress-inducing] photo that would explain apparently negative events in a less negative way.”), can cause a marked and

systematic shift in cerebral responses to the viewing of stressful stimuli. The cerebral activations associated with viewing stressful visual stimuli altered from significant limbic arousal (specifically, in the medial orbitofrontal cortex and amygdala) when no cognitive reattribution strategy was used by normal subjects, to significant prefrontal activation and an associated and essentially complete calming of limbic activity when prior training in the use of a reappraisal technique was actively applied when viewing the stimulus.

Performing a cognitive reattribution involves a quite specific mental action, namely “the cognitive transformation of emotional experience,” or actively processing the *cognitive* (or, very roughly, the “*meaningfulness*”) components of the conscious experience differently. What makes this study a genuinely landmark piece of work is that it so clearly demonstrates that the mental action of cognitively recontextualizing a visually induced emotional experience systematically alters the way the brain processes the visual stimulus – the use of cognitive reappraisal strategies which, and this is a crucial point, require *willful choice* and *effort* to mobilize, markedly diminish the responses generated by the limbic structures of what has come to be called the emotional brain. The cerebral mechanism via which this effortful action accomplishes this task involves the activation of the prefrontal cortex, in both its lateral and medial aspects.

Actively damping down limbic responses and the emotion-laden experiences and feelings they generate seems to involve the activation of prefrontal mechanisms – in the Ochsner study, among several important cerebral activity correlations, there was a highly significant one correlating ventral prefrontal activation with amygdala de-activation. Cognitive reappraisal is a particularly language based means of emotional regulation, insofar as it involves subjects, as they describe it, “mentally talking themselves through their reappraisals,” and the lateral prefrontal activations that were seen were on the left side, as is generally seen in studies involving verbal information processing. But cognitive reappraisal is of course not the only way to self-regulate one’s emotions. Another method, used by Beauregard et al. (2001), involves asking subjects to make an effort to emotionally “distance themselves” from stimuli, thus becoming “a detached observer.” This strategy was used to investigate brain changes in subjects viewing sexually arousing films.

When adult males viewed erotic scenes in a normal “non-detached” manner, they became sexually aroused and this arousal correlated with activation in the hypothalamus, an area known to play a pivotal role in sexual responses. There were also activations of the amygdala and the anterior temporal pole. When subjects, in response to the investigator’s request, distanced themselves from the erotic stimuli, both the sexual arousal and the activation of

the hypothalamus and temporal lobe structures was prevented. Instead, there was activation of the prefrontal cortex, again as in the Ochsner et al. (2002) study in both its lateral and medial aspects, but this time with all activations being in the right hemisphere. This may indicate that differing emotional self-regulation strategies have different patterns of hemispheric activation. But it is especially noteworthy that in both types of self-regulation the lateral prefrontal and medial cingulate cortices were robustly activated.

Very recent work by the Beauregard group on the self-regulation of the affective response of sadness in normal subjects (Lévesque et al. 2003) also demonstrates the critical role of prefrontal cortex activation in the suppression of sadness responses in both adults and children. Indeed, the brain area activated in all the emotional self-regulation studies done so far is the prefrontal cortex, an area of the brain also activated in studies of cerebral correlates of willful mental activity, particularly those investigating self-initiated action and the act of attending to one's own actions (Spence & Frith 1999; Schwartz & Begley 2002). There is however one aspect of willful mental activity that seems particularly critical to emotional self-regulation and seems to be the critical factor in its effective application – the factor of dispassionate self-observation that, in a rapidly growing number of clinical psychology studies, has come to be called mindfulness or mindful awareness (Segal, Williams, & Teasdale 2002).

The mental act of clear-minded introspection and observation, variously known as mindfulness, mindful awareness, bare attention, the impartial spectator, etc. is a well-described psychological phenomenon with a long and distinguished history in the description of human mental states (Nyanaponika 2000). The most systematic and extensive exposition is in the canonical texts of classical Buddhism preserved in the Pali language, a dialect of Sanskrit. Because of the critical importance of this type of close attentiveness in the practice of Buddhist meditation some of its most refined descriptions in English are in texts concerned with meditative practice (although it is of critical importance to realize that the mindful mental state does not require any specific meditative practice to acquire, and is *certainly not* in any sense a “trance-like” state). One particularly well-established description, using the name bare attention, is as follows:

Bare Attention is the clear and single-minded awareness of what actually happens *to* us and *in* us at the successive moments of perception. It is called ‘bare’ because it attends just to the bare facts of a perception as presented either through the five physical senses or through the mind ... without reacting to them.

(Nyanaponika 1973:30)

Another classic description of this mental action, this one from the Eighteenth Century British empiricist tradition, is the following:

When I endeavor to examine my own conduct . . . I divide myself as it were into two persons; and that I, the examiner and judge, represent a different character from the other I, the person whose conduct is examined into and judged of. The first is the spectator . . . The second is the agent, the person whom I properly call myself, and of whose conduct, under the character of a spectator, I was endeavoring to form some opinion . . . [in this way] we suppose ourselves the spectators of our own behavior. (Smith 1976: 112–113)

Perhaps the essential aspect of mindful observation is that you are just watching, observing all facts, both inner and outer, very calmly, clearly and closely.

This mental action may reasonably be understood as the core aspect of all emotional self-regulation, for it is the key to initiating whatever other strategies are to be utilized. Thus cognitive reappraisal, for example, requires the mental act of recognizing that one is in the appropriate situation to begin using the self-regulating reappraisal techniques that one has been practicing to better cope with emotional stress. Although it is certainly a subject for further systematic investigation, it seems a reasonable working hypothesis that mindful awareness is the key mental act whereby one monitors whether the act of cognitive reappraisal is actually being effectively performed, i.e., mindfulness is the way in which one assesses whether one is, in fact, coping more adaptively with (or at least modifying one's reactions to) an emotional stressor. In a word, bare attention is the means by which one actualizes an emotional self-regulating strategy. Analogous reasoning would apply to "distancing," "suppression" etc. Of course, as work by Gross (2002), among others (Ochsner & Feldman Barrett 2001), has demonstrated, the use of these various strategies can differ in effectiveness, depending on the nature of the stressful situation being dealt with.

A working hypothesis for ongoing investigation in human neurophysiology, based on a significant body of preliminary data, is that the mental action of mindful awareness specifically modulates the activity of the prefrontal cortex. Because of the well established role of this cortical area in the planning and willful selection of self-initiated responses (Spence & Frith 1999; Schwartz & Begley 2002) the capacity of mindful awareness, and by implication all emotional self-regulating strategies, to specifically modulate activity in this critical brain region has tremendous implications for the fields of mental health and related areas. It is of considerable interest to note that while essentially all studies concerning the effects of self-regulation and/or self-observation on brain activity in normal subjects have documented an increase of activity in prefrontal areas

during the performance of tasks of this type, in clinical states what might better be called a “normalizing” effect is seen. Thus in conditions with excessive anxiety such as OCD (Schwartz 1998) and phobia (Paquette et al. 2003), or in the more ventral areas of the brain regularly found to be overactive in depression (Brody, Saxena, Stoessel, Gillies, Fairbanks, Alborzian, Phelps, Huang, Wu, Ho, Ho, Au, Maidment, & Baxter 2001), psychological interventions have been associated with decreases in activity. Intriguingly, two studies on brain changes associated with improvements in depressive symptoms after placebo response have seen increases in activity in prefrontal areas often found to be underactive in depression (Leuchter, Cook, Witte, Morgan, & Abrams 2002; Mayberg, Silva, Brannan, Tekell, Mahurin, McGinnis, & Jerabek 2002).

Philosophical foundations of neuroscience

The problem at issue here is the nature of connection between the features of the experiments described in psychological/mentalistic terms and the features described in spatio-temporally-based physical terms. This question is an aspect of the long-standing problem of the relationship between mind and matter, which has a history dating back to the time of the ancient Greeks. The issue was rekindled by the rise of Newtonian physics during the seventeenth century, and it generated a huge body of speculation and argumentation during the second half of the twentieth century.

It is neither appropriate nor feasible try to review or explain here the complexities of contemporary philosophical opinions on this question, except to say that the reigning view is “materialism,” and that: (1), there is no agreement among its proponents as to how to make rational good sense of this doctrine (Horgan 1994); and (2), the doctrine, and its supporters, seem, nevertheless, to have strongly influenced the thinking of many neuroscientists.

The central thesis of materialism is that:

The human body is a causally complete physico-chemical system: although the body is highly susceptible to external causal influences, all physical events in the body, and all bodily movements are fully explainable in physico-chemical terms. (Horgan 1994:472)

This thesis is, from a contemporary physics point of view, very obscure: What does “causally complete physico-chemical system” mean? It seems to be referring back to the seventeenth/nineteenth century classical-physics conception of nature, which is now known to be fundamentally false. If the doctrine does indeed

assume a classical-physics-type of conception of nature, then every bodily movement would indeed be in principle fully explainable in physico-chemical terms. But no reference to mental states or events occurs in classical physics. Hence one is faced with a question that the proponents of materialism have debated at length: how does one inject causally efficacious mind into this causally complete physical description of nature that make no mention of mind.

The ingenuity of philosophers has provided many possible answers, but every proposal seems flawed to other philosophers, and debate continues.

In addition to the problem of trying to attach causally efficacious mind onto a causally complete mindless theory, there is a still more basic problem: classical physical theory is known to be fundamentally false, and in principle inapplicable to systems, such as brains, that depend sensitively upon, for example, the motions of ions. And there is also the problem that the physico-classical aspect of contemporary physical theory, by itself, does not yield any predictions about empirically accessible data. To obtain even statistical predictions in quantum theory one needs to bring in "The Observer," who is described, fundamentally, in psychological/mentalistic terms. And this "Observer" plays a *participatory role* in the quantum formulation: he is not the purely passive observer of classical physics. He enters into the dynamics in a causally efficacious way.

In view of these profound, and apparently profoundly relevant, deviations of contemporary physics from what is essentially the seventeenth century materialist creation of Isaac Newton, it is strange that twenty-first century neuroscientists should adhere so unwaveringly to that seemingly inappropriate and ill-defined doctrine based on a now-known-to-be-fundamentally-false physical theory.

What orthodox quantum theory affirms, and materialism appears to deny, is that psychologically described realities enter *fundamentally* into the scientific description of phenomena, and have, *per se*, causal effects that are *not explained* solely in terms of the physical laws that are the generalization of the physical laws of classical physical theory. These causal effects are, however, explained by using other laws of quantum physics, which have no analog in classical physics.

The big problem with materialism as a basis for neuroscience is that it elevates to primary status, by fiat, the (actually nonexistent) physical entities of classical physics, and relegates to secondary status the experiential realities that are the primary variables of pragmatic empirical science. Classical physics does not explain how functional properties come to be connected to experiential realities, and hence fails to explain the causal efficacy of our thoughts. The power of our thought to influence bodily action seems, therefore, from the standpoint

of classical physics, to be some sort of illusion, or at least unexplained mystery. Quantum theory, on the other hand, displays the mechanism of conscious control, and hence dispels the mystery. The details are given in a later section.

A causal role for mindful awareness in brain regulation

It is important for investigators in the emerging field of *Consciousness, Emotional Self-regulation and the Brain* to realize that classical models of neurobiological action fail to account for all of the mechanisms that are operating when human beings utilize self-directed strategies for the purpose of modulating emotional responses and their cerebral correlates. Specifically, the assumption that all aspects of mental activity and emotional life are ultimately explicable solely in terms of micro-local deterministic brain activity, with no superposed effects of mental effort, is neither consistent with the basic data of psychological observation nor entailed by modern physics. The simple classical model must in principle be replaced by the more accurate and functionally useful concept that the role played by the mind when observing and modulating one's own emotional states is an intrinsically active process, one in which mental action is *affecting* brain activity and not merely being affected by it. One key reason for the necessity of this change in perspective is due to the fact that a highlighting of the active role of the mind in emotional self-regulation is an intrinsic aspect of a proper understanding of what is actually happening when a person directs his or her inner resources to the challenging task of modifying emotional responses. It takes *effort* for people to do this. That is because it requires a redirection of the brain's resources away from lower level limbic responses and toward higher level prefrontal functions – and this does not happen automatically. Rather, it requires willful training and directed effort. It is deeply misleading to view these kinds of brain changes as being solely an intra-cerebral “the brain is changing itself” type of phenomenon, since the critical effect of the quantum action of mind on brain is lost when conceptualizing things in this way. The application of bare attention to one's own mental processes is an activity that is not adequately described solely in terms of brain mechanisms.

Further, as we will see in detail in the following sections of this chapter, modern concepts of theoretical physics are ideally suited to a proper understanding of the action of mindful self-observation on brain function. Classical models of physics, which view all action in the physical world as being the result of the movements of material particles, are now seriously out of date, and

no longer should be seen as providing a proper paradigm for investigating the mind-brain interface.

There are, of course, other reasons as well, involving ethical issues such as personal responsibility, a concept which is seriously undermined by rigidly applied theories of material causation – for if all causes are ultimately material causes there is no clear role for personal responsibility in the actions people take. These kinds of questions have been discussed in detail elsewhere (Stapp 2001; Schwartz & Begley 2002). For the moment let's approach this question of the need for allowing a causal role for mind in the most pragmatic of ways, and ask the question: Does it make sense to try to understand the process of emotional self-regulation solely in terms of brain mechanisms?

For at least one quite straightforward reason it seems clear that it does not. That reason is that it is intrinsically impossible to explain and describe to real people the techniques they must learn to perform emotional self-regulation strategies without using language that contains instructions about what to do with your mind, i.e., without using terms referring to mental experience, words like: feeling, effort, observation, awareness, mindfulness and so forth. When people are practicing self-regulating activities they are attending to their mental and emotional *experiences*, not merely their limbic or hypothalamic brain mechanisms. And while no scientifically oriented person denies that those brain mechanisms play a critical role in generating those experiences, precisely what the person is training himself to do is to willfully *change* how those brain mechanisms operate – and to do that absolutely requires attending to mental experience *per se*. It is in fact the basic thesis of emotional self-regulation research that the *way* in which a person directs his attention, e.g., mindfully or unmindfully, will affect both the emotional state of the person and the state of their brain.

The very acquisition of the skills required in order to change the brain in that way requires understanding what it means to observe mindfully etc., and learning those skills cannot be accomplished via the sole use of neurobiological terminology – the language of mental experience must of necessity be utilized. We can, and as therapists do, teach people all the time how to use self-observation strategies to regulate their emotional responses. A growing body of research informs us that when people learn to do that it modulates the activity of the prefrontal cortex, among other areas. But, to paraphrase the Wittgenstein quote at the outset of this chapter, saying to someone “Now modulate your prefrontal cortex,” just like that, so far makes no sense. We must actively bring into play, through training and practice, what Wittgenstein (1968) calls “a modified concept of *sensation*” to accomplish that goal of self-directed brain modula-

tion, and that modified concept of sensation *requires* the use of mind-based experiential language to learn and accomplish.

People can certainly learn how to be mindful, and when they do it changes brain function in very beneficial ways. But to effect and accomplish those brain changes requires the language of mental experience and activity in basic and irreducible ways – it can never be accomplished solely by the use of brain based language. This straightforward fact tells us that the language of neurobiology will never be sufficient for the effective self-regulation of brain activity. The language of the active mind is an absolute logical requirement. As we will now see, in the field of physics as well as in the field of psychology, the language of the mind is needed for a proper understanding of the actions of the brain.

Physics in neuroscience

Classical physics

Classical physics is a theory of nature that originated with the work of Isaac Newton in the seventeenth century and was advanced by the contributions of James Clerk Maxwell and Albert Einstein. Newton based his theory on the work of Johannes Kepler, who found that the planets appeared to move in accordance with a simple mathematical law, and in ways wholly determined by their spatial relationships to other objects. Those motions were apparently *independent of our human observations of them*.

Newton assumed that all physical objects were made of tiny miniaturized versions of the planets, which, like the planets, moved in accordance with simple mathematical laws, independently of whether we observed them or not. He found that he could explain the motions of the planets, and also the motions of large terrestrial objects and systems, such as cannon balls, falling apples, and the tides, by assuming that every tiny planet-like particle in the solar system attracted every other one with a force inversely proportional the square of the distance between them.

This force was an *instantaneous action at a distance*: it acted instantaneously, no matter how far apart the particles were located. This feature troubled Newton. He wrote to a friend “That one body should act upon another through the vacuum, without the mediation of anything else, by and through which their action and force may be conveyed from one to another, is to me so great an absurdity that I believe no man, who has in philosophical matters a competent faculty of thinking, can ever fall into it” (Newton 1687: 634).

Although Newton's philosophical persuasion on this point is clear, he nevertheless formulated his universal law of gravity without specifying how it was mediated.

Albert Einstein, building on the ideas of Maxwell, discovered a suitable mediating agent: a distortion of the structure of space-time itself. Einstein's contributions made classical physics into what is called a *local theory*: there is no action at a distance. All influences are transmitted essentially by contact interactions between tiny neighboring mathematically described "entities," and no influence propagates faster than the speed of light.

Classical physics is, moreover, *deterministic*: the interactions are such that the state of the physical world at any time is completely determined by the state at any earlier time. Consequently, according to classical theory, the complete history of the physical world *for all time* is mechanically fixed by contact interactions between tiny component parts, together with the initial condition of the primordial universe.

This result means that, according to classical physics, *you are a mechanical automaton*: your every physical action was pre-determined before you were born solely by mechanical interactions between tiny mindless entities. Your mental aspects are *causally redundant*: everything you do is completely determined by mechanical conditions alone, without reference to your thoughts, ideas, feelings, or intentions. Your intuitive feeling that your mental intentions make a difference in what you do is, according to the principles of classical physics, a false and misleading illusion.

There are two possible ways within classical physics to understand this total incapacity of your mental side – your stream of consciousness – to make any difference at all in what you do. The first way is to consider your thoughts, ideas, and feelings to be epiphenomenal by-products of the activity of your brain. Your mental side is then a causally impotent sideshow that *is produced*, or *caused*, by your brain, but that *produces* no reciprocal action back upon your brain. The second way is to contend that your mental aspects are the *very same things* as certain of motions of various tiny parts of your brain.

Problems with classical physics

William James (1890:138) argued against the first possibility, epiphenomenal consciousness, by arguing that "*The particulars of the distribution of consciousness, so far as we know them, points to its being efficacious.*" He noted that consciousness seems to be "an organ, superadded to the other organs which maintain the animal in its struggle for existence; and the presumption of course is that it helps him in some way in this struggle, just as they do. But it cannot

help him without being in some way efficacious and influencing the course of his bodily history.” James said that the study described in his book “will show us that consciousness is at all times primarily a *selecting agency*.” It is present when choices must be made between different possible courses of action. He further mentioned that “It is to my mind quite inconceivable that consciousness should have *nothing to do* with a business to which it so faithfully attends” (1890:136).

If consciousness has no effect upon the physical world then what keeps a person’s mental world aligned with his physical situation: what keeps his pleasures in general alignment with actions that benefit him, and pains in general correspondence with things that damage him, if pleasure and pain have no effect at all upon his actions?

These liabilities of the notion of epiphenomenal consciousness lead most thinkers to turn to the alternative possibility that a person’s stream of consciousness is *the very same thing* as some activity in his brain: consciousness is an “emergent property” of brains.

A huge philosophical literature has developed arguing for and against this idea. The primary argument against this “emergent-identity theory” position, *within a classical physics framework*, is that within classical physics the full description of nature is in terms of numbers assigned to tiny space-time regions, and there appears to be no way to understand or explain how to get from such a restricted conceptual structure, which involves such a small part of the world of experience, to the whole. How and why should that extremely limited conceptual structure, which arose basically from idealizing, by miniaturization, certain features of observed planetary motions, suffice to explain the totality of experience, with its pains, sorrows, hopes, colors, smells, and moral judgments? Why, given the known failure of classical physics at the fundamental level, should that richly endowed whole be explainable in terms of such a narrowly restricted part?

The core ideas of the arguments in favor of an identity-emergent theory of consciousness are illustrated by Roger Sperry’s example of a “wheel” (Sperry 1992:267). A wheel obviously does something: it is causally efficacious; it carries the cart. It is also an *emergent property*: there is no mention of “wheelness” in the formulation of the laws of physics, and “wheelness” did not exist in the early universe; “wheelness” *emerges* only under certain special conditions. And the macroscopic wheel exercises “top-down” control of its tiny parts. All these properties are perfectly in line with classical physics, and with the idea that “a wheel is, precisely, a structure constructed out of its tiny atomic parts.” So why

not suppose “consciousness” to be, like “wheelness”, an emergent property of its classically conceived tiny physical parts?

The reason that consciousness is not analogous to wheelness, within the context of classical physics, is that the properties that characterize wheelness are properties that are *entailed*, within the conceptual framework of classical physics, by properties specified in classical physics, whereas the properties that characterize consciousness, namely the way it feels, are *not entailed*, within the conceptual structure provided by classical physics, by the properties specified by classical physics.

This is the huge difference-in-principle that distinguishes consciousness from things that, according to classical physics, are constructible out of the particles that are postulated to exist by classical physics.

Given the state of motion of each of the tiny physical parts of a wheel, as it is conceived of in classical physics, the properties that characterize the wheel – e.g., its roundness, radius, center point, rate of rotation, etc., – are specified within the conceptual framework provided by the principles of classical physics, which specify only geometric-type properties such as changing locations and shapes of conglomerations of particles, and numbers assigned to points in space. But given the state of motion of each tiny part of the brain, as it is conceived of in classical physics, the properties that characterize the stream of consciousness – the painfulness of the pain, the feeling of the anguish, or of the sorrow, or of the joy – are not specified, within the conceptual framework provided by the principles of classical physics. Thus it is possible, within that classical physics framework, to strip away those feelings without disturbing the physical descriptions of the motions of the tiny parts. One can, within the conceptual framework of classical physics, take away the consciousness without affecting the locations and motions of the tiny physical parts of the brain. But one cannot, within the conceptual framework provided by classical physics, take away the wheelness of the wheel without affecting the locations and motions of the tiny physical parts of a wheel.

Because one can, within the conceptual framework provided by classical physics, strip away the consciousness without affecting the physical behavior, one cannot rationally claim that the consciousness is the *cause* of the physical behavior, or is *causally efficacious* in the physical world. Thus the “identity theory” or “emergent property” strategy fails in its attempt to make consciousness efficacious, within the conceptual framework provided by classical physics. Moreover, the whole endeavor to base brain theory on classical physics is undermined by the fact that the classical theory fails to work for phenomena that depend critically upon the properties of the atomic constituents of the

behaving system, and brains are such systems: brain processes depend critically upon synaptic processes, which depend critically upon ionic processes that are highly dependent upon their quantum nature. This essential involvement of quantum effects will be discussed in later sections.

The quantum approach

Classical physics is *an approximation* to a more accurate theory – called quantum mechanics – and quantum mechanics makes mind efficacious. Quantum mechanics *explains* the causal effects of mental intentions upon physical systems: it explains how your mental effort can produce the brain events that cause your bodily actions. Thus quantum theory converts science's picture of you from that of a mechanical automaton to that of a mindful human person. Quantum theory also shows, explicitly, how the approximation that reduces quantum theory to classical physics completely eliminates all effects of your conscious thoughts upon your brain and body. Hence, from a physics point of view, trying to understand the mind-brain connection by going to the classical approximation is absurd: it amounts to trying to understand something in an approximation that eliminates the effect you are trying to study.

Quantum mechanics arose during the twentieth century. Scientists discovered, empirically, that the principles of classical physics were not correct. Moreover, they were wrong in ways that no minor tinkering could ever fix. The *basic principles* of classical physics were thus replaced by *new basic principles* that account uniformly both for all the successes of the older classical theory and also for all the newer data that is incompatible with the classical principles.

The most profound alteration of the fundamental principles was to bring the consciousness of human beings into the basic structure of the physical theory. In fact, the whole *conception of what science is* was turned inside out. The core idea of classical physics was to describe the “world out there,” with no reference to “our thoughts in here.” But the core idea of quantum mechanics is to describe *our activities as knowledge-seeking human agents*, and *the knowledge that we thereby acquire*. Thus quantum theory involves, basically, what is “in here,” not just what is “out there.”

The basic philosophical shift in quantum theory is the *explicit* recognition that science is about *what we can know*. It is fine to have a beautiful and elegant mathematical theory about a *really existing physical world out there* that meets a lot of intellectually satisfying criteria. But the essential demand of science is that the theoretical constructs be tied to the experiences of the human scientists who devise ways of testing the theory, and of the human engineers and technicians who both participate in these tests, and eventually put the theory

to work. So the structure of a proper physical theory must involve not only the part describing the behavior of the not-directly-experienced theoretically postulated entities, expressed in some appropriate symbolic language, but also a part describing the human experiences that are pertinent to these tests and applications, expressed in the language that we actually use to describe such experiences to ourselves and each other. Finally we need some “bridge laws” that specify the connection between the concepts described in these two different languages.

Classical physics met these requirements in a rather trivial kind of way, with the relevant experiences of the human participants being taken to be direct apprehensions of various gross behaviors of large-scale properties of big objects composed of huge numbers of the tiny atomic-scale parts. And these apprehensions were taken to be *passive*: they had no effect on the behaviors of the systems being studied. But the physicists who were examining the behaviors of systems that depend sensitively upon the behaviors of their tiny atomic-scale components found themselves forced to go to a less trivial theoretical arrangement, in which the human agents were no longer passive observers, but were *active participants* in ways that contradicted, and were impossible to comprehend within, the general framework of classical physics, *even when the only features of the physically described world that the human beings observed were large-scale properties of measuring devices*. The sensitivity of the behavior of the devices to the behavior of some tiny atomic-scale particles propagates in such a way that the acts of observation by the human observers of *large scale properties of the devices* could no longer be regarded as passive. Thus the core structure of the basic general physical theory became transformed in a profound way: the connection between physical behavior and human knowledge was changed from a one-way bridge to a mathematically specified two-way bridge. This revision must be expected to have important ramifications in neuroscience, because the issue of the connection between mind (the psychologically described aspects of a human being) and brain/body (the physically described aspects of that person) has recently become a matter of central concern in neuroscience.

This original formulation of quantum theory was created mainly at an Institute in Copenhagen directed by Niels Bohr, and is called “The Copenhagen Interpretation.” Due to the profound strangeness of the conception of nature entailed by the new mathematics, the Copenhagen strategy was to refrain from making any ontological claims, but to take, instead, a purely pragmatic stance. Thus the theory was formulated *basically* as a set of practical rules for how scientists should go about their tasks of acquiring knowledge, and then using

this knowledge in practical ways. Speculations about “what the world out there is really like” were regarded as “metaphysics,” not real science.

The most profound change in the principles is encapsulated in Niels Bohr dictum that “in the great drama of existence we ourselves are both actors and spectators” (Bohr 1958:81, 1963:15). The emphasis here is on “actors”: in classical physics we were mere spectators.

Copenhagen quantum theory is about the relationships between human agents (called *participants* by John Wheeler) and the systems that they act upon. In order to achieve this conceptualization the Copenhagen formulation separates the physical universe into two parts, which are described in two different languages. One part is the observing human agent and his measuring devices. That part is described in mental terms – in terms of our instructions to colleagues about how to set up the devices, and our reports of what we then “see,” or otherwise consciously experience. The other part of nature is *the system that the agent is acting upon*. That part is described in physical terms – in terms of mathematical properties assigned to tiny space-time regions.

That approach works very well in practice. However, it seems apparent that the body and brain of the human agent, and his devices, are parts of the physical universe, and hence that a complete theory ought to be able to describe also our bodies and brains in physical terms. On the other hand, the structure of the theory depends critically also upon aspects of reality described in mentalistic language as intentional actions and experiential feedbacks.

The great mathematician and logician John von Neumann reformulated the theory in a rigorous way that places the bodies and brains of the agents, along with their measuring devices, in the physical world, *while retaining those mentalistically described properties of the agents that are essential to the structure of the theory*. It is this von Neumann formulation that provides a natural science-based account of how your mental intentions influence the activities of your brain.

Von Neumann identifies two very different processes that enter into the quantum theoretical description of the evolution of a physical system. He calls them Process I and Process II (Von Neumann 1955:418). Process II is the analog in quantum theory of the process in classical physics that takes the state of a system at one time to its state at a later time. This Process II, like its classical analog, is *local* and *deterministic*. However, Process II by itself is not the whole story: it generates physical worlds that do not agree with human experiences. For example, if Process II were the *only* process in nature then the quantum state of the moon would represent a structure smeared out over a large part

of the sky, and each human body-brain would likewise be represented by a structure smeared out continuously over a huge region.

To tie the quantum mathematics to human experience in a rationally coherent and mathematically specified way quantum theory invokes *another process*, which Von Neumann calls Process I.

Any physical theory must, in order to be complete, specify how the elements of the theory are connected to human experience. In classical physics this connection is part of a *metaphysical* superstructure: it is not part of the core dynamical description. But in quantum theory this connection of the mathematically described physical state to human experiences is part of the essential dynamical structure. And this connecting process is not passive: it does not represent a mere *witnessing* of a physical feature of nature by a passive mind. Rather, the process is active: it injects into the physical state of the system being acted upon properties that depend upon the intention of the agent.

Quantum theory is built upon the practical concept of intentional actions by agents. Each such action is expected or intended to produce an experiential response or feedback. For example, a scientist might act to place a Geiger counter near a radioactive source, and expect to see the counter either "fire" during a certain time interval or not "fire" during that interval. The experienced response, "Yes" or "No", to the question "Does the counter fire during the specified interval?" specifies one bit of information. Quantum theory is thus an information-based theory built upon the knowledge-acquiring actions of agents, and the knowledge that these agents thereby acquire.

Probing actions of this kind are performed not only by scientists. Every healthy and alert infant is engaged in making willful efforts that produce experiential feedbacks, and he/she soon begins to form expectations about what sorts of feedbacks are likely to follow from some particular kind of effort. Thus both empirical science and normal human life are based on paired realities of this action-response kind, and our physical and psychological theories are both basically attempting to understand these linked realities within a rational conceptual framework.

The basic building blocks of quantum theory are, then, a set of intentional actions by agents, and for each such action an associated collection of possible "Yes" feedbacks, which are the possible responses that the agent can judge to be in conformity to the criteria associated with that intentional act. For example, the agent is assumed to be able to make the judgment "Yes" the Geiger counter clicked or "No" the Geiger counter did not click. Science would be difficult to pursue if scientists could make no such judgments about what they were experiencing.

All known physical theories involve idealizations of one kind or another. In quantum theory the main idealization is not that every object is made up of miniature planet-like objects. It is rather that there are agents that perform intentional acts each of which can result in a feedback that may conform to a certain criterion associated with that act. One bit of information is introduced into the world in which that agent lives, according to whether the feedback conforms or does not conform to that criterion. Thus knowing whether the counter clicked or not places the agent on one or the other of two alternative possible separate branches of the course of world history.

These remarks reveal the enormous difference between classical physics and quantum physics. In classical physics the elemental ingredients are tiny invisible bits of matter that are idealized miniaturized versions of the planets that we see in the heavens, and that move in ways unaffected by our scrutiny, whereas in quantum physics the elemental ingredients are intentional actions by agents, the feedbacks arising from these actions, and the effects of our actions on the physical states that embody or carry this information.

Consideration of the character of these differences makes it plausible that quantum theory may be able to provide the foundation of a scientific theory of the human person that is better able than classical physics to integrate the physical and psychological aspects of his nature. For quantum theory describes the effects of a person's intentional actions upon the physical world, whereas classical physics systematically leaves these effects out.

An intentional action by a human agent is partly an intention, described in psychological terms, and partly a physical action, described in physical terms. The feedback also is partly psychological and partly physical. In quantum theory these diverse aspects are all represented by logically connected elements in the mathematical structure that emerged from the seminal discovery of Heisenberg. That discovery was that in order to get the quantum generalization of a classical theory one must formulate the theory in terms of *actions*. A key difference between numbers and actions is that if A and B are two actions then AB represents the action obtained by performing the action A upon the action B . If A and B are actions then generally AB is different from BA : the order in which actions are performed matters.

The intentional actions of agents are represented mathematically in Heisenberg's space of actions. Here is how it works.

Each intentional action depends, of course, on the intention of the agent, and upon the state of the system upon which this action acts. Each of these two aspects of nature is represented within Heisenberg's space of actions by an action. The idea that a "state" should be represented by an "action" may sound

odd, but Heisenberg's key idea was to replace what classical physics took to be a "being" by a "doing." I shall denote the action that represents the state being acted upon by the symbol S .

An intentional act is an action that is intended to produce a feedback of a certain conceived or imagined kind. Of course, no intentional act is sure-fire: one's intentions may not be fulfilled. Hence the intentional action puts in play a process that will lead either to a confirmatory feedback "Yes," the intention is realized, or to the result "No", the "Yes" response did not occur.

The effect of this intentional mental act is represented mathematically by an equation that is one of the key equations of quantum theory. This equation represents, within the quantum mathematics, the effect of the Process I mental action upon the quantum state S of the system being acted upon. The equation is:

$$S \rightarrow S' = PSP + (I-P)S(I-P).$$

This formula exhibits the important fact that this Process I action changes the state S of the system being acted upon into a new state S' , which is a *sum* of two parts.

The first part, PSP , represents the possibility in which the experiential feedback called "Yes" appears, and the second part, $(I-P)S(I-P)$, represents the alternative possibility "No", this feedback does not appear. Thus the intention of the action and the associated experiential feedback are tied into the mathematics that describes the dynamics of the physical system being acted upon.

The symbol P is important. It represents the fact that the Process I depends on *the intention of the agent*. The action represented by the symbol P , acting both on the right and on the left of S , is the action of eliminating from the state S all parts of S except the "Yes" part. That particular retained part is determined by the intentional choice of the agent. The symbol I is the unit operator, which is essentially multiplication by 1, and the action of $(I-P)$, acting both on the right and on the left of S , is, analogously, to eliminate from S all parts of S except the "No" parts.

Notice that Process I produces the *sum* of the two alternative possible feedbacks, not just one or the other. Since the feedback must either be "Yes" or "No = Not-Yes," one might think that Process I, which *keeps* both the "Yes" and the "No" parts, would do nothing. But that is not correct! This is a key point. It can be made quite clear by noticing that S can be written as a sum of four parts, only two of which survive the Process I action:

$$S = PSP + (I-P)S(I-P) + PS(I-P) + (I-P)SP.$$

This formula is a strict identity. The dedicated reader can quickly verify it by collecting the contributions of the four occurring terms PSP, PS, SP, and S, and verifying that all terms but S cancel out. This identity shows that the state S is a sum of four parts, *two of which are eliminated by Process I*.

But this means that Process I has a *nontrivial effect* upon the state being acted upon: it eliminates the two terms that correspond neither to the appearance of a “Yes” feedback nor to the failure of the “Yes” feedback to appear.

That is the *first key point*: quantum theory has a specific dynamical process, Process I, which specifies the effect upon a physical system of an *intention-controlled act* of a human agent.

Free choices

The second key point is this: the agent’s choices are “free choices,” *in the specific sense specified below*.

Orthodox quantum theory is formulated in a realistic and practical way. It is structured around the activities of human agents, who are considered able to freely elect to probe nature in any one of many possible ways. Bohr emphasized the freedom of the experimenters in passages such as:

The freedom of experimentation, presupposed in classical physics, is of course retained and corresponds to the free choice of experimental arrangement for which the mathematical structure of the quantum mechanical formalism offers the appropriate latitude. (Bohr 1958:73)

This freedom of action stems from the fact that in the original Copenhagen formulation of quantum theory the human experimenter is considered to stand outside the system to which the quantum laws are applied. Those quantum laws are the only precise laws of nature recognized by that theory. Thus, according to the Copenhagen philosophy, *there are no presently known laws that govern the choices* made by the agent/experimenter/observer about how the observed system is to be probed. This choice is, *in this very specific sense*, a “free choice.”

Probabilities

The predictions of quantum theory are generally statistical: only the *probabilities* that the agent will experience each of the alternative possible feedbacks are specified. Which of these alternative possible feedbacks will actually occur in response to a Process I action is not determined by quantum theory.

The formula for the probability that the agent will experience the feedback ‘Yes’ is $\text{Tr PSP} / \text{Tr S}$, where the symbol Tr represents the trace operation. This trace operation means that the actions act in a cyclic fashion, so that the right-

most action acts back around upon the leftmost action. Thus, for example, $\text{Tr } ABC = \text{Tr } CAB = \text{Tr } BCA$. The product ABC represents the result of letting A act upon B , and then letting that product AB act upon C . But what does C act upon? Taking the trace of ABC means specifying that C acts back around on A .

An important property of a trace is that the trace of any of the sequences of actions that we consider must always give a positive number or zero. Thus this trace operation is what ties the actions, as represented in the mathematics, to measurable numbers.

Von Neumann generates his form of quantum theory by recognizing that Process I describes an influence of a mentalistically described aspect of reality upon a physically described aspect, and by expanding the physically described part to include the brain that is connected to the mentalistically described stream of consciousness. Thus Process I represents, in the end, a dynamical influence of the mind of an agent upon his brain

But if the agent is *free* to choose which action to take, and if the *intention* of that action, represented by P , affects the state being acted upon, then the agent's free mental choice of intention influences the state S being acted upon, which in Von Neumann quantum theory is his/her brain.

This is the important conclusion: Orthodox (von Neumann) quantum theory has a Process I action that, on the one hand, is need to tie the theory to human experience, and that, on the other hand, produces a dynamical effect upon the state of the brain.

It is worthwhile to reflect for a moment on the ontological aspects of Von Neumann quantum theory. Von Neumann himself, being a clear thinking mathematician, said very little about ontology. But he called the mentalistically described aspect of the agent "his abstract 'ego'" (Von Neumann 1955:421). This phrasing tends to conjure up the idea of a disembodied entity, standing somehow apart from the body/brain. But another possibility is that consciousness is an *emergent property* of the body-brain. Notice that some of the problems that occur in trying to defend this idea of emergence within the framework of classical physical theory disappear when one accepts the validity of quantum theory. For one thing, one no longer has to defend against the charge that the emergent property, consciousness, has no "genuine" causal efficacy because anything it does is done already by the physically described process, independently of whether the psychologically described aspect emerges or not. If quantum theory is accepted then the causal efficacy of our thoughts is no illusion: it's the real thing!

Another difficulty with "emergence" in a classical physics context is in understanding how the motion of a set of miniature planet-like objects, careening

through space, can *be a painful experience*. But the essential nature of the “stuff” represented by a quantum event is experiential, and the mathematically described state that stores the fact that this experience has occurred is not material, it is informational. Hence there is no longer any clash with intuition: the concept of the physical stuff has changed in a way that eliminates the problem.

In this connection, Heisenberg remarked:

The conception of the objective reality of the elementary particles has thus evaporated not into the cloud of some obscure new reality concept, but into the transparent clarity of a mathematics that represents no longer the behavior of the particle but rather our knowledge of this behavior. (Heisenberg 1958)

By closing in this way the conceptual gap between mental and physical properties the acceptance of quantum theory opens the way to a less problematic version of the emergent-identity theory of mind.

This pragmatic re-conceptualization of the nature of science, and of the nature of the physical universe and its connection to human thoughts, should, it would seem, be equally important in neuroscience. This is because the basic problem in neuroscience is essentially the very same problem as the one of atomic physics, namely the problem of linking, in a practically useful way, the spacetime-imbedded mathematical description of a physical system being acted upon to the psychologically described aspects of the human agents. The problem in both cases, and in science in general, is to link, in practically useful ways, the psychological language that we human beings use to communicate our normal experiences from one to another to the mathematical language of physics and physiology. Matter-based classical physics does not provide a practically useful linkage between these two languages, but agent-based, action-based quantum physics does.

According to quantum theory, consciousness is, in effect, a *causally efficacious reality* that is connected to physical brain processes in a non-local, non-reducible, non-redundant, non-illusory, and non-trivial way.

Process I is non-local because each operator P must (due to its boundedness) act not at a point, but rather over some extended region (such as a macroscopic part of a brain). It is non-reducible because it is a real dynamical process that is neither part of, nor entailed by, the local-deterministic mechanical Process II. It is non-redundant, non-illusory, and non-trivial for the same reason. By dealing directly with a *dynamically formulated* linkage – Process I – between the psychological language that describes the actions and knowledge of the agents and the mathematical representation of the state of the system being acted upon, quantum theory provides a pragmatic foundation for a solu-

tion to the mind-brain problem of neuroscience that is both analogous to, and an extension of, the pragmatic solution devised by physicists for the problem of creating a scientific theory of atomic phenomena.

The quantum state of a human brain is, of course, a very complex thing. But its main features can be understood by considering first a classical conception of the brain, and then folding in some key features that arise already in the case of the quantum state of a single particle, or object, or degree of freedom.

States of a simple harmonic oscillator

One of the most important examples of a quantum state is the one corresponding to a pendulum, or more precisely, to what is called a “simple harmonic oscillator.” Such a system is one in which there is a restoring force that tends to push the center of the object to a single “base point” of lowest energy, and in which the strength of this restoring force is directly proportional to the distance of the center point of the object from this base point.

According to classical physics any such system has a state of lowest energy. In this state the center point of the object lies motionless at the base point. In quantum theory this system again has a state of lowest energy, but the center point is not localized at the base point: it is represented by a *cloudlike* spatial structure that is spread out over a region that extends to infinity. However, the amplitude of this cloudlike form has the shape of a bell: it is largest at the base point, and falls off in a prescribed manner as the distance of the center point from the base point increases.

If one were to squeeze this state of lowest energy into a more narrow space, and then let it loose, the cloudlike form would first explode outward, but then settle into an oscillating motion. Thus the cloudlike spatial structure behaves rather like a swarm of bees, such that the more they are squeezed in space the faster they move, and the faster the squeezed cloud will explode outward when the squeezing constraint is released. These visualizable properties extend in a natural way to many-particle cases.

The double-slit experiment

An important difference between the behavior of the quantum cloudlike form and the somewhat analogous *classical probability distribution* is exhibited by the famous *double-slit experiment*. If one shoots an electron, an ion, or any other quantum counterpart of a tiny classical object, at a narrow slit then if the object passes through the slit the associated cloudlike form will fan out over a wide angle. But if one opens two closely neighboring narrow slits, then what passes through the slits is described by a probability distribution that is

not just the sum of the two separate fanlike structures that would be present if each slit were opened separately. Instead, at some points the probability value will be *twice the sum* of the values associated with the two individual slits, and in other places the probability value drops nearly to zero, even though both individual fanlike structures give a large probability value at that place. These *interference* features of the quantum cloudlike structure make that structure logically different from a classical-physics probability distribution, for in the classical case the probabilities arising from the two slits would simply add, due to the fact that, according to classical principles, the particle must pass through one slit or the other, and the fact that some other slit is also open should not matter very much.

Quantum theory deals consistently with this interference effect, as well as all the other non-classical properties of these cloudlike structures.

Nerve terminals, ion channels, and the need to use quantum theory

Some neuroscientists who study the relationship of consciousness to brain process believe that classical physics will be adequate for that task. That belief would have been reasonable during the nineteenth century, but now, in the twenty-first, it is rationally untenable: quantum theory must in principle be used because the behavior of the brain depends sensitively upon ionic and atomic processes, and these processes involve quantum effects.

To study quantum effects in brains within an orthodox (i.e., Copenhagen or Von Neumann) quantum theory one must use the Von Neumann formulation. The reason is that *Copenhagen* quantum theory is formulated in a way that leaves out the quantum dynamics of the human observer's body and brain. But Von Neumann quantum theory takes the physical system *S* upon which the crucial Process I acts to be the brain of the agent. Thus Process I then describes an interaction between a person's stream of consciousness, described in mentalistic terms, and the activity in his brain, described in physical terms. That interaction drops completely out when one passes to the classical approximation. Hence ignoring quantum effects in the study of the mind-brain connection means, according to the basic principles of physics, ignoring the dynamical connection one is trying to study. One must *in principle* use quantum theory. But there is then the quantitative issue of how important the quantum effects are.

To explore that question we now consider the quantum dynamics of nerve terminals.

Nerve terminals

Nerve terminals are essential connecting links between nerve cells. The way they work is reasonably well understood. When an action potential traveling along a nerve fiber reaches a nerve terminal a host of ion channels open. Calcium ions enter through these channels into the interior of the terminal. These ions migrate from the channel exits to release site on vesicles containing neurotransmitter molecules. The triggering effect of the calcium ions causes these contents to be dumped into the synaptic cleft that separates this terminal from a neighboring neuron, and these neurotransmitter molecules influence the tendencies of that neighboring neuron to “fire.”

The channels through which the calcium ions enter the nerve terminal are called “ion channels.” At their narrowest points they are less than a nanometer in diameter (Cataldi, Perez-Reyes, & Tsien 2002). This extreme smallness of the opening in the ion channels has profound quantum mechanical importance. The consequence is essentially the same as the consequence of the squeezing of the state of the simple harmonic oscillator, or of the narrowness of the slits in the double-slit experiments. The narrowness of the channel restricts the lateral spatial dimension. Consequently, the lateral velocity is forced by the *quantum uncertainty principle* to become large. This causes the cloud associated with the calcium ion to *fan out* over an increasing area as it moves away from the tiny channel to the target region where the ion will be absorbed as a whole, or not absorbed, on some small triggering site.

This spreading of the ion wave packet means that the ion may or may not be absorbed on the small triggering site. Accordingly, the vesicle may or may not release its contents. Consequently, the quantum state of the vesicle has a part in which the neurotransmitter is released and a part in which the neurotransmitter is not released. This quantum splitting occurs at every one of the trillions of nerve terminals.

What is the effect of this *necessary* incursion of the cloud-like quantum character of the ions into the evolving state of the brain?

A principal function of the brain is to receive cues from the environment, to form an appropriate plan of action, and to direct and monitor the activities of the brain and body specified by the selected plan of action. The exact details of the plan will, for a classical model, obviously depend upon the exact values of many noisy and uncontrolled variables. In cases close to a bifurcation point the dynamical effects of noise might even tip the balance between two very different responses to the given cues, e.g., tip the balance between the ‘fight’ or ‘flight’ response to some shadowy form.

The effect of the independent superpositions of the “release” or “don’t release” options, coupled with the uncertainty in the timing of the vesicle release at each of the trillions of nerve terminals will be to cause the quantum mechanical state of the brain to become a smeared out superposition of different macro-states representing different alternative possible plans of action. As long as the brain dynamics is controlled wholly by Process II – which is the quantum generalization of the Newtonian laws of motion of classical physics – all of the various alternative possible plans of action will exist in parallel, with no one plan of action singled out as the one that will actually occur. Some other process, beyond the local deterministic Process II, is required to pick out one particular real course of physical events from the smeared out mass of possibilities generated by all of the alternative possible combinations of vesicle releases at all of the trillions of nerve terminals. That other process is Process I, which brings in the action of the mind of the agent upon his brain.

This explanation of why quantum theory is pertinent to brain dynamics has focused on individual calcium ions in nerve terminals. That argument pertains to the *Process II component* of brain dynamics.

The equally important *Process I component* of the brain dynamics, which brings the mind of the agent into the dynamics, must be analyzed in terms of a completely different set of variables, namely certain *quasi-stable macroscopic degrees of freedom*. These specify the brain structures that enjoy the stability or persistence, and the causal connections, needed to represent intentional actions and expected feedbacks.

The states of the brain that will be singled out by the actions P that specify the form of a Process I action will be more like the lowest-energy state of the simple harmonic oscillator discussed above, which tends to endure for a long time, or like the states obtained from such lowest-energy states by spatial displacements and shifts in velocity. Such states tend to endure as oscillating states, rather than immediately exploding. In other words, in order to get the needed stability properties the projection operators P corresponding to intentional actions should be constructed out of *oscillating states of macroscopic subsystems of the brain*, rather than out of sharply defined spatial states of the individual particles. The pertinent states will be functionally important brain analogs of a collection of oscillating modes of a drumhead, in which large collections of particles of the brain are moving in a coordinated way that will lead on to further coordinated activity.

In summary, the *need to use quantum theory* in brain dynamics arises from the dispersive quality of *Process II* action at the level of the ionic, and electronic, and atomic components of the brain. Hence *that* analysis is carried out

at the individual-particle level. However, the opposing integrative and selective action, Process I, which brings in the mental (i.e., psychologically described) aspect involves a completely different set of variables. Process I is specified by an operator P that singles out a quasi-stable large-scale pattern of brain activity that is the brain correlate of a particular mental intention.

It should be mentioned here that the actions P are *non-local*: they must act over extended regions, which can, and are expected to, cover large regions of the brain. Each conscious act is associated with a Process I action that coordinates and integrates activities in diverse parts of the brain. A conscious thought, as represented by the Von Neumann Process I, effectively grasps as a whole an entire quasi-stable macroscopic brain activity.

Choices of the Process I actions

It has been emphasized that the choices of which Process I actions actually occur are "free choices," in the sense that they are not specified by the currently known orthodox laws of physics. On the other hand, a person's intentions surely depend upon his brain. This means that we need to understand the process that determines the choice of P , which, within the framework of contemporary physical theory, is a free choice. In other words, the laws of contemporary quantum theory, although highly restrictive, are not the whole story: there is still work to be done. Hypotheses must be formulated and tested.

According to the theory, each experience is associated with the occurrence of a Process I event. As a simple first guess, let us assume, following a suggestion of Benjamin Libet and other psychologists, that the occurrence of a Process I action is triggered by a "consent" on the part of the agent, and that the rapidity with which consent is given can be increased by "mental effort."

To get a definite model, let $\{P\}$ be the set of actions P that correspond to possible mental intentions. Then let $P(t)$ be the "most probable P in $\{P\}$, where the probability is defined by brain state $S(t)$. In equations this most probable P in $\{P\}$ would be the P in $\{P\}$ that maximizes $\text{Tr } PS(t)P / \text{Tr } S(t)$. The first hypothesis will be that the Process I event specified by $P(t)$ will occur if and only if a "consent" is given at time t .

To make mind efficacious it is assumed that "consent" depends on the *mental realities* associated with $P(t)$, and that "consent" can be given with a rapidity that is increased if the mental evaluation includes a feeling of effort. This simplest model makes the choice of the Process I action dependent both upon the physical state of the agent's brain, and also upon the mental realities associated with that action.

It is assumed, here, that the consent associated with “hearing a nearby clap of thunder” is essentially passive: it will occur unless attention is strongly focused elsewhere. The important input of the mental aspect arises from the *effortful* focusing of mental attention on some intention.

Quantum theory *explains* how such a mental effort can strongly influence the course of brain events. Within the Von Neumann framework this potentially very strong effect of mind upon brain is an automatic consequence of a well-known and well studied feature of quantum theory called The Quantum Zeno Effect.

The Quantum Zeno Effect

If one considers only passive consents, then it is very difficult to identify any clean empirical effect of this intervention, apart from the production of low-level awareness. In the first place, the empirical averaging over the “Yes” and “No” possibilities tends to wash out all measurable effects. Moreover, the passivity of the mental process means that we have no independent self-controlled mental variable.

But the study of effortful and intentionally controlled attention brings in two empirically accessible variables, the intention and the amount of effort. It also brings in the important physical Quantum Zeno Effect. This effect is named for the Greek philosopher Zeno of Elea, and was brought into prominence in 1977 by the physicists Misra and Sudarshan (1977). It gives a name to the fact that repeated and closely-spaced intentional acts can effectively hold the “Yes” feedback in place for an extended time interval that depends upon the *rapidity at which the Process I actions are happening*. According to our quantum model, this rapidity is controlled by the amount of effort being applied.

This Quantum Zeno Effect is, from a theoretical point of view, a very clean consequence of the Von Neumann theory. It was first identified *theoretically*, and the theoretical predictions were later confirmed in many experimental contexts. The first confirmations were in the realm of atomic and molecular physics.

Consider an atom that has absorbed a photon of energy. That energy has kicked one of the atom’s electrons into what’s called a higher orbital, kind of like a super-massive asteroid kicking Mercury into Venus’s orbit, and the atom is said to be “excited.” But the electron wants to go back where it came from, to its original orbital, which it can do if the atom releases a photon. When the atom does so is one of those chance phenomena, like when a radioactive atom will decay: the atom has some chance of releasing a photon (and allowing the

electron to return home) within a given period of time. Physicists can measure whether the atom is still in its initial state or not. If they carry out such measurements repeatedly and rapidly, physicists have found, we can keep the atom in its initial state. This is the Quantum Zeno Effect: such a rapid series of observations locks a system into that initial state. The more frequent the observations of a quantum system, the greater the suppression of transitions out of the initial quantum state. Taken to the extreme, observing continuously whether an atom is in a certain quantum state keeps it in that state forever. For this reason, the Quantum Zeno Effect is also known as the watched pot effect. The mere act of rapidly asking questions of a quantum system freezes it in a particular state, preventing it from evolving as it would if we weren't peeking. Simply observing a quantum system suppresses certain of its transitions to other states.

How does it work? Consider this experiment. An ammonia molecule consists of a single atom of nitrogen and three atoms of hydrogen. The arrangement of the four atoms shifts over time because all the atoms are in motion. Let's say that at first the nitrogen atom sits atop the three hydrogens, like an egg nestled on a tripod (The nitrogen atom has only two options, to be above or below the trio. It cannot be in between.). The wave function that describes the position of the nitrogen is almost all concentrated in this configuration; that is, the probability of finding the nitrogen at the apex is nearly 100 percent. Left to its own devices, the wave function would shift as time went by, reflecting the increasing probability that the nitrogen atom would be found below the hydrogens. But before the wave function shifts, we make an observation. The act of observation causes the wave function (which, again, describes the probability of the atom being in this place or that one) to collapse from several probabilities into a single actuality. This is all standard quantum theory, the well-established collapse of the wave function following an observation.

But something interesting has happened. "The wave function has ceased oozing toward the bottom," as Sudarshan and his colleague Rothman explained (Rothman & Sudarshan 1998) "it has been 'reset' to the zero position. And so, by repeated observations at short intervals, . . . one can prevent the nitrogen atom from ever leaving the top position." If you rapidly and repeatedly ask a system, *are you in this state or are you not?*, by making observations designed to ascertain whether or not the nitrogen atom is where it began, the system will not evolve in the normal way. It will become, in a sense, frozen. An answer of "Yes" to the posed question [in this case: Is the nitrogen atom on top?] will become fixed and unchanging. The state will be forced to stay longer within the realm that provides a "Yes" answer.

Quantum Zeno has been verified experimentally many times. One of the neatest confirmations came in a 1990 study at the National Institute of Standards and Technology. There, researchers measured the probability that beryllium ions would decay from a high-energy to a low-energy state. As the number of measurements per unit time increased, the probability of that energy transition fell off; the beryllium atoms stayed in their initial, high-energy state because scientists kept asking them, "So, have you decayed yet?" The watched pot never boiled. As Sudarshan and Rothman concluded, "One really can stop an atomic transition by repeatedly looking at it."

This effect is a very special case of the general fact that if a sequence of similar Process I events occur rapidly [on the time scale of the macroscopic oscillations associated with the associated actions P] then the "Yes" outcome can be held in place in the face of strong Process II mechanical forces that would tend to quickly produce the "No" feedback. This means that agents whose efforts can influence the rapidity of Process I actions would enjoy a survival advantage over competitors that lack this feature, for they could sustain beneficial activities longer than their Process I deprived competitors. This gives the leverage needed to link mind to natural selection, and also the leverage needed to allow us to link our minds to our physical actions. For these efforts will then have intention-controlled physical effects, and this linkage can be discovered, and integrated into behavior, by trial and error processes.

Support from psychology

The empirical evidence cited above for the actual existence of the theoretically mandated Quantum Zeno Effect comes from physics experiments. But, according to the theory, the effect should manifest itself also in the realm of psychology.

A person's experiential life is a stream of conscious experiences. The person's experienced "*self*" is *part* of this stream of consciousness: it is not an extra thing that is outside or apart from the stream. In James's words "*thought is itself the thinker*, and psychology need not look beyond" (James 1890:401). The "*self*" is a slowly changing "fringe" part of the stream of consciousness. It provides a background cause for the central focus of attention.

The physical brain, evolving mechanically in accordance with the local deterministic Process II does most of the necessary work, without the intervention of Process I. It does its job of creating, on the basis of its interpretation of the cues provided by the senses, a suitable response. But, due to its quantum nature, the brain necessarily generates an amorphous mass of overlapping and conflicting templates for action. Process I acts to extract from this jum-

bled mass of possibilities a dynamically stable configuration in which all of the quasi-independent modular components of the brain act together in a maximal mutually supportive configuration of non-discordant harmony that tends to prolongs itself into the future and produce a characteristic subsequent feedback. This is the preferred “Yes” state PSP that specifies the form of the Process I event. But the quantum rules do not assert that this preferred part of the prior state *S* necessarily comes into being: they assert, instead, that if this process is activated – say by some sort of “consent” – then this “Yes” component PSP will come into being with probability

$$\text{Tr PSP/Tr } S.$$

And the rate at which consents are given is assumed to be increasable by mental effort.

The phenomena of “will” is understood in terms of this effortful control of Process I, which can, by means of the Quantum Zeno Effect, override strong mechanical forces arising from Process II, and cause a large deviation of brain activity from what it would be if no mental effort were made.

Does this quantum-physics-based conception of the connection between mind and brain explain anything in the realm of psychology?

Consider some passages from “Psychology: The Briefer Course”, written by William James. In the final section of the chapter on attention James (1892: 227) writes:

I have spoken as if our attention were wholly determined by neural conditions. I believe that the array of things we can attend to is so determined. No object can catch our attention except by the neural machinery. But the amount of the attention which an object receives after it has caught our attention is another question. It often takes effort to keep mind upon it. We feel that we can make more or less of the effort as we choose. If this feeling be not deceptive, if our effort be a spiritual force, and an indeterminate one, then of course it contributes coequally with the cerebral conditions to the result. Though it introduce no new idea, it will deepen and prolong the stay in consciousness of innumerable ideas which else would fade more quickly away.

In the chapter on will, in the section entitled “Volitional effort is effort of attention” James (1892:417) writes:

Thus we find that we reach the heart of our inquiry into volition when we ask by what process is it that the thought of any given action comes to prevail stably in the mind.

and later

The essential achievement of the will, in short, when it is most 'voluntary,' is to attend to a difficult object and hold it fast before the mind. ... Effort of attention is thus the essential phenomenon of will.

Still later, James says:

Consent to the idea's undivided presence, this is effort's sole achievement. ... Everywhere, then, the function of effort is the same: to keep affirming and adopting the thought which, if left to itself, would slip away.

This description of the effect of mind on the course of mind-brain process is remarkably in line with *what had been proposed independently from purely theoretical considerations of the quantum physics of this process*. The connections specified by James are *explained* on the basis of the same dynamical principles that had been introduced by physicists to explain atomic phenomena. Thus the whole range of science, from atomic physics to mind-brain dynamics, is brought together in a single rationally coherent theory of an evolving cosmos that consists of a physical reality that is constituted not of matter but of an action-based reality that determines propensities or tendencies for Process I events to occur.

In the quantum theory of mind-brain being described here there are two separate processes. First, there is the unconscious mechanical brain process called Process II. As discussed at length in the book, *Mind, Matter, and Quantum Mechanics* (Stapp 1993:150), this brain processing involves dynamical units that are represented by complex patterns of neural activity (or, more generally, of brain activity) that are "facilitated" (i.e., strengthened) by use, and are such that each unit tends to be activated as a whole by the activation of several of its parts. The activation of various of these complex patterns by cross referencing – i.e., by activation of several of its parts – coupled to feed-back loops that strengthen or weaken the activities of appropriate processing centers, appears to account for the essential features of the mechanical part of the dynamics in a way that in many cases is not greatly different from that of a classical model, except for the creation of a superposition of a host of parallel possibilities that according to the classical concepts could not exist simultaneously.

The second process, Von Neumann's Process I, is needed in order to select what actually happens from the continuum of alternative possibilities generated by Process II.

Process I, which is connected to conscious awareness and to what actually happens, has itself two modes. The first involves mere passive consent, and temporally isolated events; the second involves mental effort, and a sequence

of temporally closely spaced events that bring importantly into play the Quantum Zeno Effect. The first mode involves passive processing, and exploits the massively parallel processing capacities of process II, whereas the second mode involves an effortfully sustained sequence of Process I events.

Active Process I intervention has, according to the quantum-physics-based theory described here, a distinctive form. It consists of a sequence of intentional actions the rapidity of which can be increased with effort. Effort-induced speed-up of the rate of occurrence of these events can, by means of the quantum Zeno effect, keep attention focused on an intention.

Effort can, by increasing the number of intentional events per second, increase the input of mental processing into brain activity.

An extended discussion of non-trivial agreement of these features with a large body of data from the field of the psychology of attention is described in Stapp (2001).

Mental action in neuroscience

Scientists in different fields are to some extent free to choose what sort of models or theories they use to organize, explain, understand, and predict the observed features of the data in their field, and to guide their further inquiries. On the other hand, the ideal of *the unity of science* gives precedence to models that mesh with the basic principles of physics, or at least do not contradict them.

On the basis of this ideal it would seem that, for experiments that deal directly with the effects of self-directed mental intentions upon brain activity, the preferred theoretical framework would be one based upon contemporary physical theory, rather than one based upon an approximation that, according to contemporary physical theory, systematically eliminates from the more exact theory some important effects of mental intention upon the brain that seem to be exactly of the kind needed to account for the neuroscience data.

The conceptual structure of the more accurate quantum model seems eminently better suited to the analysis of this data than models based on the classical approximation. The quantum treatment not only *retains* a mechanism that coherently accounts for the data, it is also much simpler. For, just as in the treatment of atomic systems, it brings directly into the *dynamical* description of the phenomena a representation of the *phenomenological* description of those phenomena. It ties the mentalistic language that we human beings use to communicate to each other the facts about the phenomena into the physical description of the phenomena.

The foregoing account of the quantum approach is general, but somewhat abstract. To make it more tangible and concrete let us consider the experiments of Ochsner et al. (2002) with particular attention to the following four key questions:

1. How does the quantum mechanism work in this case, in comparison to what the classical account would say?
2. How do we account for the rapid changes occurring in large neural circuits involving millions of neurons during conscious and voluntary regulation of brain activity?
3. How does consciousness “know” where and how to interact in the brain in order to produce a specific psychological effect?
4. Is consciousness localized, and, if so, how and in what sense; or does it lie, instead, “outside of space”?

Reduced to its essence the experiment consists first of a training phase in which the subject is taught how to distinguish, and respond differently to, two instructions given while viewing emotionally disturbing visual images: ATTEND (meaning passively “be aware of, but not try to alter, any feelings elicited by”) or REAPPRAISE (meaning actively “reinterpret the content so that it no longer elicits a negative response”). The subjects then perform these mental actions during brain data acquisition. The visual stimuli, when passively attended to, activate limbic brain areas and when actively reappraised activate prefrontal cerebral regions.

From the classical materialist point of view this is essentially a conditioning experiment, where, however, the “conditioning” is achieved via linguistic access to cognitive faculties. But how do the cognitive realities involving “knowing,” “understanding,” and “feeling” arise out of motions of the miniature planet-like objects of classical physics, which have no trace of any experiential quality? And how do the vibrations in the air that carry the instructions get converted into feelings of understanding? And how do these feelings of understanding get converted to effortful actions, the presence or absence of which determine whether the limbic or frontal regions of the brain will be activated.

Within the framework of classical physics these connections between feelings and brain activities are huge mysteries. The classical materialist claim is that *someday* these connections will be understood. But the basic question is whether these connections will ever be understood in terms of a physical theory that is known to be false, and that, moreover, is false in ways that, according to contemporary physical theory, systematically exclude the causes of the correlations between the psychological and physiological aspects of the mind-brain

system that these neuropsychology experiments reveal. Or, on the other hand, will the eventual understanding of this linkage accord with the causal linkage between mental realities and brain activities that orthodox (Von Neumann) contemporary physical theory entails.

There are important similarities and also important differences between the classical and quantum explanations of the experiments of Ochsner et al. (2002). In both approaches the particles in the brain can be conceived to be collected into nerves and other biological structures, and into fluxes of ions and electrons, which can all be described reasonably well in essentially classical terms. However, in the classical description the dynamics are well described in terms of the local deterministic classical laws that govern these classical quantities, insofar as they are precisely defined.

Quantum theory asserts, however, that the condition that these classical quantities be *precisely defined* is unrealistic. Heisenberg's uncertainty principle asserts that this assumption is not justified: one must accept at least some small amount of cloudlike uncertainty. But small uncertainties rapidly grow into larger uncertainties. The discussion of the ionic motions in nerve terminals exemplifies this growth of uncertainty: the state of the brain rapidly fans out into a state that encompasses many possible experiential states.

This incursion into the dynamics of growing uncertainties renders the classical approach basically incomplete: it can never lead to well defined experiential states, except by actually violating the quantum laws.

[There is a well-known and powerful process in quantum theory that strongly influences this expansion of the state of the brain into a state that encompasses many alternative experiential possibilities. It is called "environmental decoherence." The interactions of the brain with its environment makes the state *S* of the brain into what is called a "mixture." This means that the interference effects between significantly different classically describable possibilities becomes markedly attenuated. That effect is, however, already completely accounted for in the Von Neumann state *S* of the brain: environmental decoherence in no way upsets or modifies the Von Neumann theory described here. Indeed it makes that theory basically accessible to neuroscientists by converting the complex mathematical concept of a quantum state into a structure that can be visualized as simply a collection of virtual classically conceived states: the quantum state of the brain is effectively transformed by environmental decoherence effects into an ensemble of classically describable potentialities that become converted to experiential realities by a Process I action. Thus the quantum brain dynamics become much easier both to conceive and to describe because the environmental decoherence effect allows classical language and im-

agery to be validly used in an important way. But it has never been shown to obviate the need for Process I.]

One could, despite violating the quantum laws, try to pursue a quasi-classical calculation. This would be a classical-type computation with the quantum-mandated uncertainties folded in as probability distributions, and with certain classically describable brain states identified as the “neural correlates” of the various possible experiential states. One could then produce, in principle, the same general kinds of statistical predictions that quantum theory would give.

This sort of quasi-classical approach would, in fact, probably give results very similar to quantum theory for situations arising from “passive attention.” For in these cases mind is acting essentially as a passive witness, in a way that is basically in line with the ideas of classical physics.

But quantum theory was designed to deal with the other case, in which the conscious action of an agent – to perform some particular probing action – enters into the dynamics in an essential way. Within the context of the experiment by Ochsner et al. (2002), quantum theory provides, via the Process I mechanism, an explicit means whereby the successful effort to “rethink feelings” actually causes – by catching and actively holding in place – the prefrontal activations critical to the experimentally observed deactivation of the amygdala and orbitofrontal cortex. The resulting *intention-induced modulation* of limbic mechanisms that putatively generate the frightening aversive feelings associated with passively attending to the target stimuli is the key factor necessary for the achievement of the emotional self-regulation seen in the active cognitive reappraisal condition. Thus, within the quantum framework, the causal relationship between the mental work of mindfully reappraising and the observed brain changes presumed to be necessary for emotional self-regulation is *dynamically* accounted for. Furthermore, and crucially, it is accounted for in ways that fully allow for communicating to others the means utilized by living human experimental subjects to attain the desired outcome. The classical materialist approach to these data, as detailed earlier in this chapter, by no means allows for such effective communication. Analogous quantum mechanical reasoning can of course be utilized *mutatis mutandis* to explain the data of Beauregard et al. (2001) and related studies of self-directed neuroplasticity (see Schwartz & Begley 2002).

Quantum theory resolves, within a scientific context, the problem of the connection between the psychologically and physically described features of the mind-brain system by, first, taking the linguistic/semantic modes of expression that scientists use to communicate experimental conditions and facts

to one another, and to others, to be descriptions of psychological realities that, in essence, are what they seem to be, and do what they seem to do, and, second, specifying the dynamical equation that determines how these psychologically described realities act upon physical brain states.

The second question is: How do we account for the rapid changes induced by mental effort in large brain circuits?

The answer is that the non-local operator P that represents the intention singles out a large quasi-stable and functionally important brain state that is likely to produce the expected feedbacks. Large intention-controlled functionally effective brain activities are singled out and linked to mental effort through *learning*, which depends upon the fact that the mental efforts, per se, have physical consequences. A "Yes" outcome actualizes such a functional state. Thus quantum theory provides the mathematical machinery for linking mentalistically described functional properties to the physically described macroscopic properties of the brain that implements them. No comparable linkage is provided by classical physics, which would appear to be constitutionally ill-equipped to accommodate causally efficacious mental actions.

The third question is: How does consciousness "know" where and how to interact in the brain in order to produce a specific psychological effect?

The answer is that felt intentions, per se, have physical consequences, and thence experiential consequences. Hence an agent can learn, by trial and error, how to select an intentional action that is likely to produce a feedback that fulfills an intention.

The fourth question is: Is consciousness localized, and, if so, how and in what sense; or does it lie, instead, "outside of space"?

Each conscious event is associated with a Process I action that involves an action P that is necessarily non-local, for mathematical reasons. Moreover, the "Yes" part must have the functional properties needed to set in motion the brain-body activity that is likely to produce the intended feed-back experience. Thus each conscious action would, in order to meet these requirements, act over some functionally characterized extended portion of the brain. [In fact, for reasons that go well beyond the scope of this chapter, this event also induces effects in faraway places: these effects are the causes, within the Von Neumann ontology, of the long-range non-local effects associated with the famous theorem of John Bell (1964).]

Conclusions and summary

The key point of this entire chapter is simply this: neuropsychology is greatly simplified by accepting the fact that brains must in principle be treated quantum mechanically. Accepting that obvious fact means that the huge deferred-to-the-future question of how mind is connected to a classically described brain must, in principle, be replaced by the already resolved question of how mind is connected to a quantum mechanically described brain. That shift resolves the problem of accounting for the dynamic linkage between linguistic/semantic modes of communication used in setting up experiments involving cognitive control of emotions and the physical description of the brains of subjects, by adopting the same pragmatic solution that atomic physicists adopted when faced with this *same* problem of accounting coherently for the effects of *mentalistically described human intentional actions upon the physically described systems that those actions act upon*. This solution is encapsulated in Process I. The benefits of adopting the pragmatic quantum approach may be as important to progress in neuroscience as they were in atomic physics.

References

- Beauregard, Mario, Levesque, Johanne, & Bourgouin, Pierre (2001). Neural Correlates of the Conscious Self-Regulation of Emotion, *Journal of Neuroscience*, 21, RC165, 1–6.
- Bell, John (1964). On the Einstein, Podolsky, Rosen Paradox. *Physics*, 1, 195–201.
- Bohr, Niels (1958). *Atomic Physics and Human Knowledge*. New York: Wiley.
- Bohr, Niels (1963). *Essays 1958/1962 on Atomic Physics and Human Knowledge*. New York: Wiley.
- Brody, A. L., Saxena, S., Stoessel, P., Gillies, L. A., Fairbanks, L. A., Alborzian, S., Phelps, M. E., Huang, S.-C., Wu, H.-M., Ho, M. L., Ho, M. K., Au, S. C., Maidment, K. & Baxter, L. R. (2001). Regional brain metabolic changes in patients with major depression treated with either paroxetine or interpersonal therapy. *Archives of General Psychiatry*, 58, 631–640.
- Byl, N. N. & McKenzie, A. (2000). Treatment effectiveness for patients with a history of repetitive hand use and focal hand dystonia: A planned, prospective follow-up study. *Journal of Hand Therapy*, 13, 289–301.
- Cataldi, Mauro, Perez-Reyes, Edward, & Tsien, Richard W. (2002). Difference in apparent pore sizes of low and high voltage-activated Ca^{2+} channels. *Journal of Biol. Chem.*, 277, 45969–45976.
- Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology*, 9, 281–291.
- Heisenberg, W. (1958). The representation of nature in contemporary physics. *Daedalus*, 87, 95–108.

- Horgan, Terence E. (1994). Physicalism. In Samuel Guttenplan (Ed.), *A companion to the philosophy of mind* (pp. 471–479). Oxford: Blackwell.
- James, William (1890). *The principles of psychology, Vol. I*. New York: Dover.
- James, William (1892). Psychology: The briefer course. In *William James: Writings 1879–1899*. New York: Library of America (1992).
- Leuchter, A. F., Cook, I. A., Witte, E. A., Morgan, M., & Abrams, M. (2002). Changes in brain function of depressed subjects during treatment with placebo. *American Journal of Psychiatry*, 159, 122–129.
- Lévesque, J., Joanette, Y., Paquette, V., Mensour, B., Beaudoin, G., Leroux, J.-M., Bourgouin, P., & Beaugard, M. (2003) Neural circuitry underlying voluntary self-regulation of sadness. *Biological Psychiatry*, 53, 502–510.
- Mayberg H. S., Silva, J. A., Brannan, S. K., Tekell, J. L., Mahurin, R. K., McGinnis, S., & Jerabek, P. A. (2002). The functional neuroanatomy of the placebo effect. *American Journal of Psychiatry*, 159, 728–737.
- Misra, B. & Sudarshan, E. C. G. (1977). The Zeno's paradox in quantum theory. *Journal Math Phys.*, 18, 756–763.
- Musso, M., Weiller, C., Kiebel, S., Muller, S. P., Bulau, P., & Rijntjes, M. (1999). Training-induced brain plasticity in aphasia. *Brain*, 122, 1781–1790.
- Newton, Isaac (1687). *Principia Mathematica*. [Newton's Principia, Florian Cajori (Ed.) (1964). Berkeley: University of California Press.]
- Nyanaponika Thera (2000). *The vision of Dhamma: Buddhist writings of Nyanaponika Thera*. Seattle, WA: BPS Pariyatti Editions.
- Nyanaponika Thera (1973). *The heart of Buddhist meditation*. York Beach, ME: Samuel Weiser.
- Ochsner, K. N. & Feldman Barrett, L. (2001). A multi-processperspective on the neuroscience of emotion. In G. Bonnano & T. J. Mayne (Eds.), *Emotion: Current issues and future directions* (pp. 38–81). New York: Guilford.
- Ochsner, K. N., Silvia, A. Bunge, James, J. Gross, & John D. E. Gabrieli (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience*, 14 (8), 1215–1229.
- Paquette, V., Lévesque, J., Mensour, B., Leroux, J.-M., Beaudoin, G., Bourgouin, P. & Beaugard, M. (2003). "Change the mind and you change the brain": Effects of cognitive-behavioral therapy on the neural correlates of spider phobia. *NeuroImage*, 18, 401–409.
- Rothman, T. & Sudarshan, E. C. G. (1998). *Doubt and Certainty*. Reading, MA: Perseus Books
- Schwartz, J. M., Stoessel, P. W., Baxter, L. R. Jr, Martin, K. M., & Phelps, M. E. (1996). Systematic changes in cerebral glucose metabolic rate after successful behavior modification treatment of obsessive-compulsive disorder. *Archives of General Psychiatry*, 53 (2), 109–113.
- Schwartz, J. M. (1998). Neuroanatomical aspects of cognitive-behavioural therapy response in obsessive-compulsive disorder: An evolving perspective on brain and behavior. *British Journal of Psychiatry*, 173 (suppl. 35), 39–45.
- Schwartz, J. M. & Begley, Sharon (2002). *The mind and the brain: Neuroplasticity and the power of mental force*. New York: Harper Collins.

- Segal, Zindel V., Williams, J. Mark G., & Teasdale, John D. (2002) *Mindfulness-based cognitive therapy for depression*. New York: Guilford Press.
- Smith, Adam (1976). Raphael, D. D. & Macfie, A .L. (Eds.). *The theory of moral sentiments* (pp. 112–113). New York: Oxford University Press.
- Spence, S. S. & Frith, C. (1999). Towards a functional anatomy of volition. In B. Libet, A. Freeman, & K. Sutherland (Eds.), *The volitional brain: Towards a neuroscience of free will* (pp. 11–29). Thorverton, UK: Imprint Academic.
- Sperry, R. W. (1992). Turnabout on consciousness: A mentalist view. *Journal of Mind & Behavior*, 13 (3), 259–280.
- Stapp, Henry P. (1993). *Mind, matter, and quantum mechanics*. New York: Springer-Verlag.
- Stapp, Henry P. (2001). Quantum theory and the role of mind in nature. *Found. of Phys.*, 11, 1465–1499.
- Von Neumann, John (1955). *Mathematical foundations of quantum theory*. Princeton: Princeton University Press.
- Wittgenstein, Ludwig (1968). *Philosophical Investigations* (3rd edition). Translated by G. E. M. Anscombe. New York: Macmillan.

Appendix

The culture of behaviorism and the conflict in philosophy of science (by Jeffrey Schwartz and Mario Beauregard)

The question might well arise: what is controversial about the assertion that the mental act of mindfully observing one's own emotional experience can in and of itself change the brain's responses to stressful stimuli? A brief overview of the relevant background assumptions of mainstream research in neuropsychology and cognitive neuroscience may help place this question in a clearer historical and philosophical perspective. (For further details and references concerning the issues discussed in this section, see: Honderich 1995; Chalmers 1996; Schwartz & Begley 2002).

The regnant world view for twentieth century philosophy (which was based on classical physics) was narrowly materialistic. With regard to the mind/brain question, this world view posited that mental phenomena are ultimately explicable in terms of the behavior of material particles. This perspective also tacitly assumed that all material interactions are in principle observable by the five senses, given appropriate technical advances in magnification, spatial and temporal resolution, etc. This view thus resulted in the generally unstated assumption that only phenomena observable and measurable within the realm of the five senses count as relevant for scientific investigation, i.e., all phenomena having causal significance for science should at least potentially be observable by the five senses, given appropriate technical advances. To properly understand the nature of the current controversy it is important to firmly grasp that, because of these assumptions, what all forms of materialism deny is the basic intuition that the process of conscious observation, *per se*, *does something* to the experience that is observed and to the brain states related to that experience. Because the process of mindful observation, as an experiential process in and of itself, is not reducible in principle to externally observable material particles, such experiential phenomena, *per se*, can

not have the power to effect change in the brain or anything else – for materialism, only physical matter has causal efficacy. In the final analysis what materialists are claiming is that, regardless of how real it seems, it is mere fantasy to believe that the quality of attention you choose to direct (e.g., mindful or not mindful) actually makes a difference in the quality of your perceived observation, much less in the brain mechanisms related to that observation.

In psychology the classic elaboration of this outlook is behaviorism, which limits the realm of investigation of humans to precisely that of all other phenomena, namely externally observable five-sense data. This view which denies the causal efficacy, and, in its most austere form even the reality of internally experienced phenomena, is still very much part of the cognitive neuroscience's culture. Behaviorism reduces all emotional experience to their physically observable manifestations. On this view a description like "anxious" tells us only facts about things like a person's blood pressure, muscle tension, and observable acts in certain kinds of situations, i.e., emits a shriek and/or certain types of language-based auditory phenomena when confronted by a spider – it tells us nothing of causal significance about any inner experience the person is having. A newer and more developed version of this perspective involves adding data about the state of the brain obtained by functional brain imaging and related investigational tools to the description. But it is critical to note that in principle this changes nothing of importance from a behaviorist's basically materialist perspective – all it does is add a new source of externally describable five sense data to the explanatory story. The inner experience of the person whose brain is imaged still has no role to play in the causal chain. It is just that the linguistic and other behaviors of the person are now explained in terms of brain data instead of only externally observable behavioral data – inner mental experience is still, as in classical behaviorism, viewed as causally unrelated to any scientifically meaningful phenomena. A philosophical position related to this perspective has been called eliminativism, basically the view that all common language or "folk psychology" descriptions of mental experience should be eliminated and replaced by description using neuroscientific language.

Most people find eliminativism impossible to believe – the word "belief" itself being an example of "folk psychology" that seems unreasonable to dispense with. One common approach used by philosophers attempting to maintain the position that all aspects of the mind are explicable solely in terms of material causes, while not dispensing with all reference to common psychological language, is to define mental states solely in terms of the functions they seem to serve. These functions are claimed to be revealed in verbal behavior and the relations between such utterances and the behavioral activity (including brain activities) that can be observed to precede and follow them. This view is generally known as functionalism. A closely related perspective, known as identity theory or the emergent property position, is discussed in detail in the physics section of this chapter due to its intrinsic relationship to the world-view of Newtonian mechanics.

The biggest problem with these kind of explanations, all of which are closely wedded to classical materialist (as opposed to quantum) notions of physical causation, is that they deny in principle that conscious mental experiences *per se* are the real causes of anything that science is concerned with explaining. For those who think the awareness of an emotional state is a fundamentally real conscious experience that plays a crucial role in its regulation, and that the mental act of regulating an emotion causes changes in how observable brain states operate, this is a huge problem. These materialist views are also extremely counter-

intuitive, since our most basic experience informs us that our choice of perspective regarding how we apprehend our mental states makes a real difference in how we respond to them. For those who insist that only material causes matter such intuitions must be denied as illusory. Fortunately, as we have attempted to demonstrate in the body of this chapter, physics as it is currently understood and practiced, i.e., quantum physics, offers a precise scientifically grounded solution to this apparent conflict between the observed reality of our emotional experience and the requirements of rigorous data-based explanation.

A word about the meaning of the term data may begin to point us toward a more pragmatic perspective on these issues. For the materialist *all* possible data *must* in principle be provided, at least potentially, by the five senses (perhaps aided by technical advances in data acquisition) and their contact with the external world. But the way in which the experiments on the neuropsychology of emotional self-regulation are actually designed and performed belies this perspective: the use of language and other modes of communication that refer to phenomena that are intrinsically experiential and non-reducible to materialist terminology (e.g., Actively reappraise this scene; Be mindful now, etc.) are a critical and irreplaceable part of the experimental instructions, training and set-up. From the materialist's point of view the only aspects of the entire experimental set-up that count as real are the brain data itself, any observable bodily phenomena, and the actual physical sounds of the verbal reports the subjects make to the investigators, as recorded by marks on rating scale sheets, etc. The concept that the subjects *did something* with their minds *per se* that actually influenced their emotional responses and the related brain data collected is strictly prohibited by the materialist perspective. The idea that the subjects actually *experienced* any internal change in feeling state on account of their mental acts is considered entirely out-of-bounds. Only what is externally observable counts as real in any ultimate sense. One is reminded of the old anecdote about two strict behaviorists who meet at a conference. After imbibing some alcoholic beverage they proceed to a hotel room and engage in intensive copulatory activity. Pulses soar, blood surges, engorges, and subsides. At the conclusion of the physiological excitation process, one turns to the other and sighs, "Well, I know it was great for you. How was it for me?"

Chalmers, David J. (1996). *The conscious mind: In search of a fundamental theory*. New York: Oxford University Press.

Honderich, Ted (Ed.). (1995). *The Oxford companion to philosophy*. New York: Oxford University Press.

CHAPTER 8

EEG biofeedback (“Neurofeedback”) and affective disorders

J. Peter Rosenfeld and Elsa Baehr

Introduction

Organization of this chapter

After a definition of the term *Neurofeedback*, and a brief discussion of its underlying principles, this chapter will review the evidence that at least some examples of operant EEG conditioning – such as the voluntary control of brain waves – are not trivially mediated by familiar phenomena. Then, a presentation of examples of the functional significance of neurofeedback will be given; one experimental and one clinical illustration will finally be followed by an example of the limits of this new clinical protocol.

Definition with underlying principles

The term *Neurofeedback* is a recently coined shorthand term for operant conditioning of neural activity. Nowadays, it is frequently used in clinical contexts. In general, the neural activity can be any form of individual or population measures of the activities of neurons, and indeed there have been reports of operant conditioning of action potentials from single cortical neurons (Fetz & Finnochio 1971) as well as more frequent reports of conditioning of spontaneous EEG (Kamiya 1969) and its time-locked derivatives, i.e., event-related potentials (ERPs) from animal brains (Fox & Rudell 1968) or human scalps (Rosenfeld, Rudell, & Fox 1969). Operant conditioning of any neural event means that a subject is rewarded for (voluntarily) changing the future probability of the specific event. Obviously, this is the prevailing operant model of neurofeedback. This model is close to the original formulation of operant conditioning as articulated by Skinner in the 1930s: training a rat to press a

bar is simply a matter of setting up a reinforcement contingency such that any time the bar is pressed, the rat is positively reinforced or rewarded with, say, a food pellet. If the rat is motivated (e.g., hungry), he will learn the association between response and reward and will eagerly press the bar. Operant or voluntary control in humans is often also by trial and error, perhaps aided with a bit of coaching. When we learn a new word processor program, it is not always obvious how to achieve an effect, such as bolding. We try a few things, perhaps coached by icons, and we learn whatever works, such as the association between the “B” icon press and the bolding effect. Unfortunately, the neural mechanism of this simple trial-and-error learning process remains as unknown as in Skinner’s time, even for the most simple of operant responses. In the neurofeedback situation, we assume that a certain desired brain wave pattern is like a bar press, and we reward the subject following emission of these target neural events. As in operant behavioral conditioning, the target behaviors – neural or ordinary – have to be in the animal’s ordinary behavioral repertory prior to training. Indeed in our first studies of evoked potential amplitude training described below, we would observe the subject for one or more days of baseline with no rewards just to collect the amplitude distribution. Then we would reward animals for increasing the probability of one tail of this *normal* distribution of pre-training amplitudes. That is, the amplitude had to be within the normal repertory of amplitudes. How the animals learned to shift their distributions for reward remains a mystery. What was critical to us was to eliminate some obvious trivial mechanisms (this is discussed in the next section). In some human clinical situations, matters become slightly more complicated, as operant neural training may be preceded and/or accompanied by relaxation training, and/or various other therapies. These other components of a neurofeedback training package probably have non-specific effects, and may be unnecessary.

There may be one additional complication regarding the mechanism of operant neural conditioning vs. operant conditioning of familiar behaviors (bar presses), and that complication merits noting. This is the issue of what fundamental event gets conditioned, specifically. Do subjects learning to change the amplitude of an EEG wave have direct, non-trivial control over that wave? Or do the subjects change something else which influences or is reflected in that EEG event? If, for example, a visual evoked potential is changed by the subject’s learning to close his eyes or stare into the light source, this is mediated conditioning. Indeed, it is trivially mediated, as discussed in the next section. On the other hand, the conditioning could be mediated, but in a novel, non-trivial fashion. Or it could be unmediated and direct. With ordinary behavioral

conditioning, one can readily identify the target behavior (e.g., the bar press) and assume that the subject can experience the behavior also that results in the reward. If not the behavior, then the somatic sensory feedback from the behaving limb, its efferent control and/or collateral feedback discharge. With neural events, there is no known physiological feedback from the conditioned brain part which sends information to awareness systems. The learning mechanism becomes an even deeper conondrum.

The trivial mediation issue

S. S. Fox and Rudell's use of operant controlled neural events (OCNE) was introduced as a new methodology for clarifying neural coding of behavior. However, their initial 1968 paper in *Science* caused interest in the neuroscience community not so much because it was a new approach to neural coding, but because prior to this report, it was widely believed that the pattern of components in the photically evoked ERPs in the cat's cortex was not subject to self-regulation, but represented the hard wired, sequential summations of post-synaptic potentials in the visual cortex in response to a consistent high intensity stroboscopic light source. The cats of Fox and Rudell dramatically changed their brain responses following a regimen of training in which they received a small amount of milk (pumped directly into their mouths) following emission of responses of low, pre-training probability. Thus, reactions to this report included some nay-saying. For example, it was noted that the conditioned cats were free to move about in their conditioning chambers and thus able to voluntarily alter their photic ERPs by (uninterestingly) changing their receptor orientation to the light source. Or they could execute either phasic or tonic movements whose somatosensory feedback could, via reticular formation and its widespread reticulocortical connections, alter the photic ERPs (also uninteresting and not novel). A series of papers followed (summarized by Rosenfeld 1990) which ultimately ruled out all possible sources of such trivial mediation of operant controlled ERPs. In particular, Rosenfeld, Hetzler, Birkel, Antoinetti, and Kowatch (1976) demonstrated that in total darkness, rats could be trained (via rewarding brain stimulation in the lateral hypothalamus) to systematically alter the amplitude of early and late components of the visual ERP elicited by mild shock to the optic chiasm, each shock presented at random intervals. It had been thus clearly shown that organisms could voluntarily control internal neural activity utilizing some novel mechanism. This was a novel operant response that could be used to test the generality of putative laws of learn-

ing derived from experiments on other (usually voluntary motor) systems (see Rosenfeld et al. 1976). (Incidentally, even in our earliest studies, we informally visually observed both cats and people during the operant ERP conditioning and never saw any sign of trivial mediation.)

In retrospect, such self-regulation of brain activity in a non-trivial manner could have been anticipated. In Rosenfeld et al. (1969), humans instructed to sit still and alter acoustic ERPs explained their successful ability to generate such alterations by referring to use of their imaginations. We knew nothing about how imagining activity was encoded in EEG and how it might influence externally evoked activity, and so we tended to write off these responses, (imagining activity was also not a popular element of any hypothesis at a place such as Kenneth Spence's highly behaviorally oriented department of Psychology where our early work was done). Nevertheless it can be simply stated today that the key assumption of any model of operant neural conditioning is that there must be an input to the conditioned neuronal organization from another neuronal organization over which a subject has direct control. We take as given the fact that individuals can control the contents of their imaginations to a large extent from moment to moment. The fact that individuals can alter ERPs in a non-trivial manner and report using imagination to do so means that the neuronal organizations mediating imagination activity must be connected to the altered neuronal populations. This need not be the only mechanism of operant neural control or neurofeedback.

Functional significance: Experimental studies

The fact that individuals could alter visual ERPs in a non-trivial manner begged the question of functional significance. After all, the sizeable changes in the amplitudes of visual ERPs that were being produced by operant conditioning meant that the patterns of post-synaptic potential integration giving rise to EEG and ERP phenomena must themselves be changing. The neurons generating such post-synaptic potentials were likely to be functioning neurons in the visual cortex. Thus one could wonder to what extent vision was being influenced by operant control of visual ERPs. We next addressed ourselves to this question of functional significance, however for various reasons (see Rosenfeld 1990), we moved from the visual modality to the pain modality. Therefore, instead of recording from visual cortex, we recorded from the somatic sensory cortex (a terminus of major pain systems) and instead of evoking potentials by stimulating the optic tracts, we delivered *non-painful* shocks to the trigeminal

tract, the neural pathway which transmits orofacial and dental pain information to the central nervous system. Clearly, high-level shocks to this tract would produce pain. This path was our ERP-eliciting locus, and had to be stimulated several hundred times, every few seconds during a daily training run. The level of stimulation used had to be kept at non-painful levels and so was adjusted to be just high enough to evoke to-be-conditioned potentials in the somatic sensory cortex. To measure the effects of operantly conditioning these trigeminally evoked, cortical ERPs on pain perception, we slowly increased the heat in a heater attached to the rats' faces. We assumed that when the rat started rubbing at the heaters, the heat was approaching painful levels and we immediately shut off the heat, recording the time from heat onset to first face rub as the pain tolerance index. The results (Dowman & Rosenfeld 1983, 1985), to our amazement, were that all rats with histologically confirmed correct electrode placements were readily able to increase as well as decrease (depending on the reinforcement contingency we set) the amplitude of the selected ERP component, *and the consequence of this neural training was a systematic change in the pain tolerance index. Also, the degree of this change was "clinically significant"* since it corresponded to the change produced by a moderate dose of morphine.

These results did stimulate us to explore whether they would generalize to human clinical situations, and we did initiate such work (Rosenfeld, Silvia, Weitkunat, & Dowman 1985; Weitkunat & Rosenfeld 1986), however we did not carry it forward for various reasons. One reason was that we had demonstrated effects in animals *with experimentally induced pain*. Human pain in clinical situations is very different. Clinical pain tends to be tonic in nature, unlike our rapidly increasing heat bursts in rats which tended to peak in 5 seconds. Also, chronic clinical pain is also often accompanied by psychological stress and/or anxiety and/or depression. Our results in rats did serve the purpose of demonstrating functional significance, if not a novel clinical modality, and so we decided to leave it to others to explore the clinical pain situation. There was a theoretical issue also. The rationale for the somatic sensory evoked potential training effect on pain was our knowledge that there existed neurons, called "wide dynamic range neurons," in somatic sensory cortex, which were activated both by innocuous somatic sensation, such as our non-noxious shocks to the trigeminal tract, as well as by pain such as that produced by our facial heaters. We reasoned that if the operant conditioning procedure could change the states of these neurons during the training session, their states would still be altered when the actually painful heat burst was applied immediately after the last daily training trial, leading to altered pain tolerance. This worked, as we have seen, but we had no idea how long this altered state would last beyond

the end of the training session. This was another loose end that we felt needed tying up prior to a foray into the clinical pain problem. However, our major reservation was that our approach had been indirect in that the ERPs evoked by non-noxious stimulation were themselves not a pain index, yet their alteration was (not unreasonably) expected to lead to pain change. We decided that before a clinical application of operant neural control was attempted, we would wait until someone found a robust indicator of some human pathology.

Functional significance: Clinical application for depression

We did not have a long wait. Indeed Richard J. Davidson and colleagues had commenced a program of study of the neural mechanisms of emotion at about the time we had been training ERP changes in the rat somatic sensory cortex. Davidson's highly influential program of research has dealt with many aspects of emotion, theoretical and applied (see Davidson 1995, and other chapters in this volume by Fox & Lane), but for us, the key papers were those by Henriques and Davidson (1990, 1991). These papers showed that, as predicted by the Davidson group's other work and theorization, depression was characterized by a characteristic frontal cortical asymmetry in comparison to the EEG from a non-depressed control group. Normal frontal cortical asymmetry tended to produce roughly equal amounts of right and left cortical activation, or more left than right activation. Depression correlated with a reduced activation on the left side. This was expected and predicted since many other studies from this laboratory had demonstrated that manipulations tending to produce negative affect also led to a reduction in left frontal activation and an increase in right frontal activation. The EEG results, in turn, were predicted from Davidson's model in which (to oversimplify) there is a positive, approach emotion system in the left frontal cortex, but a negative, withdrawal emotional system in the right frontal cortex. Now having a direct neural correlate of affect, we decided to see whether or not the EEG asymmetry could be operantly conditioned, and if so, to determine the emotional consequence, if any, in a clinical situation.

In Davidson's studies and in ours to be described, activation asymmetry was indexed (inversely) by EEG Alpha (8–12 Hz) power. Thus an active cortex would have a paucity of alpha, and an idling cortex would have excess alpha (the reasons for the inverse indexing are in Rosenfeld 2000). There are two formulae which have been used to index moment to moment alpha asymmetry: $A[1] = \log R - \log L$ where R and L are right and left alpha power or magnitude;

$A[2] = (R - L)/(R + L)$. As these *Asymmetry scores* correlate at .98 (Rosenfeld 2000) even though they are not mathematically equivalent, both formulae may be used interchangeably for moment to moment assessment of alpha asymmetry, where the higher the score, the greater the relative ratio of right to left alpha, the lower the ratio of right to left activation, and presumably, the greater the positive affect. In summarizing, for patients, asymmetry results for a session, as well as for research, we found that the mean $A[1]$ or mean $A[2]$ scores were not as good as a percentage (PCT) score we developed: this was simply the percent of time that $A[2]$ was greater than zero. In one session, we had found that the PCT scores discriminated depressed and normal persons better than the mean $A[2]$ score, as might be expected, since a mean $A[1]$ or $A[2]$ index would be much more influenced by extreme values (Baehr, Rosenfeld, Baehr, & Earnest 1998) than would the PCT index. Our neurofeedback training method in these early studies was lifted from our work with human ERPs (Rosenfeld et al. 1969): we would observe the alpha power in one or more baseline sessions, and then reward subjects with a pleasant tone sound for producing $A[1]$ scores 0.7 standard deviations greater than the baseline mean. Our methods with clinical cases are similar and detailed in Rosenfeld (2000).

Our first study in the operant conditioning (neurofeedback) of frontal alpha asymmetry was reported by Rosenfeld, Cha, Blair, and Gotlib (1995). In this study in which we were working with normal subjects from the introductory psychology participant pool and whose training time was, therefore, limited, we subjected the subjects to only three days of training (most neurofeedback regimens in current use call for upwards of 30–40 sessions). Nevertheless, 9 of 13 subjects showed significant (doubling) of their baseline $A[1]$ scores. This encouraged our plan to transpose the paradigm into the clinical situation. In the meantime, an independent lab (however with our software) extended our work by demonstrating that the asymmetry changes could be generated, and that these changes could be made bidirectionally, and that there were emotional consequences: subjects trained to increase $A[1]$ showed greater positive affect to happy and neutral films than did subjects trained to decrease $A[1]$, as would be predicted from Davidson's model (Allen & Cavendar 1996).

These results were certainly encouraging with respect to our clinical ambitions, however since no patients with real affective disorders were involved, the studies were merely preliminary. Nevertheless, these studies were very relevant to a theoretical issue with which Davidson's group and many other workers have long been grappling: the state-trait issue. Henriques and Davidson (1990, 1991) had shown, as we have seen, that in one study, never depressed persons had $A[1]$ scores much higher than currently depressed persons; in another

study they showed that never depressed persons had A[1] scores also much greater than previously depressed but *currently remitted, i.e., non-depressed persons*. They saw these data as supporting the notion that alpha asymmetry was a trait marker rather than a state marker; i.e., persons with the depressed asymmetry pattern had an innate “affective style” which made them vulnerable to depressive episodes in response to the stresses of life. We actually extended these findings by comparing all three groups within one study and observed that currently depressed and previously depressed but *currently remitted, i.e., non-depressed* persons both had lower A[1] scores than never depressed persons (Gotlib, Ranganath, & Rosenfeld 1998). This was consistent with the Henriques and Davidson studies, and the trait model of activation asymmetry. However, the neurofeedback studies immediately dislodged us from the pure trait position, because we reasoned that if the asymmetry pattern were fixed and innate, it would seem impossible to change it appreciably with any intervention. Indeed, the Henriques and Davidson studies, as well as the Gotlib et al. study, could be explained without a trait model: it could be the case that the normal asymmetry pattern is present in all individuals prior to a first depressive episode. Once this episode occurs, the pattern is changed and tends to linger. In this formulation, the asymmetry pattern is a result, not an antecedent of depression. However, it could be countered that the *range* of asymmetry values in an individual is fixed, and neurofeedback can produce changes only within the narrower individual range. The range is what may vary among individuals and the range may show properties of a trait. Our clinical studies with real patients, as described below, however, will indicate that neurofeedback can produce EEG and emotional changes which shift depressed patients’ asymmetry scores well out of the depressive range as affect is improved. There is much other evidence that alpha asymmetry is a state marker in that it varies with the nasal cycle (Quinn 1998), and with simple passage of time over days, and that its value on a given day predicts affective response in psychotherapy (Rosenfeld, Baehr, Gotlib, & Ranganath 1996). All these results are summarized in detail in Rosenfeld (2000).

In order to deal with issues involving magnitude of change in patients or controls, it became necessary to develop our PCT index (see above) and to compare its values in patients vs. normals. Previous studies of this type had utilized either A[1] or A[2] indices. Baehr et al. (1998) undertook precisely this novel study involving 13 currently depressed out-patients and 11 individuals free of depressive affect. It was the first time PCT as a dependent variable was studied in this way. All diagnoses were based on DSM-IV interviews and on scores on the Beck Depression Inventory (BDI – a score of 10 was the

Table 1.

PCT at end of therapy	PCT follow-up	(years later)	BDI at end	BDI follow-up
(1) 84	86	(1 year)	3	3
(2) 86	66	(3 years)	4	4
(3) 69	69	(5 years)	2	3

cut-off for depression). We found that PCT scores <56 always characterized depressed persons, and that scores >59 characterized non-depressed affect. Between values of 56 and 59, were PCT scores from 4 of the 24 cases (17%), 2 depressed subjects and 2 non-depressed subjects. Of course there were significant *group* differences in A[2], PCT, and BDI, which supported the Henriques and Davidson (1990, 1991) and Gotlib et al. (1998) studies, however only the PCT score (and not the A[2] score) nicely discriminated *individual* members of the two groups.

The next step was, finally, to attempt the neurofeedback therapy involving operant conditioning of the A[2] score in clinically depressed out-patients (from the private clinical practice of the present second author, E.B.). These patients were all on medications with doses stabilized months before the beginning of the neurofeedback treatment (see Rosenfeld 2000, where details of the training protocol and patient demographics may also be found along with the results for four individual cases.) Psychotherapy sessions alternated with pure neurofeedback training sessions. Figure 1 below shows summary data. The figure shows that the neurofeedback protocol led to changes in the PCT scores from about 50, $sd = 8.7$ (i.e., well within the depressed range) to about 71, $sd = 7.7$ (well into to range of normal affect). The corresponding BDI scores fell from 28 ($sd = 13.4$) to 4.7 ($sd = 6.2$). The DSM-IV diagnoses after neurofeedback also indicated clear recovery from depression.

Since this initial study, two additional patients from the practice of Elsa Baehr were put successfully through the asymmetry protocol (making a total of six), and an independent investigator also reported success, for the first time, with an adolescent patient (Earnest 1999). The next question to investigate, which had not yet been posed, concerned the issue of long term results. That is, how long after the last therapy session do the positive effects – both EEG and clinical – endure? Baehr, Rosenfeld, and Baehr (2002) were able to examine three of the original six patients tested at one, three, and five years, respectively, after the last neurofeedback training session. The results for the three cases are as shown in Table 1.

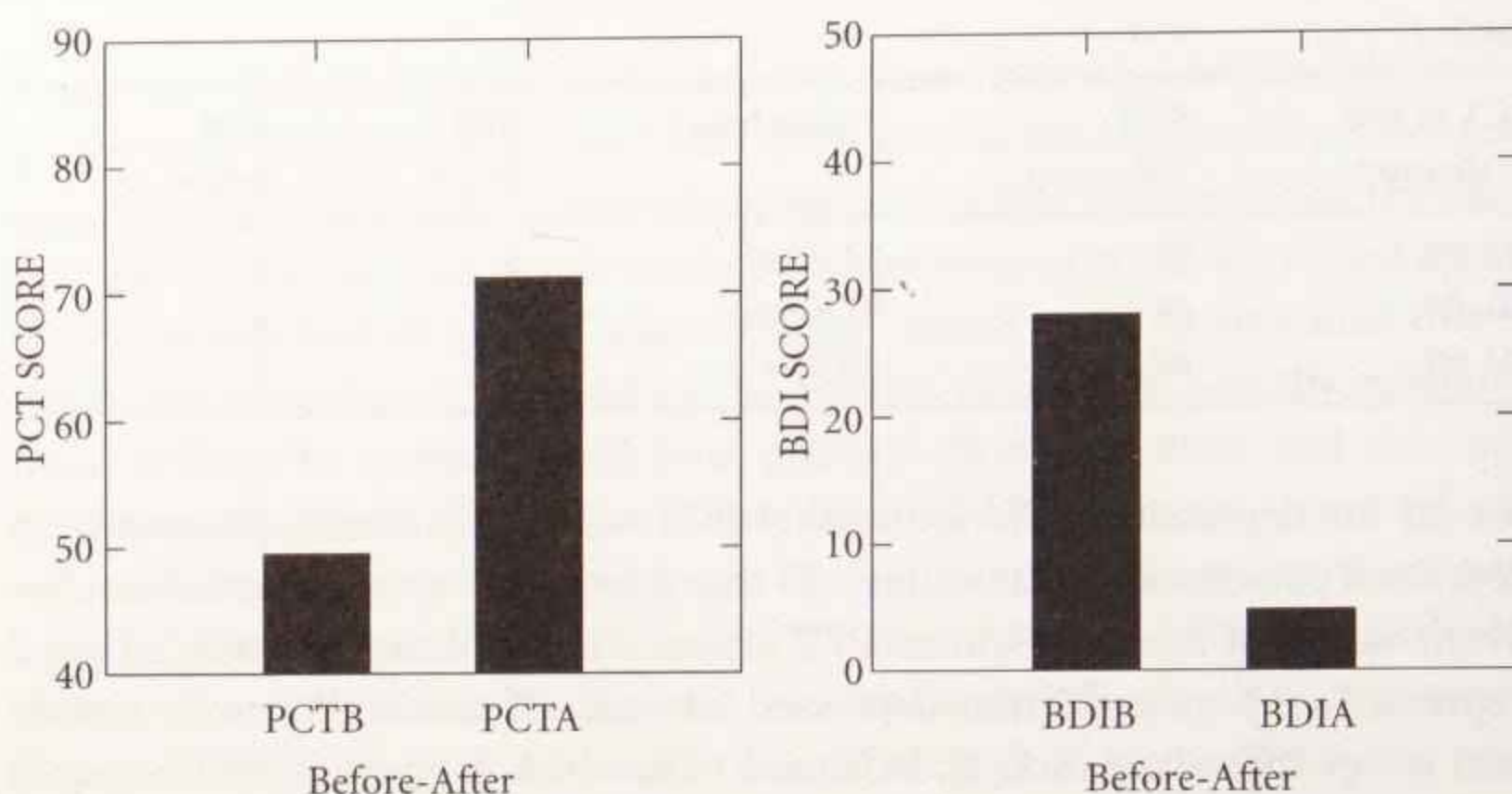


Figure 1. Mean PCT and BDI scores in a group of 5 previously depressed patients, before and after training with the alpha asymmetry protocol.

It is noted that the mean PCT score for these cases before therapy was 51.3 and the mean BDI score was 35.5. Clearly, the neurofeedback protocol appears to result in PCT and BDI scores well out of the depressive ranges of >58 and <10 , respectively, and these values hold up after 1–5 years (Table 1).

These results allow us to suggest that it is possible, with a specific neurofeedback paradigm, to alter neural correlates of depression in a human patient, which, in turn, results in long-enduring emotional changes. Only the lack of a solid control group tempers this suggestion so that it cannot yet be a conclusion. We want to be able to say that it is the *learning of a specific change in the EEG which produces the psychological change*. However, given only the data so far collected, we cannot say this. This is because it could be objected that it is the patient contact and/or the psychotherapy component of the treatment package and/or the charisma of the therapist which produces the lifting of the depressive affect. This could happen in various possible ways: the psychotherapeutic component of the experience could be a corrective emotional experience which results directly in positive affect which is then reflected, as expected, in the EEG; or, alternatively, the entire therapy experience could directly alter the A[2] scores, resulting in improved affect. In either case, it could be that *any* neurofeedback paradigm would have resulted in the same positive outcome. An ideal control would be a group just like the one treated with the asymmetry protocol, however given some other neurofeedback reinforcement contingency operating on a neural activity which should not affect the relative

left-right frontal cortical activation ratio. We have not run such a control group at this point, although it is noted that some of the original depressed patients had been in other therapy treatments (showing no affective improvement) for years prior to use of the asymmetry training protocol. Indeed one such patient had been in various forms of pharmacotherapy and psychotherapy – including one other ineffective neurofeedback therapy – for 12 years with one of us (E.B.) prior to showing any effects with the asymmetry training protocol.

An important finding in these clinical studies, even without a control group, is the fact that large changes in both PCT score and clinical function (as measured by BDI score and psychiatric interview) could be generated under experimental control, because these findings bear directly on the state-trait issue raised above. These findings showed that functionally effective changes in the PCT score – not just small changes within an innate range – could be generated by imposing the protocol. Even if it were the case that individuals are each born with an innate and characteristic asymmetry range, the fact that this range can be dramatically changed on a long-term basis with use of a clinically effective treatment protocol (including various components plus neurofeedback) suggests that the asymmetry score is not a rigid trait. However, we are *not* attempting to argue that once the neurofeedback protocol has been successfully applied and the PCT significantly changed, it cannot regress again, as illustrated in the next section.

Premenstrual Dysphoric Disorder (PMDD)

We have recently had the opportunity to study five cases of depressed women who were also suffering from *Premenstrual Dysphoric Disorder* (PMDD). This syndrome was first officially listed in DSM-IV (Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition 1994). A woman who has PMDD experiences *severe depression*, irritability and/or mood swings, which interfere with functioning. The symptoms begin 7–10 days prior to menstruation during what is called the luteal phase of the menses. Sometimes PMDD has been conceptualized as an extreme form of PMS (premenstrual syndrome), but it is known that the symptoms of PMDD are much more severe, and that PMDD has specific metabolic correlates (Endicott 2001). One of these women had been dose-stabilized on Zoloft, another on Wellbutrin, and a third on Prozac. Two others were untreated medically. All were run through the Alpha asymmetry training protocol. All these women initially presented with PCT scores well into the depressed range (<50), but as they were treated with neurofeed-

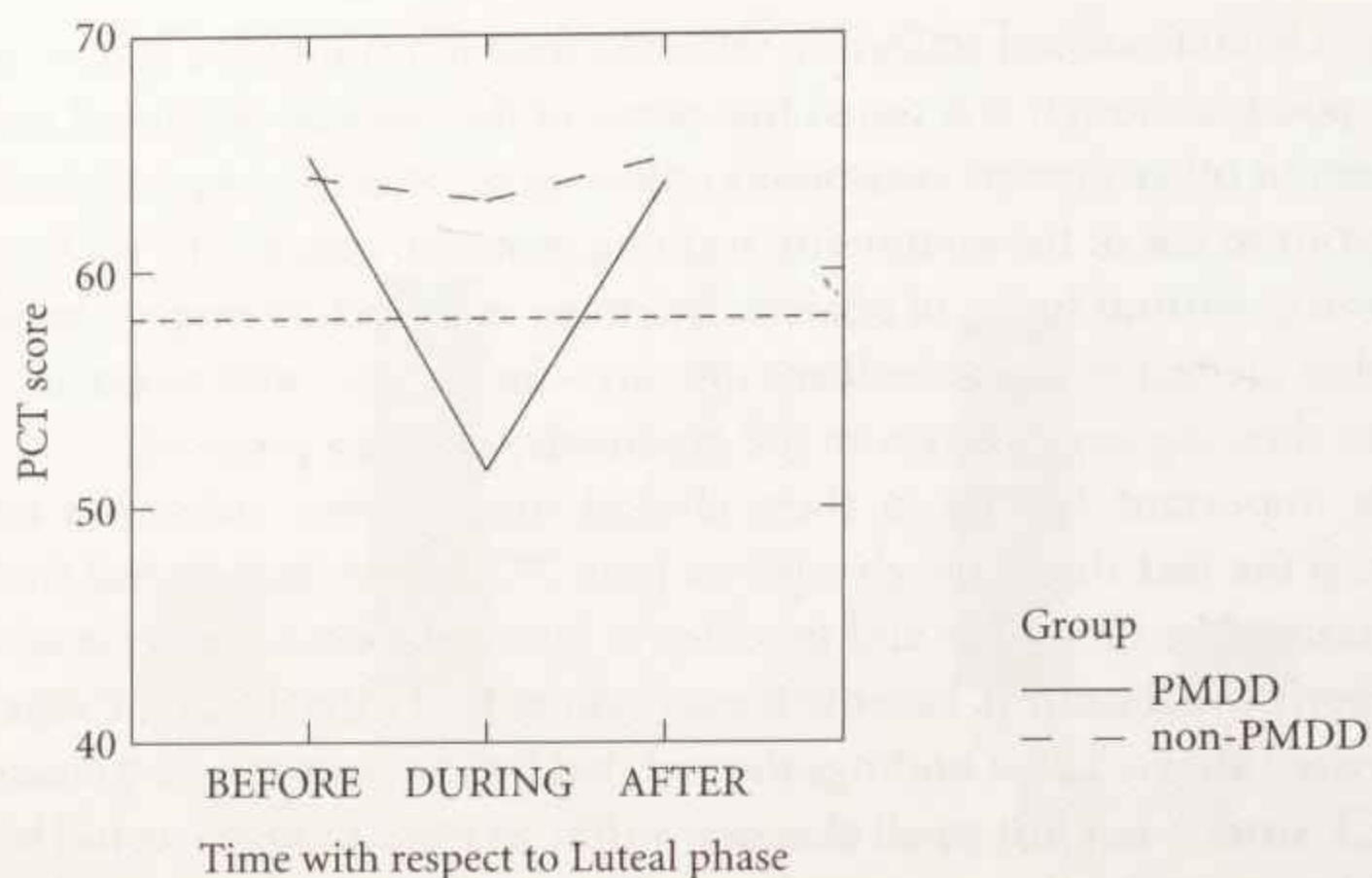


Figure 2. PCT scores of PMDD patient group and non-PMDD patient control groups before, during, and after the Luteal phases of their menstrual cycles.

back, they made significant gains, well out of the depressed range. They mostly remained in the non-depressed range and showed correspondingly normal affect, however during the luteal phase of their cycles, they each showed profound regressions as seen in Figure 2, in which a group of five demographically matched control cases (one on Zoloft, one on Hypericin, three unmedicated) are superimposed. These control cases also presented with depression, but were not diagnosed with PMDD. Only one cycle's worth of data is shown here, however data from second cycles are virtually identical. These data indicate two important points: (1) Although the alpha asymmetry training is effective at dramatically altering PCT scores and affect, these conditioned changes are not invulnerable to life's vicissitudes, such as severe PMDD luteal symptoms. (2) Aside from these relatively formal observations with PMDD cases, we have noted informally that previously depressed, non-PMDD patients in final successful phases of asymmetry training will show occasional regressions (in PCT and affect) always closely traceable to negative life events.

One concludes that the asymmetry protocol is a promising treatment modality for affective disorders, and that the measure itself (PCT) may be a useful diagnostic index for affect – it certainly discriminates PMDD cases and other women. The first conclusion must be tempered by our lack of systematic control data without which we do not know whether or not the therapeutic effects are specific to the asymmetry protocol. The second conclusion (about

diagnostics) must be tempered by our presentation of only five PMDD cases; given the rareness of PMDD it will take some time to gather more data. One additional conclusion which seems pretty safe to put forth is that our use of the asymmetry protocol has indicated that frontal cortical activation asymmetry is not simply a hard-wired, immutable trait, but is a reasonable state index and may be altered under operant control.

Summary

In this chapter, the term *Neurofeedback* was defined as a voluntary change in brain activity resulting from rewarding of target neural events, as in operant conditioning. It was shown with one example, the conditioning of evoked potential amplitude, that the conditioning is not trivially mediated. Then, via a literature review, it was shown that operant control of evoked potential amplitude in somatic sensory cortex produced changes in experimental pain threshold in animals. A clinical application of neurofeedback was then presented: the treatment of depression via the operant conditioning of left-right frontal cortical activation. Finally, it was noted that some sources of depression, such as the extremely negative affect and its EEG correlate experienced by women with premenstrual dysphoric disorder during the luteal phase of their menses, cannot be controlled either with neurofeedback and/or medication.

References

- Allen, J. B. & Cavendar, J. H. (1996). Biofeedback alters EEG asymmetry. *Psychophysiology*, 33, (suppl): S17 (abstract).
- Baehr, E., Rosenfeld, J. P., Baehr, R., & Earnest, C. (2002). Clinical use of an alpha asymmetry neurofeedback protocol in the treatment of mood disorders: Follow-up study one to five years post-treatment. *Journal Neurotherapy*.
- Baehr, E., Rosenfeld, J. P., et al. (1998). Comparison of two EEG asymmetry indices in depressed patients vs. normal controls. *Int. Journal Psychophysiol.*, 31, 89–93.
- Davidson, R. J. (1995). Cerebral asymmetry, emotion, and affective style. In R. J. Davidson & K. Hugdahl (Eds.), *Brain asymmetry*. Cambridge: MIT Press.
- Dowman, R. J., Rosenfeld, J. P., & Heinricher, M. (1983). Operant conditioning of trigeminally evoked cortical potentials: Correlated effects on facial nociception. *Brain Research*, 269, 111–118.
- Dowman, R. & Rosenfeld, J. P. (1985). Operant conditioning of somatosensory evoked potential (SEP) in rats. I. Specific changes in SEP amplitude and a naloxone-reversible, somatotopically specific change in facial nociception. *Brain Research*, 333, 201–212.

- Earnest, C. (1999). Single case study of EEG asymmetry biofeedback for depression: And independent replication in an adolescent. *Journal Neurotherapy*, 3, 28–35.
- Endicott, J. T. E. N. (2001). Diagnosis and psychopharmacologic treatment of premenstrual dysphoric disorder. *The Economics of Neuroscience*, 3 (6), 44–47.
- Fetz, E. E. & Finnochio, D. V. (1971). Operant conditioning of specific patterns of neural and muscular activity. *Science*, 174, 431–435.
- Fox, S. S. & Rudell, A. P. (1968). Operant controlled neural event: Formal and systematic approach to electrical coding of behavior in brain. *Science*, 162, 1299–1302.
- Gotlib, I. A., Ranganath, C., & Rosenfeld, J. P. (1998). Frontal EEG alpha asymmetry, depression, and cognitive functioning. *Cognition and Emotion*, 12, 449–478.
- Henriques, J. B. & Davidson, R. J. (1990). Regional brain electrical activities discriminate between previously depressed and healthy control subjects. *Journal of Abnormal Psychology*, 99, 22–31.
- Henriques, J. B. & Davidson, R. J. (1991). Left frontal hypoactivation in depression. *Journal of Abnormal Psychology*, 100, 535–545.
- Kamiya, J. (1969). Operant control of the EEG Alpha rhythm and some of its reported effects on consciousness. In C. Tart (Ed.), *Altered states of consciousness* (pp. 507–517). New York: John Wiley & Sons.
- Quinn, L. (1998). Hemispheric EEG asymmetry, emotion, and the nasal cycle. Unpublished PhD Thesis, California School of Professional Psychology, San Diego California.
- Rosenfeld, J. P. (1990). Applied psychophysiology and biofeedback of event-related potentials (brain waves): Historical perspective, review, future directions. *Biofeedback and Self-Regulation*, 15, 99–120.
- Rosenfeld, J. P. (2000). An EEG biofeedback protocol for affective disorders. *Clinical Electroencephalography*, 31, 7–12.
- Rosenfeld, J. P., Baehr, E., Baehr, R., Gotlib, I., & Ranganath, C. (1996). Daily changes in frontal alpha asymmetry correlate with changes in affect in therapy sessions. *Int. Journal Psychophysiology*, 23, 37–41.
- Rosenfeld, J. P., Cha, G., Blair, T., & Gotlib, I. (1995). Operant control of left-right frontal alpha power differences. *Biofeedback and Self-Regulation*, 20, 241–258.
- Rosenfeld, J. P., Hetzler, B. E., Birkel, P., Antoinetti, D., & Kowatch, R. (1976). Operant conditioned potentials, centrally evoked at random intervals. *Behavioral Biology*, 16, 305–317.
- Rosenfeld, J. P., Rudell, A. P., & Fox, S. S. (1969). Operant control of neural events in humans. *Science*, 165, 821–823.
- Rosenfeld, J. P., Silvia, R., Weitkunat, R., & Dowman, R. (1985). Operant control of human somatosensory evoked response alters experimental pain perception. *Advances in Neurology*, 9, 343–348 (Raven Press).
- Weitkunat, R. & Rosenfeld, J. P. (1986). Pain control by biofeedback of somatosensory evoked potentials. In Vinck et al. (Eds.), *Topics in behavioral medicine* (pp. 31–47). Berwyn: Swets North America Inc.

CHAPTER 9

Consciousness, emotional self-regulation, and the psychosomatic network

Relevance to oral biology and medicine

Francesco Chiappelli, Paolo Prolo, Elaina Cajulis,
Scott Harper, Elaine Sunga, and Edna Concepcion

Introduction: Consciousness and volitional control

From the viewpoint of philosophy, whence we should begin since it may be considered the master of all sciences (*scientia ancilla philosophiae*), the interpretation of consciousness has ranged from the “sense-certainty” of Heraclitus, to Plato’s myth of the Cave. Here, the “soul” was said to progress a self-edifying journey from an initial state similar to that of a prisoner trapped at the lowest level of knowing and being in a world of hellish visions of sheer illusion. By an act of volition, the “soul-prisoner” became free, and commenced the long and arduous ascent out of the cave (equivalent perhaps to today’s “emotional self-regulation” – *vide infra*). The “soul” eventually emerged into the clarity of the heavenly intelligible world - consciousness. In modern philosophy, Hegel described consciousness as rising through a series of historical levels. It preceded the view proposed by Kant, who revolutionized Modern philosophical thought by defending that it is the representation that makes the object possible, rather than the reverse. Kant argued that it is the consciousness of the object, rather than the object itself, that makes the representation of the object possible: that is, the human mind is the active originator of experience, through consciousness, not merely the passive recipient of perception. In the kantian view, perceptual inputs are processed, recognized, and integrated by the conscious mind, lest they be categorized as noise. Hence the twentieth century biologists, headed by Polany, described consciousness as a fundamental

set of phenomena by which information is processed by the central nervous system (CNS).

Contemporary scientific views of consciousness speak of a complex set of processes designed to combine information about attributes of the present multi-modal sensory environment with relevant elements of the past (John 2002). These processes are likely to be mediated by neural correlates/networks in the cortex and other areas of the CNS, and to be directed by changes in certain electromagnetic dynamics in those regions, which are, to some extent at least, under individual control or volition (Beauregard, Lévesque, & Bourgouin 2001). Consciousness relates, in today's view, to awareness of the world, feelings of control over one's behavior and mental state (i.e., volition and a sense of agency), and sustenance of self. Focal, or executive, consciousness serves to control details of awareness, and is closely associated with volitional acts; that is, the set of voluntary abilities that permits us to select among competing items, to correct error and to regulate emotions. Consciousness involves, and serves to regulate cognition and emotion.

Information is continuously fractionated into distinct features, which are processed locally by different brain regions relatively specialized for extracting these disparate components and globally by interactions among these regions. Information processing occurs at a certain level of synchronization within neuronal populations and of coherence among multiple brain regions. Therefore, significant random fluctuations and deviations, which would constitute local and global negative entropy, are minimized or prevented. The CNS transforms, more often than not under some degree at least of volitional control, these apparently asynchronous and mis-distributed neuronal discharges into seamless global subjective consciousness (John 2002). Whereas the act of volition plays a critical role in many – perhaps most – aspects of consciousness, a considerable amount of conscious processing occurs in the CNS without attention, a prerequisite of volitional consciousness.

A complete theory of consciousness must, therefore, explain why and how some cognitive and cerebral representations can be temporarily or permanently inaccessible to consciousness. It must discuss what is the range of possible conscious contents, how they map onto specific cerebral circuits, and whether or not generic neural processes underlie them. In view of the association of consciousness with cognition, memory, and emotions, including the regulation of emotions by the self, its neuro-anatomical localization has been proposed to include not only the prefrontal cortex, anterior cingulate and the areas that connect them, but limbic structures as well. Taken together, these lines of evidence converge to suggest an important role of conscious-

ness in the interaction between the psyche and the health – disease continuum. Such an important role must be taken into account in a well-founded theory of consciousness. That, in turn, should lead to a better understanding of the role of consciousness and of self-regulation of emotions in the modulation of the psycho-neuroendocrine-immune network (Grof 1998). The remainder of this chapter examines the intertwined roles of consciousness and self-regulation of emotions – in effect an aspect of psychological control – in psychosomatic disease.

The psychosomatic network: Psychoneuroimmunology

It was Descartes¹ who clearly distinguished the functions of the body (*res extensa*) from the functions of the mind (*res cogitans*). His principal contribution led to the characteristic reductionism of modern science. The cognitive schism that resulted led to the division of the fields we find in the Western medicine today.

The current area of “systems biology” attempts to override this dichotomy. Indeed, the modern concept of psychiatry as a distinct branch from neurology is relatively recent and dates from the late nineteenth century, when positivistic theories suggested that everything could be scientifically explained. Researchers devoted their interest to many diseases, whose clinical manifestations could be clearly identified, but whose etiology remained unknown. In the earlier part of the twentieth century, existentialism – whose major advocates were Brentano in the 1880’s and Heidegger in the 1900’s – and the concept of phenomenology – originated by Husserl in 1905 – sought to consider human awareness as accounts of the objective reality and the subjective response. These philosophical domains influenced early attempts to classify mental disorders. Kraepelin and his collaborators proposed a phenomenology that is still considered the basis for today’s manual of mental diseases, the diagnostic and statistical manual of mental disorders (DSM) of the American Psychiatric Association. It characteristically rests on a naturalistic description of symptoms, which has substantially evolved with the contributions of biological psychiatry.

Freud proposed a psycho-dynamic theory based on the *conscious*, the *sub-conscious*, and the *inconscious* (ego, super-ego and id). The model lacked a biological framework, and precluded the assignment of the patients into a classification system of symptoms and disease, but it rested on the notion that early life events influence consciousness, and therefore vulnerability to mental and physical illnesses. The division between neurology and psychiatry broadened,

with the latter drawn away from the biological underpinnings of medicine toward the social sciences. In more recent decades, however, the emergence of psychoneuroendocrinology, biological psychiatry and the neurosciences have witnessed the application and implications of molecular biology, proteonomics and genomics to psychiatry. This evolution now positions psychiatry in general, and psychosomatic medicine in particular as a new frontier in medicine, which may soon contribute to the elucidation, among other phenomena, of the fundamental molecular principles of Freud's consciousness (Schoore 1997).

A critical bridge in this evolution is undoubtedly the establishment of psychosomatic medicine, and the understanding of the role of socio-cognitive, psycho-emotional, physio-behavioral, and biological factors (i.e., cellular, biochemical, molecular) in human health and disease. In its present conception, psychosomatic medicine seeks to integrate these fundamental fields of science in order to obtain a fuller description of the biological substrates that promote health, or that lead to disease. The fundamental goal of psychosomatic medicine is to promote education and improved wellness. Techniques to observe and analyze normal human behavior, as well as psycho-pathological patterns and psychiatric states, and to design experimental and clinical protocols for manipulating and acting upon certain psycho-biological processes are proper to the study of psychosomatic medicine. In brief, this frame of reference acknowledges that all forms of human disease are related to alterations in the interaction among genetic, endocrine, nervous, immune and psychic (including consciousness, volitional control, cognitive and emotional) factors. The fundamental concepts, first championed by Speransky (cf., "neuro-dystrophic processes") (Speransky 1943) and Engle (Engle 1960), were later precisely outlined by Solomon (cf., "psychoneuroimmunology") (Solomon & Moos 1964; Solomon 1987).

The domain of inquiry of psychoneuroimmunology pertains to:

... the complex bidirectional interactions between the CNS (mediating both psychic and biologic processes) and the immune system (not only responsible for resistance to infectious diseases and cancer but also serving newly recognized bioregulatory functions)... the field reinforces the view that all disease is multifactorial and biopsychosocial in onset and course – the result of interrelationships among specific etiologic (e.g., bacteria, viruses, carcinogens), genetic, endocrine, nervous, immune, emotional, and behavioral factors. (Solomon 1987:1)

Recent thorough reviews of the field established the broad implications of psychoneuroimmunology for the basic biological sciences and medicine (Kiecolt-

Glaser, McGuire, Robles, & Glaser 2002; Eskandari & Sternberg 2002), as well as oral biology and medicine (Chiappelli, Abanomy, Hodgson, Mazey, Messadi, Mito, Nishimura, & Spigleman 2001).

Psychological strain and anxiety, as in dental anxiety, often arises as the real or perceived lack of fit of the person within the extrinsic (cf., "outer world of the individual") or intrinsic environment (cf., "the person's inner world") (Chiappelli et al. 2002). The perception, or consciousness of lack of fit is directly related to those acts under volitional control, which are directed at enhancing or establishing person-environment fit. From the psychosomatic perspective, the individual's perception is critical (Solomon 1987), and the perception of lack of fit is a significant psycho-emotional stress (Chiappelli et al. 2001).

Stress is a complex construct (Lazarus² 2000), which can be simply interpreted either as beneficial (i.e., *eustress*) or as potentially damaging or threatening (i.e., *distress*). The perception of fit of the person within the environment is dictated in part by the person's consciousness about social roles, which represent an intertwined set of perceived or actual rights and responsibilities, his/her personality, social involvement, cultural background, legal status, and hierarchical position. Individuals with different resilience to the demands of the environment, and the associated anxiety, have different inner motivational strengths (cf., volitional control) to overcome the strain of fit they may perceive or they may actually experience (i.e., consciousness) on a daily basis (i.e., daily hassles). The person's motivation rests on fundamental attributions (Weiner 1985):

- Locus: where does the cause of the lack of fit originate?
- Stability: how stable or unstable is the situation that brings about the lack of fit?
- Globality: is the lack of fit particular to the situation or generalizable to all situations?

In a similar context, Bandura's notion of "locus of control" (i.e., how much control is there on the part of the individual over the situation?) critically determines appraisal of fit, and the effect of stress on health via the immune system (Kaplan 1991). One's conscious awareness of control is a dominant aspect of psychosomatic medicine in general, and of psychoneuroimmunology in particular. Four domains of psychological control have been explicated, which can be noted as cognitive control (i.e., thinking and reflecting about the aversive event), informational control (i.e., learning and gathering information about the event), behavioral control (i.e., acting upon certain effects of the event),

and retrospective control (i.e., *post-facto* assessment and evaluation of the event and its consequences) (Taylor, Lichtman, & Wood 1984). It is possible and even probable that they represent specific constructs of the meta-construct of volitional control, which may independently impact upon health. Cognitive control appears to be the most effective predictor of psycho-emotional adjustment (cf., psychobiologic resiliency), particularly with respect to cancer of the breast (Taylor et al. 1984).

Undoubtedly, attributions and perceived responsibility, as manifestations of consciousness and volitional control, come to form the essential elements of an important model of psychological coping with stress (Chiappelli et al. 2001). Knowledge and appraisal of given situations, consciously or subconsciously, cognitively or instinctively, are additional critical elements of perceptions of fit, and of consequential emotions, psychological well-being, and the behavioral manifestations thereof.

Maldaptation of the person in the environment is often cause for psychological turmoil, with serious psychosomatic *sequelae*. Indeed, certain environmental factors have been identified since the time of Hippocrates as putative causative factors in the pathogenesis and in the exacerbation of a wide variety of diseases, from cardiovascular disease to psychiatric disorders. Immune surveillance processes, specifically the production of certain proinflammatory cytokines, can be directly stimulated by negative emotions and stressful experiences and indirectly stimulated by chronic or recurring infections. Accordingly, distress-related immune dysregulation may be one core mechanism behind a diverse set of health risks associated with negative emotions (Kiecolt-Glaser et al. 2002). Most immune effects are generally associated to the general expression of a certain affect (e.g., happiness, sadness, anger, anxiety), rather than the specific quality of any given mood state (e.g., clinical depression) (Futterman, Kemeny, Shapiro, & Fahey 1994).

Conversely, the perception of being well-adapted is beneficial to an individual's life in time (Cowen 1991). Psychosomatic wellness is the product of an active, conscious, volitional, and continuously evolving balance of psychobiological challenges: psychic, psycho-social and psycho-emotional triggers that together modulate physiological, cellular, and molecular responses, which involve the endocrine and the immune systems. Biological triggers on the other hand act to modulate CNS-mediated psychic response (i.e., cognitive and emotional). For example, consciousness about emotions and volitional control over emotional expression generates balance in the neuropeptide-receptor network and a functional healing system. Emotional expression is also a marker for psycho-spiritual vitalization, and further research should evaluate links be-

tween energy-based models of health and neuropeptide-receptor-based models under the rubric of an informational paradigm (Pert, Dreher, & Ruff 1998).

In brief, the spectrum of human emotions, from depression to joy and laughter, has been associated with significant changes in functional aspects of immune surveillance. Cognitive challenges, as well as awareness of self, of others, and of the surrounding environment at present and in the future also seem to impact upon cellular and humoral immunity. Relaxation and meditation, as well as cognitive-behavioral stress management and support groups, may beneficially impact neuroendocrine-mediated immune regulation in part by the biological mechanisms detailed below. This field is still characterized, however, by a dearth of controlled clinical trials and experimental research specifically directed at elucidating the effect of psychosocial interventions in a variety of conditions in medicine and dentistry (Kemeny & Gruenewald 2000). Evidence-based research (Chiappelli & Prolo 2002; Chiappelli, Prolo, Newman, Cruz, Sunga, Concepcion, & Edgerton 2003) must systematically address this domain of research to establish the best available evidence of the influence of emotions and emotional self-regulation on the modulation of immune function by the neuro-endocrine systems (Prolo, Chiappelli, Fiorucci, Dovio, Sartori, & Angeli 2002a).

Immune tissues are extensively innervated by the sympathetic nervous system. Both primary and secondary lymphoid organs receive such innervation, which enter the parenchyma of the tissue in association with the vasculature (arterial, venous, lymphatic), and which projects into the parenchyma to form synapse-like junctions with lymphoid and myeloid cells. This innervation is functional, and changes in immune responsiveness follow pharmacological manipulation of noradrenergic sympathetic or peptidergic nerves. The presence, distribution, and coexistence of various peptides, neuroendocrine markers and enzymes of the catecholamine pathway in nerves supplying lymphoid tissues in a variety of mammalian species confirm physiological relevance. Changes during development and aging in sympathetic innervation of lymphoid organs further suggest that the sympathetic nervous system and catecholaminergic peptide levels play a significant role in age-associated immune dysregulation. In brief, lymphoid organs and immune tissues receive innervation by fibers containing dopamine- β -hydroxylase and/or tyrosine hydroxylase, neural markers like protein gene product 9.5, synaptophysin, neurofilament, and a varied spectrum of peptides. The prominent peptides are tachykinins (substance P, neurokinin A), calcitonin gene-related peptide (CGRP), neuropeptide Y (NPY), and vasoactive intestinal polypeptide/peptide histidine isoleucine (VIP/PHI). Tachykinins and CGRP, and tyrosine hydrox-

ylase and NPY generally co-exist in the innervation patterns of the lymphoid organs. A minor proportion of fibers however shows co-existence of NPY and tachykinins, or of VIP/PHI and tachykinins. Opioid innervation of lymphoid organs is variable. Little is known of what directs the innervation to specific compartments within the lymphoid tissues, but classic receptor binding studies demonstrate a wide variety of target cells that possess β -adrenoceptors and receptors for neuropeptides and for a variety of hormones on cells of the immune system (Chiappelli et al. 2001).

The principal elements of the psychological and the biological framework of the psychosomatic network in general and of stress research in particular involve the cross-talk between the psychoneuroendocrine and the immune systems. For example, the hypothalamic-pituitary-adrenal (HPA) axis plays pivotal role in maintaining physiological homeostasis, including immune regulation, under both basal and challenge conditions. The HPA axis and the immune system participate in a key regulatory cascade, which constitutes the '*allostatic load*' (Solomon 1987; Chrousos & Gold 1992; McEwen 2000; Chiappelli et al. 2001; Kiecolt-Glaser et al. 2002; Chiappelli & Cajulis 2003).

Cytokines, a large family of polypeptide mediators classically associated with the regulation of immunity and inflammation, modulate CNS-driven and, more specifically, hypothalamus-originating responses (e.g., emotions, appetite, fever, endocrine regulation). The major proinflammatory cytokines, interleukin (IL)- 1β and tumor necrosis factor (TNF)- α , together with IL-6, regulate the "*sickness behavior*", as well as the activation of the HPA axis. The HPA response leads to a complex cascade of events that includes sympathetic-adrenal-medullary outcomes (i.e., the locus coeruleus/norepinephrine system), and that results in the production of pro-opiomelanocortin (POMC)-derived peptides (i.e., adrenocorticotrophic hormone [ACTH]), and cortisol. Feedback regulation dampens hypothalamic and pituitary output (Chiappelli et al. 2001). The HPA axis involves a network of central and peripheral components, consisting centrally of corticotropin releasing hormone (CRH) and urocortin (a CRH-related peptide), and peripherally of ACTH and cortisol.

The actions of CRH in the brain and in the periphery are mediated through a family of specific receptors, whose isoforms are encoded by two distinct genes (i.e., CRHR1 α &1 β , CRHR2 α , 2 β and 2 γ). In *situ* hybridization revealed CRH receptor type 1 in cells of the anterior pituitary, in the granular cell layer of the dentate gyrus, in the pyramidal cell region of the hippocampus, and in cells of the piriform and cerebral cortex areas. Biochemical data indicated that CRH receptor activation increases intracellular calcium and cAMP levels. Furthermore, the DNA cAMP responsive element and the calcium responsive element,

both variants of the basic palindromic motif TGACGTCA, was shown to be lacking from the promoter region of the POMC gene in the anterior pituitary (Wong, Licinio, Pasternak, & Gold 1994).

The cloning of CRH receptors and their localization in the brain have led to the development of receptor antagonists of potential use in the treatment of certain conditions (e.g., major depression). For instance, antalarmin is a novel class I CRH receptor antagonist that blunts the effect of CRH on pituitary ACTH, but it does not affect the adrenal response to an acute stressor (Wong, Webster, Spokes, Phu, Ehrhart-Bornstein, Bornstein, Park, Rice, Chrousos, Licinio, & Gold 1999). In addition, leptin, characterized less than a decade ago as the cytokine product of the *ob* gene (Zhang, Proenca, Maffei, Barone, Leopold, & Friedman 1994), is now known to provide information to the hypothalamus about energy homeostasis by translating the metabolic status of the peripheral adipocyte to the brain. Plasma levels of leptin are pulsatile and present diurnal variations that are inversely correlated with cortisol concentrations (Prolo, Wong, & Licinio 1998). Indeed, since leptin levels are highest between midnight and the early morning hours, and lowest noon to mid-afternoon, it is possible and even probable that the nocturnal rise in leptin is associated with the suppression of appetite during sleep. Leptin has not only an adipostatic function, but rather it appears to be a stress-related hormone, which might be important to survival. Moreover, whereas leptin concentrations and the HPA axis are closely linked together by an inverse relation, leptin is also a trophic factor for the reproductive system in terms of a dynamic relation between it and HP-gonadal axis hormones: leptin affects reproduction by acting directly at the dorsomedial and the ventromedial nuclei of the hypothalamus, at the pituitary gland, and at the ovary (Prolo et al. 1998).

Another CRH receptor ligand in mammals, urocortin, elicits a robust ACTH response after peripheral administration by interacting with both classes of CRH receptors. The actions of CRH are modulated by the CRH-binding protein, which serves as a decoy for bioactive CRH. CNS levels of CRH lead to a series of coordinated events that are psychobiologically adaptive to stress. They include the activation not only of the HPA axis, but also of the autonomic activity, which in turn leads to increased blood glucose levels, and increased heart rate and blood pressure. CRH is a biological modulator of arousal (i.e., consciousness), avoidance and anxiety, and acts to depress feeding behavior, sexual activity and sleep (Munck & Guyre 1986; Prolo et al. 1998; Chiappelli et al. 2001).

CRH secreted in the hypophyseal circulation binds to class I CRH receptors on cells of the anterior pituitary, and elicits the activation of transcription fac-

tors for the transcription of POMC mRNA. This gene is translated and spliced into several peptides, including ACTH and β -endorphin. POMC expression is under the regulatory control of arginine-vasopressin (AVP), which is released from the hypothalamus parvocellular division of the paraventricular nucleus, and from the magnocellular neurons of the supraoptic nucleus. Physiological regulation of the HPA axis depends upon the the CRH/AVP ratio (Scott & Dinan 1998).

Cortisol and other corticosteroid hormones act via intracellular receptors. The binding of the hormone to its receptor leads to a conformational change in the receptor and its dissociation from its binding protein. Translocation signals induce a dimerization of the receptor complex; the dimer enters the nucleus and binds to glucocorticoid-responsive elements on the chromatin DNA, regulating transcription. Type I and type II corticosteroid receptors have been cloned: type I receptors preferentially binds to aldosterone, and type II receptors preferentially bind to dexamethasone, a synthetic corticosteroid. Corticosterone downregulates type II receptors and upregulates type I receptors; by contrast, mineralocorticoids down regulate both types of receptors, while spironolactone, a type I receptor antagonist, has the opposite effect. That these effects on binding are accompanied by changes in mRNA levels suggests a role for steroids in the turnover rate of their own receptors in the physiological regulation of the HPA axis (Lupien & McEwen 1997).

In the CNS, type I glucocorticoid receptors are more widely expressed than type II receptors, which are primarily found in the paraventricular nucleus and other hypothalamic nuclei, as well as in brainstem monoaminergic nuclei. Glucocorticoid action has been extensively studied in the hippocampus, which expresses both Type I and Type II receptors, because it has an important role in memory, and putatively in consciousness. The physiological responses of hippocampal neurons to corticosteroid administration range from modulation of excitability to regulation of neurogenesis and programmed cell death in the dentate gyrus, and atrophy of the Hammon's horn (Lupien & McEwen 1997). The brain, and in particular the hippocampus shows signs of atrophy in response to elevated cortisol and traumatic stress (Sapolsky, Krey, McEwen, & Sapolsky 1986). Brain imaging techniques permit visualization of which structures are most affected. A modulatory role for glucocorticoids is also evident in the amygdala, which is involved in the emotionally influenced process of learning and memory. The basolateral and medial nuclei are implicated both in memory for emotional events and in the modulation of memory storage, which is modulated by glucocorticoids. Both Type I and Type II recep-

tors are present in the central and median nuclei of the amygdala (Lupien & McEwen 1997).

Immune activation causes production of nitric oxide (NO), which has a variety of effects on neuroendocrine regulation, particularly during the response to inflammatory stressors. For example, NO can modulate the release of CRH, and it can stimulate or inhibit specific brain functions. Indeed, the role of NO in brain repair is a highly promising area of active investigation at the present time (Merrill & Murphy 1997).

Exposure to an antigenic stimulation activates the production of pro-inflammatory cytokines (i.e., IL-1 β , IL-6, TNF- α), which in turn stimulate CRH secretion in the hypothalamus. CRH activates the HPA axis, whose final product, cortisol, suppresses the production of pro-inflammatory cytokines and of other cytokines engaged in T proliferation and activation (i.e., TH1 cytokines: IL-2, interferon[IFN]- γ). CRH also activates the sympathetic-adrenomedullary (SAM) system, which, by means of the sympathetic innervation of lymphoid organs, tends to subdue immune responses. SAM further modulates heart rate, blood pressure, and a variety of physiological responses. Nervous and immune systems share specific recognition molecules for signals that originate in both systems. The information substances are polypeptides and their receptors. They comprise the multi-directional information exchange network whereby the brain and the nervous system function, including mood and emotion, are integrated with the immune and the endocrine system. Disruption of any component of this circuit results in alteration in the susceptibility to inflammation and autoimmunity (Chiappelli et al. 2001).

Glucocorticoids not only turn off the immune system, but act as fine and powerful modulators of cellular immunity by specifically dampening pro-inflammatory and TH1 responses, and seemingly leaving unaltered TH2-type responses (i.e., antibody production) (Munck & Guyre 1986). Glucocorticoids also enhance and favor the expression of certain cytokine receptors (e.g., IL-1R, IL-2R, IL-4R, IL-6R) at the molecular level by means of glucocorticoid-induced expression of signaling molecules common to several cytokine receptors (e.g., gp130). Increased concentrations of the specific receptors contribute to containing the excess cytokines that they are in the process of suppressing. That is to say, glucocorticoids act in a very specific manner to induce a net cytokine imbalance, a shift in the TH1/TH2 ratio, and an enhancement, a synergism even at times of the effects of the remaining cytokines. Similar connections between other hormones (e.g., endogenous opioids, prolactin, melatonin, gonadal steroids) and immune-related events are becoming increasingly well characterized, and further support the notion that psychoneuroendocrine re-

sponses and immune responses are intertwined (Solomon 1987; Chiappelli et al. 2001; Kiecolt-Glaser et al. 2002).

Furthermore, the neuroendocrine system shows a circadian pattern dependent on the rest-activity/sleep-wake cycle. Immune effectors appear to be regulated in the same way. These temporal organizations have functional implications for the regulation of psychoneuroendocrine-immune events. For example, ACTH and cortisol levels correlate directly with the active period both in diurnal and in nocturnal animals. In contrast, cerebrospinal fluid levels of CRH and pro-inflammatory cytokines, which also presents diurnal variations, shows higher levels in the evening and lowest levels early in the morning that are the opposite of plasma cortisol levels (Prolo et al. 2002a). We, for the first time, observed distinct patterns of circadian fluxes by over a dozen cytokines (i.e., pro-inflammatory, TH1, TH2, chemokines) in whole saliva from normal healthy young adult men and women with no oral lesions. Administration of a stressful stimulus under experimental conditions (e.g., cold pressor test) led to significant changes in the levels of certain pro-inflammatory cytokines (e.g., IL-6), but not others at 30 min post-stimulus. We also were the first to note significant differences in salivary cytokines (e.g., TNF-alpha) and in the levels of Amyloid-beta in patients with aging-dementia of the Alzheimer's type, compared to age-matched control subjects (cf., Table 1) (Prolo, Chiappelli, Harper, Neagos, Fiala, Angeli, & Dovio 2003). In order to understand fully human neuroendocrine-immune functions, detailed circadian studies are required in the future with as many evaluation points as possible, under rigorous study conditions not only to examine plasma hormone concentrations, and, through the use of deconvolution methods, secretion rates, but also to assess sequence-dependent patterns, by assessment of parameters such as approximate entropy, which quantifies the orderliness of sequential measures, such as hormonal and cytokine time series.

Relevance to oral biology and medicine

The relevance of the psychosomatic network in general and of psychoneuroimmunology to oral biology and medicine is increasingly recognized and well accepted (Chiappelli et al. 2001). Psychoneuroendocrine and immune processes work in concert to insure the maintenance of health and to promote the healing of the hard (i.e., periodontal) and of the soft mucosa of the oral cavity (i.e., cheeks, lips, tongue, soft palate). Both mucosa are richly innervated by sympathetic fibers that travel with the vasculature from the stellate and the

Table 1. TNF- α and IL-6 Levels (in ng/ml) in Serum and Saliva in patients with probable Alzheimer's disease ($n = 15$), and age-matched control subjects

	TNF- α		IL-6	
	Controls	Patients	Controls	Patients
Serum	20.44 $\pm$ 3.04	27.71 $\pm$ 7.78*	0.41 $\pm$ 0.31	0.42 $\pm$ 0.40
Saliva	9.38 $\pm$ 3.81	18.14 $\pm$ 2.97*	1.98 $\pm$ 1.39	2.86 $\pm$ 2.64

(*: $p < 0.05$)

superior cervical ganglia. Both mucosa are also innervated by parasympathetic fibers that run intertwined with the cranial nerves that serve the oral cavity (e.g., trigeminal, lingual, hypoglossal). Lymphatics from the dental pulp direct lymphocytic migration to and from the pulp cavity as mediated by neuropeptides (Patel, Park, Lin, Chiappelli, & Huang 2003). The hard and soft mucosa are also richly supplied by the lymphatic system, and drain into the sub-mental and the sub-mandibular chain of lymph nodes, and thus into the deep crevical chains of nodes. Together, these channels permit, modulate and mediate extensive, directed and regulated migration of myeloid and lymphoid immune cells into and out of the oral mucosa. Saliva produced by the parotid, the sublingual and the other multiple minor salivary glands scattered throughout the oral cavity, contains significant levels of endocrine (e.g., cortisol) and immune products (e.g., cytokines). Whereas the assessment of cortisol in saliva was criticized in the past, it is now widely accepted. Salivary cortisol was rejected principally on the grounds that that it may fail to reflect endogenous circadian patterns, that it may not provide an adequate index of 24 hour cortisol, and that it may not manifest appropriate responses to hypothalamic-pituitary-adrenal tests. Further, it was argued that salivary collections may be contaminated with plasma from ulcers in the oral mucosa and gingival inflammation. Significant methodological developments have contributed to establish salivary levels of cortisol as a valid and reliable reflection of the unbound serum levels (Aardal-Eriksson, Karlberg, & Holm 1998). Our data show that a variety of cytokines can be reliably assessed in saliva from normal and from patient populations. For instance, we have shown that the mean tumor TNF- α levels, but not IL-6, are higher in serum and in saliva sample from patients with the Alzheimer's disease, compared to age-matched control subjects (Table 1).

Several pathologies of the soft and the hard oral mucosa are believed to have psychosomatic underpinnings. For example, oral lichen planus (OLP), a relatively common mucocutaneous immunological disease, results from an abnormal immune response of unknown cause, in which changes in the antigenicity of cell surface epithelial cells cause them to be recognized as foreign

Table 2. Clinical criteria for OLP diagnosis

Non-ulcerative lesions	
Reticular	lacelike keratotic patterns
Atrophic	lacelike keratotic patterns, and keratosis and erythema, few ulcerations
Ulcerative lesions	
Erosive	acelike keratotic patterns with ulcers, white patches (leukoplakia) and solid white lesions
Bullous	lacelike keratotic patterns with aggressive ulcers, leukoplakia and solid white lesions, small to large vesicles with thin walled <i>bullae</i>

(i.e., akin to an autoimmune disorder). The most common type of lesions in OLP is a reticular pattern of slightly raised, interlacing white keratotic streaks, termed Wickham’s striae. Lesions may also be painful and ulcerated. Indeed, lesions of OLP wax and wane over several decades. During the course of the disease, OLP lesions can manifest as reticular (i.e., lacelike keratotic patterns, “striae of Wickham”), atrophic (reticular keratosis and erythema, papules and plaques), erosive (atrophic mucosa with shallow ulcers), and bullous (large thin-walled bullae) (Table 2). OLP is observed predominantly in women at or beyond middle-age. Lesions are typically multiple and bilateral, primarily in the buccal aspect of the soft mucosa and in the sublingual regions. Lesions in the vestibular aspect of the lips, the hard and soft palate mucosa, and the alveolar ridge/gingiva are rarer. Patients experience significant discomfort, which can interfere with eating and lower quality of life (Prolo, Chiappelli, Cajulis, Bauer, Spackman, Romeo, Carrozzo, Gandolfo, & Christensen 2002b; Chiappelli & Cajulis 2003).

OLP lesions are invaded by lymphoid leukocytes that express the cluster of differentiation (CD) # 3, which recognizes T cells, and characteristically lacks CD20+ cells (B cells), immunoglobulin or complement. OLP lesions are identified by degeneration of the basal cell layer, and the presence of apoptotic basal keratinocytes. From a histopathological viewpoint, OLP lesions show degeneration of the basal cell layer and hyperkeratinization, dense subepithelial lymphocytic infiltrate, extensive epithelial basal cell destruction, and basement membrane branching and duplication. Apoptotic basal keratinocytes form homogenous eosinophilic colloid globules with traces of fragmented DNA. Microscopic clefts are evident between the epithelium and the connective tissue. Para-keratosis, epithelial hyper-keratosis, occasional slight acanthosis and basal epithelial cell destruction co-manifest with infiltration of mast cells and of

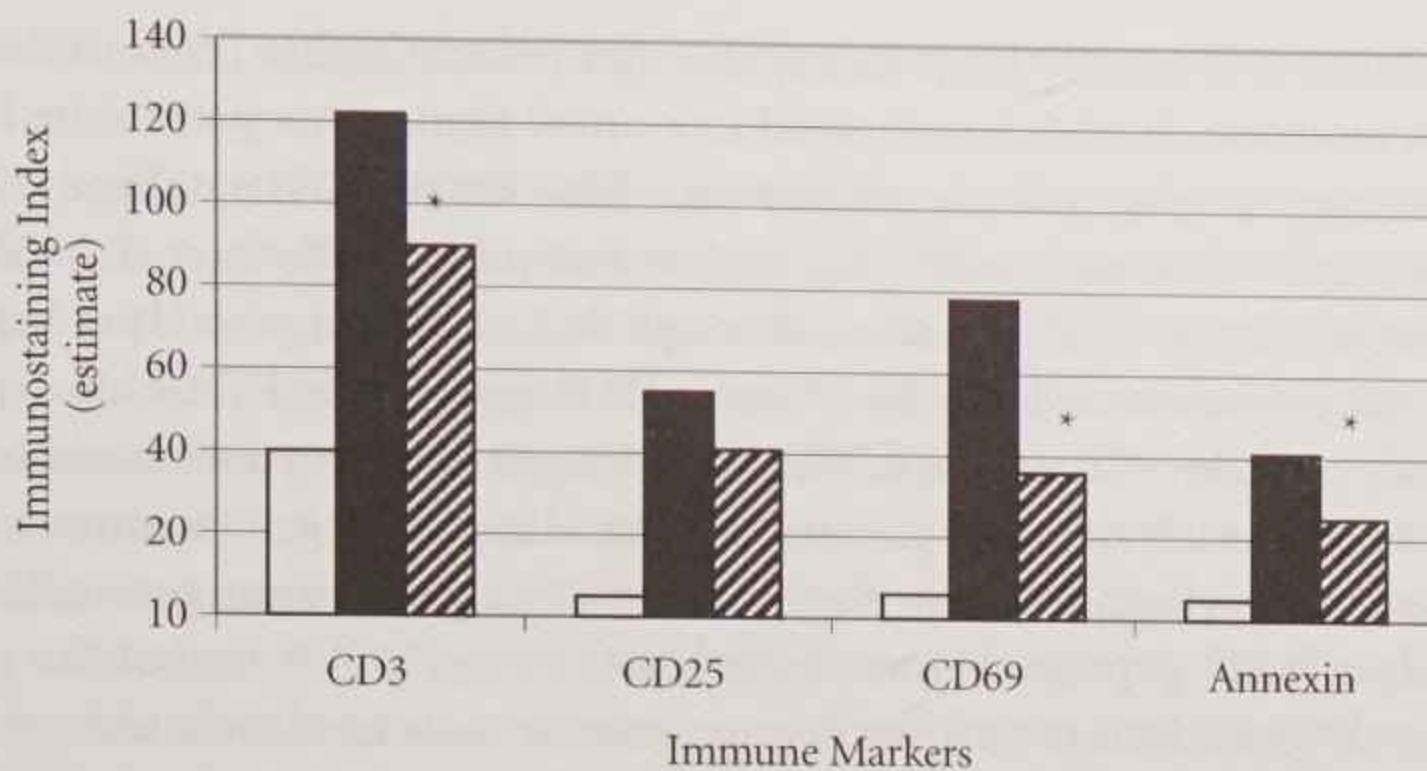


Figure 1. Comparative low magnification immunostaining of T cell markers and Annexin in normal and OLP tissue sections. Frozen sections of normal oral mucosa, erosive OLP lesions, and reticular OLP lesions were stained with anti-CD3 (A), anti-CD25 (B), anti-CD69 (C), and anti-Annexin (D). The sections were counterstained with methyl green. Estimate counts of cells that expressed definite membrane-associated positive staining were obtained, and the medians plotted, for the immune membrane markers CD3, CD25 and CD69. Estimate of the percentage of immunopositive cell material was obtained for Annexin, which does not immunostain discretely cell membranes, but presents a more diffuse immunostain on the cellular matrix (*: $p < 0.05$, X^2 test for comparisons of categorical data).

leukocytes in the submucosa, with greater direct mast cell-nerve interaction compared to normal tissue (Prolo et al. 2002b).

The invading population of CD3+ T cells is composed of CD4+ and of CD8+ subsets. CD4+ lymphocytes outnumber CD8+ T cells by up to three-fold. CD8+ T cells strategically line primarily the epithelial-mesenchymal interface. Both CD4+ and CD8+ lymphocytes that infiltrate OLP lesions express the memory phenotype (CD45RO+) that express the $\beta 1$ integrin, CD29+, and that are therefore essentially non-migratory. A large proportion, albeit not all T cells found in reticular or erosive OLP lesions, expresses markers of T cell activation. Clinically more severe erosive lesions may contain significantly more numerous activated lymphoid cells, compared to the symptomatically less severe reticular lesions (Fig. 1). The percent of peripheral blood CD4+CD45RO+ cells in patients with OLP is 66% of the circulating leukocyte population, compared to 49% in control subjects. The percent of naïve CD4 cells (CD4+CD45RA+) that infiltrate the OLP lesion epithelium is close to 25%, but are insignificant in normal oral soft mucosa. T lymphocytes and

Langerhans cells in OLP biopsies express significantly higher levels of the adhesion molecule, ICAM-1, compared to control tissue. One percent or fewer OLP lesional lymphocytes are positive for mRNA for the TH1 cytokine, IFN- γ , and that these cells are found lining the basal membrane (Prolo et al. 2002b).

The etiology of OLP is unclear, although there may be a genetic predisposition since expression of HLA-Bw57 favors OLP onset, while HLA-Dq1 expression may provide resistance to it. The initial trigger of OLP lesions could be the result of chemically-induced cellular trauma, allergens (e.g., cinnamon), mechanical trauma (e.g., Köebner phenomenon), or a putative antigen, including possibly a “self” peptide. Human hepatitis C virus (HCV), one of the principal pathogenic factors for liver disease, may be associated with leukoplakia, a putative precursor of the pathogenesis of OLP. A genetic predisposition for co-morbidity of HCV infection and OLP may be associated with the expression of the HLA-Dr6 allele. OLP lesions often exacerbate with pharmacological treatments with nonsteroidal anti-inflammatory drugs, anti-rheumatics, angiotensin-converting enzyme inhibitors, anti-hypertensives, antibiotics or β -blockers. OLP lesions exacerbation may also have psychosomatic underpinnings (Prolo et al. 2002b; Chiappelli & Cajulis 2003).

Lesions in OLP may have increased risk for malignant transformation. Leukoplakia may signify a non-specific early stage of OLP. As defined by the World Health Organization, leukoplakia is a predominantly white lesion of the oral mucosa that cannot be scraped off, and is not definable as any other disease or lesion. It is the most common premalignant lesion of the oral cavity. The pathogenesis of leukoplakia is unclear but its transformation to cancer may result from changes of apoptotic signal transduction proteins in epithelial cells (Klaassen and Braakhuis 2002).

The relationship between OLP and leukoplakia points to a common etiology, including tobacco use and abuse. However, while earlier evidence may have suggested an etiological role for smoking on the incidence of OLP, more recent evidence is less convincing, although a significant decrease in incidence of leukoplakia may follow tobacco cessation. Over 5% of precursor lesions of leukoplakias undergo malignant transformation. It is likely that the presence of bio-transformation enzymes in tobacco are responsible for the oral epithelium's susceptibility to carcinogens in tobacco. A proposed mechanism of action also includes metabolically activating or detoxifying tobacco agents and also opposing influences from oxidative stress. One study suggests several mechanisms by which smokeless tobacco usage contributes to oral cancer development, including metabolism of carcinogenic agents such as

N-nitroamines, and inhibition of p-53 mediated apoptosis (Banoczy, Glinter, & Dombi 2001).

Indeed, tobacco has profound and widely recognized effects upon the psychosomatic network in general and psychoneuroimmune surveillance in particular, both systemically and in the oral cavity. Suffice to recall that tobacco use significantly alters the psychoneuroendocrine and immune interaction and communication system outlined above. For example, whereas basal levels of plasma cortisol are similar in most instances for both smokers and nonsmokers, following smoking, the cortisol levels increase significantly among smokers only to levels that are significantly higher compared with the nonsmokers. Mean prolactin curves are higher among nonsmokers compared with smokers, but growth hormone levels are similar in both groups. Following dexamethasone administration (1 mg orally, 14 hours), smokers suppress, as non-smoker controls, plasma cortisol levels to undetectable levels. There are significantly more salivary IgA, IgA2 and J-chain in smokers, compared to nonsmokers, and levels of salivary lysozyme and lactoferrin are typically significantly lower in smokers (Gregory, Kindle, Hobbs, & Malmstrom 1991). Taken together, these data indicate that tobacco use and abuse can significantly alter the psychosomatic network. Oral manifestations of tobacco use, including leukoplakia, OLP and carcinoma, are significant health risks.

Conclusion

In conclusion, that any component of the psychosomatic network can be viewed as a biological system apart from interdependence from other physiological systems is now recognized as absurd: feedback loops exist within the immune system, and between the immune system and the rest of the body. These loops are essential for the survival of the organism, because they include direct communication of components of the immune and lymphoid system with the central and the peripheral nervous system, as well as communication and interdependence between endocrine and immune responses. This set of complex and fine interactions signifies the fundamental nature of psychosomatic cross-talk, starting from consciousness and volition, and leading to the physiological modulation of cellular immune surveillance events at the molecular level.

Recent progress in the fundamental cartography of the human psyche, of the nature and architecture of psychoneuroendocrine regulation, and of immune surveillance mechanisms lead to a better understanding of psycho-

somatic disorders. That, in turn, leads to a better understanding of the role of consciousness and of self-regulation of emotions in the modulation of the psycho-neuroendocrine-immune network (Grof 1998). This emerging understanding of psychoneuroendocrine-immune relationships that are fundamental to the psychosomatic network could lead to novel therapeutic models, designed to integrate a more comprehensive knowledge of the role of spirituality in health and disease, and of our conscious sense of reality in the process of healing, particularly with respect to oral manifestations of psychosomatic disturbances.

Summary

Psychosomatic medicine seeks to elucidate the fundamental role of psychosocio-cognitive and emotional factors in the modulation of biological responses that determine health and disease. In its present conception, the fundamental model of psychosomatic medicine is one of prevention aimed at promoting education and wellness.

In this chapter, we discuss the relationship between consciousness and volitional control, and psycho-emotional stress from the viewpoint of the fit of the person within the environment. We also explore the meaning of allostatic load in the context of psychoneuroimmune interactions.

Psychoneuroimmunology is the scientific pursuit of the relations between psycho-physiological and immuno-physiological processes and mechanisms. The spectrum of human emotions, from stress to depressed moods and major depression, to joy and laughter, are associated with significant changes in neuroendocrine-mediated immune regulation of functional aspects of immune surveillance. Consciousness of self, of others, and of the surrounding environment, as well as volitional control impact upon cellular and humoral immunity. Relaxation, meditation, and cognitive-behavioral stress management also impact beneficially upon psychoneuro-immune regulation.

The complex inter-relationship between the psychoneuroendocrine and the immune system is important for the maintenance of health systemically, as well as locally. In this chapter, we provide examples of psychoneuroendocrine-immune regulation in the oral cavity. In particular, we examine the case of Oral Lichen Planus, a disease of the soft mucosa. This condition, first described by E. Wilson in 1859, is a T-cell-mediated inflammatory disease whose lesions lack B cells, NK cells, plasma cells, or immunoglobulin. We established a clear relationship between changes in hypothalamic-pituitary-adrenal regu-

lation, systemic markers of cellular immunity and mood states, with clinical stages of this condition (i.e., atrophic vs. erosive vs. bullous lesions).

The field of psychosomatic medicine in general and or psychoneuroimmunology in particular is characterized by a dearth of controlled clinical trials and experimental research aimed at elucidating the effect of psychosocial interventions in a variety of conditions in oral biology and medicine. We propose that evidence-based research must systematically examine the pertinent literature to establish the best available evidence with respect to the influence of consciousness, emotions and emotional self-regulation on the modulation of psychoneuroendocrine-immune interactions.

Acknowledgements

The authors wish to thank the Diagnostic Product Co., the Amgen Co., the John D. and Catherine T. MacArthur Foundation, the Kettering Foundation, the Harbor-UCLA Research & Education Institute, the UCLA Norman Cousins Program on Psychoneuroimmunology, the UCLA Center for the Interdisciplinary Study of Immunological Diseases, the UCLA School of Dentistry, and the National Institutes of Health (AI 07126; CA16042, DA07683, DA10442) for funding and supporting various aspects of the research discussed here. The authors also wish to thank the students and the collaborators who have contributed to this body of research throughout the years.

Notes

1. Renatus Cartesius (aka, Descartes) French philosopher and scientist (31 March 1596 – 11 February 1650) "*Discours de la méthode*" (1637).
2. Richard S. Lazarus, a major force in contemporary stress and psychosomatic research, passed away on 24 November 2002 at the age of 80. A professor at UC Berkeley for close to five decades, he was world-renown for his work on stress as "*daily hassles*". He often quoted Hamlet: "... for there is nothing either good or bad, but thinking makes it so..."

References

- Aardal-Eriksson, E., Karlberg, B. E., & Holm, A. C. (1998). Salivary cortisol—an alternative to serum cortisol determinations in dynamic function tests. *Clin. Chem. Lab. Med.*, 36 (4), 215–222.

- Banoczy, J., Gliner, Z., & Dombi, C. (2001). Tobacco use and oral leukoplakia. *Journal Dent. Educ.*, 65, 322–327.
- Beauregard, M., Levesque, J., & Bourgouin, P. (2001). Neural correlates of conscious self-regulation of emotion. *Journal Neuroscience*, 21, 1–6.
- Chiappelli, F., Abanomy, A., Hodgson, D., Mazey, K. A., Messadi, D. V., Mito, R. S., Nishimura, I., & Spigleman, I. (2001). Clinical, experimental and translational psychoneuroimmunology research models in oral biology and medicine. In Robert Ader, Robert Cohen, & David Felten (Eds.), *Psychoneuroimmunology, III*, Chapter 64 (pp. 645–670). Academic Press.
- Chiappelli, F., Prolo, P., Newman, M., Cruz, M., Sunga, E., Concepcion, E., & Edgerton, M. (2003). Evidence-based practice in dentistry: Benefit or hindrance. *Journal Dent. Res.*, 82, 6–7.
- Chiappelli, F. & Prolo, P. (2002). Evidence-based dentistry for the 21st century. *Gen Dent.*, 50, 270–273.
- Chiappelli, F. & Cajulis, O. S. (2003). Psychological views on “stress-related oral ulcers”. *Quintessence*.
- Chrousos, G. P. & Gold, P. W. (1992). The concepts of stress and stress system disorders. Overview of physical and behavioral homeostasis. *JAMA*, 4, 267 (9), 1244–1252.
- Cowen, E. L. (1991). In pursuit of wellness. *Am Psychol*, 46, 404–408.
- Engle, G. L. (1960). A unified concept of health and disease. *Persp Biol Medicine*, 3, 459–485.
- Eskandari, F. & Sternberg, E. M. (2002). Neural-immune interactions in health and disease. *Ann. NY Acad. Sci.*, 966, 20–27.
- Futterman, A. D., Kemeny, M. E., Shapiro, D., & Fahey, J. L. (1994). Immunological and physiological changes associated with induced positive and negative moods. *Psychosom Medicine*, 56, 499–511.
- Gregory, R. L., Kindler, J. C., Hobbs, L. C., & Malmstrom, H. S. (1991). Effect of smokeless tobacco use in humans on mucosal immune factors. *Arch. Oral Biol.*, 36, 25–31.
- Grof, S. (1998). Human nature and the nature of reality: conceptual challenges from consciousness research. *J. Psychoact. Drugs*, 30, 343–357.
- Gross, J. J. & Levenson, R. W. (1993). Emotional suppression: Physiology, self-report, and expressive behavior. *Journal Person. Social. Psychol.*, 64, 970–986.
- John, E. R. (2002). The neurophysics of consciousness. *Brain Res. Rev.*, 39, 1–28.
- Kaplan, H. B. (1991). Social psychology of the immune system: A conceptual framework and review of the literature. *Soc. Sci. Med.* 33 (8), 909–923.
- Kemeny, M. E. & Gruenewald, T. L. (2000). Affect, cognition, the immune system and health. *Prog. Brain. Res.*, 122, 291–308.
- Kiecolt-Glaser, J. K., McGuire, L., Robles, T. F., & Glaser, R. (2002). Psychoneuroimmunology and psychosomatic medicine: Back to the future. *Psychosom. Medicine*, 64, 15–28.
- Klaassen, I. & Braakhuis, B. J. (2002). Anticancer activity and mechanism of action of retinoids in oral and pharyngeal cancer. *Oral Oncol.*, 38, 532–542.
- Lazarus, R. S. (2000). Toward better research on stress and coping. *Am. Psychol.*, 55 (6), 665–673.
- Lupien, S. J. & McEwen, B. S. (1997). The acute effects of corticosteroids on cognition: integration of animal and human model studies. *Brain Res. Brain Res. Rev.*, 24, 1–27.

- McEwen, B. S. (2000). Allostasis and allostatic load: Implications for neuropsychopharmacology. *Neuropsychopharmacol.*, 22, 108–124.
- Merrill, J. E. & Murphy, S. P. (1997). Regulation of gene expression in the nervous system by reactive oxygen and nitrogen species. *Metab. Brain. Dis.*, 12, 97–112.
- Munck, A. & Guyre, P. M. (1986). Glucocorticoid physiology, pharmacology and stress. *Adv. Exp. Med. Biol.*, 196, 81–96.
- Pert, C. B., Dreher, H. E., & Ruff, M. R. (1998). The psychosomatic network: foundations of mind-body medicine. *Alt. Ther. Health Medicine*, 4, 30–41.
- Patel, T., Park, S. H., Lin, L. M, Chiappelli, F., & Huang, G. T. J. (2003). Subtsance P, not calcitonin gene-related peptide induces interleukin-8 by cells from the human dental pulp. *Oral Surg, Oral Med, Oral Pathol, Oral Radiol Endod.* In press.
- Prolo, P., Wong, M.-L., & Licinio, J. (1998). Molecules in focus: Leptin. *Int. J. Biochem. Cell. Biol.*, 30 (12), 1285–1290.
- Prolo, P., Chiappelli, F., Fiorucci, A., Dovio, A., Sartori, M. L., & Angeli, A. (2002a). Psychoneuroimmunology: New avenues of research for the 21st Century. *Ann. NY. Acad. Scien.*, 966, 400–408.
- Prolo, P., Chiappelli, F., Cajulis, E., Bauer, J., Spackman, S., Romeo, H., Carrozzo, M., Gandolfo, S., & Christensen, R. (2002b). Psychoneuroimmunology in Oral Biology and Medicine: The Model of Oral Lichen Planus. *Ann. NY. Acad. Scien.*, 966, 429–440.
- Prolo, P., Chiappelli, F., Harper, S. R., Neagos, N., Fiala, M., Angeli, A., & Dovio, A. (2003). Profile of cytokines and Amyloid-beta in saliva: Putative markers of probable Alzheimer's Disease. *Brain Aging*.
- Sapolsky, R. M., Krey, L. C., & McEwen, B. S. (1986). The neuroendocrinology of stress and aging: The glucocorticoid cascade hypothesis. *Endocr. Rev.*, 7, 284–301.
- Scott, L. V. & Dinan, T. G. (1998). Vasopressin and the regulation of hypothalamic-pituitary-adrenal axis function: Implications for the pathophysiology of depression. *Life Sci.*, 62, 1985–1998.
- Schore, A. N. (1997). A century after Freud's project: Is a rapprochement between psychoanalysis and neurobiology at hand? *Journal Am. Psychoanal. Assoc.*, 45 (3), 807–840.
- Speransky, A. D. (1943). *Basis for theory in medicine*. International Publisher, New York.
- Solomon, G. F. (1987). Psychoneuroimmunology: Interactions between central nervous system and immune system. *J. Neuroscien. Res.*, 18, 1–9.
- Solomon, G. F. & Moos, R. H. (1964). Emotions, immunity, and disease. *Arch. Gen. Psychiat.*, 11, 657–674.
- Taylor, Sh. E., Lichtman, R. R., & Wood, J. V. (1984). Attributions, beliefs about control, and adjustment to breast cancer. *J. Pers. Social. Psychol.*, 46, 489–502.
- Weiner, B. (1985). An attributional theory of achievement motivation and emotion. *Psychol. Rev.*, 92 (4), 548–573.
- Wong, M.-L., Licinio J., Pasternak, K. I. , & Gold, P. W. (1994). Localization of corticotropin-releasing hormone (CRH) receptor mRNA in adult rat brain by in situ hybridization histochemistry. *Endocrinol.*, 135, 2275–2278.

- Wong, M.-L., Webster, E. L., Spokes, H., Phu, P., Ehrhart-Bornstein, M., Bornstein, S., Park, C.-S., Rice, K. C., Chrousos, G. P., Licinio, J., & Gold, P. W. (1999). Chronic administration of the non-peptide CRH type 1 receptor antagonist Antalarmin does not blunt hypothalamic-pituitary-adrenal axis responses to acute immobilization stress. *Life Sci.*, 65 (4), PL53-8.
- Zhang, Y., Proenca, R., Maffei, M., Barone, M., Leopold, L., & Friedman, J. M. (1994). Positional cloning of the mouse obese gene and its human homologue. *Nature*, 372, 425-432.

Name index

A

Aksan N. 47
Alain C. 136
Anderson S. W. 172
Arnstein A. F. 108

B

Bachorowski J.-A. 20, 23
Baehr E. 245–248
Bandura A. 183
Baron R.M. 49
Barrett L. F. 92, 93
Bates A. T. 136, 137
Baumeister R. F. 12
Beauregard M. 109, 138, 163,
166–168, 170, 172, 175, 185, 195,
196, 198–200, 232, 254
Bechara A. 101
Blakemore S. J. 101
Blatt S. J. 93
Bohr N. 211, 212, 216
Bonnano G. A. 2, 7–9, 11, 14–16, 18,
20, 24
Braver T. S. 134, 135
Brazhnik E. S. 145
Bush G. 150
Buzsáki G. 142, 144

C

Calkins S. D. 35–39, 41, 42, 44–48,
50–52, 67, 68
Camras L. A. 72
Canon W. B. 124, 125
Carter C. S. 133, 134
Carver C. S. 10, 11

Casey B. J. 177
Casey R. 69
Cassidy J. 36
Chess S. 42, 43
Chiappelli F. 257–260, 263–266, 268
Claparede E. 88

D

Damasio A. 100–102, 106, 110, 171
Davidson R. J. 244–246
Dias R. 170
Dikman Z. V. 138

E

Einstein A. 206, 207
Eisenberg N. 37, 51, 65, 66, 68
Ekman P. 3, 6, 94
Elliot R. 151

F

Falkenstein M. 129–131, 145
Farthing G. W. 100
Fellons J. M. 104
Fodor J. A. 102
Fox N. A. 37, 50, 67, 174, 175
Fox S. S. 239, 241
Frijda N. 7, 8, 99, 165
Frith C. D. 99, 135, 136, 150, 170

G

Gabriel M. 140–142, 145, 151
Garavan H. 134
Gehring W. J. 129, 130, 132, 136,
151
Gemba H. 129

Gevins A. S. 133
Gilliom M. 48
Greenberg M. A. 19, 21, 110
Gross J. J. 3, 7, 16, 17, 19, 24, 74, 77,
164, 165, 176, 201

H

Hajcak G. 137
Hariri A. 164, 167, 178
Heisenberg W. 214–216, 218, 231
Holroyd C. B. 145
Horgan T. E. 202

I

Izard C. E. 2, 5–8, 76

J

James W. 207, 226–228
Johannes S. 136, 138
Johnson S. 99

K

Kahana M. J. 143
Kaszniak A. 94, 95
Kennedy-Moore E. 18
Kenney D. A. 49
Khanna S. 146, 147
Kiehl K. A. 134, 138
Kopp C. 36, 40, 41, 49, 50, 130, 131,
135, 174

L

Lane R. D. 88, 90–95, 97, 98,
108–110, 112
Larsen R. J. 10
Lavadas E. 102, 105
LeDoux J. E. 103, 104
Lévesque J. 101, 168–170, 198, 200,
254
Lewis M. 173
Linehan M. M. 75, 78
Loevinger J. 93
Luu P. 131–133, 138–140, 142–145,
151, 163

M

MacDonald A. W. 128
MacLean P. D. 104
Mangelsdorf S. 41
Mathalon D. H. 135, 136
Maxwell J. C. 206
Menson V. 134
Mishkin M. 140, 142
Misra B. 224
Morgan R. E. 145, 146

N

Nauta W. J. H. 124, 148, 152, 163
Newton I. 203, 206, 207

O

Ochsner K. N. 164, 178–181, 186,
198, 200, 230, 232
Öhman A. 103
Owren M. J. 20, 23

P

Paquette V. xi, 163, 184, 195, 198,
202
Paré D. 142, 143
Parkinson B. 23
Piaget J. 90, 93
Porges S. W. 37, 50
Posner M. I. 175
Pribram K. H. 124, 125, 127, 148,
152, 163

R

Rainville P. 97, 111
Rau J. 94
Rosenfeld J. P. 241–247
Roskilde C. E. 129
Rothbart M. K. 40, 41, 43, 44, 49,
50, 66, 175
Rothman T. 225
Russell J. A. 8

S

- Safran J. D. 19
Schaefer S. M. 164, 177, 181
Scheier M. F. 10, 11
Schwartz J. M. xi, 1, 14, 15, 74,
90–95, 176, 184, 195, 196, 198,
200–202, 205, 232, 236
Shergill S. S. 112
Shima K. 150
Shipman K. 65, 71–73, 76
Skinner B. F. 239
Sperry R. 208
Stapp H. P. 228, 229
Stemmer B. 131
Stifter C. A. 46
Sudarshan E. 224, 225
Swick D. 133

T

- Thomas A. 42, 43
Thompson R. A. 36, 51, 75, 165
Thorpe S. J. 129

Tomkins S. S. 6

Tucker D. M. 132, 149–151, 163 (*see*
also Luu)

Turken A. 133

U

Ullsperger M. 134

Vanderwolf C. H. 146

Vogeley K. 99

Von Neumann J. xi, 212, 213, 217,
220, 223, 228, 231, 233

W

Wan R.-Q. 147

Watanabe K. 128

Watson J. C. 18

Winnicott D. W. 22

Wittgenstein L. 195, 205

Y

Yakovlev P. I. 175

Subject index

A

Aggression 6, 38, 45, 66, 185

cultural differences 185

Amygdala 99, 101–105, 109, 114,
115, 140, 143, 144, 147–149,
152, 163, 167–171, 175–181,
199, 232, 262, 263

arousal 104, 199

aversive stimuli 105

basal ganglia and dopamine
148

basolateral nucleus 262

central nucleus 263

discriminative avoidance learning
140, 147

emotional self-regulation 109,
163, 167–171, 175–181, 199,
232

fear 103

implicit memory 102

limbic-neocortical interaction
104

medial nucleus 262, 263

memory 140, 262

motivation and motor control
148

neocortical-amygdala pathway
103

Post-traumatic stress disorder
(PTSD) 114

thalamo-amygdala pathway
103

theta rhythm 142–144

Anterior cingulate cortex 96–101,
106–111, 113, 114, 137, 138,

140, 141, 144, 149, 152, 163,
167–169, 172, 173, 175, 176,
178, 181, 186, 254

affect and attention 138

attention 96–98, 100

autonomic control 96, 107, 168

background feelings 106

cognitive division vs. affective
division 111

conscious experience of emotion
97, 98

depression 113

detection of emotional signals
169

discriminative approach learning
140, 141

discriminative avoidance learning
140, 141, 144

emotional self-regulation 109,
138, 163, 167–169, 172, 173,
175, 176, 178, 181, 186, 254

heart rate variability (HRV)
108

maternal behavior 96

motor responses 97

omission of reward 149

pain 96, 111, 137, 138, 152

phenomenal awareness 106

Post-traumatic stress disorder
(PTSD) 114

reflective awareness 106

response selection 96

sadness 101, 111

stress 149

visceral regulation 101

vocalization 96

- Anger 6, 18, 48, 65–67, 69–71, 76, 258
 emotion dysregulation 65–67, 69–71, 76
 temperament 48
 Anxiety 63, 64, 70, 139, 185, 202, 257, 258, 261
 children 63, 64, 70
 dental anxiety 257
 Arousal 37, 38, 41, 62, 64, 65, 75, 76, 143, 261
 Attributional processes 64
- B**
- Basal ganglia 105
 emotional gestures and expression 105
 Behaviorism 123, 236, 237
 classical and operant conditioning 123
 learning 123
 motivation 123
 reward 123
 Brainstem 105, 149
 automatic generation of emotional responses 105
 dopamine 145
- C**
- Cancer 74, 256, 258, 269
 breast cancer 258
 carcinoma 269
 Caregiving 36, 37, 39–42, 47, 51, 75
 early experiences 36
 emotions 37
 emotional self-regulation 40–42
 parents 39
 unresponsive caregivers 75
 Central nervous system (CNS) 196, 254, 256, 258, 260
 hypothalamic functions 260
 information processing 254
 immune system 256
 Cerebellum 99, 102, 183
 implicit memory 102
 Cerebral cortex 100, 260
 Cingulate motor area 150
 reward 150
 Classical physics 196, 202–211, 213, 214, 217–219, 222, 232, 236–238
 conscious mental experiences 205, 237
 consciousness 206–209
 determinism 206
 eliminativism 237
 functionalism 237
 identity theory 208, 209, 218, 237
 materialism 202, 203, 230, 236–238
 mind-brain question 196, 236–238
 philosophical foundations of neuroscience 202, 203
 Cocaine 145 146 147
 mood 147
 Cognition 5, 6, 40, 88, 89, 147, 254
 emotion 6, 88, 89, 147, 254
 emotional self-regulation 40
 Cognitive neuroscience 87, 123, 152, 153, 236
 Cognitive psychology 123, 126, 152
 action monitoring 126
 attention 123
 executive functions 126, 152
 information processing 123
 memory 123
 perception 123
 Conscious awareness 89
 Conscious mind 253
 Consciousness 75, 166–168, 170, 200, 206, 209, 217, 218, 220, 226, 227, 229, 232, 233, 253–258
 cognition 254
 emotion 254, 256
 executive consciousness 254
 health-disease continuum 255

memory 254
 phenomenology 229, 255
 psycho-dynamic theory 255
 theory 254
 volition 75, 166–168, 170, 200,
 227, 232, 253, 254, 256–258

D

Dementia of the Alzheimer's type
 (DAT) 264
 Amyloid-beta 264
 cytokines 264
 Depression 69, 70, 74, 76, 113–115,
 139, 202, 244–249, 258, 259,
 261, 270
 adulthood 74, 76
 childhood 69, 70
 cognitive-behavioral therapy
 (CBT) 114
 EEG cortical asymmetry
 244–249
 psychotherapy 247–249
 selective serotonin reuptake
 inhibitor (SSRI) 114
 Development 23, 35–52, 87, 91, 93,
 173, 174
 attention 43
 cognitive component 36
 cognitive development 87, 91,
 93, 174
 emotional self-regulation 23,
 35–52, 173, 174
 intentionality 174
 motor component 36, 41
 physiological component 36
 self-consciousness 173, 174
 social development 174
 Diseases 255, 258
 cardiovascular diseases 258
 mental 255
 physical 255
 proinflammatory cytokines 258
 psychiatric disorders 258
 Disgust 96

Distal behavioral indices and effects
 44, 47, 48, 52
 Distress 40, 41, 44, 46, 49, 64, 66,
 74–77, 109, 174, 257
 infant 40, 41, 174
 toddlerhood 46

E

Electroencephalogram (EEG) 44,
 133, 143, 174
 Emotion 1–12, 15–20, 22, 35–37, 38,
 40–42, 44–47, 49–52, 61–66,
 70–77, 87, 89, 91–95, 97,
 100, 101, 104, 106, 110, 111,
 138, 147, 164, 165, 170, 171,
 174, 244, 246, 261
 adaptation 1, 8, 9, 15, 16, 38,
 42, 47, 49, 51, 52, 61, 63, 70,
 75–77, 164, 171,
 affective style 246
 appraisal 4, 6, 7, 15, 179
 autonomic nervous system 3, 5,
 8, 15, 37, 46, 50, 87, 89, 261
 biological view 3, 5–8
 blunted affect 147
 bodily state 101, 110
 components 3, 9, 38, 76, 164
 decision-making 1, 4
 definition 1, 8, 164
 discrete emotions 7, 8
 drive states 6
 emotional awareness 90–94,
 103, 104
 environment 2, 35, 38, 41, 42,
 45, 49, 64, 73, 75, 89, 171
 evaluations 89
 expressivity 65, 75
 facial expressions 5, 6
 feelings 87, 89, 91, 92, 94, 95,
 97, 100, 101, 106, 111, 115
 generation 3, 5, 104, 110, 179
 goals 1, 2, 10–12, 18, 22, 61–63,
 75, 89, 93
 intentions 1
 Izard's multi-system model 5–8

- learning 1
- motivation 6, 7
- needs 1
- negative emotions 17–19, 36, 38, 40, 41, 44–46, 63, 65, 66, 71–73, 75, 138, 139, 164, 165, 170, 174, 244,
- non-linear dynamic systems 8
- personality 15, 16
- positive emotions 36, 51, 63, 66, 74, 75, 165, 244
- primary vs. secondary 19
- survival value 20
- reason 1
- social view 3
- suffering 164
- valence 3
- Emotional Stroop Task 101, 112, 114
- Emotion dysregulation 35, 61, 63, 65–78, 88, 113–115
 - adulthood 73–75
 - Attention Deficit/Hyperactivity Disorder (ADHD) 69
 - attentional control 65, 66, 76
 - avoidance 73, 261
 - blunted affect 63
 - Borderline Personality Disorder (BPD) 75, 77
 - childhood 65–73, 76–78
 - child maltreatment 71, 72, 76
 - coping 65, 66, 71, 73, 74, 76, 77
 - cortisol 67
 - deficits 67, 69, 76, 78
 - Dialectical Behavior Therapy (DBT) 78
 - emotional lability 63, 71, 72, 75
 - environmental stressors 70,
 - externalizing problems 65, 66, 68
 - intense emotional experiences 68
 - internalizing problems 65, 66, 68
 - interpersonal difficulties 63, 70, 71
 - negative thoughts 73, 74, 77
 - Oppositional Defiant Disorder (ODD) 69
 - parental influence 68, 71, 72
 - psychological interventions 78
 - psychopathology 35, 61, 63, 65, 71, 74, 75, 78
 - social difficulties 66
 - vagal tone 67
- Emotional intelligence 90, 106
- Emotional self-regulation 1–4, 10, 13–22, 35, 36, 38, 42–45, 48–50, 52, 61–64, 72, 75–77, 87, 123, 139, 165, 170, 172, 173, 178–180, 197, 199, 201, 204, 205, 238, 254
 - anger 165
 - anticipatory regulation 2, 19, 20
 - anxiety 165
 - beliefs 10
 - conscious regulation 2, 14
 - control regulation 2, 13, 35, 178
 - control skills 20, 21, 36, 38, 41, 42, 45, 49, 50, 62, 75
 - culture 17
 - deficit 44, 45
 - definition 3, 35, 36, 62, 63, 165
 - depression 165, 170, 172, 173
 - desires 10
 - effortful control 43
 - emotional components regulated 64, 165
 - emotional dissociation 14–16
 - emotion dynamics 165
 - emotion generation 165
 - emotion recognition 94
 - emotional expression 16–19
 - exploratory regulation 2, 21, 22
 - impulse control 93
 - impulsive aggression and violence 165
 - individual differences 36, 38

- inhibition 171
 - marital conflict 13
 - moral development 165
 - process model 165
 - psychotherapy 165
 - rationalization 165
 - reappraisal 19, 109, 165, 178–180, 199, 201
 - response inhibition 178
 - self 254
 - self-awareness 171
 - self-guidance 171
 - social adjustment 37, 61, 62, 76
 - social affiliation 171
 - social judgment 171
 - socialization 165
 - strategies 14–22, 38–42, 45, 48, 52, 64, 72, 77, 165
 - style 44
 - suppression 93, 165–173, 175, 178
 - unconscious regulation 2, 14
 - values 10
 - volitional control of emotions 75
- Event-related potentials (ERPs) 131, 143, 145, 239–242
- visual ERPs 240–242
 - acoustic ERPs 242
- Explicit processes 87, 89–91, 102, 104–106
- explicit emotion 90, 91, 104, 105, 106
- Extrinsic factors 42, 76
- F**
- Fear 6, 38, 50, 51, 64, 165
- fearful children 50
- Films 101, 166, 168, 245,
- erotic 166
 - happy 245
 - neutral 166, 168, 245
 - sad 101, 168
- Frustration 38, 46, 47
- Functional magnetic resonance imaging (fMRI) 98, 99, 108, 128, 133, 134, 138, 163, 164, 166–169, 172, 175, 177, 178, 185, 186
- Functionalism theory 61
- G**
- Genes 259–262, 266
- chromatin DNA 262
 - DNA 260
 - fragmented DNA 266
 - mRNA 262
 - ob gene 261
 - protein gene product 95, 259
- Global negative entropy 254
- H**
- Happiness 96, 258
- Health 256, 258, 259, 270
- cognitive-behavioral stress management 270
 - emotional self-regulation 259
 - healing system 258
 - healing and spirituality 270
 - psycho-emotional adjustment 258
 - psychological well-being 258
 - psychosomatic wellness 258
 - psychospiritual vitalization 258
 - relaxation and meditation 259, 270
 - resiliency 258
 - wellness 256
 - volition 258
- Heroin
- mood 147
- Hippocampus 99, 102, 140–144, 146, 147, 260, 262
- action regulation 141
 - atrophy 262
 - cortisol 262
 - dentate gyrus 260, 262
 - explicit memory 102
 - GABAergic interneurons 146

- Hammon's horn 262
- memory 140, 262
- neurogenesis 262
- opiates 147
- pain 147
- pyramidal neurons 146
- theta rhythm 142–144, 146–147
- Homeostasis 9–11, 13, 21–23, 123–127, 139, 146, 148–150, 152
 - action regulation 126
 - allostasis 125–127, 139, 146, 148, 149, 152
 - body temperature 124, 125
 - control theory 123
 - corollary discharge 124
 - cybernetic mechanisms 124, 126, 152
 - expectancies 126
 - feedback 124, 149
 - feed-forward mechanisms 9, 124, 148, 149
 - frontal neocortex 124
 - goals 124, 139, 148, 149
 - homeostatic bias 139
 - homeostatic set-points 125, 148, 149
 - internal states 124, 125, 150
 - limbic cortex 124
 - mood regulation 10
 - monitoring 126
 - motivation 148, 149
 - motivational cybernetics 149
 - negative feedback loop 9
 - planning 124
 - physiological self-regulation 123–126
 - servomechanisms 124
 - stress 124
 - thirst 124
- Hormones 263
 - gonadal steroids 263
 - melatonin 263
 - opioids 263
 - prolactin 263
- Hypothalamic-pituitary-adrenal (HPA) axis 125, 149, 260–263, 270
 - allostatic load 260, 270
 - gonadal hormones 261
- Hypothalamus 105, 149, 150, 163, 167, 168, 171, 175, 176, 181, 262, 263
 - automatic generation of emotional responses 105
 - CRH secretion 263
 - emotional self-regulation 163, 167, 168, 171, 175, 176, 181
 - magnocellular division 262
 - paraventricular nucleus 262
 - parvocellular division 262
 - supraoptic nucleus 262
- I
- Implicit processes 87–91, 95, 102, 104–106
 - attention 88
 - implicit emotion 87–91, 95, 102, 104–106
 - memory 88
 - perception 88
 - priming 88
 - problem-solving 88
- Insula 98, 99, 101, 106, 110, 149
 - attention to emotional responses 98
 - phenomenal awareness 106
 - sadness 101
 - visceromotor and viscerosensory functions 149
- Interpersonal relationships 62, 63
 - parent-child interaction 62
- Intrinsic factors 41, 75
- J
- Joy 62, 259, 270
- L
- Laughter 259, 270

Levels of emotional awareness 87,
90–95, 97–101, 106, 109,
110, 112, 113, 115
action tendencies 90, 91, 99
background feelings 87, 95, 97,
100, 101, 106, 109, 110, 112,
113, 115
blends of emotion 90, 91
blends of blends of emotional
experience 91
emotional arousal 109, 115
intentions 99
Levels of Emotional Awareness
Scale (LEAS) 91–95
metacognition 97, 111
Perception of Affect Task (PAT)
94
peripheral awareness 100
phenomenal awareness 97, 110,
112, 115
reflective awareness 95, 97, 98,
110, 112, 113, 115
schemata 90, 94, 95
self 91, 92, 99
single emotions 91
Lymphatic system 265

M

Medial prefrontal cortex 98–101,
106–108, 114, 176, 178–182,
186
attention to emotional responses
98
autonomic regulation 107
background feelings 106
depression 114
emotional self-regulation 176,
178–182, 186
heart rate 107, 108
representations of self and other
99
somatic marker hypothesis 101
vagus tone 108
Medial septum
 β -endorphine 147

pain 146
Mental state 254
Mind-brain interaction 182–186,
196
agentic factors 183, 185
brain domain 183
brain processes 183
cognitive-behavioral therapy
(CBT) 184, 185
complex dynamic systems 183
emotional impulses 185
genetic determinism 185
Homo sapiens 185
information 183
intentionality 183
mammalian brain 185
mind domain 183
mind processes 184
moral values 185
obsessive-compulsive disorder
(OCD) 184
self-agency 183
self-consciousness 183–186
self-guidance 183
self-regulation 183
spider phobia 184
spirituality 185
subjectivity 183
synthesis rate of serotonin 184
transduction 184
unconscious modules 182
Modularity 102–104
Motor cortex 97, 183

N

Neocortex
priming 102
Neural basis of emotional
self-regulation 37, 41, 44, 50,
109, 113, 138, 163, 164,
166–182, 184–186, 198–200,
254
adults 163, 166–173, 177–181,
186

- amygdala 109, 163, 167–171, 175–181, 199
- anterior cingulate cortex 109, 138, 163, 167–169, 172, 173, 175, 176, 178, 181, 186, 254
- anterior temporal pole 109, 167, 169, 171, 176, 181, 199
- brain maturation 41, 50, 174, 175, 177
- brain stem 170, 171, 176
- children 163, 173–177, 186
- development 37
- diencephalon 170
- dorsolateral prefrontal cortex 109, 163, 173, 198
- emotion rating scale 166, 168,
- emotional awareness 170, 178
- endocrine and autonomic expression of emotion 167–169, 171, 176, 181
- executive functions 167
- frontal lobes 37, 50
- frontal lobe syndrome 171
- frontolimbic circuit 163
- hypothalamus 109, 163, 167, 168, 171, 175, 176, 181, 199, 200
- inhibitory control 171, 175
- insula 169, 171, 181
- lateral prefrontal cortex 167, 170, 171, 173, 176, 178, 180–182, 184–186
- limbic structures 163, 175, 232, 254
- medial prefrontal cortex 176, 178–182, 186
- metacognition 167, 176, 184
- midbrain 169, 176, 181
- moral behavior 172
- orbitofrontal cortex 163, 168, 170, 171, 173, 176, 179–182, 186, 232
- paralimbic structures 163, 170
- prefrontal cortex 163, 167, 170, 174, 175, 178, 186, 200, 201, 254
- pseudopsychopathic syndrome 171
- reflective conscious awareness 176
- sadness 163, 168–170, 172, 175, 176, 181, 186, 200
- self-awareness 172
- sexual arousal 138, 163, 166–168, 181, 198, 199
- social self-regulation 172
- spinal cord 170
- subjective experience 166–169, 175
- suppression 166–173, 175, 176, 181, 200
- theory-of-mind 176
- vagal tone 109, 113
- ventrolateral prefrontal cortex 169, 170, 173, 176, 180
- viscerosensory information 170
- volition 166–168, 170, 200
- Neurofeedback (EEG biofeedback) 239–251
 - alpha asymmetry training protocol 246–251
 - asymmetry pattern 244
 - auditory system 242
 - definition 239
 - depression 246–249
 - EEG alpha power 244
 - evoked potential amplitude training 240
 - imagination 242
 - in animals 241–243
 - in humans 242, 245
 - operant conditioning of action potentials 239
 - operant EEG conditioning 239
 - operant neural training 240
 - pain 242, 243
 - psychotherapy 247–249
 - somatic sensory cortex 242

trivial mediation issue 240
 visual system 241
 Neuronal populations 254
 coherence 254
 synchronization 254
 Neuropsychology 236
 Nitric oxide (NO) 263
 neuroendocrine regulation 263

O

Operant conditioning 239, 240
 reinforcement contingency 240
 reward 240
 trial-and-error learning 240
 Oral biology and medicine 257,
 264–270
 acanthosis 266
 apoptotic basal keratinocytes
 266
 basal epithelial cell destruction
 266
 B cells 266, 270
 carcinoma 269
 cortisol 269
 cranial nerves 265
 cytokines 265
 hard and soft mucosa of the oral
 cavity 264–266, 270
 hyper-keratosis 266
 leukocytes 267
 leukoplakia 268
 lymphocytes 267, 268
 lymphoid leucocytes 266
 NK cells 270
 OLP lesions 266–268, 270
 oral lichen planus (OLP)
 265–268
 para-keratosis 266
 salivary cortisol 265
 salivary glands 265
 superior cervical ganglia 265
 T cells 266, 267, 270
 tobacco 268, 269

Orbitofrontal cortex 106, 115, 129,
 149, 163, 168, 170, 171, 173,
 176, 179–182, 186, 199, 232
 amygdala 106
 emotional self-regulation 163,
 168, 170, 171, 173, 176,
 179–182, 186
 error responsive cells 129
 omission of reward 149
 phenomenal awareness 106
 reinforcement 129
 somatic sensations 106

P

Parahippocampal gyrus 184, 185,
 198
 contextual fear memory 184
 Parents 43, 47, 48, 63, 71
 expectations 43
 parental guidance 48, 63
 parenting practices 43, 47, 48
 parental support 71
 Parietal cortex 102, 106
 background feelings 102, 106
 Peers 42
 Peptides 259–265
 adrenocorticotrophic hormone
 (ACTH) 260, 261, 264
 antalarmin 261
 arginine-vasopressin (AVP) 262
 β -adrenoreceptors 260
 β -endorphin 262
 calcitonin gene-related peptide
 (CGRP) 259
 cAMP 260
 cloning 261, 262
 corticotrophin releasing
 hormone (CRH) 260, 261, 263,
 264
 CRH receptor type 1, 260
 cytokines 260, 263
 feedback regulation 260
 IL-6 260, 263, 265
 interleukin (IL)-1 β 260, 263
 intracellular calcium 260

- neurokinin A 259
- neuropeptide Y (NPY) 259, 260
- pituitary gland 260, 261
- pro-opiomelanocortin (POMC)-derived peptides 260, 262
- sickness behaviour 260
- substance P 259
- tachykinins 259, 260
- tumor necrosis factor (TNF)- α 260, 265
- urocortin 260, 261
- vasoactive intestinal polypeptide/peptide histidine isoleucine (VIP/PHI) 259, 260
- Pictures 94, 98, 112, 177–179
 - fearful 178
 - International Affective Picture System (IAPS) 98, 177–179
 - negative and neutral 177
 - pleasant, unpleasant and neutral 98
- Placebo response 202
- Positron emission tomography (PET) 96, 98, 107, 167, 173
 - regional cerebral blood flow (rCBF) 96, 98, 167, 173
- Posterior cingulate cortex 148, 151
 - discrimination learning 140
- Post-synaptic potentials 242
- Post-traumatic stress disorder (PTSD) 113–115
- Prefrontal cortex 101, 108
 - memory and dopamine 108
 - dorsolateral prefrontal cortex and strategic control of task performance 128
 - ventrolateral prefrontal cortex and sadness 101
- Premenstrual Dysphoric Disorder (PMDD) 249–251
 - luteal phase and symptoms 249–251
 - neurofeedback treatment 249–251
- Proximal behavioral indices and effects 44, 47, 48, 52
- Psychological control 257
 - behavioral control 257
 - cognitive control 257
 - informational control 257
 - retrospective control 257
- Psychological self-regulation 2, 12, 22, 40, 41, 64, 67, 70
 - behavioral inhibition 41, 64, 67, 70
 - development 40
 - frontal cortex 67
- Psycho-neuroendocrine-immune network 255, 263, 264, 269, 270
 - consciousness 255
 - self-regulation of emotions 255
- Psychosomatic network 184, 255, 256, 258, 259, 269–271
 - biological factors 256
 - cellular and humoral immunity 259
 - emotional factors 270
 - feedback loops 269
 - genetic factors 256
 - informational paradigm 259
 - lymphoid system 269
 - maladaptation 258
 - mind-body dichotomy 255
 - neuroendocrine-mediated immune regulation 259
 - neuropeptide-receptor network 258, 259
 - physio-behavioral factors 256
 - psycho-emotional factors 256
 - psychoneuroendocrinology 256
 - psychoneuroimmunology 255
 - psycho-socio-cognitive factors 270
 - psychosomatic medicine 256, 270, 271
 - psychosomatic disturbances 270
 - systems biology 255

Q

- Quantum mechanics 196, 204, 210–234, 238
 - attention 227
 - consciousness 217, 218, 220, 226, 232, 233
 - Copenhagen Interpretation 210, 211, 216, 220
 - double-slit experiment 219, 221
 - emotional self-regulation 230, 232
 - environmental decoherence 231
 - free choices 216, 223
 - harmonic oscillator 219, 221
 - human agency 210, 212–216, 232, 234
 - human participation 202, 212
 - ion channels 220, 221
 - mind 210
 - mind-brain question 196, 210, 211, 228, 232, 234, 238
 - nerve terminals 220, 221
 - neurotransmitter 221
 - non-locality 223, 233
 - phenomenology 229
 - probabilities 216
 - Process I 212, 213, 215–218, 220, 222, 223, 226–229, 231, 232
 - Process II 212, 218, 222, 226–228
 - quantum state 212, 219, 231
 - Quantum Zeno Effect 224–229
 - self 226
 - uncertainty principle 221, 231
 - vesicles 221, 222
 - volition 227, 232
 - Von Neumann formulation 212, 217, 220, 223, 224, 231, 233

R

- Recall-induced emotion 96
 - anterior cingulate cortex 96

- mid-cingulate cortex 96

S

- Sadness 6, 51, 62, 63, 69, 71, 76, 96, 101, 163, 165, 168–170, 172, 175, 176, 163, 168–170, 172, 175, 176, 181, 186, 258
- Self 92, 254, 259, 270
 - awareness of self 259, 270
 - emotion 92
- Self-directed neuroplasticity 196–198, 200–202, 204, 205, 236, 238
 - attentional refocusing 198
 - cognitive-behavioral therapy (CBT) 198
 - cognitive reframing 197, 198
 - mental effort 196, 204
 - mentalistic terminology 196, 198, 205
 - mindful awareness (mindfulness) 200, 201, 204, 236, 238
 - neurophysiological terminology 196, 198
 - obsessive-compulsive disorder (OCD) 198, 202
 - phobia 198, 202
 - willfully directed attention 197
- Self-regulation 126–153, 206
 - action monitoring 127, 128, 130, 131, 135–137, 148, 150
 - action regulation 126, 140–142, 148, 151
 - adaptive goals 128
 - affective control 137
 - allostatic mechanisms 125, 126, 127, 139, 146, 148, 149, 152
 - anterior cingulate cortex 126–135, 137, 139, 140, 143–145, 147–149, 151, 152
 - anxiolytic drug 138
 - attention for action 127
 - cingulate motor area 134
 - cognitive control 143
 - conflict monitoring 133–135

- correct-related negativity 136
- corticolimbic mechanisms 127
- cybernetic servomechanisms 127
- depression 139
- discriminative avoidance learning 144, 145
- dopamine 145, 146, 148
- error feedback 131, 133
- error-related negativity (ERN) 127, 130–133, 135–139, 143–145, 150, 151
- executive functions 127, 149
- expectancies 126
- frontal cortex 148
- frontal lobes 127, 150
- frontal networks 137
- frontolimbic cortices 148
- medial frontal cortex 134
- medial frontal negativity (MFN) 127, 132, 133, 139, 148, 150, 151
- midline theta oscillations 133, 143
- mismatch 130
- motivation 148, 151
- motivational set-points 128
- motives and actions 150
- negative feedback 139
- neurocybernetic mechanisms 139
- obsessive-compulsive disorder and monitoring deficits (OCD) 135–137
- opiates 146
- Papez circuit 148
- Parkinson's disease 145
- placebo 138
- positive feedback 139
- psychomotor poverty 136, 137
- schizophrenia and monitoring deficits 135, 136
- Stroop interference task 127, 128
- supplementary motor area 132, 134
- theta rhythm 142, 145, 146
- ventral limbic networks 148
- visceral and motivational circuits 137
- visceromotor control system 149
- Sexual activity 261
- Siblings 42
- Skin conductance response 89, 103
 - amygdala 103
- Sleep 261, 264
- Socialization 42, 72, 76, 77
 - emotion dysregulation 72, 76
 - social rules 77
- Somatosensory cortex 102, 106, 137
 - background feelings 102, 106
 - pain 137
- Stress 67, 257, 258, 260–264, 270
 - coping 258
 - corticosteroid receptors 262
 - cortisol 67, 260–264
 - cultural background 257
 - cytokines 264
 - environment 257
 - glucocorticoids 262, 263
 - hierarchical position 257
 - immune dysregulation 258
 - legal status 257
 - leptin 261
 - locus of control 257
 - motivation 257
 - negative emotions 258
 - perception of fit 257
 - personality 257
 - rights and responsibilities 257
 - social involvement 257
- Striatum 102, 105
 - nucleus accumbens 105
 - reward stimuli 105
 - skills and habits 102
- Sympathetic nervous system 259, 260, 263, 265
 - catecholamine pathway 259

dopamine-hydroxylase 259
 locus coeruleus 260
 lymphoid cells and organs 259,
 263, 265
 myeloid cells 259, 265
 neuroendocrine markers and
 enzymes 259
 neurofilament 259
 norepinephrine 260
 parenchyma 259
 sympathetic-adrenal-medullary
 responses 260, 263
 synaptophysin 259
 tyrosine hydroxylase 259

T

Temperament 9, 10, 24, 35, 38,
 41–52, 67, 68
 biological basis 43, 52
 emotional self-regulation 9, 10,
 24, 35, 38, 41–52
 proneness-to-distress 45
 reactivity 44, 50, 68
 types 42
 Temporal pole 98, 106

attention to emotional responses
 98

phenomenal awareness 106

Thalamus 105, 140, 143

anterior nucleus 143

automatic generation of
 emotional responses 105

discriminative avoidance learning
 140

V

Ventral cingulate cortex 98

attention to emotional responses
 98

Ventral tegmental area (VTA) 129,
 145

dopamine 129

errors and rewards 129

Visual association cortex 184

Volition 75, 166–168, 170, 200, 227,
 232, 253, 254, 256–258

W

Withdrawal 45, 50

Working memory 143, 178

In the series ADVANCES IN CONSCIOUSNESS RESEARCH (AiCR) the following titles have been published thus far or are scheduled for publication:

1. GLOBUS, Gordon G.: *The Postmodern Brain*. 1995.
2. ELLIS, Ralph D.: *Questioning Consciousness. The interplay of imagery, cognition, and emotion in the human brain*. 1995.
3. JIBU, Mari and Kunio YASUE: *Quantum Brain Dynamics and Consciousness. An introduction*. 1995.
4. HARDCASTLE, Valerie Gray: *Locating Consciousness*. 1995.
5. STUBENBERG, Leopold: *Consciousness and Qualia*. 1998.
6. GENNARO, Rocco J.: *Consciousness and Self-Consciousness. A defense of the higher-order thought theory of consciousness*. 1996.
7. MAC CORMAC, Earl and Maxim I. STAMENOV (eds): *Fractals of Brain, Fractals of Mind. In search of a symmetry bond*. 1996.
8. GROSSENBACHER, Peter G. (ed.): *Finding Consciousness in the Brain. A neurocognitive approach*. 2001.
9. Ó NUALLÁIN, Seán, Paul MC KEVITT and Eoghan MAC AOGÁIN (eds): *Two Sciences of Mind. Readings in cognitive science and consciousness*. 1997.
10. NEWTON, Natika: *Foundations of Understanding*. 1996.
11. PYLKKÖ, Pauli: *The Aconceptual Mind. Heideggerian themes in holistic naturalism*. 1998.
12. STAMENOV, Maxim I. (ed.): *Language Structure, Discourse and the Access to Consciousness*. 1997.
13. VELMANS, Max (ed.): *Investigating Phenomenal Consciousness. Methodologies and Maps*. 2000.
14. SHEETS-JOHNSTONE, Maxine: *The Primacy of Movement*. 1999.
15. CHALLIS, Bradford H. and Boris M. VELICHKOVSKY (eds.): *Stratification in Cognition and Consciousness*. 1999.
16. ELLIS, Ralph D. and Natika NEWTON (eds.): *The Caldron of Consciousness. Motivation, affect and self-organization – An anthology*. 2000.
17. HUTTO, Daniel D.: *The Presence of Mind*. 1999.
18. PALMER, Gary B. and Debra J. OCCHI (eds.): *Languages of Sentiment. Cultural constructions of emotional substrates*. 1999.
19. DAUTENHAHN, Kerstin (ed.): *Human Cognition and Social Agent Technology*. 2000.
20. KUNZENDORF, Robert G. and Benjamin WALLACE (eds.): *Individual Differences in Conscious Experience*. 2000.
21. HUTTO, Daniel D.: *Beyond Physicalism*. 2000.
22. ROSSETTI, Yves and Antti REVONSUO (eds.): *Beyond Dissociation. Interaction between dissociated implicit and explicit processing*. 2000.
23. ZAHAVI, Dan (ed.): *Exploring the Self. Philosophical and psychopathological perspectives on self-experience*. 2000.
24. ROVEE-COLLIER, Carolyn, Harlene HAYNE and Michael COLOMBO: *The Development of Implicit and Explicit Memory*. 2000.
25. BACHMANN, Talis: *Microgenetic Approach to the Conscious Mind*. 2000.
26. Ó NUALLÁIN, Seán (ed.): *Spatial Cognition. Selected papers from Mind III, Annual Conference of the Cognitive Science Society of Ireland, 1998*. 2000.
27. McMILLAN, John and Grant R. GILLET: *Consciousness and Intentionality*. 2001.

28. ZACHAR, Peter: *Psychological Concepts and Biological Psychiatry. A philosophical analysis*. 2000.
29. VAN LOOCKE, Philip (ed.): *The Physical Nature of Consciousness*. 2001.
30. BROOK, Andrew and Richard C. DeVIDI (eds.): *Self-reference and Self-awareness*. 2001.
31. RAKOVER, Sam S. and Baruch CAHLON: *Face Recognition. Cognitive and computational processes*. 2001.
32. VITIELLO, Giuseppe: *My Double Unveiled. The dissipative quantum model of the brain*. 2001.
33. YASUE, Kunio, Mari JIBU and Tarcisio DELLA SENTA (eds.): *No Matter, Never Mind. Proceedings of Toward a Science of Consciousness: Fundamental Approaches*, Tokyo, 1999. 2002.
34. FETZER, James H.(ed.): *Consciousness Evolving*. 2002.
35. Mc KEVITT, Paul, Seán Ó NUALLÁIN and Conn MULVIHILL (eds.): *Language, Vision, and Music. Selected papers from the 8th International Workshop on the Cognitive Science of Natural Language Processing*, Galway, 1999. 2002.
36. PERRY, Elaine, Heather ASHTON and Allan YOUNG (eds.): *Neurochemistry of Consciousness. Neurotransmitters in mind*. 2002.
37. PYLKKÄNEN, Paavo and Tere VADÉN (eds.): *Dimensions of Conscious Experience*. 2001.
38. SALZARULO, Piero and Gianluca FICCA (eds.): *Awakening and Sleep-Wake Cycle Across Development*. 2002.
39. BARTSCH, Renate: *Consciousness Emerging. The dynamics of perception, imagination, action, memory, thought, and language*. 2002.
40. MANDLER, George: *Consciousness Recovered. Psychological functions and origins of conscious thought*. 2002.
41. ALBERTAZZI, Liliana (ed.): *Unfolding Perceptual Continua*. 2002.
42. STAMENOV, Maxim I. and Vittorio GALLESE (eds.): *Mirror Neurons and the Evolution of Brain and Language*. 2002.
43. DEPRAZ, Natalie, Francisco VARELA and Pierre VERMERSCH.: *On Becoming Aware. A pragmatics of experiencing*. 2003.
44. MOORE, Simon and Mike OAKSFORD (eds.): *Emotional Cognition. From brain to behaviour*. 2002.
45. DOKIC, Jerome and Joelle PROUST: *Simulation and Knowledge of Action*. 2002.
46. MATHEAS, Michael and Phoebe SENGERS (ed.): *Narrative Intelligence*. 2003.
47. COOK, Norman D.: *Tone of Voice and Mind. The connections between intonation, emotion, cognition and consciousness*. 2002.
48. JIMÉNEZ, Luis: *Attention and Implicit Learning*. 2003.
49. OSAKA, Naoyuki (ed.): *Neural Basis of Consciousness*. 2003.
50. GLOBUS, Gordon G.: *Quantum Closures and Disclosures. Thinking-together post-phemenology and quantum brain dynamics*. 2003.
51. DROEGE, Paula: *Caging the Beast. A theory of sensory consciousness*. 2003.
52. NORTHOFF, Georg: *Philosophy of the Brain. The 'Brain problem'*. 2003.
53. HATWELL, Yvette, Arlette STRERI and Edouard GENTAZ (eds.): *Touching for Knowing. Cognitive psychology of haptic manual perception*. 2003.

54. BEAUREGARD, Mario (ed.): *Consciousness, Emotional Self-Regulation and the Brain*. 2004.
55. PERUZZI, Alberto (ed.): *Mind and Causality*. n.y.p.
56. GENNARO, Rocco J. (ed.): *Higher-Order Theories of Consciousness. An Anthology*. n.y.p.



3 2815 00865 0049

RC 343 .C725 2004

Consciousness, emotional
self-regulation, and the

DATE DUE

FEB 16 2006

JUL 13 2010

AUG 30 2010

WITHDRAWN
THE COLLEGE AT BROCKPORT
DR. J. M. WILSON MEMORIAL LIBRARY

During the last decade, the study of emotional self-regulation has blossomed in a variety of sub-disciplines belonging to either psychology (developmental, clinical) or the neurosciences (cognitive and affective). *Consciousness, Emotional Self-Regulation and the Brain* gives an overview of the current state of this relatively new scientific field. Several areas are examined by some of the leading theorists and researchers in this emerging domain. Most chapters seek to either present theoretical and developmental perspectives about emotional self-regulation (and dysregulation), provide cutting edge information with regard to the neural basis of conscious emotional experience and emotional self-regulation, or expound theoretical models susceptible of explaining how healthy individuals are capable of consciously and voluntarily changing the neural activity underlying emotional processes and states. In addition, a few chapters consider the capacity of human consciousness to volitionally influence the brain's electrical activity or modulate the impact of emotions on the psychoneuroendocrine-immune network. This book will undoubtedly be useful to scholars and graduate students interested in the relationships between self-consciousness, emotion, the brain, and the body.

John Benjamins Publishing Company

ISBN 90 272 5188 6 (Eur) / 1 58811 459 7 (US)