

2 Neuroscience Approaches in Social 3 and Personality Psychology

4 David M. Amodio *and* Eddie Harmon-Jones

5 Abstract

6 Social neuroscience is an interdisciplinary approach to studying the mind and behavior, noted for its
7 appreciation for the dynamic interactions of situational and dispositional processes as they relate to
8 neural and biological mechanisms. In this chapter, we describe the methodological approach of social
9 neuroscience and review research that has applied this approach to address the interplay of the person
10 and situation in the domains of social cognition, attitudes, emotion and motivation, intergroup
11 relations, and personality. We provide critical discussion of how neuroscience may contribute to
12 classic questions in personality and social psychology, and we describe how the social neuroscience
13 approach promotes the integration of dispositional and situational accounts of the mind and behavior.

14 **Keywords:** social neuroscience, emotion, attitudes, intergroup, review, psychophysiology, cognitive
15 neuroscience, personality and neuroscience

16 *Neuroscience Approaches to Social 17 and Personality Psychology*

18 The recent interest in neural and biological compo-
19 nents of social and personality processes may seem
20 new, but is actually a return to form for the field of
21 social and personality psychology. Founding psy-
22 chologists such as James and Wundt were trained in
23 physiology, and they approached issues of thought,
24 emotion, memory, and perception with a firm belief
25 that mental processes were rooted in the brain. To
26 these psychologists, the mind and the brain were
27 symbiotic, and a consideration of neural processes,
28 along with dispositional and interpersonal processes,
29 provided a natural and comprehensive approach to
30 understanding the mind and behavior. Early psy-
31 chological research on social processes, such as bluff-
32 ing to other players during a poker game (Riddle,
33 1925) or responding to socially taboo words and
34 phrases (Darrow, 1929), incorporated psychophysi-
35 ological measures to complement behavioral obser-
36 vations. Indeed, early research on intergroup inter-
37 actions and prejudice used physiological measures to

38 examine anxiety during an interracial encounter 38
(Rankin & Campbell, 1955). Hence, physiological 39
measures have long served as an important compo- 40
nent of an integrated approach to social-personality 41
psychology. 42

43 As the field of psychology developed, a shift 43
toward behaviorism effectively vanquished the role 44
of the mind and brain from the study of behavior, 45
and the field of neuroscience branched away from 46
psychology. Although research on neural function 47
continued at lower levels of analysis (e.g., cellular 48
and molecular), it no longer made contact with the 49
higher-level processes of social and personality psy- 50
chology. When a focus on mental function reemerged 51
during the cognitive revolution, it was generally 52
held separate from neural and biological function 53
(with some notable exceptions, e.g., Cacioppo & 54
Petty, 1983; Frith, Morton, & Leslie, 1991; 55
Gazzaniga, 1985; Shapiro & Crider, 1969). 56
However, with recent advances in brain imaging 57
technology, research on cognitive neuroscience 58
has surged over the past decade, making increasing 59

1 contact with questions of social cognition (Ochsner
2 & Lieberman, 2001). In this way, social and person-
3 ality psychologists have begun to reintegrate neural
4 and biological approaches into the range of methods
5 used to understand the social mind and behavior.

6 In this chapter, we review the contemporary social
7 neuroscience approach to social-personality psychol-
8 ogy. This general approach began to appear with
9 increasing frequency in the laboratories of social psy-
10 chologists, cognitive neuroscientists, and neurolo-
11 gists during the late 1990s. The term “social
12 neuroscience” was coined in print by Cacioppo and
13 Berntson (1992; see also Carlston, 1994) and tends
14 to describe the broad enterprise of examining the
15 interplay of social and physiological levels of analysis.
16 More recent formulations by Ochsner and Lieberman
17 (2001) and Klein and Kihlstrom (1998) incorpo-
18 rated ideas from cognitive neuroscience and neuropsy-
19 chological patient literatures, respectively, prompt-
20 ing new aspects of social neuroscience referred to as
21 “social cognitive neuroscience” and “social neuropsy-
22 chology.” Over the past decade, social neuroscience
23 has been the subject of several dedicated research
24 conferences, culminating in the formation of the
25 Social and Affective Neuroscience Society in 2008
26 and the Society for Social Neuroscience in 2010.
27 Whereas social neuroscience was seen as a novelty at
28 social-personality meetings merely a decade ago, it is
29 now fully integrated into the social/personality psy-
30 chologist’s methodological repertoire.

31 The present volume highlights the interplay of
32 social and personality factors in studies of the mind
33 and behavior. The social neuroscience approach fits
34 this theme well. Integrative at its core, social neuro-
35 science encompasses the study of personality and
36 individual differences as well as situational and envi-
37 ronmental effects, as they interface with cognitive
38 processes and neural mechanisms. In this chapter,
39 we use the term “social neuroscience” to refer to this
40 general integrative approach, although it might just
41 as easily be called “social-personality neuroscience.”

42 In what follows, we begin with an overview of the
43 methodological approaches used in social neurosci-
44 ence. We then describe social neuroscience research
45 across major areas of social and personality psychol-
46 ogy, with a focus on how neuroscience and physiolog-
47 ical approaches pertain to social-personality theory.

48 **The Social Neuroscience Approach**

49 Broadly speaking, social neuroscience refers to
50 an integrative approach that can be applied to any
51 scientific question concerning social processes and
52 the brain. However, the types of questions that are

addressed with this approach vary considerably 53
across disciplines. To the social psychologist, *social* 54
neuroscience refers to an interdisciplinary research 55
approach that integrates theories and methods of 56
neuroscience (and other biological fields) to address 57
social psychological questions. To a cognitive neuro- 58
scientist, it often refers to research that addresses 59
questions about the neural substrates of social 60
processes, with a focus on understanding neural 61
function. To an animal behaviorist, social neurosci- 62
ence research may address questions about the 63
neural and hormonal mechanisms associated with 64
low-level social behaviors, such as dominance and 65
affiliation. Thus, many “social neuroscience” studies 66
examine questions outside the typical purview of 67
social-personality psychology, and therefore it is 68
helpful for consumers of this literature to carefully 69
consider the question asked by a particular study. In 70
this section, we describe the two main types of ques- 71
tions asked in human social neuroscience and their 72
corresponding methodological approaches. We also 73
describe the critical role of reverse inference in draw- 74
ing conclusions from neuroscience findings. 75

Brain Mapping Approach 76

Brain-mapping studies ask “Where in the brain 77
is _____?” For example, Where in the brain is fear? 78
Where is episodic memory? Where is love? Where is 79
the self? Human brain mapping is a cornerstone of 80
modern cognitive neuroscience. It concerns the 81
mapping of basic psychological processes to particu- 82
lar regions of the brain. 83

Early forms of brain mapping involved the prob- 84
ing of exposed brain tissue by a neurosurgeon while 85
a patient reported his or her experience. Today, rela- 86
tively noninvasive neuroimaging measures, such as 87
functional magnetic resonance imaging (fMRI), are 88
often used for a similar purpose. In cognitive neuro- 89
science, this approach is used to map relatively low- 90
level psychological processes such as basic forms of 91
sensation, perception, and specific aspects of learn- 92
ing and memory. As a general rule, lower-level cog- 93
nitive processes can be mapped more directly onto 94
specific physiological responses than more complex 95
high-level processes. 96

In social psychology, researchers have attempted 97
to map very high-level psychological processes, such 98
as social emotions, the self-concept, trait impres- 99
sions, and political attitudes, onto the brain as well. 100
This is where things get trickier. For example, to 101
study the neural basis of romantic love, researchers 102
have scanned participants’ brains while they viewed 103
pictures of strangers versus their significant others 104

1 (Aron, Fisher, Mashek, Strong, Li, & Brown, 2005).
 2 Similarly, to study the neural basis of the self,
 3 researchers have scanned the brain while subjects
 4 judged whether trait adjectives described them
 5 versus another person (Kelley et al., 2002; Mitchell,
 6 Banaji, & Macrae, 2005). Such studies apply the
 7 same logic to identifying the neural substrates of
 8 very high-level processes as neuroscientists have
 9 applied in the neural mapping of very low-level pro-
 10 cesses, such as edge detection in vision. Although
 11 high-level psychological ascriptions of brain activity
 12 may have heuristic value, they may risk obscuring
 13 the important low-level mechanisms that the
 14 observed brain activations likely represent.

15 A defining feature of the brain mapping approach
 16 is that it seeks to create a valid mapping of psycho-
 17 logical processes onto a pattern of neurophysiologi-
 18 cal responses. Pure brain-mapping studies are
 19 undertaken with few prior assumptions about the
 20 psychological function of a brain region—indeed,
 21 the point of such studies is to establish ideas about
 22 function through the process of induction across
 23 multiple studies using a variety of conceptually simi-
 24 lar tasks and manipulations. This approach is poten-
 25 tially useful for generating new ideas about
 26 commonalities in the cognitive processes that may
 27 underlie two otherwise distinct psychological func-
 28 tions. For example, some researchers have observed
 29 that social exclusion and physical pain activate a
 30 common region of the anterior cingulate cortex
 31 (figure 6.1; among many other nonoverlapping
 32 areas) and concluded that social and physical pain
 33 share some common psychological features
 34 (Eisenberger, Lieberman, & Williams, 2003; but see
 35 Somerville, Heatherton, & Kelley, 2006). Although
 36 this approach does not tell us exactly how or why
 37 social and physical forms of pain might be related,
 38 simply because the true function of the neural activ-
 39 ity is difficult to discern, it nevertheless provokes
 40 new ideas about potential relationships between psy-
 41 chological processes. But because brain activations
 42 alone are usually ambiguous with regard to their spe-
 43 cific psychological functions, they do not always pro-
 44 vide a reliable index of a psychological variable. Thus,
 45 the brain-mapping approach is not appropriate for
 46 testing hypotheses about the relationship between
 47 two psychological variables or the effects of an exper-
 48 imental manipulation on a psychological variable.

49 **Hypothesis Testing Approach**

50 The hypothesis testing approach in social neurosci-
 51 ence is used to test relationships between psychological
 52 variables. This approach begins with the assumption

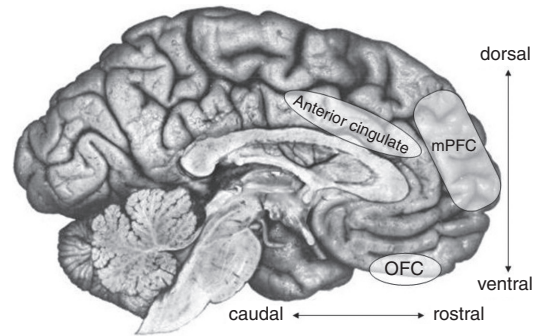


Fig. 6.1 Medial aspect of the left hemisphere of the brain. mPFC = medial prefrontal cortex, OFC = orbital frontal cortex.

that a particular brain region reflects a specific
 53 psychological process. In this regard, it does not concern
 54 brain mapping, but instead relies on past research
 55 to have already established the validity of neural indi-
 56 cators. For example, a social psychologist who studies
 57 intergroup prejudice might hypothesize that implicit
 58 racial bias is rooted in mechanisms of classical fear
 59 conditioning (Amodio, Harmon-Jones, & Devine,
 60 2003). To test this hypothesis, one might measure
 61 brain activity in the amygdala (figure 6.2)—a struc-
 62 ture implicated in fear conditioning in many previ-
 63 ous studies—while a participant completes a
 64 behavioral task designed to elicit implicit racial bias.
 65 In this case, the construct validity of the neural mea-
 66 sure of fear conditioning (amygdala activity) is
 67 already reasonably established (but see Amodio &
 68 Ratner, 2011a), and the question concerns not the
 69 meaning of brain activations, but experimental effects
 70 among psychological variables. It is the hypothesis
 71 testing approach of social neuroscience that is of
 72 primary interest to social-personality psychologists. 73

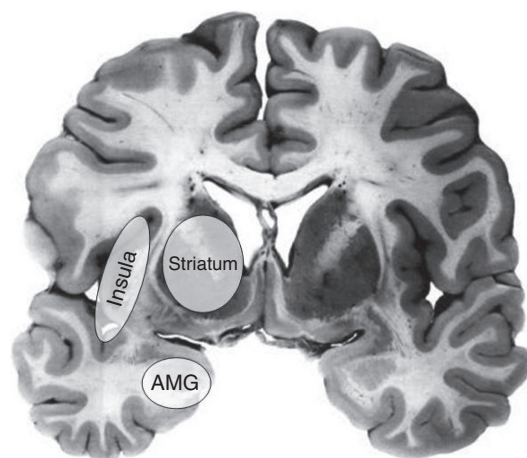


Fig. 6.2 View of coronal slice through brain, with structures on the left side labeled. AMG = amygdala.

1 Whereas brain-mapping studies may inform our
2 understanding of the brain, hypothesis-testing stud-
3 ies attempt to inform psychological theories of the
4 mind.

5 Critically, brain mapping and psychological
6 hypothesis testing approaches should not be com-
7 bined within a single experiment; major inferential
8 problems occur as a result (Amodio, 2010a). This is
9 because a test of a psychological hypothesis assumes
10 that the mapping of a psychological variable to a
11 neural structure is already established (e.g., that the
12 neural measure has construct validity). The brain
13 mapping approach is used to establish the mapping
14 between a psychological variable and neural struc-
15 ture (i.e., to establish construct validity of the neural
16 measure). When these approaches are combined,
17 there is a risk of defining the neural operationaliza-
18 tion of a psychological construct on the basis of
19 whether it supports one's theoretical hypothesis—
20 an example of tautological inference.

21 To illustrate, imagine that a researcher wants to
22 test the hypothesis that empathy involves self-reflec-
23 tion. Given previous research linking self-judgments
24 to activity in the medial frontal cortex (mPFC,
25 figure 6.1), he decides to use a measure of mPFC
26 during an empathy task (e.g., viewing pictures of
27 people in pain) to index the engagement in self-
28 reflection. However, the region of mPFC that has
29 been linked to the self is rather large and idiographic
30 across individuals and, this area has been associated
31 with several other psychological responses, includ-
32 ing mentalizing, evaluation, and self-regulation.
33 Therefore, it is difficult to know whether observed
34 mPFC activity on a given task represents self-reflec-
35 tion or some other process. This ambiguity under-
36 mines the construct validity of mPFC activity as a
37 measure of the “self”.

38 To deal with this problem, the researcher might
39 simply examine a correlation between mPFC activ-
40 ity and scores on an empathy questionnaire to see
41 which, if any, portions of the task-related mPFC
42 activity might relate to empathy. But this can com-
43 pound the problem. Given his hypothesis that
44 empathy involves self-reflection, the researcher
45 might simultaneously infer that any mPFC region
46 correlated with the questionnaire must be the “self”
47 region (establishing the construct) and that “self”
48 activity is indeed associated with greater empathy
49 (testing the psychological hypothesis). This blurs
50 the important steps of establishing construct valid-
51 ity (brain mapping) and internal validity (hypothe-
52 sis testing). Because the construct is validated on the
53 basis of the hypothesis-testing correlation analysis,

the logic of the test is circular (Amodio, 2010a; 54
Barrett, 2009). This analytical approach is fairly 55
common in social neuroscience research, primarily 56
because the social psychological processes of interest 57
are complex and difficult to localize. Nevertheless, 58
this approach is problematic, and consumers of 59
social/personality neuroscience should be aware of 60
such practices and cautious of their use. 61

Reverse Inference 62

When considering the two general approaches 63
described above, the issue of *reverse inference* is often 64
a concern. Reverse inference refers to a form of rea- 65
soning used heavily in social and cognitive neurosci- 66
ence to infer the psychological meaning of a brain 67
activation based on previous findings (Poldrack, 68
2006). In some studies (e.g., simple brain-mapping 69
studies), a psychological process is manipulated and 70
the resulting pattern of brain activity is observed. 71
The inference that the psychological manipulation 72
produced the brain activity may be described as a 73
forward inference, in that the brain activity clearly 74
follows from the manipulation. The inference is 75
based on the known validity of the manipulation. 76
By contrast, the inference of a psychological process 77
from an observed pattern of brain activity is a *reverse* 78
inference. In this case, the precise meaning of the 79
brain activation is ambiguous and inferred from 80
other studies that have used a particular manipula- 81
tion to activate the same area. The practice of reverse 82
inference becomes increasingly problematic to the 83
extent that the source of inference—in this case, a 84
brain activation—could reflect different psychologi- 85
cal processes (Cacioppo et al., 2003; Poldrack, 86
2006). In studies of low-level vision, reverse infer- 87
ence is a comparatively lesser problem (but still a 88
serious issue). For example, retinotopic mapping of 89
stimuli onto primary visual cortex provides a rela- 90
tively constrained index of basic visual processing. 91
But as psychological variables become more com- 92
plex, as they do with social and personality pro- 93
cesses, the mapping between a particular brain 94
region and a psychological process becomes less cer- 95
tain. In these cases, reverse inference can be a serious 96
problem. 97

All cognitive and social neuroscience studies rely 98
on reverse inference. That is, to the extent that a 99
neural activation is interpreted as reflecting a psy- 100
chological process, the use of reverse inference is 101
unavoidable. However, researchers can take steps to 102
bolster the strength of a reverse psychological infer- 103
ence by enhancing the construct validity of a neural 104
indicator and the strength of their experimental 105

1 designs, such as through the careful use of theory,
2 converging evidence from other studies (including
3 animal research), and the use of behavioral tasks
4 that provide valid manipulations of a construct and
5 interpretable behavioral data.

6 ***What Types of Social and Personality*** 7 ***Questions Are Amenable to a Neuroscience*** 8 ***Analysis?***

9 First and foremost, the brain is a *mechanism*, and an
10 extremely complex one at that. Hence, neuroscience
11 models and methods are especially useful for the
12 study of psychological mechanisms, such as those
13 involved in action control, perception, and atten-
14 tion. Psychological phenomena that are not mecha-
15 nisms, but correspond more closely to appraisals
16 (e.g., attitudes and beliefs), subjective psychological
17 states, abstract psychological structures (e.g., the
18 self), and high-level representation, may be less
19 amenable to a neuroscience level of analysis. For a
20 social/personality psychologist who is considering
21 the potential benefit of a neuroscience approach,
22 the most critical issue is whether one's question
23 concerns basic psychological mechanisms. Can
24 the components of one's mechanistic model be
25 described in terms of low-level functions, such as
26 perception, sensation, low-level cognition, and low-
27 level motivation? If so, then neuroscience models
28 may be particularly useful. If the psychological phe-
29 nomena of interest cannot be conceptualized at a
30 low level of analysis, but rather are most meaningful
31 at a high level of construal (e.g., the self), then it
32 may be more difficult to make valid inferential
33 connections between psychological theory and the
34 brain.

35 **Methods of Social and Personality** 36 **Neuroscience**

37 Contemporary social neuroscience makes use of a
38 wide range of methods that are often used in com-
39 bination with the more traditional tools of person-
40 ality and social psychology. In addition to new
41 technologies for measurement, social neuroscience
42 methodology relies on the use of careful experimen-
43 tal designs, valid manipulations of psychological
44 states and processes, and careful inference and inter-
45 pretation. Here, we describe the most prominent
46 methods currently used in the field and briefly dis-
47 cuss their relative advantages as they relate to exper-
48 imental designs, issues of construct validity, and
49 psychological inference. A more detailed descrip-
50 tion of methods in neuroscience approaches to
51 social and personality psychology is provided by

Harmon-Jones and Beer (2009), and recent discus- 52
sions of inference and validity in social neuroscience 53
can be found in Amodio (2010a), Barrett (2009), 54
and Cacioppo et al. (2003). 55

Early studies taking a social neuroscience 56
approach primarily used peripheral physiological 57
measures, such as electrocardiogram (e.g., heart 58
rate), galvanic skin response (i.e., skin conductance, 59
a measure of sympathetic activation vis-à-vis palm 60
sweating), and electromyography (e.g., measures of 61
facial muscle activity related to emotional expres- 62
sions). More recently, neuroimaging measures have 63
become popular. The two most common neuroim- 64
aging techniques include functional magnetic reso- 65
nance imaging (fMRI), which measures the flow of 66
oxygenated blood in the brain, and electroencepha- 67
lography (EEG), which measures electrical activity 68
produced from the firing of neuron populations. 69
EEG is used to examine event-related potentials 70
(ERPs), which represent a burst of EEG activity in 71
response to a discrete event, such as a stimulus pre- 72
sentation or subject response. fMRI yields high spa- 73
tial resolution and thus is optimal for determining 74
the location of activity within the brain. But because 75
it assesses slow-moving blood flow, its temporal 76
resolution is slow. By contrast, EEG/ERP yields 77
high temporal resolution and is thus optimal for 78
assessing the timing of a neural process, but its spa- 79
tial resolution is comparatively poor. Given their 80
relative strengths, researchers may select fMRI or 81
EEG methods to suit their particular question, or 82
use both approaches in complementary studies 83
within a program of research. Neuroimaging and 84
psychophysiological approaches may also be com- 85
bined with measures of hormones, immune factors, 86
and DNA, for example, to provide convergent evi- 87
dence for a physiological process of interest. 88
However, as with traditional methods in social-per- 89
sonality psychology, the utility of these measures 90
depends on the quality of the question, the experi- 91
mental paradigm, and careful interpretation. 92

93 **Major Content Areas of Social Neuroscience**

94 In this section, we provide a broad review of the
95 social neuroscience literature in the areas of social
96 cognition and the self, attitudes, emotion and moti-
97 vation, intergroup relations, and individual differ-
98 ences. Although our review distinguishes these five
99 areas of research for convenience, their content
100 overlaps substantially. In each area, we will integrate
101 methods and levels of analysis, and we will discuss
102 how research in each area has contributed to social/
103 personality psychology theories.

1 *Social Cognition and the Self*

2 The earliest studies conducted at the intersection of
3 social psychology and neuroscience examined basic
4 processes of automaticity and control, as well as the
5 processing involved in perceiving the self and other
6 people. Together, these areas of research have laid
7 the foundation for social neuroscience studies of
8 more complex social-personality processes.

9 **AUTOMATIC AND CONTROLLED PROCESSING**

10 Theories of automatic and controlled processes rep-
11 resent a cornerstone of modern social cognition.
12 Mechanisms of automaticity and control also con-
13 stitute a central topic in cognitive psychology, and
14 thus a large body of cognitive neuroscience research
15 has been devoted to their elucidation. These pro-
16 cesses have been studied primarily in two different
17 broad research literatures on memory and cognitive
18 control.

19 *Automaticity*

20 Research on systems of learning and memory is par-
21 ticularly relevant to social-personality theories of
22 automaticity and implicit processes. Traditional
23 models of learning and memory often distinguish
24 between neural correlates of explicit (declarative)
25 and implicit (nondeclarative) memory processes,
26 and neuroscience research suggests these forms of
27 memory reflect distinct neural substrates (Squire &
28 Zola, 1996). Although implicit and automatic refer
29 to different properties of a process (i.e., degree of
30 awareness vs. degree of intentionality), implicit
31 forms of memory, such as classical fear conditioning
32 and procedural memory (i.e., skill or habit learning),
33 have important automatic characteristics and thus
34 are relevant to the present discussion. Studies of
35 nonhuman animals have identified the amygdala as
36 a critical structure in the learning and expression of
37 fear conditioning (LeDoux, Iwata, Cicchetti, &
38 Reis, 1988; Fendt & Fanselow, 1999), a role that has
39 been corroborated in fMRI studies of fear condi-
40 tioning in humans (LaBar, Gatenby, Gore, LeDoux,
41 & Phelps, 1998). The neuroscience research suggests
42 that automatic fear-related responses function some-
43 what independently from other types of automatic
44 responses.

45 Other research on procedural memory has
46 focused on the basal ganglia, a set of interconnected
47 structures that include the striatum (figure 6.2; cau-
48 date, putamen, and nucleus accumbens), globus pal-
49 lidus, and their dopaminergic inputs from the
50 midbrain (substantia nigra and ventral tegmentum).
51 Research has identified regions of basal ganglia as

being critical for implicit skill learning, such as when
52 subjects learn response associations that are embed-
53 ded implicitly in a task procedure (Foerde, Knowlton,
54 & Poldrack, 2006; Squire & Zola, 1996). These
55 forms of learning and memory more closely resem-
56 ble the type of automatic processes studied in the
57 social and personality psychology literature, in that
58 they drive actions that may be activated and imple-
59 mented without conscious awareness or intention. It
60 is notable that, in recent years, studies of economic
61 decisions and reward learning have also focused on
62 the role of basal ganglia, but as a substrate for error
63 prediction or the computation of “value” (e.g., Hare,
64 O’Doherty, Camerer, Schultz, & Rangel, 2008).
65 Research has yet to resolve these different interpreta-
66 tions. However, given the strong anatomical connec-
67 tivity between the basal ganglia with motor cortices
68 and PFC regions linked to goal representations, the
69 interpretation that the basal ganglia are centrally
70 involved in goal driven behaviors (including auto-
71 matic responses) remains plausible.

72
73 In the social cognition literature, automaticity is
74 often demonstrated on sequential priming tasks,
75 when a prime word is shown to facilitate the catego-
76 rization of an associated target word (Gaertner &
77 McLaughlin, 1983; Dovidio, Evans, & Tyler, 1986).
78 This idea was originally adapted from cognitive psy-
79 chology research on semantic associations between
80 words, such as “bread-butter” or “doctor-nurse”
81 (Meyer & Schaneveldt, 1971, 1976). In fMRI stud-
82 ies, semantic priming has been associated with activ-
83 ity in left posterior PFC (figure 6.3; e.g., Blaxton
84 et al., 1996; Demb et al., 1995; Raichle et al., 1994;
85 Wagner, Gabrieli, & Verfaellie, 1997) and temporal
86 cortex (Rissman, Eliassen, & Blumstein, 2003; 86
87 Schacter & Buckner, 1998; Squire, 1992), as well as

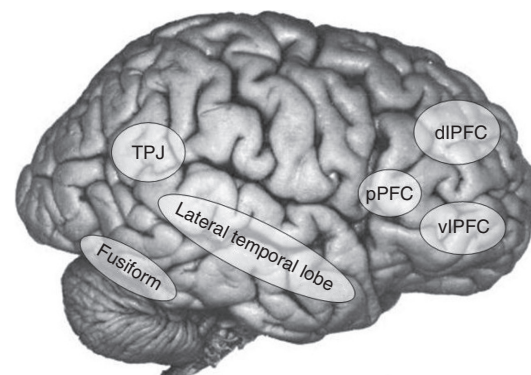


Fig. 6.3 Lateral aspect of the right hemisphere of the brain. dlPFC = dorsolateral prefrontal cortex, vlPFC = ventrolateral prefrontal cortex, pPFC = posterior prefrontal cortex, TPJ = temporo-parietal junction.

1 reduced activity in regions linked to attention (e.g.,
2 in the parietal cortex, figure 6.3; Gabrieli, 1998).
3 Given other research implicating the left PFC in
4 approach-related motivation and action tendencies
5 (Harmon-Jones, 2003), this pattern of neural cor-
6 relates suggests a link between automatic semantic
7 processes and goal-driven behavior (Amodio, 2008),
8 consistent with the idea that “thinking is for doing”
9 (Fiske, 1992).

10 Together, the neuroscience research on fear-con-
11 ditioning, procedural memory and reward, and
12 semantic associations has helped to distinguish dif-
13 ferent types of automatic processes. In doing so,
14 they clarify the functions of automaticity and shed
15 new light on how automatic processes operate and
16 change. These advances have already begun to
17 inspire new theories of implicit social cognition
18 (e.g., Amodio & Ratner, 2011b).

19 *Control*

20 Cognitive neuroscience research on mechanisms of
21 control has examined subjects’ brain activity while
22 they completed classic cognitive control tasks, such
23 as the Stroop color naming task, the Eriksen Flankers
24 Task, or the Go/No-Go task. Tasks such as these
25 typically include two main types of trials, which
26 require either a high or low degree of control. In one
27 set of trials, subjects make responses that are facili-
28 tated by either existing semantic associations (e.g.,
29 the color-naming Stroop task), perceptual cues (e.g.,
30 the Eriksen Flankers task), or expectancy (e.g., the
31 Go/No-Go). Responses on these trials are thought
32 to benefit from automatic processing. On other
33 trials, the participant must override the automatic
34 influence in order to deliver the correct task response.
35 Control-related behavior on these tasks has been
36 consistently associated with activity in the anterior
37 cingulate cortex (ACC) and PFC regions of the
38 brain.

39 In particular, the ACC responds to instances
40 when a prepotent, or automatic, response is incon-
41 sistent with the task goal, such as when the text of a
42 color word interferes with one’s goal to name the
43 ink color (Carter et al., 1998; MacDonald, Cohen,
44 Stenger, & Carter, 2000). Botvinick, Braver, Barch,
45 Carter, and Cohen (2001) proposed that the ACC
46 serves a *conflict monitoring* function, such that it is
47 involved in detecting conflict between alternative
48 response tendencies and, when conflict arises, it sig-
49 nals regions of the PFC involved in implementing
50 one’s intended response over other tendencies.
51 Consistent with the conflict monitoring hypothesis,
52 anatomical research on monkeys has revealed that

the ACC is strongly interconnected with motor 53
structures as well as PFC regions associated with 54
high-level representations of goals and actions 55
(Miller & Cohen, 2001). Some researchers have 56
extended this theorizing to suggest that ACC activ- 57
ity on control tasks may simply reflect a distress 58
signal or social pain (Eisenberger et al., 2003; 59
Inzlicht, McGregor, Hirsh, & Nash, 2009). 60
However, most cognitive control tasks that elicit 61
ACC activity do not involve distress or pain, com- 62
plicating this interpretation. Finally, several studies 63
have shown that conflict-related ACC activity 64
occurs in the absence of awareness (Berns, Cohen, 65
& Mintun, 1997; Nieuwenhuis, Ridderinkhof, 66
Blom, Band, & Kok, 2001), suggesting that it rep- 67
resents a preconscious component of control that 68
initiates the engagement of more deliberative com- 69
ponents. The findings of cognitive neuroscience 70
research on conflict monitoring and control have 71
been applied to questions regarding self-regulation 72
in social-personality contexts such as stereotyping 73
and prejudice (Amodio, Shah, Sigelman, Brazy, & 74
Harmon-Jones, 2004; Amodio, Kubota, Harmon- 75
Jones, & Devine 2006; Amodio, Master, Yee, & 76
Taylor 2008; Bartholow, Dickter, & Sestir, 2006), 77
individual differences related to anxiety and neu- 78
roticism (Amodio, Master, et al., 2008; Robinson, 79
Ode, Wilkowski, & Amodio, 2007), political orien- 80
tation (Amodio, Jost, Master, & Yee, 2007), religi- 81
osity (Inzlicht et al., 2009), and social exclusion 82
(Eisenberger et al., 2003). 83

84 Although several regions of the PFC are activated
85 during attempts at response control, a general pattern
86 has been observed in the literature whereby left-sided
87 PFC regions are associated with the implementation
88 of intended actions, whereas right-sided PFC activity
89 has been associated with the intentional inhibition of
90 action (Aron, 2007). It is notable that EEG and fMRI
91 research is almost always conducted on right-handed
92 participants, and observed hemispheric asymmetries
93 in patterns of control are likely related to the lateral
94 specialization of hand (and foot) dominance
95 (Harmon-Jones, 2006).

96 In sum, cognitive neuroscience research on control
97 has highlighted different subcomponents of
98 controlled processing. Although aspects of these
99 subcomponents have been considered in previous
100 social and personality psychology theories (e.g.,
101 Wegner, 1994; Wegener & Petty, 1997), the neuro-
102 science literature has provided important clarifications
103 of these components, as well as methods for assess-
104 ing aspects of control that function rapidly and
105 without conscious awareness.

1 PERCEPTION OF THE SELF AND OTHERS

2 *The Self*

3 The self is one of social-personality psychology's ear- 54
 4 liest and most enduring constructs (see Crocker & 55
 5 Canevello, chapter 11, this volume) and, not sur- 56
 6 prisingly, it was among the first constructs to be 57
 7 examined in neuroscientific studies of social psy- 58
 8 chological processes (e.g., Craik et al., 1999; Klein, 59
 9 Loftus, & Kihlstrom, 1996). Most of this research 60
 10 has examined brain activity associated with self- 61
 11 reflection and judgments about the self in compari- 62
 12 son to judgments of others (see also Uleman & 63
 13 Saribay, chapter 14, this volume). Using positron 64
 14 emission tomography (PET) to measure cerebral 65
 15 blood flow, Craik et al. (1999) found that judg- 66
 16 ments of trait words as relating to the self versus 67
 17 others were associated with large activations in the 68
 18 mPFC. In a similar study that used fMRI, Kelley 69
 19 et al. (2002) found that reflections on one's own 70
 20 traits activated a region of ventral mPFC to a greater 71
 21 extent than did reflecting on the traits of another 72
 22 person (in this study, George W. Bush). The finding 73
 23 of ventral mPFC activity in response to self-related 74
 24 judgments has been replicated in several studies 75
 25 (e.g., Gutchess, Kensinger, & Schacter, 2007; 76
 26 Heatherton et al., 2006; Kircher et al., 2002; Pfeifer,
 27 Lieberman, Dapretto, 2007; Saxe, Moran, Scholz,
 28 & Gabrieli, 2006; Schmitz, Kawahara-Baccus, &
 29 Johnson, 2004; Turner, Simons, Gilbert, Frith, &
 30 Burgess, 2008; Zhang, Lawson, Guo, & Jiang,
 31 2006; Zhu, Zhang, Fan, & Han, 2007). Studies of
 32 other aspects of the self, such as agency and self-
 33 discrepancies, have observed regions of the brain
 34 typically involved in more general aspects of visual
 35 perception, conflict monitoring, and cognitive control
 36 (Blakemore, Oakley, & Frith, 2003; Farrer
 37 et al., 2008). Thus, brain activations during self-
 38 related judgments likely pertain to the process of
 39 self-reflection, but not to other aspects of self-related
 40 processing.

41 Although neuroimaging research on the self is a
 42 popular area of research in social cognitive neurosci-
 43 ence, the findings of this area of research must be
 44 considered in light of some important interpreta-
 45 tional concerns. For example, in Kelley et al. (2002),
 46 the activity level of the mPFC during judgments of
 47 the self and other was lower than activity during a
 48 baseline condition, in which participants viewed a
 49 fixation cross on the computer display. That is, self-
 50 related processing is often associated with a deacti-
 51 vation in mPFC activity relative to baseline. This
 52 below-baseline effect is observed in most studies of
 53 the self, and it represents a major interpretational

problem. If mPFC activity reflects “self” processing,
 why would this region be more activated when sub-
 jects stare at a fixation point than when they are
 explicitly reflecting on the self? This observation has
 led some researchers to suggest that research partici-
 pants spontaneously focus on the self when at rest
 (to a greater extent than when instructed to think
 about the self), and this observation has prompted
 theories about a baseline “default” network of brain
 activity that supports thoughts about the self and
 other people (e.g., Gusnard, Akbudak, Shulman, &
 Raichle, 2001; Gusnard & Raichle, 2001). Although
 the idea that humans reflect on the self and others
 by default is interesting, it is inconsistent with daily
 diary research showing that, when probed at random
 points during the day and asked to report on what
 one was doing at the moment, participants rarely
 (8% of 4,700 responses) reported that they were
 engaged in some form of self-reflection
 (Csikszentmihalyi & Figurski, 1982). Ultimately, a
 construct like “the self” is a very broad and complex
 construct that may not be easily localized to a
 circumscribed set of neural structures.

Perceiving Faces

Information about conspecifics and social relation-
 ships is eminent in perception and cognition, and
 the initial stage of social processes often begins with
 face perception (see Uleman & Saribay, chapter 14,
 this volume). Research on visual perception suggests
 that some components of the visual system are spe-
 cialized for seeing faces, and these face-specialized
 processes have been localized in the fusiform gyrus
 in fMRI studies (figure 6.1; Kanwisher, McDermot,
 & Chun, 1997). Although the idea of a specialized
 face area has been debated, with some arguing that
 fusiform responses to faces reflect expertise rather
 than a “face module” (Haxby, Hoffman, & Gobbini,
 2000), the finding that this region responds to faces
 more than to other objects is consistent.

Faces are also known to elicit a characteristic
 ERP component that peaks 170 ms after the presen-
 tation of a face. This “N170” component is consis-
 tently larger to faces than to nonface stimuli that are
 matched on other visual dimensions (Bentin,
 Allison, Puce, Perez, & McCarthy, 1996; Carmel &
 Bentin, 2002), making it a valuable neural marker
 of the engagement of low-level face-specific percep-
 tion processes marking the encoding of facial fea-
 tures (e.g., Eimer, 2000). The N170 is believed to
 reflect activity in multiple temporal-occipital struc-
 tures linked to face processing (Deffke et al., 2007),
 including the fusiform (figure 6.1; e.g., Haxby, 105

1 Hoffman, & Gobbini, 2000; Puce, Allison, Gore,
2 & McCarthy, 1995) and other temporal regions
3 (e.g., Desimone, 1991; Perrett, Rolls, & Caan,
4 1982). Together, fMRI and ERP studies of face per-
5 ception have shown that faces are perceived very
6 quickly and at relatively low levels in the hierarchy
7 of visual processing. These studies also establish a
8 way to measure online face perception processes
9 without requiring behavioral or self-reported
10 responses.

11 *Mentalizing and Theory of Mind*

12 Once an object is perceived as a face, a perceiver
13 rapidly begins to evaluate and infer information
14 about the individual (Todorov, Said, Engell, &
15 Oosterhof, 2008). The process of inferring another
16 person's unique motives and perspectives is referred
17 to as *mentalizing*, which is the process underlying
18 one's *theory of mind* (Frith & Frith, 1999). Early
19 studies of mentalizing in the brain used PET to
20 measure activity involved in inferences about the
21 thoughts of other people or characters. These stud-
22 ies were motivated in part by an effort to under-
23 stand autism and its links to deficits in theory of
24 mind (Frith, 1989; Baron-Cohen, Tager-Flusberg,
25 & Cohen, 1993). In this line of research, theory of
26 mind is best characterized by tasks involving false
27 belief or deceptive intent—tasks on which success-
28 ful performance depends on one's ability to take
29 another person's perspective. In an early study on
30 this topic, Fletcher et al. (1995) examined brain
31 activity while normal subjects read a set of short sto-
32 ries. Mentalizing stories involved jokes or lies as a
33 literary device—that is, they made sense to the
34 extent that the reader understood that a character
35 was the victim of a lie or joke. Hence, the stories
36 required an understanding of a character's false
37 belief. Control stories did not rely on such devices,
38 but rather involved straightforward physical descrip-
39 tions. Although several brain regions were activated
40 by these stories, only the mPFC was uniquely more
41 active during the mentalizing stories. A similar set
42 of mentalizing activations were observed in another
43 study when subjects viewed movies of people show-
44 ing deceptive intent (Grezes, Frith, & Passingham,
45 2004).

46 Castelli, Happe, Frith, and Frith (2000) con-
47 nected their findings with the attribution literature
48 in social psychology by measuring brain activity
49 while participants viewed a set of videos inspired
50 by the famous Heider and Simmel (1944) anima-
51 tions, in which three shapes moved in an anthropo-
52 morphic fashion that implied human interaction.

The authors found that the viewing of this type of 53
animation also elicited mPFC activity, compared 54
with control videos in which the movement of the 55
shapes was not interpreted anthropomorphically. 56
More recent work has linked the process of disposi- 57
tional attribution to activity in the mPFC (Harris, 58
Todorov, & Fiske, 2005; cf. Heider, 1958). Since 59
these initial studies, a large body of research has 60
associated activity of the mPFC with a range of tasks 61
involving mentalizing and complex aspects of 62
person perception (Amodio & Frith, 2006; Frith & 63
Frith, 1999; Saxe, Carey, & Kanwisher, 2004). 64
These tasks also typically elicit activity in regions of 65
the superior temporal lobe (or temporal-parietal 66
junction) and the temporal poles—regions associ- 67
ated with the perception of biological motion and 68
to conceptual representations of social information, 69
respectively (Frith & Frith, 1999). Converging find- 70
ings from the developmental literature corroborate 71
the idea that mPFC development underlies the 72
emergence of theory of mind abilities in children 73
(Bunge, Dudukovic, Thomason, Valdy, & Gabrieli, 74
2002). 75

76 Since the initial finding that mentalizing acti-
77 vated regions of mPFC, researchers have asked
78 whether other forms of person perception might
79 involve the same brain regions. A series of studies by
80 Mitchell, Macrae, and colleagues proposed that
81 social-cognitive aspects of person perception, such
82 as the ascription of trait attributes to a person, might
83 also activate areas of mPFC (even if they do not nec-
84 essarily require mentalizing). For example, when
85 subjects judged noun-adjective word pairs that
86 described a person, compared with those describing
87 an inanimate object, activity was found in regions
88 of interest within the mPFC, as well as areas of the
89 temporal cortex and the temporal-parietal junction
90 (figure 6.3; Mitchell, Heatherton, & Macrae, 2002).
91 This pattern of activity has been seen across several
92 studies using similar tasks (e.g., Mitchell et al.,
93 2005; Mitchell, Macrae, & Banaji, 2006). Other
94 researchers have observed activity in similar regions
95 when simply viewing faces in an easy memory task
96 (Gobbini, Leibenluft, Santiago, & Haxby, 2004),
97 demonstrating that activity in this region to faces
98 does not necessarily imply the inference of traits.
99 Some research has found that viewing and making
100 trait judgments of unfamiliar faces or dissimilar
101 people is associated with activity in more dorsal
102 regions of the mPFC, whereas more familiar and/or
103 similar faces are associated with activity in more
104 ventral regions, near areas activated by self-reflection
105 (Gobbini et al., 2004; Mitchell et al., 2006). It is

1 notable, however, that the mPFC is a large region of
2 cortex, and the specific locus of person-related activ-
3 ity varies considerably across studies (Gilbert et al.,
4 2006).

5 As with neuroimaging studies of the self, activity
6 associated with forming impressions of both people
7 and inanimate objects is typically lower than base-
8 line mPFC activity (e.g., Mitchell, Macrae, &
9 Banaji, 2004). If the process of person perception is
10 truly located in the mPFC, then the data imply that
11 subjects engage more strongly in person perception
12 during baseline periods (i.e., viewing a fixation
13 cross) than when they are explicitly engaged in the
14 person perception process. This explanation assumes
15 that people naturally reflect on others when at rest
16 (presumably while also thinking about the self; but
17 see Csikszentmihalyi & Figurski, 1982). As with
18 interpretations of the mPFC as reflecting activation
19 of the self, this issue complicates the idea that the
20 mPFC is the neural substrate of person perception.
21 Other researchers have proposed that this region
22 serves a domain-general process of coordinating
23 one's responses with complex (e.g., externally
24 guided) plans (Amodio & Frith, 2006; Amodio
25 et al., 2006), such as when a research subject pre-
26 pares for an upcoming trial during intertrial inter-
27 vals (when mPFC activity is usually highest). This
28 interpretation of mPFC function helps to account
29 for the broad range of findings involving this
30 region.

31 Although the potential contribution of mapping
32 abstract constructs like “the self” and “social cogni-
33 tion” to theories of social-personality psychology
34 has yet to be established, fMRI research on mental-
35 izing, person perception, and the self has inspired
36 interesting debates about the processes through
37 which a person judges another's thoughts or inten-
38 tions. For example, “simulation” theory posits that
39 people consider how they would respond in the
40 other person's situation and then respond to that
41 person accordingly. “Theory” theory posits that
42 people have an implicit theory of how a person
43 would respond in a particular situation and, rather
44 than reflecting on the self, form their perceptions
45 based on this theory. As evidence for “theory” theory,
46 some researchers have noted that brain activity asso-
47 ciated with self and other judgments is related to
48 different regions of the mPFC, implying that people
49 do not activate representations of the self when con-
50 sidering the responses of others (Saxe, 2005). Other
51 researchers have argued in favor of simulation
52 theory, based on the observation that judgments of
53 similar or familiar others activate a region close to

areas activated by self-reflection, (Mitchell, 2005). 54
Although a lively debate, these interpretations are 55
tentative because they have relied primarily on 56
reverse inferences about the function of brain areas 57
(cf. Poldrack, 2006), assuming that these regions 58
of the mPFC truly represent the “self” and “social 59
cognition”. Indeed, the strongest evidence in 60
such debates comes from behavioral studies, which 61
provide more clearly interpretable data (Saxe, 62
2005). 63

Empathy 64

Empathy is broadly defined as concern for another's 65
welfare (Batson, 1991). By some definitions, empa- 66
thy involves experiencing another's perspective and 67
affective response (Lamm, Batson, & Decety, 2007; 68
see also Castano, chapter 17, this volume). As with 69
mentalizing and theory of mind, empathy is com- 70
plex and involves a broad set of neural and psycho- 71
logical processes associated with affect, perception, 72
social cognition, self-regulation, mimicry, and 73
action (Decety, 2010). Building on neuroscience 74
studies of mentalizing, research on the neural sub- 75
strates of empathy have focused primarily on the 76
role of the mPFC (Decety, 2010; Rameson & 77
Lieberman, 2009). Many studies have examined 78
empathy by measuring brain activity while a subject 79
views another person experiencing pain. For exam- 80
ple, Singer et al. (2004) used fMRI to measure brain 81
activity while participants experienced a painful 82
stimulus or viewed a loved one receiving the same 83
stimulus. A set of structures, including the rostral 84
ACC and anterior insula, were active in both condi- 85
tions, relative to baseline. Rostral ACC activity was 86
greater in response to observing a loved one's pain 87
among subjects with higher scores on a trait empa- 88
thy scale. Other research has found similar brain 89
regions to be more strongly activated when partici- 90
pants observed racial ingroups experiencing pain 91
than racial outgroups (Xu, Zuo, Wang, & Han, 92
2009). There are also suggestive findings from lesion 93
patient studies, in which damage to the ventrome- 94
dial PFC and ACC are associated with impaired 95
empathy (Shamay-Tsoory, Tomer, Berger, & 96
Aharon-Peretz, 2003). Given that the ACC is 97
involved in a wide range of processes involving 98
expectancy violation, these findings may reflect 99
some aspect of expectancy violation or concern 100
when either the self or another person is subjected 101
to pain, rather than suggesting that empathy is 102
related to the experience of pain per se. Overall, this 103
body of research has focused primarily on the brain 104
mapping of empathic processes. 105

1 Related to work on empathy, a “mirror neuron”
 2 system has been proposed as a brain network
 3 devoted to understanding other people through
 4 their actions (Iacoboni & Dapretto, 2006). The
 5 mirror neuron idea originated from single-unit
 6 recording in the macaque premotor cortex, in which
 7 the same neuron fired when the monkey moved its
 8 arm toward a food reward and when it watched an
 9 experimenter move its arm toward the reward
 10 (Gallese, Fadiga, Fogassi, & Rizzolatti, 1996).
 11 “Mirror neuron” is not a literal term, in the sense
 12 that no single neuron can be described as providing
 13 a mirroring function. Rather, “mirror neurons” refer
 14 loosely to areas of the brain that are activated both
 15 when an individual observes the behavior of another
 16 person, and when one performs the same behavior.
 17 Brain regions implicated in the “mirror neuron”
 18 network include premotor cortex, inferior frontal
 19 cortex, superior temporal sulcus, anterior insula,
 20 and the amygdala (Iacoboni & Dapretto, 2006;
 21 Rizzolatti & Sinigaglia, 2010), although the pat-
 22 terns and locations of activity in these regions vary
 23 considerably from study to study. Although the
 24 notion that we relate to other people by represent-
 25 ing their actions and mental states in the same way
 26 we represent our own actions and states has intuit-
 27 ive appeal, more recent theoretical analyses have
 28 questioned the plausibility of mirror neurons as
 29 mechanisms of action understanding (Decety, 2010;
 30 Hickock, 2009; Saxe, 2005; Vivona, 2009). Aside
 31 from questions about the neural substrates, the fact
 32 that so many social interactions often require comple-
 33 mentary responses (e.g., when conversing or
 34 dancing), rather than mimicry, calls into question
 35 the idea that human social behavior is rooted in
 36 a mirroring system. Hence, more research will be
 37 needed to assess the utility of the mirror neuron
 38 idea.

39 *Humanization*

40 Humanization refers to the process of seeing another
 41 person as possessing the characteristics unique to
 42 the human species, including the human rights
 43 associated with being a member of society. Hence,
 44 dehumanization refers to the process of denying a
 45 person these qualities (Haslam, 2006). High status
 46 groups and members of one’s ingroup are typically
 47 perceived as possessing these qualities; low-status
 48 individuals and members of the outgroup are often
 49 seen as lacking these qualities (Leyens et al., 2001;
 50 Leyens et al., 2003). The process of “humanization”
 51 is also associated with empathy and mentalizing,
 52 but whereas humanization processes typically

applies only to people, empathy and mentalizing 53
 may also relate to nonhumans and inanimate 54
 objects, as a form of anthropomorphism (Epley, 55
 Waytz, & Cacioppo, 2007). Research on dehuman- 56
 ization has connected the neuroscience work on 57
 mentalizing and the mPFC to the topic of inter- 58
 group relations (Harris & Fiske, 2009). For exam- 59
 ple, a study by Harris and Fiske (2006) demonstrated 60
 that mPFC activity was greater when participants 61
 viewed pictures of valued others (e.g., members of 62
 the ingroup, people of high social status) compared 63
 with “dehumanized” individuals, such as drug 64
 abusers and homeless people. 65

Why the mPFC? 66

What is the significance of the mPFC as it relates to 67
 the self, person perception, and mentalizing? 68
 Mitchell argued (2009) that the fact that these 69
 “social” processes all activate the same general brain 70
 region indicates that social psychology is a “natural 71
 kind,” meaning that social psychological processes 72
 have a unique and privileged place in neural activity. 73
 Taking a different approach to this issue, Amodio 74
 and Frith (2006) considered the neuroanatomical 75
 properties of the mPFC in its relation to social cog- 76
 nition. They noted that the mPFC is a highly 77
 interconnected region of brain uniquely situated 78
 to integrate information about internal processes 79
 (e.g., motor responses, visceral states) and higher- 80
 level representations of goals, reward contingencies, 81
 and complex expectancies. In their analysis, 82
 the mPFC is thought to be involved in any 83
 processes that involve a complex interplay of inter- 84
 nal states and the tracking of abstract external 85
 contingencies for one’s response—a type of process 86
 exemplified by social cognition. According to 87
 this view, the mPFC is not the neural instantiation 88
 of the self or social cognition, and “social psychol- 89
 ogy” is not a natural kind in the brain. Rather, 90
 thinking about the self and others is most likely 91
 to involve the neural and cognitive processes sup- 92
 ported by this region. More research is needed to 93
 understand the significance of the mPFC for social 94
 cognition. 95

To date, social neuroscience research on social- 96
 cognitive processes, as has those reviewed above, has 97
 primarily focused on brain mapping. In this way, it 98
 has been primarily exploratory and used to elucidate 99
 the understanding of neural function. How has this 100
 research informed social-personality psychology? 101
 Neuroscience studies of cognitive control have 102
 informed social psychological theories by delineating 103
 different components of the self-regulatory process. 104

1 Similarly, neuroscience research on learning and
 2 memory systems has provided a useful framework
 3 for understanding automatic and implicit forms of
 4 social cognition (Amodio, 2008; Amodio & Ratner,
 5 2011b). By contrast, the contribution of extant
 6 brain-mapping studies of the self and person percep-
 7 tion for theories of social-personality psychology
 8 remains less clear.

9 Attitudes

10 The attitude is a foundational construct in the field
 11 of social psychology (Thurstone, 1928; see also
 12 Ajzen, chapter 15 this volume), and attitudes were
 13 the subject of the earliest psychophysiological stud-
 14 ies of social psychological processes. Rankin and
 15 Campbell (1955) measured changes in skin con-
 16 ductance levels (i.e., galvanic skin response) while
 17 White participants in their study interacted with
 18 White and Black experimenters. Skin conductance
 19 responses, which reflect activity of the sympathetic
 20 nervous system, were interpreted as instances of
 21 heightened anxiety. Although participants in their
 22 study reported similar liking for the two experi-
 23 menters, they exhibited larger skin conductance
 24 responses to Black than White experimenters on
 25 average. The authors interpreted the results as evi-
 26 dence for negative attitudes toward African
 27 Americans—the first evidence of implicit racial
 28 bias.

29 The attitude construct is complex, and although
 30 it is often defined simply as liking/disliking, the psy-
 31 chological and physiological processes that give rise
 32 to an attitude are very complicated. The classic
 33 tripartite model of attitudes includes cognitive,
 34 affective, and behavioral components. However, at
 35 the physiological level of analysis, an attitude likely
 36 corresponds to several other processes, such as moti-
 37 vational tendencies, action representations, changes
 38 in attention, and basic motor tendencies, among
 39 others. For this reason, it is very difficult—and per-
 40 haps inadvisable—to map the high-level construct
 41 of an attitude onto specific physiological systems.
 42 Indeed, there are ongoing debates on whether an
 43 “attitude” is properly conceptualized as an abstract
 44 appraisal or a psychological mechanism (Fazio,
 45 2007; Harmon-Jones, Harmon-Jones, Amodio, &
 46 Gable, in press; Schwarz, 2007). To the extent that
 47 an attitude is not defined as a mechanism, a neuro-
 48 science analysis of attitudes may not be useful. If
 49 a researcher is simply interested in assessing a sub-
 50 ject’s affective response to an object, peripheral
 51 physiological responses may provide more useful
 52 indicators.

The use of physiological methods to examine
 attitudes was developed significantly by the research
 of Cacioppo and his colleagues. These methods
 allowed researchers to assess changes in a partici-
 pants’ response unobtrusively and often uncon-
 sciously, using a range of physiological indicators
 including skin conductance (Cacioppo & Sandman,
 1978), heart rate (Cacioppo, Sandman, & Walker,
 1978), facial EMG (Cacioppo & Petty, 1979), EEG
 spectral power (Cacioppo, Petty, & Snyder, 1979),
 and ERPs, among others. This early social psy-
 chophysiology research on attitudes focused on the
 psychological processes involved in persuasion,
 which leads to attitude change. Of special interest
 was the role of information processing. For example,
 Cacioppo and colleagues focused on asymmetries in
 parietal cortical activity associated with semantic
 versus nonsemantic processing of a stimulus (e.g.,
 Cacioppo, Petty, & Quintanar, 1982). This focus
 contrasts with other researchers’ focus on hemi-
 spheric asymmetries in frontal cortical brain regions
 that were associated with emotion and motivation
 (Davidson & Fox, 1982). Other research examined
 subtle and implicit affective responses to pro- versus
 counterattitudinal messages by measuring subtle
 changes in facial expressions during message
 processing (Cacioppo & Petty, 1979). Cacioppo
 and Petty’s early use of psychophysiological meth-
 ods to study attitudes and persuasion blazed the
 trail for future neuroscience approaches in social
 and personality psychology.

Peripheral physiological measures have been
 especially useful for assessing the affective compo-
 nent of attitudes, by way of assessing arousal (e.g.,
 Rankin & Campbell, 1955). However, skin con-
 ductance measures are limited because they cannot
 distinguish between positive versus negative
 responses. In order to draw stronger inferences
 about the valence of a physiological response, facial
 EMG may be used to measure activity in muscle
 groups associated with different facial expressions
 linked to emotion (Dimberg, 1982; Cacioppo &
 Petty, 1979). Activity of the corrugator supercillii
 muscle, located just above and running parallel to
 the brow, is commonly described as brow-furrow-
 ing, and it is associated with many forms of negative
 affect, including anger and disgust. Activity of the
 zygomaticus major muscle, which runs across
 the cheek from the corners of the mouth to the
 cheekbones, is related to smiling, and it is associated
 with many forms of positive affect. Thus, EMG
 provides information about an emotional expres-
 sion, from which researchers may infer valence,

1 approach/withdrawal orientation, or any other psy-
2 chological process linked to a facial expression.

3 Although emotional expressions can usually be
4 observed directly, EMG allows researchers to quan-
5 tify their activity with precision, providing continu-
6 ous online assessments of affective response without
7 requiring an overt response from the participant.
8 Facial EMG has also been shown to be sensitive to
9 microexpressions—changes in facial muscle activity
10 that are imperceptible to the naked eye (Cacioppo,
11 Bush, & Tassinari, 1992). Other research has used
12 EMG to measure subtle emotional responses to
13 subliminally presented stimuli. Dimberg, Thunberg,
14 and Elmehed (2000) demonstrated changes in
15 EMG measures of the zygomaticus and corrugator
16 responses to subliminally presented pictures as a
17 result of mere exposure. Harmon-Jones and Allen
18 (2001) also used facial EMG to demonstrate that
19 repeated presentations of a neutral stimulus caused
20 an increase in zygomatic activity, as well as more
21 positive attitudes, as a way to examine the role of
22 affect in the mere exposure effect.

23 NEURAL ASSESSMENTS OF ATTITUDES

24 The first brain-based assessments of attitudes used
25 measures of frontal cortical activity to assess affect-
26 tive responses to different stimuli and situations.
27 This earlier research showed that responses to posi-
28 tive stimuli were associated with greater left-sided
29 PFC activity, whereas responses to aversive stimuli
30 were associated with greater right-sided PFC activ-
31 ity (Davidson, Ekman, Saron, Senulis, & Friesen,
32 1990; Davidson & Fox, 1982). Subsequent research
33 by Harmon-Jones and his colleagues (Harmon-
34 Jones, 2003; Harmon-Jones & Allen, 1998) clar-
35 ified that, at the physiological level of analysis,
36 responses to positive and negative stimuli were orga-
37 nized according to approach-withdrawal processes
38 rather than to positive-negative valence. This
39 research raises questions about whether the concept
40 of “valence” is meaningfully represented at the
41 neural or physiological level of analysis.

42 ERP methods have also been used to examine
43 attitudes. Early psychophysiology research observed
44 that unexpected stimuli, such as a loud tone among
45 a series of soft tones in an oddball task, reliably elicit
46 a positive-going ERP response approximately 300–
47 500 ms after the deviant stimulus, maximal over
48 parietal scalp sites (Squires, Donchin, Herning, &
49 McCarthy, 1977; Sutton, Braren, Zubin, & John,
50 1965). This ERP component was named the P300,
51 for its positive polarity and its approximate peak at
52 300 ms. However, given that the latency of its peak

varies considerably across tasks, it is often referred
53 to as the P3 (because it is the third positive-going
54 peak following the stimulus) or *late positive potential*
55 (LPP). The P3 was interpreted as reflecting the
56 engagement of attention, associated with a surprise
57 response, and the updating of one’s mental set
58 (Donchin, 1981). More recent research has linked
59 the LPP to activity of norepinephrine systems in
60 response to an unexpected event (Nieuwenhuis,
61 Aston-Jones, & Cohen, 2005), suggesting the LPP
62 reflects a complex set of processes associated with
63 attention and arousal.
64

Cacioppo, Crites, Berntson, and Coles (1993)
65 noted that the LPP component, in conjunction
66 with the oddball task, could be used to assess indi-
67 viduals’ evaluations of attitude objects. For example,
68 the authors found that a negative “oddball” stimu-
69 lus that appeared within a series of positive objects
70 elicited an LPP response, much like a high-pitched
71 tone amongst a series of low-pitched tones. Other
72 research showed that the magnitude of the evalua-
73 tive oddball effect varied as a function of its evalua-
74 tive extremity, such that extremely negative trait
75 words elicited larger LPP amplitudes than moder-
76 ately negative trait words, when they were embed-
77 ded within a series of positive words (Cacioppo,
78 Crites, Gardner, & Berntson, 1994; Crites &
79 Cacioppo, 1996; Ito, Larsen, Smith, & Cacioppo,
80 1998). These LPP effects were also found to be sen-
81 sitive to individual differences in attitudes, such that
82 they were responsive to subjects’ ideographically
83 liked versus disliked objects (Crites & Cacioppo,
84 1996). The LPP/oddball method of attitude assess-
85 ment has even been shown to reveal people’s true
86 attitudes when they attempted to misreport the atti-
87 tude (Crites, Cacioppo, Gardner, & Berntson,
88 1995) or when implicit evaluations differed from
89 explicit task instructions (Ito & Cacioppo, 2000).
90 This technique of examining attitudes by measuring
91 the LPP during the oddball task has since been used
92 to assess a variety of attitudes, including intergroup
93 attitudes (as described below).
94

Hemispheric asymmetries in ERP responses have
95 also been related to attitudes. In one study, Cunning-
96 ham, Espinet, DeYoung, and Zelazo (2005) mea-
97 sured the LPP while participants made evaluative
98 (good vs. bad) and nonevaluative (abstract vs. con-
99 crete) judgments about socially relevant concepts.
100 The concepts were then rated for goodness and bad-
101 ness. Concepts rated as “bad” caused greater LPPs
102 over the right frontal hemisphere, while concepts
103 rated “good” caused greater LPPs over the left
104 frontal hemisphere. Similarly, van de Laar, Licht, 105

1 Franken, and Hendriks (2004) found that cocaine-
 2 addicted individuals, but not nonaddicted individu-
 3 als, showed larger positive slow wave responses over
 4 the left (but not right) frontal cortex to cocaine-
 5 related photographs as compared to neutral photo-
 6 graphs. Along similar lines, Gable and Harmon-Jones
 7 (2010) observed greater left than right frontal LPPs
 8 to pictures of desserts, and Ohgami et al. (2006)
 9 found ERP evidence that reward cues caused greater
 10 left frontal cortical activity.

11 With the introduction of functional neuroimaging
 12 tools with good spatial resolution, such as fMRI,
 13 researchers have begun to explore the specific neural
 14 systems that underlie attitude processes. Whereas
 15 peripheral physiology measures provide indicators of
 16 preference, studies of brain activity linked to atti-
 17 tudes often seek to address questions about mecha-
 18 nism. This raises the question of whether an attitude
 19 represents a mechanism, per se, or an appraisal of an
 20 object. As noted above, a neuroscience analysis of
 21 the attitude construct is informative to the extent
 22 that it reflects mechanism. Initial fMRI studies of
 23 attitudes focused on brain mapping. For example,
 24 Cunningham, Johnson, Gatenby, Gore, and Banaji
 25 (2003) measured brain activity while subjects viewed
 26 and evaluated pleasant versus unpleasant words.
 27 They observed greater amygdala activity to negative
 28 than positive words. However, later findings sug-
 29 gested this effect was driven by the arousal associated
 30 with particular words and not the valence, per se (cf.
 31 Whalen, 1998), whereas the valence of the words
 32 was associated with activity in the insula, among
 33 other regions (Cunningham, Raye, & Johnson,
 34 2004). Although these findings do not address psy-
 35 chological questions about attitudes, they begin to
 36 map out the neural correlates of evaluation.

37 Research in neuroeconomics has examined how
 38 people evaluate goods and money and how they
 39 process potential rewards (Fehr & Camerer, 2007;
 40 Glimcher, Camerer, Fehr, & Poldrack, 2009). By
 41 engaging subjects in economic games, often with
 42 real cash rewards, these studies of attitudes provide
 43 a higher degree of ecological validity than measures
 44 of word judgments. Much of this research has found
 45 greater activation in regions of the basal ganglia,
 46 such as the ventral striatum and nucleus accumbens,
 47 when participants viewed high-value stimuli. Similar
 48 brain activations have been observed during the
 49 evaluation of people (Delgado, Frank, & Phelps,
 50 2005). Although some researchers have interpreted
 51 these activations as reflecting the neural computa-
 52 tion of value, the broader literature on basal ganglia
 53 function suggests that these neural activations reflect

the learning and coordination of motor plans linked
 to reward-driven actions (Alexander, DeLong, &
 Strick, 1986). This line of research on evaluations is
 useful because it links evaluation processes more
 directly to goals and behavior, whereas contempo-
 rary social psychology research on attitudes focuses
 on the representation of attitudes in cognition, with
 less emphasis on links to behavior.

Emotion and Motivation

Affect and emotion range from relatively low-level
 aspects of reward and punishment; to discrete basic
 emotions, such as joy, anger, and fear; to highly
 complex emotion processes such as guilt, jealousy,
 compassion, and schadenfreude (see Clore &
 Robinson, chapter 13, this volume), and they are
 almost always intertwined with motivation. Al-
 though some research has attempted to study
 brain activations unique to complex emotional
 responses (e.g., Moll, Zahn, Oliveira-Souza,
 Krueger, & Grafman, 2005), interpretation of such
 findings is difficult (Barrett & Wager, 2006). In-
 deed, a meta-analysis of brain activations in response
 to manipulations of different emotional states failed
 to reveal clear patterns of activity for specific emo-
 tional states (Phan, Wager, Taylor, & Liberzon, 2002).
 Although one could interpret this finding as evi-
 dence against mechanistic distinctions among dif-
 ferent emotions, this pattern of null results likely
 reflects the limitations of trying to assess fine-
 grained processes using comparatively coarse mea-
 sures (e.g., fMRI) and methods (e.g., meta-analysis).
 Like attitudes, emotion constructs correspond to a
 complex set of more basic processes at the neuro-
 physiological level of analysis and, therefore,
 common distinctions in the subjective experience of
 emotions should not cleanly map onto the organi-
 zation of the brain and physiology. For this reason,
 most progress has been made in studying the neural
 processes associated with lower-level emotion pro-
 cesses, such as motivational, attentional, and auto-
 nomic processes, and basic mechanisms underlying
 fear and reward responses.

At the physiological level of analysis, emotional
 processes overlap substantially with motivational
 processes. In the affective neuroscience literature, it
 is generally assumed that approach motivation and
 responses to rewards involve a positive affective
 system, whereas avoidance motivation and responses
 to punishments involve a negative affective system.
 Indeed, motivational dispositions toward approach
 and withdrawal are often associated with emotions.
 However, an emotion is not a single “thing,” but

1 rather a multicomponent process made up of basic
2 processes such as feelings of pleasure or displeasure,
3 facial/body expressions, particular appraisals, and
4 particular action plans and activation states (Frijda,
5 1993). Moreover, these components are not per-
6 fectly correlated with one another (Lang, 1995).

7 Approach and withdrawal motivational processes
8 likely involve neural *systems* rather than specific
9 brain structures. However, a systems-level analysis
10 of approach-withdrawal motivation has yet to be
11 thoroughly investigated due to the empirical diffi-
12 culties of mapping the precise timecourse of these
13 microprocesses. In addition, much of the research
14 on the neuroscience of emotion and motivation has
15 proceeded under a “brain mapping” approach rather
16 than a “hypothesis testing” approach, as defined
17 earlier. Consequently, this review will focus on brain
18 regions that have received the most research atten-
19 tion. These are the amygdala, nucleus accumbens/
20 ventral striatum, the orbitofrontal cortex, and the
21 prefrontal cortex. While considering the reviewed
22 research, it is important to keep in mind the diffi-
23 culty of making one-to-one associations between
24 psychological processes and physiological processes.
25 For example, if fMRI data indicate amygdala activ-
26 ity in response to viewing a face, it is almost impos-
27 sible to claim that this activation reflects a certain
28 psychological variable like fear, given that different
29 subnuclei of the amygdala support a wide range of
30 psychological variables, including uncertainty
31 (Whalen, 1998), positive affect (Anderson et al.,
32 2003), and motivational relevance (Cunningham,
33 Van Bavel, & Johnsen, 2008).

34 *Perception of Motivational Relevance*

35 Many of the stimuli that arouse motivation are per-
36 ceived via the visual or auditory system. Novel and
37 significant events attract our attention and engage
38 the orienting response, and they are typically associ-
39 ated with approach and avoidance behavior
40 (Thorndike, 1911) or the emotion evoked by an
41 event (Bradley, 2009; Maltzman, 1979). The pro-
42 cesses of orienting and attending have been posited
43 to “stem from the activation of defensive and appet-
44 itive motivational systems that evolved to protect
45 and sustain the life of the individual” (Bradley,
46 2009, p. 1). In this way, attention, emotion, and
47 motivation are inextricably linked. Emotion is often
48 theoretically defined, fundamentally, as a disposi-
49 tion to act, or to behave effectively to events that
50 threaten or promote life (Frijda, 1986; Lang, 1985).
51 The associated motivational tendencies are realized
52 in general systems of approach and avoidance, with

approach processes often acting to promote survival 53
and avoidance processes often acting to prevent 54
threats to well-being. Some theorists suggest that 55
judgments of positivity reflect approach motivation, 56
judgments of negativity reflect avoidance motiva- 57
tion, and judgments of arousal index the intensity 58
of activation or motivation (Bradley, 2009). 59
Although this may often be the case, the relation- 60
ship between emotional valence and motivational 61
direction (i.e., approach motivation is positive) is 62
not always so direct. For instance, anger, a nega- 63
tively valenced emotion, is often associated with 64
approach motivation (Carver & Harmon-Jones, 65
2009). We return to this point later. 66

Fear Processing

67 As noted above, human neuroimaging research has 68
converged with the animal research to reveal that the 69
amygdala is important for processing fear. For 70
instance, the amygdala region is more activated by a 71
neutral stimulus paired with an aversive event (con- 72
ditioned stimulus) compared to another neutral 73
stimulus that does not predict an aversive event 74
(LaBar et al., 1998). Moreover, amygdala activation 75
correlates with the degree of skin conductance 76
response (an indication of arousal) to the condi- 77
tioned stimulus (LaBar et al., 1998). Going beyond 78
these correlations, research has revealed that patients 79
with lesions of the right, left, or bilateral amygdala 80
do not demonstrate a conditioned response as mea- 81
sured by skin conductance, even though they 82
respond normally to the unconditioned (aversive) 83
stimulus (Bechara et al., 1995). These results fit well 84
with the animal research demonstrating that the 85
amygdala—specifically, the central nucleus of the 86
amygdala—plays a critical role in fear conditioning. 87

88 Interestingly, although the amygdala is impor- 88
tant for the acquisition of fear, as measured implic- 89
itly by skin conductance to fear-conditioned stimuli, 90
it does not appear to be important for the acquisi- 91
tion of fear learning measured explicitly. Individuals 92
who suffer bilateral amygdala damage acquire 93
explicit knowledge about the relationship between 94
the conditioned stimulus and the aversive uncondi- 95
tioned stimulus (Gazzaniga, Irvy, & Mangun, 96
2002). This type of explicit knowledge is associated 97
with the hippocampus (Squire & Zola-Morgan, 98
1991). Individuals with a damaged hippocampus 99
but intact amygdala show normal skin conductance 100
response to conditioned stimuli but no explicit 101
knowledge of the relationship between the condi- 102
tioned stimulus and unconditioned stimulus 103
(Bechara et al., 1995). 104

1 More recent human neuroimaging research has
 2 revealed that the amygdala becomes activated in
 3 response to a variety of emotive stimuli in addition
 4 to fear-provoking ones (Whalen, 1998). For
 5 instance, experiments have revealed that positive
 6 stimuli also evoke greater amygdala activity than
 7 neutral stimuli (Breiter et al., 1996). Other studies
 8 have independently manipulated valence and inten-
 9 sity and found that amygdala activity is more associ-
 10 ated with processing affective intensity than with
 11 processing any specific valence (Anderson et al.,
 12 2003). Consistent with results obtained from these
 13 studies, Whalen (1998) observed that the amygdala
 14 is generally associated with vigilance to motivation-
 15 ally relevant stimuli (see also Anderson & Phelps,
 16 2001; Cunningham et al., 2008). Although these
 17 findings appear to contradict the fear conditioning
 18 literature, they likely reflect the role of a different
 19 part of the amygdala—the basal nucleus—which
 20 supports the initiation of appetitive and goal-driven
 21 responses to stimuli, such as rewards (Holland &
 22 Gallagher, 1999; LeDoux, 2000). Contemporary
 23 fMRI methods lack the resolution to sufficiently
 24 discern the subnuclei in the amygdala, and this
 25 sometimes leads to confusion over interpretations of
 26 the amygdala as relating to motivational and emo-
 27 tional responses such as fear (e.g., freezing), with-
 28 drawal, and approach.

29 Along these lines, research has revealed that
 30 extraversion is correlated directly with amygdala
 31 activation to positive (relative to negative) emo-
 32 tional pictures and that neuroticism is correlated
 33 directly with amygdala activation to negative (rela-
 34 tive to positive) emotional pictures (Canli et al.,
 35 2001). Extraversion has also been found to correlate
 36 directly with amygdala activation to happy (relative
 37 to fearful) faces (Canli, Sivers, Whitfield, Gotlib, &
 38 Gabrieli, 2002). Similarly, individual differences in
 39 promotion focus (sensitivity to gains) is associated
 40 with amygdala activation to positive (relative to
 41 negative) word stimuli, whereas individual differ-
 42 ences in prevention focus (sensitivity to losses) is
 43 associated with amygdala activation to negative (rela-
 44 tive to positive) word stimuli (Cunningham, Raye,
 45 & Johnson, 2005). These findings demonstrate that
 46 the amygdala is responsive to both appetitive and
 47 aversive stimuli, although this pattern likely reflects
 48 the roles of different subnuclei of the amygdala.

49 ***Reward Processing***

50 In humans, fMRI research has linked the anticipa-
 51 tion of rewards to activity in regions of the basal
 52 ganglia, such as the nucleus accumbens. For

instance, Knutson, Wimmer, Kuhnen, and
 Winkielman (2008) found that anticipation of
 viewing rewarding stimuli (e.g., erotic female nudes,
 viewed by heterosexual men) increased nucleus
 accumbens activity and financial risk taking.
 Nucleus accumbens activity also increased in antici-
 pation of making a risky decision, that is, a relatively
 high-risk (\$1.00) as compared to a low-risk (\$0.10)
 financial gamble. Moreover, the risk taking was par-
 tially mediated by increases in nucleus accumbens
 activation.

Research on dopamine and the nucleus accum-
 bens have revealed differential mechanisms associ-
 ated with the “wanting” and “liking” of a reward.
 Specific subregions of the nucleus accumbens, in
 combination with specific neurotransmitters, are
 involved in “liking” or postgoal positive affect. For
 instance, microinjection of morphine, which acti-
 vates opioid receptors, into posterior and medial
 regions of the accumbens shell increases positive
 affective reactions to sweet tastes (Peciña & Berridge,
 2000). Other research has revealed that the nucleus
 accumbens is critical in regulating effort-related
 functions, such that lever pressing schedules that
 require minimal work are unaffected by accumbens
 dopamine depletions, whereas lever-pressing sched-
 ules that require greater work are impaired by
 accumbens dopamine depletions (Salamone,
 2007).

The orbital frontal cortex (figure 6.1; OFC) is
 another brain region that is widely implicated in
 studies of reward contingencies, such as in reversal
 learning (Schoenbaum, Setlow, & Ramus, 2003),
 and in the self-monitoring of emotional responses.
 In reversal learning, an animal is taught that
 responding to one cue produces reward, whereas
 acting similarly to another cue produces nonreward
 or punishment. After the animal learns to respond
 correctly, the experimenter switches the cue-out-
 come associations, and the animal must learn to
 change its behavior. During cue-outcome learning
 across reversals, the OFC is activated (O’Doherty,
 Critchley, Deichmann, & Dolan, 2003).

The reversal-learning function of the OFC relates
 to the process of self-monitoring in humans. Self-
 monitoring is defined as the ability to evaluate one’s
 behavior in the moment in reference to higher-order
 goals or the reactions of other people (Prigatano,
 1991; Stuss, 1991; Stuss & Benson, 1984). This
 is the process “by which individuals evaluate
 their behavior in the moment to make sure that
 the behavior is consistent with how they want
 to behave and how other people expect them to

1 behave” (Beer, John, Scabini, & Knight, 2006,
 2 p. 872). Individuals with OFC damage have an
 3 impaired ability to prioritize solutions to interpersonal
 4 problems (Saver & Damasio, 1991), a tendency
 5 to greet strangers in an overly familiar manner
 6 (Rolls, Hornak, Wade, & McGrath, 1994), and to
 7 behave with disruptive manners in hospital settings
 8 (Blair & Cipolotti, 2000). They also tease strangers
 9 inappropriately and are more likely to disclose
 10 unnecessary or inappropriate personal information
 11 when answering questions (Beer, Heerey, Keltner,
 12 Scabini, & Knight, 2003; Kaczmarek, 1984). This
 13 self-monitoring perspective on OFC is consistent
 14 with the previously reviewed research on the emotional
 15 functions of this region when functional
 16 accounts of emotion are considered.

17 *PFC Asymmetries Associated with* 18 *Emotion and Motivation*

19 The asymmetric involvement of prefrontal cortical
 20 regions in positive affect (or approach motivation)
 21 and negative affect (or withdrawal motivation) was
 22 suggested over 70 years ago by observations of persons
 23 who had suffered damage to the right or left
 24 anterior cortex (Goldstein, 1939). Later research
 25 supported these observations using the Wada test,
 26 which involves injecting amytal, a barbiturate derivative,
 27 into one of the internal carotid arteries, suppressing
 28 the activity of one hemisphere. Amytal injections
 29 in the left side were found to produce depressed
 30 affect, whereas injections in the right side
 31 produced euphoria (Alema, Rosadini, & Rossi,
 32 1961; Perria, Rosadini, & Rossi, 1961; Rossi &
 33 Rosadini, 1967; Terzian & Cecotto, 1959). These
 34 effects were interpreted as reflecting the release of
 35 one hemisphere from contralateral inhibitory influences.
 36 According to this view, activation in the right
 37 hemisphere, when not inhibited by the left hemisphere,
 38 caused depression; and disinhibited left
 39 hemisphere caused euphoria.

40 Subsequent studies appeared to confirm these
 41 results, finding that persons who had suffered left
 42 hemisphere damage or lesions tended to show
 43 depressive symptoms (Black, 1975; Gasparrini,
 44 Satz, Heilman, & Coolidge, 1978; Gainotti, 1972;
 45 Robinson & Price, 1982), whereas persons who had
 46 suffered right hemisphere lesions tended to show
 47 manic symptoms (Gainotti, 1972; Robinson &
 48 Price, 1982; Sackeim et al., 1982). Other research
 49 has revealed asymmetries underlying appetitive and
 50 avoidant behaviors in nonhuman animals, in species
 51 ranging from great apes and reptiles (Deckel,
 52 Lillaney, Ronan, & Summers, 1998; Hopkins,

Bennett, Bales, Lee, & Ward, 1993) to chicks 53
 (Güntürkün et al., 2000), amphibians (Rogers, 54
 2002), and spiders (Ades & Ramires, 2002). 55

56 More recent research suggests that in humans
 57 these affect-related asymmetric activations are often
 58 specific to the frontal cortex. This research often
 59 uses asymmetric activation in right versus left frontal
 60 cortical areas as a dependent variable, usually
 61 assessed by EEG recordings. Frontal cortical asymmetry
 62 is assessed by comparing activation levels
 63 between homologous areas on the left and right
 64 sides. Difference scores are widely used in this
 65 research, and their use is consistent with the amytal
 66 and lesion research described above that suggests
 67 that asymmetry may be the key variable, with one
 68 hemisphere inhibiting the other hemisphere.

69 Much of this evidence has been obtained with
 70 EEG measures of brain activity, or more specifically,
 71 power (root mean square of the voltage amplitude)
 72 of activity within the alpha frequency band (8-13
 73 Hz) of the EEG. Research has revealed that alpha
 74 power is inversely related to regional brain activity
 75 using hemodynamic measures (Cook, O’Hara,
 76 Uijtdehaage, Mandelkern, & Leuchter, 1998) and
 77 behavioral tasks (Davidson, Chapman, Chapman,
 78 & Henriques, 1990). Source localization of EEG
 79 signals (Pizzagalli, Sherwood, Henriques, &
 80 Davidson, 2005) and fMRI results (Berkman &
 81 Lieberman, 2010) obtained in emotion-frontal
 82 asymmetry studies converge in suggesting that the
 83 dorsolateral PFC (figure 6.3) is responsible for these
 84 alpha asymmetry effects. These findings are further
 85 corroborated by evidence from studies of transcranial
 86 magnetic stimulation, discussed later (Schutter,
 87 2009; Schutter, van Honk, d’Alfonso, Postma, & de
 88 Haan, 2001).

89 *Trait Affective Styles and Resting Frontal* 90 *Cortical Asymmetry*

91 Depression has been found to relate to resting frontal
 92 asymmetric activity, with depressed individuals
 93 showing relatively less left than right frontal brain
 94 activity (Jacobs & Snyder, 1996; Schaffer, Davidson,
 95 & Saron, 1983), even when in remission status
 96 (Henriques & Davidson, 1990). Other research has
 97 revealed that trait positive affect in healthy subjects
 98 is associated with greater left than right frontal cortical
 99 activity, whereas trait negative affect is associated
 100 with greater right than left frontal activity
 101 (Tomarken, Davidson, Wheeler, & Doss, 1992).

102 Subsequent studies observed that trait behavioral
 103 activation, construed as approach motivation
 104 (Carver & White, 1994), was related to greater

1 left than right frontal activity at resting baseline
 2 (Amodio, Master, et al., 2008; Harmon-Jones &
 3 Allen, 1997; Sutton & Davidson, 1997). These
 4 studies suggested that asymmetric frontal cortical
 5 activity could be associated with motivational direc-
 6 tion instead of affective valence. However, avoid-
 7 ance and approach motivation are mostly associated
 8 with negative and positive affect, respectively
 9 (Carver & White, 1994), and consequently, the
 10 interpretation is clouded. Similarly, the finding that
 11 promotion (versus prevention) forms of regulatory
 12 focus are associated with greater relative left (versus
 13 right) frontal activation at baseline (Amodio et al.,
 14 2004) could be interpreted from either the motiva-
 15 tional direction or affective valence view. That is, the
 16 distinction between emotional valence with motiva-
 17 tional direction has been somewhat ambiguous
 18 given the methods used in past research, and
 19 researchers have tended to interpret relatively greater
 20 left than right frontal cortical activity as reflecting
 21 greater approach motivation and positive affect, and
 22 relatively greater right than left frontal cortical activ-
 23 ity as reflecting greater withdrawal motivation
 24 and negative affect. Although these claims fit well
 25 into dominant emotion theories that associate posi-
 26 tive affect with approach motivation and negative
 27 affect with withdrawal motivation (Lang, 1995;
 28 Watson, 2000), they do not fit so well with known
 29 physiological structure and function, as discussed
 30 below.

31 *State Affect and Asymmetric Frontal Activity*

32 Research has also demonstrated that asymmetric
 33 frontal brain activity is associated with state emo-
 34 tional responses. For instance, Davidson and Fox
 35 (1982) found that 10-month-old infants exhibited
 36 increased left frontal activation in response to a film
 37 clip of an actress generating a happy facial expres-
 38 sion as compared to a sad facial expression. Frontal
 39 brain activity has been found to relate to facial
 40 expressions of positive and negative emotions, as
 41 well. For example, Ekman and Davidson (1993)
 42 found increased left frontal activation during volun-
 43 tary facial expressions of smiles of enjoyment. Coan,
 44 Allen, and Harmon-Jones (2001) found that volun-
 45 tary facial expressions of fear produced relatively less
 46 left frontal activity.

47 Some positive affects are lower in approach moti-
 48 vation, whereas others are higher in approach moti-
 49 vation. An important question remains regarding
 50 the findings relating affective valence to asymmetric
 51 frontal cortical activity: Do positive affects of any
 52 approach motivational intensity cause increases in

relative left frontal activation? An experiment by
 Harmon-Jones, Harmon-Jones, Fearn, Sigelman,
 and Johnson (2008) addressed this issue by manip-
 ulating positive affect and approach motivation
 independently while measuring frontal EEG.
 Participants who wrote about positive/high-
 approach and positive/low-approach events both
 reported elevated positive affect, but only the posi-
 tive/high-approach manipulation produced an
 increase in left-frontal EEG. These results support
 the hypothesis that it is the approach motivational
 component of positive affective experiences, and
 not the positive valence per se, that causes greater
 relative left frontal cortical activation (as measured
 by EEG).

Anger and Asymmetric Frontal Cortical Activity

The experiment by Harmon-Jones et al. (2008),
 described above, suggests that approach motivation
 and positive affect are not perfectly associated with
 each other. More convincing evidence for the dis-
 sociation of affective valence and motivational
 direction (e.g., positive affect = approach motiva-
 tion) comes from research on anger. Anger is a neg-
 atively valenced emotion that evokes behavioral
 tendencies of approach (e.g., Darwin, 1872; Ekman
 & Friesen, 1975; Plutchik, 1980; Young, 1943).
 Anger is associated with attack, particularly offen-
 sive aggression (e.g., Berkowitz, 1993; Blanchard &
 Blanchard, 1984; Lagerspetz, 1969). Offensive
 aggression can be distinguished from defensive
 aggression, which is associated with fear. Other
 research also suggested that anger was associated
 with approach motivation (e.g., Izard, 1991; Lewis,
 Alessandri, & Sullivan, 1990; Lewis, Sullivan,
 Ramsay, & Alessandri, 1992). More recent studies
 examined whether trait behavioral approach or
 behavioral approach sensitivity (BAS) related to
 anger-related responses. Several studies have found
 that trait BAS, as assessed by Carver and White's
 (1994) scale, is positively related to state and trait
 anger (Carver, 2004; Harmon-Jones, 2003; Smits
 & Kuppens, 2005). Therefore, the emotion of anger
 provides a critical test case to disentangle interpre-
 tations of frontal cortical asymmetry as relating to
 valence or motivational direction.

In an initial test of this hypothesis, Harmon-
 Jones and Allen (1998) assessed trait anger using the
 Buss and Perry (1992) questionnaire and assessed
 asymmetric frontal activity by examining baseline,
 resting EEG activity. In this study of adolescents,
 trait anger related to higher left frontal activity and
 lower right frontal activity at baseline. Asymmetric

1 activity in other brain regions did not relate to anger,
 2 a finding that has been observed in all subsequent
 3 observations of this effect. Replications have revealed
 4 that these results were not due to anger being evalu-
 5 ated as a positive feeling (Harmon-Jones, 2004),
 6 and this general effect has been replicated other
 7 laboratories (e.g., Hewig, Hagemann, Seifert,
 8 Naumann, & Bartussek, 2004; Rybak, Crayton,
 9 Young, Herba, & Konopka, 2006). Other research
 10 has manipulated asymmetrical frontal cortical activ-
 11 ity using transcranial magnetic stimulation and
 12 found that the disruption of right PFC increased
 13 approach responses to angry faces compared with
 14 disruption of the left PFC (d'Alfonso, van Honk,
 15 Hermans, Postma, & de Haan, 2000; van Honk &
 16 Schutter, 2006).

17 Researchers have also tested the motivational
 18 direction model of frontal cortical asymmetry by
 19 manipulating state anger. Harmon-Jones and
 20 Sigelman (2001) found that individuals who were
 21 insulted evidenced greater relative left frontal activ-
 22 ity than individuals who were not insulted.
 23 Additional analyses revealed that within the insult
 24 condition, reported anger and aggression were posi-
 25 tively correlated with relative left frontal activity.
 26 Neither of these correlations was significant in the
 27 no-insult condition. Harmon-Jones, Peterson, and
 28 Harris (2009) conceptually replicated the above
 29 research and extended it by showing that anger and
 30 jealousy in responses to social rejection caused
 31 increased relative left frontal activity. This pattern of
 32 state anger and approach motivation has been con-
 33 ceptually replicated in other labs as well (e.g.,
 34 Jensen-Campbell, Knack, Waldrip, & Campbell,
 35 2007; Verona, Sadeh, & Curtin, 2009).

36 Considered as a whole, EEG asymmetry studies
 37 of emotion and motivation have provided a critical
 38 test of how emotions are organized in the brain.
 39 Although the subjective appraisal of emotion is usu-
 40 ally described in terms of valence (e.g., pleasant vs.
 41 unpleasant), this large body of research demon-
 42 strates that at the neural and physiological level of
 43 analysis, emotional responses are organized in terms
 44 of their approach versus withdrawal motivational
 45 orientation (Harmon-Jones et al., in press).

46 *Emotion Regulation*

47 The idea that people often seek to regulate their
 48 emotional response dates back to classic theories of
 49 Freud and Descartes, who suggested that reason is
 50 needed to overcome passion (in Freud's terms, the
 51 ego must adjudicate the sway of the id). Con-
 52 temporary views on emotion regulation have

53 similarly focused on the role of cognitive control in
 54 modulating lower-level emotional processes
 55 (Davidson, Jackson, & Kalin, 2000). In social cog-
 56 nitive neuroscience, researchers have focused on
 57 regions of the PFC as the neural substrate of control
 58 and on regions of the subcortex, such as the
 59 amygdala, as the substrate of emotion, particularly
 60 negative emotion (Ochsner & Gross, 2005). For
 61 example, in many emotion regulation studies, sub-
 62 jects view aversive (vs. neutral) images during an
 63 fMRI scan. On some trials, subjects are instructed
 64 to simply view the image. On other trials, subjects
 65 may be instructed to decrease their affective response
 66 to the image. In one study using this method,
 67 Ochsner, Bunge, Gross, and Gabrieli (2002) found
 68 that the simple viewing of aversive images was asso-
 69 ciated with increased activity in the amygdala.
 70 When subjects attempted to reduce their affective
 71 response, activations in lateral prefrontal cortex
 72 (PFC, figure 6.3) were observed and interpreted as
 73 reflecting the engagement of control, whereas
 74 amygdala activity on such trials did not differ from
 75 baseline (Ochsner et al., 2002). Several other stud-
 76 ies have observed similar effects (e.g., Banks, Eddy,
 77 Angstadt, Nathan, & Phan, 2007; Cunningham
 78 et al., 2004; Ochsner et al., 2002; Lieberman,
 79 Hariri, Jarcho, Eisenberger, & Bookheimer,
 80 2005; Lieberman et al., 2007; Phan et al., 2005;
 81 Urry et al., 2006).

82 As more direct evidence for the down-regulation
 83 hypothesis, researchers have observed a negative
 84 correlation between activity in the lateral PFC
 85 during emotion regulation trials and amygdala
 86 activity during baseline (no-regulation) trials (e.g.,
 87 Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003;
 88 Ochsner et al., 2002). This negative correlation is
 89 typically interpreted as demonstrating the direct
 90 "down-regulation" of emotion, in the direction of
 91 the PFC acting on the amygdala. There are prob-
 92 lems with this interpretation, however. Despite
 93 observed correlations between regions of lateral
 94 PFC and the amygdala, these regions share very few
 95 anatomical connections, and of these, most run
 96 from the amygdala to the PFC (Ghashghaei &
 97 Barbas, 2002). Thus, the idea that the PFC "down-
 98 regulates" emotion may be incorrect, or at least
 99 inconsistent with neuroanatomy.

100 If the PFC does not directly down-regulate emo-
 101 tion, how might it otherwise play a role? One recent
 102 study suggests that PFC activity interpreted as con-
 103 trolling emotion may actually reflect the control
 104 of eye gaze (van Reekum et al., 2007). The authors
 105 found that, when instructed to reduce one's affective

1 response to aversive images, subjects' gaze avoided
 2 the most aversive parts of the image, as measured by
 3 eye-tracking sensors. Interestingly, increases in PFC
 4 activity (and decreases in amygdala activity) associ-
 5 ated with the voluntary reduction of negative affect
 6 were statistically explained by these changes in eye
 7 gaze. This important finding suggests that PFC acti-
 8 vations previously interpreted as reflecting the direct
 9 down-regulation of affect may actually reflect an
 10 indirect mechanism, whereby the control of atten-
 11 tion away from the aversive stimulus lessens the
 12 emotional impact. The notion of an indirect path-
 13 way for emotion regulation is consistent with previ-
 14 ous physiological studies (Gross & Levenson, 1993,
 15 1997).

16 Neuroscience research on emotion regulation
 17 highlights the idea that psychological conceptions
 18 of emotion do not map cleanly onto specific under-
 19 lying neural structures (Amodio, 2008; Cacioppo
 20 et al., 2003). Rather, psychological constructs of
 21 emotions (e.g., subjective feelings) more likely
 22 reflect a summary appraisal of several interacting
 23 physiological mechanisms that function in concert
 24 to promote adaptive responses to an emotion-elic-
 25 iting situation. If so, then questions about the neural
 26 correlates of emotion and emotion regulation may
 27 need to be reconsidered and perhaps replaced by
 28 questions about more basic processes of motivation,
 29 attention, arousal, and action that support the adap-
 30 tive functions associated with subjective experiences
 31 of emotion.

32 **Intergroup Processes**

33 One of the most active areas in the field of social
 34 neuroscience examines prejudice, stereotyping, and
 35 intergroup relations (for a review, see Amodio,
 36 2008). The area has provided fertile ground for
 37 social neuroscience research because it concerns the
 38 confluence of multiple psychological processes
 39 across multiple levels of analysis. That is, researchers
 40 can simultaneously examine issues of automaticity
 41 and control, emotion and cognition, motivation,
 42 attitudes, and a range of individual differences, all
 43 within the context of social cognition and social
 44 behavior, and with broad societal implications (see
 45 also Tropp & Molina, chapter 22, this volume).

46 **Seeing Groups**

47 How early is a person's social category perceived in
 48 the person perception process? Several studies have
 49 examined this question by testing whether faces of
 50 ingroup and outgroup members are perceived dif-
 51 ferently in basic visual processes. A study by Golby,

Eberhardt, Chiao, and Gabrieli (2001) observed 52
 greater activity in the fusiform gyrus in response to 53
 ingroup than outgroup faces, and this difference 54
 predicted later recognition of the faces. Several stud- 55
 ies have also examined this question using the N170 56
 component of the ERP while participants viewed 57
 faces of Black and White people in the context of 58
 various tasks. However, the findings have been 59
 mixed, with some studies observing no differences 60
 (Caldara, Rossion, Bovet, & Hauert, 2004; Caldara 61
 et al., 2003; Wiese, Stahl, & Schweinberger, 2009), 62
 some finding larger effects of visual processing for 63
 the ingroup (Ito & Urland, 2005) and others for the 64
 outgroup (Walker, Silvert, Hewstone, & Nobre, 65
 2008). These inconsistent findings are likely due to 66
 task differences. Also, few extant studies have con- 67
 trolled for important low-level visual factors (e.g., 68
 luminance and contrast) in their stimuli, and there- 69
 fore more research is needed to understand what 70
 aspects of group membership might contribute to 71
 N170 effects. 72

73 In an effort to address these methodological
 74 issues, Ofan, Rubin, and Amodio (in press) pre-
 75 sented White participants with "two-tone" faces of
 76 Black and White people. These two-tone faces were
 77 created by transforming grayscale images into
 78 images consisting only of pure white or black pixels.
 79 Images of White and Black faces were then equated
 80 for luminance and contrast by equating the propor-
 81 tion of black and white pixels in each face. In addi-
 82 tion, the authors proposed that differences in face
 83 processing might relate to participants' degree of
 84 implicit racial bias. Participants with stronger pro-
 85 White bias, as assessed using a sequential evaluative
 86 priming task, may perceive Black outgroup faces as
 87 less normative compared with White ingroup faces.
 88 Given past work showing that N170 amplitudes are
 89 larger in response to non-normative faces (e.g.,
 90 Halit, de Haan, & Johnson, 2000), the authors pre-
 91 dicted that participants with stronger pro-White
 92 implicit bias would exhibit larger N170s to Black
 93 faces than to White faces. Indeed, this pattern was
 94 found (Ofan et al., in press). These results suggest
 95 that social group differences are indeed registered at
 96 the earliest stages of visual face processing, but that
 97 the effect of group differences on these processes
 98 varies as a function of the context and the perceiver's
 99 goals and attitudes.

100 **Brain Mapping of Racial Bias**

101 Physiological research on responses to race provided
 102 the first evidence that implicit forms of racial bias
 103 may differ from explicit self-reports (Rankin &

1 Campbell, 1955; Vanman, Paul, Ito, & Miller,
2 1997). The earliest neuroscience studies of inter-
3 group bias attempted to map this implicit response
4 to the brain. At the time of these early neuroimag-
5 ing studies, new findings were emerging from
6 the study of fear conditioning in nonhuman
7 animals that identified the amygdala as a key neural
8 structure involved in the fear response (Davis &
9 Whalen, 1998; LeDoux, 2000). Given other
10 research suggesting that fear-related responses might
11 underlie observations of implicit racial bias (Fazio,
12 Jackson, Dunton, & Williams, 1995; Greenwald,
13 McGhee, & Schwartz, 1998), the amygdala seemed
14 to be a logical candidate for the neural substrate
15 of prejudice.

16 A pair of initial studies by Hart et al. (2000) and
17 Phelps et al. (2000) used fMRI to measure partici-
18 pants' brain activity while viewing faces of ingroup
19 and outgroup members. Both studies used blocked
20 designs, such that participants would view a series
21 of White faces in one block and a series of Black
22 faces in a different block. Hart et al. (2000) included
23 both White and African American participants,
24 whereas Phelps et al. (2000) included only White
25 participants. In both studies, the authors focused on
26 the amygdala as the proposed substrate of racial
27 prejudice. Each face was viewed for several seconds,
28 and measures of brain activity were averaged across
29 the entire block, as a function of race. Although
30 these studies did not allow for inferences about the
31 "implicitness" of a response to race, given that the
32 blocked task design allowed for slow and potentially
33 deliberative responses, it was assumed that the
34 amygdala only operates in an implicit manner, and
35 thus any differences in amygdala activity to ingroup
36 versus outgroup faces would indicate a neural form
37 of implicit bias.

38 Interestingly, neither study found a significant
39 effect for ingroup versus outgroup race in amygdala
40 responses. Rather, the effects were more complex.
41 Hart et al. (2000) observed that although passive
42 viewing of both ingroup and outgroup faces both
43 elicited a similar degree of amygdala activity,
44 amygdala responses to ingroup faces habituated
45 more quickly than activity to outgroup faces. In
46 Phelps et al. (2000), subjects viewed faces and indi-
47 cated whether each face was the same or different
48 from the previous trial. Although the authors did
49 not observe an effect for race in amygdala activity,
50 individual differences in amygdala response to Black
51 versus White faces from the fMRI measure
52 were correlated with subjects' scores on a Black-
53 White IAT and also their degree of startle-eyeblink

modulation to Black versus White faces. None of
54 these measures were associated with explicit self-
55 report measures of racial attitudes. These patterns of
56 results were consistent with the idea that the
57 amygdala may be involved in implicit responses to
58 race. By suggesting a neural correlate of implicit
59 bias, these studies provided a foothold for future
60 research to consider how mechanisms of learning
61 and memory associated with the amygdala might be
62 involved in implicit intergroup processes. 63

64 ***Social-Personality Research on Intergroup*** 65 ***Bias and the Brain***

66 From a social psychological perspective, identifying
67 a neural substrate of implicit racial bias was interest-
68 ing because it could offer new clues as to how
69 implicit racial biases are learned, activated, expressed
70 in behavior, and potentially altered or extinguished.
71 Furthermore, such research could help to clarify
72 important individual differences in people's abilities
73 to respond without prejudice. For example, it could
74 help us understand whether egalitarians respond
75 without prejudice because they are very effective in
76 regulating their responses or because they do not
77 have biased responses in the first place. To address
78 these questions, Amodio et al. (2003) conducted a
79 study in which they used the startle-eyeblink
80 method to examine fear-related amygdala responses
81 to race.

82 Amodio et al.'s (2003) study was designed to
83 address multiple goals. First, the authors sought to
84 identify a uniquely affective form of implicit racial
85 bias. Previous theories assumed that implicit bias
86 reflected associations between a target group and
87 related attributes within a semantic network (Fazio
88 et al., 1995; Greenwald & Banaji, 1995). This type
89 of theory explained observations of priming between
90 target groups and judgments of stereotypic and
91 evaluative words. Interestingly, priming effects for
92 evaluative word associations were frequently inter-
93 preted as an affective form of bias. However, it is
94 difficult to make this claim based on a measure of
95 semantically based word associations. Amodio et al.
96 (2003) noted that although the amygdala is associ-
97 ated with fear-related affective responses, it is not
98 able to process semantic information (see also
99 Amodio & Devine, 2006). Therefore evidence for
100 differences in amygdala activity to Black versus
101 White faces using the startle-eyeblink measure
102 would provide strong evidence for an affective basis
103 of implicit bias. By using a startle-eyeblink index of
104 amygdala activity, the authors could also assess
105 rapid changes in amygdala activity in response to

Au: There is no
Davis &
Whalen 1998
in the
references.
Might 2001 be
correct?

1 an ingroup versus outgroup face, which would
 2 strengthen the interpretation that the amygdala
 3 response reflects “automatic” processing. Further-
 4 more, the startle eyeblink method assesses activity
 5 of the amygdala’s central nucleus—the region spe-
 6 cifically involved in the learned fear response. By
 7 contrast, fMRI measures of amygdala activity
 8 cannot distinguish activity in the central nucleus
 9 from other regions that are not associated with fear.
 10 Thus, the startle eyeblink measure provided the best
 11 method for linking implicit racial bias to fear and
 12 threat-related affect.

13 Amodio et al.’s (2003) second goal was to under-
 14 stand why some people with sincere low-prejudice
 15 attitudes nevertheless show anti-Black bias on
 16 implicit measures, whereas other low-prejudice
 17 people do not. Previous research had shown that
 18 among people with strong internal motivations to
 19 respond without prejudice, those who were also
 20 very worried about appearing prejudiced in front of
 21 others exhibited high levels of implicit racial bias
 22 (e.g., on the IAT), whereas those who were not wor-
 23 ried about social pressures exhibited low levels of
 24 implicit bias (Devine, Plant, Amodio, Harmon-
 25 Jones, Vance, 2002). Thus, Amodio et al. (2003)
 26 asked whether the subset of low-prejudice people
 27 who did not show implicit affective bias in behavior
 28 either (1) did not have a bias in the first place or (2)
 29 were biased but were very effective in regulating
 30 their bias.

31 To address these questions, participants viewed
 32 pictures of White, Black, and Asian faces. Some
 33 trials included a startle probe at 400 ms following
 34 picture onset, which assessed amygdala responses
 35 prior to the opportunity for controlled processing,
 36 and other trials included a startle probe at 4000 ms,
 37 assessing amygdala activity after an opportunity for
 38 controlled processing. At 400 ms, startle-eyeblink
 39 responses revealed greater amygdala activity to Black
 40 than White faces, but only among people with low
 41 internal motivation to respond without prejudice
 42 (i.e., high-prejudice subjects). At 4000 ms, there was
 43 greater amygdala activity to Black versus White faces
 44 for people with low internal motivations, as well as
 45 those with high internal motivation who were also
 46 very concerned about external social pressures.
 47 Subjects with strong internal motivations who did
 48 not worry about external social pressures—the ones
 49 who showed low implicit bias in other research—did
 50 not show a different startle response to Black versus
 51 White faces. These data suggested that indeed, some
 52 low-prejudice people did not show any signs of
 53 implicit affective bias to begin with, which might

54 explain why they often do not show bias on behav- 54
 55 ioral measures such as the IAT (Devine et al., 2002). 55
 56 Although other low-prejudice people, who worry 56
 57 about external pressures, do show signs of bias in 57
 58 their amygdala activity, a second study found that 58
 59 they are effective at controlling their expression of 59
 60 bias in more deliberative behaviors (Amodio et al., 60
 61 2003, Study 2). Thus, this research was the first to 61
 62 show significant differences in amygdala activity to 62
 63 Black versus White faces, and this effect was moder- 63
 64 ated by individual differences. This research also pro- 64
 65 vided evidence for a uniquely affective form of 65
 66 implicit bias, and it suggested that some low-preju- 66
 67 dice people possess implicit negative affective asso- 67
 68 ciation with racial outgroups, whereas other 68
 69 low-prejudice people do not. 69

70 By linking the amygdala to implicit affective 70
 71 racial associations in several studies, this body of 71
 72 research suggested that affective racial bias may 72
 73 reflect a form of fear conditioning. Therefore, 73
 74 knowledge about how fear responses are learned, 74
 75 expressed, and potentially unlearned could be 75
 76 applied to the topic of implicit affective intergroup 76
 77 bias. For example, fear-conditioned responses are 77
 78 learned rapidly, often after a single experience, and 78
 79 they are expressed primarily in autonomic responses 79
 80 and nonverbal behaviors (such as freezing and 80
 81 avoidance). Such associations are also very difficult 81
 82 and perhaps impossible to extinguish (Bouton, 82
 83 1994; Schiller, Freeman, Mitchell, Uleman, & 83
 84 Phelps, 2009); instead of being unlearned, new 84
 85 learning is needed to override the effects of older 85
 86 fear-conditioned associations. Interestingly, most 86
 87 theories of implicit social cognition suggest that 87
 88 implicit associations are learned slowly, only after 88
 89 repeated exposure (e.g., Smith & DeCoster, 89
 90 2000)—an idea that is inconsistent with fear condi- 90
 91 tioning. Nevertheless, behavioral studies have shown 91
 92 that measures of implicit evaluative bias predict 92
 93 nonverbals associated with freezing and avoidance 93
 94 (Amodio & Devine, 2006; Dovidio, Kawakami, 94
 95 Johnson, Johnson, & Howard, 1997; Fazio et al., 95
 96 1995), consistent with the models of amygdala- 96
 97 based learning. Finally, the fact that evaluative biases 97
 98 are so difficult to extinguish is also consistent with 98
 99 research suggesting that fear-conditioned associa- 99
 100 tions are relatively permanent. 100

101 Since these initial studies, several event-related 101
 102 fMRI studies have observed that amygdala activity 102
 103 is greater to outgroup than ingroup faces under 103
 104 some conditions but not others, and that these 104
 105 effects are typically subtle (Cunningham et al., 2004; 105
 106 Lieberman et al., 2005; Wheeler & Fiske, 2005; 106

1 Ronquillo et al., 2007). That is, differences in
 2 amygdala activity to Black versus White faces tend
 3 to emerge only in the absence of a minimally
 4 demanding task. As tasks become more demanding,
 5 such as when searching for a “dot” on the image,
 6 when imagining whether the target likes a particular
 7 vegetable, or when attempting to match the face to
 8 written group labels, amygdala activations tend not
 9 to be found (Lieberman et al., 2005; Wheeler &
 10 Fiske, 2005). Other research suggests that amygdala
 11 effects are lessened when the outgroup face is not
 12 looking directly at the subject (i.e., has averted gaze;
 13 Richeson, Todd, Trawalter, & Baird, 2008).

14 It is also notable that amygdala activity to Black
 15 versus White faces may reflect participants’ concern
 16 about appearing prejudiced in such studies. That is,
 17 participants are usually aware that a task concerns
 18 reactions to race after a few trials of viewing Black
 19 and White faces. To the extent that a participant is
 20 worried about showing prejudice toward Black
 21 faces, each Black face serves as a threat stimulus—
 22 that is, a trial on which the subject may reveal bias,
 23 either to others or to the self. Thus, it is possible that
 24 amygdala responses to race are due to this anxiety,
 25 rather than to previously learned affective associa-
 26 tions. Other research has shown that the amygdala
 27 responds to motivationally relevant stimuli, such as
 28 to rewarding stimuli (Holland & Gallagher, 1999)
 29 or to one’s ingroup members (e.g., in the context of
 30 an implied competition; Van Bavel, Packer, &
 31 Cunningham, 2008), although these responses may
 32 reflect the basal nucleus rather than the central
 33 nucleus of the amygdala. These findings suggest
 34 some alternative explanations for observations of
 35 amygdala activity associated with responses to race,
 36 highlighting the complexity of these processes and
 37 the need for careful interpretations and additional
 38 research.

39 *Stereotyping*

40 Whereas much research has examined the neural
 41 correlates of race-related affect and evaluation, very
 42 little has investigated social stereotypes. Stereotypes
 43 are believed to represent cognitive structures stored
 44 in memory that represent a set of attributes associ-
 45 ated with a social group (Devine, 1989). Amodio
 46 and Devine (2006; see also Amodio, 2008; Amodio
 47 & Mendoza, 2010) noted that stereotypes are rooted
 48 in mechanisms of semantic memory and selection,
 49 which are associated with neural activity in the tem-
 50 poral lobes and lateral posterior PFC (e.g.,
 51 Brodmann areas 45 and 47). Behavioral and neuro-
 52 science research on semantic learning systems has

uncovered the dynamics of how such associations 53
 are learned and expressed in behavior, and by access- 54
 ing this literature, researchers can apply findings 55
 from the memory literature to understand stereo- 56
 typing processes (Amodio, 2008; Amodio & Ratner, 57
 2011b). For example, whereas affective associations 58
 are learned quickly and are relatively indelible, 59
 semantic associations may be learned and unlearned 60
 through a process of repeated pairings and nonpair- 61
 ings. Semantic learning systems are more likely to 62
 be expressed in trait impressions, goal representa- 63
 tions, and goal-driven behaviors, and thus they are 64
 more likely to emerge in verbal responses (Amodio 65
 & Devine, 2006). 66

Some fMRI studies have examined neural activ- 67
 ity associated with the completion of stereotyping 68
 tasks (Knutson, Mah, Manly, & Grafman, 2007; 69
 Mitchell, Ames, Jenkins, & Banaji, 2009; Quadflieg 70
 et al., 2009), but these studies have not explored the 71
 mechanisms of stereotypes per se. Rather, these 72
 studies have focused on brain activity associated with 73
 more general aspects of task completion, such 74
 as response conflict and inhibition, or face percep- 75
 tion. In a brain lesion study, patients with mPFC 76
 damage did not show bias on a male versus female 77
 IAT (Milne & Grafman, 2001). However, it is likely 78
 that this mPFC damage interfered with the general 79
 process of response conflict that drives the IAT 80
 effect, rather than representations of stereotype knowl- 81
 edge (a function typically ascribed to the PFC and 82
 temporal lobes). Thus, the neural mechanisms of 83
 stereotyping remain largely unstudied, although 84
 researchers have already applied findings from the 85
 broader cognitive neuroscience literature on seman- 86
 tic selection and representation to help understand- 87
 ing the representation and functions of stereotypes 88
 (Amodio, 2008; Amodio & Ratner, 2011b). 89

90 *Control and the Regulation of* 91 *Intergroup Bias*

Given that racial stereotypes and implicit evalua- 92
 tions may be automatically activated, regulatory 93
 processes are needed to reduce their expression in 94
 behavior. Social-personality psychologists have 95
 begun to apply findings from neuroscience studies 96
 of cognitive control to understand how expressions 97
 of intergroup bias may be regulated (Amodio, 98
 Devine, & Harmon-Jones, 2007; Amodio & 99
 Devine, 2010). Amodio et al. (2003; see also 100
 Richeson et al., 2003) proposed that PFC regions 101
 were likely involved in the control of behavioral 102
 responses to race, but that control operated on 103
 behavior rather than the putative source of bias in 104

1 the amygdala (indeed, behavioral expressions of bias
2 were reduced despite sustained amygdala responses
3 to race). Indeed, Amodio et al. (2003) found that
4 participants who are highly motivated to respond
5 without prejudice are generally unable to control
6 the activation of affective responses to race directly,
7 but rather are highly effective at controlled behav-
8 ioral expression of bias. This finding suggests that
9 the mechanisms of control are complex, and that
10 research was needed to understand the different
11 ways in which control functions to guide intergroup
12 behavior.

13 *Detecting the Need for Control*

14 Despite holding nonprejudiced beliefs, many self-
15 avowed egalitarians still often express stereotypes
16 and affective biases in their behavior. In light of
17 recent neuroscience models positing that control
18 involves two major components, conflict monitor-
19 ing and regulative control (Botvinick et al., 2001),
20 Amodio et al. (2004) wondered whether such “slips”
21 might reflect a failure to detect the need for control
22 by the conflict monitoring system, or rather a fail-
23 ure to implement control once its need has been
24 detected. The authors measured EEG in low-preju-
25 dice participants while they completed the weapons
26 identification task—a sequential priming task that
27 requires enhanced control on some trials to override
28 the influence of automatic stereotypes. The use of a
29 behavioral “control” task was critical because it (1)
30 clearly manipulated the engagement of controlled
31 processing and (2) provided behavioral indicators of
32 both automatic bias and the degree of successful
33 control—both of which are needed to interpret
34 associated brain activity as being involved in
35 control.

36 Amodio et al. (2004) focused on two ERP mea-
37 sures of ACC activity—the error-related negativity
38 component and the N2 component associated with
39 successful control—and found that both were more
40 strongly activated on trials where control over ste-
41 reotypes was needed (see also Bartholow et al., 2006;
42 Correll, Urland, & Ito, 2006). Importantly, height-
43 ened ACC activity was observed both when control
44 succeeded and when it failed. In the case of failure,
45 the finding suggested that when low-prejudice
46 people respond with unwanted stereotypes, it is the
47 result of failing to implement a controlled response
48 rather than a failure to detect that control is needed.
49 A conceptually related ERP study by Bartholow
50 et al. (2006) found that alcohol administration
51 selectively impaired the regulative component of
52 control without affecting the conflict monitoring

component in the process of stereotyping inhibi- 53
tion, providing further support for the distinction 54
between these two components of control in the 55
regulation of intergroup bias. 56

Amodio, Harmon-Jones, and Devine (2008) 57
conducted a follow-up study to address why some 58
low-prejudice people—those who have strong inter- 59
nal motivation to respond without prejudice but are 60
also very concerned about external social pressures— 61
are especially prone to unintended expressions of 62
bias. The authors asked whether these individuals 63
were less sensitive to conflicts between activated ste- 64
reotypes and egalitarian response goals. Consistent 65
with this hypothesis, these high-internal/high-exter- 66
nal motivation subjects showed lower levels of ACC 67
activity on task trials that required control over 68
automatic stereotypes, similar to subjects reporting 69
high-prejudice beliefs. By contrast, participants who 70
were highly internally motivated and unconcerned 71
about external pressures showed strong ACC 72
responses when stereotype control was needed. A 73
second study showed that these groups did not 74
differ in domain-general forms of control and that 75
the observed differences were specific to racial ste- 76
reotypes (as expected, given that groups were deter- 77
mined by their motivations to respond without 78
prejudice). Other research suggests that mechanisms 79
for engaging control in response to external cues 80
(e.g., cues from other people) involve more rostral 81
regions of the ACC and mPFC, in contrast to ACC- 82
related mechanisms involved in control based on 83
internal cues (Amodio et al., 2006). This finding 84
suggests that when controlling responses according 85
to external social cues, people rely on mentalizing 86
and social cognition processes associated with the 87
mPFC in other research (Amodio & Frith, 2006; 88
Mitchell et al., 2006). 89

Implementing Control 90

Many theories in social and cognitive psychology 91
posit that, once the need for control is detected, 92
other mechanisms are engaged to implement a con- 93
trolled response (Devine, 1989; Shiffrin & 94
Schneider, 1977). Neuroscience research has impli- 95
cated the PFC in this function (Botvinick et al., 96
2001; Kerns et al., 2004; Badre & Wagner, 2007). 97
However, the specific target of control is not always 98
clear—it could be the emotional response, the ste- 99
reotype itself, the expressed behavior, the way a 100
person is perceived, or some other process. 101
Neuroscience can help clarify this issue, as an analy- 102
sis of the neuronal circuitry of the PFC provides 103
clues about the targets of control. In particular, this 104

1 circuitry suggests that the PFC modulates goal-
2 directed action processes as well as the modulation
3 of sensory input and perceptual processing (Miller
4 & Cohen, 2001). By contrast, the PFC has few con-
5 nections to the amygdala, and to the extent that the
6 amygdala is the source of implicit affective bias, it is
7 unlikely to be a direct target of control.

8 Several studies have observed patterns of PFC
9 activity associated with responses to race, but their
10 role in response control has not been clear. In the
11 earliest example, Richeson et al. (2003) found that
12 subjects who showed greater PFC activity when
13 viewing Black versus White faces in one experimen-
14 tal session were more likely, in a subsequent experi-
15 mental session, to perform more poorly on a
16 cognitive control task following a stressful interracial
17 interaction. The authors reasoned that subjects
18 who spontaneously engaged control when viewing
19 faces were also more likely to engage control during
20 an interracial interaction, which in turn interfered
21 with their performance on the Stroop task. A study
22 by Cunningham et al. (2004) measured brain activ-
23 ity to faces of Black and White people using fMRI.
24 Participants simply indicated whether faces appeared
25 on the right or left side of the visual field. Like
26 Richeson et al. (2003), Cunningham et al. (2004)
27 assumed that people spontaneously engage some
28 form of control during passive face viewing. Indeed,
29 the ACC and several regions of the PFC were more
30 active to Black faces than White faces, although
31 some PFC regions were more active to White faces
32 than Black faces. Similarly, a study by Lieberman
33 et al. (2005) found a reduction in amygdala activity
34 and an increase in some PFC regions (e.g., the right
35 ventrolateral PFC, figure 6.3) when participants
36 viewed Black versus White faces and were instructed
37 to match the faces to written labels.

38 Taken together, these studies are provocative,
39 suggesting a possible role of the PFC in the control
40 of racial bias. However, without the manipulation
41 of response control or a behavioral measure of control,
42 interpretations of these studies are unclear.
43 That is, the PFC is involved in a wide range of func-
44 tions, and therefore one cannot necessarily infer the
45 engagement of “control” from the observation of
46 PFC activity alone, as this would be a poorly sup-
47 ported reverse inference. A second issue is that
48 researchers often assume that the target of control is
49 the amygdala (i.e., the putative source of implicit
50 bias). Indeed, Cunningham et al. (2004) and
51 Lieberman et al. (2005) each found a region of lat-
52 eral PFC (a different region in each paper) that was
53 inversely correlated with an index of amygdala

activity to Black versus White faces. These authors
54 interpreted this correlation as evidence for a neural
55 mechanism of prejudice control. However, these
56 regions of lateral PFC are known to have few, if any,
57 anatomical connections to the amygdala (Ghashghaei
58 & Barbas, 2002), and therefore the interpretation of
59 this “down-regulatory” effect is likely to be incor-
60 rect. For example, it is possible that observed PFC
61 activations were associated with task demands and
62 changes in attention that were indirectly associated
63 with a reduction in amygdala responses (as in
64 Wheeler & Fiske, 2005).
65

66 Other research has examined the role of the PFC
67 in modulating behavioral intentions and the per-
68 ceptual processing of race in a way that is more con-
69 sistent with PFC anatomy. Previous work by
70 Amodio et al. (2003) observed that controlled pro-
71 cesses operated on behavior, but not directly on
72 amygdala activity. In a later study, Amodio et al.
73 (2007) demonstrated that increased left PFC activ-
74 ity was associated with the behavioral intention to
75 engage in prejudice-reducing behaviors. More
76 recently, Amodio (2010a) proposed that mecha-
77 nisms of control promote intentional behavior by
78 modulating attention to and perception of cues that
79 control is needed. For example, Monteith’s (1993)
80 self-regulation model posits that, once the goal to
81 control intergroup responses is formed, an individ-
82 ual becomes vigilant to cues that control is needed,
83 such as the presence of an outgroup member (see
84 also Richeson & Trawalter, 2008). Amodio (2009)
85 predicted that control-related PFC activity would
86 serve to allocate greater attentional resources to
87 the perception of outgroup faces, which in turn
88 would facilitate better response control (i.e., more
89 accurate responding despite any biasing effects
90 of racial stereotypes). Indeed, while White partici-
91 pants in the study by Amodio (2010a) completed
92 the weapons identification task, greater left PFC
93 activity throughout the task predicted larger
94 attentional ERP responses to Black versus White
95 face primes just 180 ms after a face appeared, as
96 well as greater response control. Furthermore,
97 the effect of PFC activity on response control was
98 mediated by attentional ERP responses to Black
99 faces, among low-prejudice participants. These find-
100 ings are consistent with anatomical models of the
101 PFC, as well as behavioral research showing that
102 people are effective at controlling their actions and
103 perceptual attention, but ineffective at directly
104 controlling their thoughts or emotions (Gross &
105 Levenson, 1993; Wegner, Schneider, Carter, &
106 White, 1987).

1 *Intergroup Emotion and the Brain*

2 Research on intergroup emotion examines responses
3 such as love, hate, threat, disgust, and guilt in an
4 intergroup context, and their implications for social
5 interactions and discrimination (Mackie & Smith,
6 1998; Fiske, Cuddy, Glick, & Xu, 2002; Devine,
7 Monteith, Zuwerink, & Elliot, 1991). Other
8 research has focused on the role of intergroup anxi-
9 ety in interracial interactions (Stephan & Stephan,
10 1985). Several social neuroscience studies have been
11 conducted to probe these processes further. However,
12 as noted above, the study of emotion (and affect) at
13 the neural level of analysis is challenging because
14 emotions and affect typically describe psychological
15 states rather than mechanisms. When one begins to
16 describe mechanisms associated with an emotion, a
17 lower level of analysis that involves motivational,
18 attentional, and/or autonomic functions is often
19 necessary. Thus, social neuroscience research on
20 intergroup emotion often focuses on these lower-
21 level mechanisms.

22 Several studies have examined the physiological
23 correlates of intergroup emotional responses within
24 a social interaction. Early physiological studies
25 showed that despite reports of liking for outgroup
26 members, White participants revealed heightened
27 skin conductance levels and facial EMG patterns
28 indicating negative emotion in response to out-
29 group members (Rankin & Cambell, 1955; Vanman
30 et al., 1997; Vrana & Rollock, 1998). As noted
31 above, initial cognitive neuroscience studies of
32 intergroup emotion focused on the amygdala as a
33 key substrate, on the basis of animal research link-
34 ing the amygdala to the learning and expression of
35 fear (e.g., Amodio et al., 2003; Hart et al., 2000;
36 Phelps et al., 2000). However, a simple mapping
37 of implicit bias to the amygdala is not informative
38 about the emotional quality of implicit bias per
39 se. Rather, it links the construct of implicit bias to
40 a set of mechanisms involved in the response to a
41 learned threat, such as the activation of the
42 autonomic nervous system, heightened attention
43 and perceptual vigilance, behavioral inhibition
44 and the preparation for “fight or flight” behaviors
45 (Amodio, 2008). This research suggests a mecha-
46 nism underlying implicit bias, which in turn sug-
47 gests new ideas for how implicit bias might relate to
48 behavior, learning, and other low-level psychologi-
49 cal functions. In general, though, relatively few
50 studies have examined the role of the brain in inter-
51 group emotional processes. Here we note a few
52 examples.

Role of Self-Directed Negative Affect

53 Guilt is an important intergroup emotion experi- 54
55 enced among egalitarians after responding uninten- 56
57 tionally with prejudice, and it is associated with new 58
59 goals to regulate one’s prejudices in future situations 59
60 (Monteith, 1993; Monteith, Ashburn-Nardo, Voils, 58
61 & Czopp, 2002). Amodio et al. (2007) showed that 59
62 guilt resulting from the unintentional expression of 60
63 prejudice is associated with a decrease in left PFC 61
64 activity, relative to baseline, and a reduction in 62
65 approach motivation. Other negative emotions, 63
66 such as shame, sadness, and anxiety, were not associ- 64
67 ated with changes in PFC activity in this situation. 65
68 Importantly, when an opportunity to reduce future 66
69 prejudice arose, subjects’ degree of guilt predicted a 67
70 shift toward greater left PFC activity, which was 68
71 associated with stronger approach motivation 69
72 toward behaviors designed to reduce prejudice. 70
73 Thus, this research used EEG measures of brain 71
74 activity to show that guilt is a complex intergroup 72
75 emotion that coordinates shifts from inhibition to 73
76 approach-motivated responses to promote prosocial 74
77 behavior. 75

Intergroup Anxiety

76 In actual intergroup situations, anxiety is often a 77
78 factor. Using cardiovascular assessments of threat 78
79 versus challenge appraisals, Mendes, Blascovich, 79
80 Lickel, and Hunter (2002) observed stronger threat 80
81 responses during interactions with outgroup mem- 81
82 bers on the basis of race or socioeconomic status, 82
83 compared with ingroup members. Based on earlier 83
84 research suggesting that anxiety can interfere with 84
85 performance and controlled processing (e.g., 85
86 Easterbrook, 1959; Baumeister & Showers, 1986), 86
87 prejudice researchers have proposed that intergroup 87
88 anxiety might undermine attempts to control 88
89 implicit racial bias, leading to greater expressions of 89
90 prejudice (Lambert et al., 2003; Richeson & 90
91 Trawalter, 2005). However, the mechanism through 91
92 which anxiety might interfere with control has been 92
93 unclear, particularly because self-report assessments 93
94 of anxiety are typically uncorrelated with changes in 94
95 behavioral control. 95

96 Taking a neuroscience perspective on this issue, 96
97 Amodio (2009) proposed that anxiety might affect 97
98 the control of intergroup responses through a neural 98
99 pathway that operates independently of subjective 99
100 emotional responses. That is, he noted that social 100
101 threats are typically associated with the release of 101
102 norepinephrine within the brain, which has been 102
103 shown to modulate ACC activity and conflict 103

1 monitoring processes (Aston-Jones & Cohen,
2 2005). According to this model, high norepineph-
3 rine signaling to the ACC sensitizes the conflict
4 monitoring processes, such that vigilance to conflict
5 is very high, but the ability to implement an
6 intended response becomes impaired. Through this
7 pathway, high anxiety should impair the control of
8 automatic stereotyping effects on behavior. Although
9 one cannot measure brain norepinephrine during
10 an interracial interaction, one of its downstream
11 effects is the release of the hormone *cortisol* in the
12 bloodstream, which can be measured in saliva. Thus,
13 Amodio (2009) measured salivary cortisol changes
14 in response to an interracial versus same-race inter-
15 action. As part of the interaction, participants com-
16 pleted a stereotype inhibition task that assessed
17 response control. Interaction-related increases in
18 cortisol predicted worse control during the interra-
19 cial interaction. This effect was not observed during
20 the same-race interaction. Also, although partici-
21 pants reported greater subjective anxiety in the
22 interracial interaction condition, self-reported anx-
23 iety was unrelated to cortisol levels or behavior. This
24 research applied a neuroscience model to propose a
25 specific mechanism through which intergroup anx-
26 iety might enhance some aspects of control (e.g.,
27 vigilance) but impair other aspects (e.g., response
28 implementation) during an intergroup interaction.

29 **Personality and Individual Differences**

30 Personality is the study of enduring psychological
31 dispositions and their influence on thought, emo-
32 tion, and behavior (see Fleeson, chapter 3, this
33 volume). For researchers interested in personality
34 processes, physiological processes that operate on a
35 longer timecourse, such as genes and hormones, are
36 of special interest. Some research in this area has
37 examined direct correlations between personality
38 traits, such as those comprised by the “Big Five.”
39 Increasingly, however, researchers have used neural
40 and physiological models of continuity and change
41 to help to understand the complexities of personal-
42 ity processes and individual differences. In this sec-
43 tion, we describe a just a few examples of this
44 growing area of research.

45 *Affective Style*

46 Research by Davidson and colleagues suggested that
47 enduring affective styles, associated broadly with
48 depression and anxiety, relate to different patterns
49 of neural function (Davidson & Irwin, 1999).
50 Specifically, these styles relate to differences in

51 frontal cortical asymmetries, as described in previ-
52 ous sections, which are also associated with differen-
53 tial approach versus withdrawal tendencies
54 (Davidson, 1998; see also Heller, Nitschke, &
55 Miller, 1998). Although much research has exam-
56 ined state changes in frontal EEG asymmetry to
57 study emotion and motivation, more stable, trait-
58 like components of the asymmetry (e.g., at rest or
59 baseline) have been taken to reflect a substrate of
60 affective personality style. These trait-like patterns
61 have been observed in adults, children, and nonhu-
62 man primates using a range of measures, and indi-
63 vidual differences in these styles have been linked to
64 various assessments of mental and biological health
65 (Kern et al., 2008; Rosenkranz et al., 2003). More
66 recently, trait-like patterns of activity in other brain
67 regions, including the amygdala and regions of the
68 basal ganglia have been included in an expanded
69 framework of affective style (e.g., Fox, Shelton,
70 Oakes, Davidson, & Kalin, 2008).

71 *Role of Genes in Personality* 72 *and Social Psychology*

73 Psychologists have long suspected that many per-
74 sonality traits are substantially heritable, with longi-
75 tudinal studies showing strong continuity in
76 temperament from childhood to adulthood (Caspi,
77 2003; Cramer & Block, 1998). Over the past sev-
78 eral decades, research on heritability using twin
79 designs has supported this view (Caspi, Roberts, &
80 Shiner, 2005; Plomin, DeFries, McClearn, &
81 Rutter, 1997). Across the “Big Five” personality
82 dimensions, a review of heritability estimates sug-
83 gests that these traits are approximately 50% due to
84 genetic similarities (Bouchard & Loehlin, 2001).
85 The traits neuroticism and extraversion tend to
86 show the largest heritability estimates, consistent
87 with theory and research suggesting that these fac-
88 tors are dominant over other traits (Eaves, Eysenck,
89 Martin, 1989; Tellegen et al., 1988). Significant
90 heritability has also been observed for attitudes,
91 such as toward social policy (e.g., the death penalty,
92 immigration), racial beliefs, and hobbies (e.g., doing
93 crossword puzzles) (Olson, Vernon, Harris, & Lang,
94 2001).

95 More recently, researchers have used a molecular
96 genetics approach to explore the relationships
97 between particular gene polymorphisms found in
98 DNA with personality traits (Plomin & Caspi,
99 1999). The most well known example of this candi-
100 date-gene approach is the effort to associate differ-
101 ences in emotional processing with variations in the

1 serotonin transporter gene—a gene that codes for
 2 proteins involved in the reuptake of serotonin from
 3 the synapse (Hariri & Holmes, 2006; Canli &
 4 Lesch, 2007). Lesch et al. (1996) found that trait
 5 neuroticism was associated with individual differ-
 6 ences (i.e., polymorphisms) in this gene. Other
 7 research has observed suggestive associations
 8 between gene polymorphisms related to dopamine
 9 function and traits of extraversion and sensation
 10 seeking (Smillie, Cooper, Proitsi, Powell, &
 11 Pickering, 2010), although a meta-analysis suggests
 12 that evidence for these relationships is mixed across
 13 studies (Munafò, Yalcin, Willis-Owen, Flint, 2008).
 14 Another method for garnering convergent insight
 15 into the genetic and neurotransmitter systems
 16 involved in social processes is the experimental
 17 administration of various drug challenges. These
 18 studies provide insight into the molecular involve-
 19 ment in aspects of personality and various behaviors,
 20 such as extraversion and economic decision-making
 21 (DePue, Luciana, Arbisi, Collins, & Leon, 1994;
 22 Crockett, Clark, Tabibnia, Lieberman, & Robbins,
 23 2008)

24 A growing body of evidence suggests that envi-
 25 ronmental experiences can directly and indirectly
 26 modulate the expression of DNA (Caspi et al.,
 27 2003; Champagne & Curley, 2005; Way &
 28 Gurbaxani, 2008), consistent with psychological
 29 theories that highlight the importance of both per-
 30 sonal and environmental factors. Although this
 31 approach has generated much excitement, it is nota-
 32 ble that effects in this literature have been difficult
 33 to replicate (Caspi et al., 2005; Munafò et al. 2003).
 34 It is likely that the extreme complexity involved in
 35 traversing such distal levels of analysis—from DNA
 36 to complex behaviors, traits, and mental states—
 37 remains beyond the grasp of extant theoretical
 38 models. Therefore, much of the current work in this
 39 area continues to explore gene-behavior associations
 40 in an effort to incrementally constrain our under-
 41 standing of their causal relationship, slowly pro-
 42 gressing toward a coherent genetic account of
 43 personality and behavior.

44 *Hormones and Psychological Dispositions*

45 Although genes provide a close analog to the con-
 46 cept of personality as an enduring trait, the role of
 47 hormones in individual differences and social behav-
 48 ior has received much more attention. Hormones
 49 are characterized as providing a broader “organiza-
 50 tional” function, in that they help to orchestrate the
 51 coordinated response of multiple physiological and
 52 brain mechanisms. By comparison, specific neural

53 activations are typically interpreted as reflecting very
 54 specific, low-level aspects of a psychological response.
 55 Furthermore, whereas neural processes typically
 56 relate to specific state-related responses, the effects
 57 of hormones on behavior are slower, ranging from a
 58 few seconds, in the case of hormonal responses to
 59 specific events, to the course of a lifetime, in the case
 60 of baseline hormonal function. Hormonal influ-
 61 ences in early development can set the stage for
 62 enduring dispositions in biological and mental pro-
 63 cesses. For example, prenatal exposure to sex hor-
 64 mones has been shown to have long-term
 65 implications for gender development and adult
 66 sexual behavior (Singh, Vidaurri, Zambarano, &
 67 Dabbs, 1999).

68 As with gene effects, hormone effects can vary
 69 substantially as a function of the situation. For
 70 example, testosterone levels may vary with changes
 71 in power and social status (Josephs, Sellers, Newman,
 72 & Mehta, 2006; Mazur & Booth, 1998), and indi-
 73 vidual differences in the testosterone response fol-
 74 lowing a competition predict who chooses to seek a
 75 rematch (Mehta & Josephs, 2006). Experimental
 76 administration of testosterone can also increase
 77 attention to potential social threats, such as angry
 78 faces (van Honk et al., 1999). More recent research
 79 suggests that testosterone plays a larger role in
 80 orchestrating social behavior, such that it may pro-
 81 mote greater cooperation in reciprocal social
 82 exchanges (Eisenegger et al., 2010).

83 Another steroidal hormone, cortisol, is widely
 84 studied as a physiological response to stress
 85 (Dickerson & Kemeny, 2004). Cortisol is produced
 86 by the adrenal glands following activation along the
 87 hypothalamic-pituitary-adrenal axis, and it func-
 88 tions broadly to regulate metabolism and arousal in
 89 dispositional (i.e., baseline) diurnal processes and
 90 also in response to specific arousing events. Cortisol
 91 secreted into the blood can be detected in saliva,
 92 and thus salivary cortisol concentrations may be
 93 measured non-invasively and with relatively low
 94 cost in the typical psychological laboratory
 95 (Schulthies & Stanton, 2009).

96 Cortisol reactivity in response to a stressor coor-
 97 dinates an adaptive response (e.g., fight or flight),
 98 but after chronic exposure, heightened levels of cor-
 99 tisol become maladaptive (McEwen, 1998; Sapolsky,
 100 Romero, & Munck, 2000). For example, higher
 101 baseline cortisol levels have been associated with
 102 unhealthy profiles, including perceived stress, anxi-
 103 ety, depression, and cardiovascular stress (Cohen
 104 et al., 2006; Gallagher, Reid, & Ferrier, 2009).
 105 Although cortisol reactivity in response to a stressful

1 event has been examined in different contexts, a
 2 meta-analysis by Dickerson and Kemeny (2004)
 3 suggests that it is especially sensitive to socioevalua-
 4 tive stressors, such as when a subject must give an
 5 extemporaneous speech to a panel of disapproving
 6 peer judges. Thus, cortisol research has highlighted
 7 the primacy of social interactions in human motiva-
 8 tion and stress responses. As an outcome measure,
 9 cortisol provides a useful assessment of the stress
 10 response that does not rely on self-report. Further-
 11 more, the connection between psychological distress
 12 and biological responses highlights connections
 13 between the mind and body, and it underlines the
 14 important effects that social and dispositional fac-
 15 tors have on physical health. Research on immune
 16 variables, such as proinflammatory cytokines, pro-
 17 vides a similar link that pertains even more directly
 18 to healing and illness processes (Maier & Watkins,
 19 1998; Segerstrom & Miller, 2004).

20 It is notable that the greatest power of the social
 21 neuroscience approach lies in its ability to probe
 22 mechanism, and researchers have recently begun to
 23 conceptualize hormones and immune variables as
 24 mechanism variables. For example, Amodio (2009)
 25 measured salivary cortisol as a downstream correlate
 26 of within-brain norepinephrine activity to test a
 27 mechanism through which intergroup anxiety
 28 affects the control of racial stereotypes. Maier and
 29 Watkins (1998) provided a detailed analysis of how
 30 changes in cytokines and other associated immune
 31 and endocrine variables can act as mechanisms to
 32 alter cognition, emotion, and behavior, and to pro-
 33 mote an organism's health. These examples suggest
 34 that endocrine and immune approaches will increas-
 35 ingly be used to understand psychobiological mech-
 36 anisms associated with social and personality
 37 processes in future research.

38 **The Future of Social Neuroscience in** 39 **Social-Personality Psychology**

40 Over the past decade, physiological approaches have
 41 reemerged as an important facet of social-personal-
 42 ity research, now augmented by advances in neural,
 43 pharmacological, endocrinological, immunological,
 44 and genetic approaches. This time, we think neuro-
 45 science is here to stay. Recently a novelty in social-
 46 personality circles, the neuroscience perspective is
 47 now woven into the natural discourse of social-per-
 48 sonality inquiry. Neuroscience data are increasingly
 49 integrated into the literature reviews of mainstream
 50 social-personality manuscripts, and psychophysio-
 51 logical methods complement the traditional tools of
 52 behavioral psychology, now without the fanfare of a

novelty act. Social neuroscience is also becoming 53
 more prevalent in the training of new social-personal- 54
 ity psychologists. These are healthy developments 55
 for the field. 56

The purpose of this volume is to highlight the 57
 interplay of personality and social psychological 58
 approaches. It is interesting to consider this aim 59
 from the perspective of social neuroscience, a field 60
 in which traditional boundaries between the person 61
 and the situation are reinterpreted as complex, 62
 dynamic, and inherently multilevel interactions. 63
 For example, neuroscientific models reveal how our 64
 perception of a situation is influenced by disposi- 65
 tional factors, such as personality, goals, and mental 66
 sets (following ideas from the New Look move- 67
 ment). At the same time, research on genetics shows 68
 that even our DNA may be influenced by situational 69
 factors at the time of conception, and that gene 70
 expression—often held to be the purest expression 71
 of personality—is strongly influenced by the situa- 72
 tion. Thus, from the social neuroscience perspec- 73
 tive, a dynamic interplay of personal and situational 74
 influences operates at every level of analysis. 75

In this regard, the social neuroscience approach 76
 is helping to build connections between the fields of 77
 social and personality psychology in two ways. The 78
 first way is through its influence on theory and 79
 research. As noted above, social neuroscience 80
 research reveals the dynamic symbiosis between situ- 81
 ational and personal factors that exists across levels 82
 of analysis, in line with the interactionist view that 83
 the effects of personality and the situation can 84
 only genuinely be studied in the context of each 85
 other. The second way is by bringing together 86
 researchers from different disciplines to lend their 87
 respective expertise to integrative research questions, 88
 and by promoting education and training in inter- 89
 disciplinary approaches used in social neuroscience 90
 research. 91

92 **Conclusion**

93 Although neuroscience has reemerged on the social-
 94 personality scene, it still needs to establish itself as a
 95 substantive contributor to social and personality
 96 psychology theory. Success in this endeavor will
 97 depend on researchers' ability to ground their social
 98 neuroscience research in rigorous methodology and
 99 to relate it to central questions and theories of social
 100 and personality psychology. Researchers in the field
 101 are clearly rising to this challenge. As we described
 102 in this chapter, neuroscience theories and methods
 103 have begun to shed new light on the mechanisms
 104 of person perception, emotion, stereotyping and

1 prejudice, and some aspects of personality processes.
 2 Once the functions of specific neural structures, and
 3 their associated networks, are better understood, the
 4 contributions of linking social-personality processes
 5 to the brain will be increasingly realized. We look
 6 forward to new contributions from the social neu-
 7 roscience approach in years to come.

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