Chapter 10

What Can Neuroscience, Genetics, and Physiology Tell Us About Attachment?

In the current chapter we review the literature on relationship neuroscience (Beckes & Coan, 2013; Cozolino, 2006) and its implications for attachment. Relationship neuroscience, similar to social neuroscience, brings together social and biological approaches to improve the understanding of the neurobiological basis of interpersonal behavior (Wilson, 1998). In this chapter, we describe leading principles and ideas relevant to attachment, as well as tools and methods from the natural sciences that have been incorporated into social science research and more specifically attachment. We finish the chapter by proposing some new and promising directions for future research.[1]

WHAT IS NEUROSCIENCE?

Neuroscience is an interdisciplinary area, which builds and interacts with other fields such as psychology, computer science, psychoneuroimmunology, neuroendocrinology, and genetics, to study the structure, development, and functioning of the nervous system and the brain. It involves a diverse set of techniques, such as brain imaging and genetic mapping, and draws on different sources of information, such as animal models and computer simulations. All of these tools

1. Some concerns have been raised regarding some of the findings revealed in social/cognitive/affective neuroscience. The most salient example of this is the Vul, Harris, Winkielman, and Pashler (2009) paper on exaggerated correlations in fMRI, other examples are the so-called "dead salmon" study (Bennett, Baird, Miller, & Wolford, 2010), recent work by Button et al. (2013) on the low power of imaging studies, and the concerns over the false positive rate in candidate gene studies (Duncan & Keller, 2011). That said, and as suggested by Farah (2014), it is important to distinguish between specific criticisms of particular applications or specific studies and wholesale criticisms of the entire enterprise of functional neuroimaging or social neuroscience. None of the criticisms in the studies mentioned earlier constitute reasons to reject or even drastically curtail the use of neuroimaging to understand social-personality psychology in general or relationships and attachment specifically. Rather, they should remind the reader that neuroimaging, and neuroscience more generally, like other scientific methods, is subject to various specific errors that the self-correcting process of science continues to address (see Lieberman & Cunningham, 2009; Poldrack, 2012, and others for similar claims).

Adult Attachment. http://dx.doi.org/10.1016/B978-0-12-420020-3.00010-4

can be used to improve the understanding of the role that anatomy, physiology, biochemistry, and the molecular biology of nerves and nerve tissue play in human behavior and experience in general and attachment dynamics in particular. We refer to these approaches as "neuroscience" for simplicity, but to be clear, we will focus in the chapter on a wide gamut of physiological indexes, as well as genetics, endocrinology, and immunology.

WHY NEUROSCIENCE?

What can blood flow to specific brain regions, electrical activity along the scalp, levels of chemicals in a synapse or the bloodstream, or the structure of one's double helix, tell us about abstract concepts such as love, relationship security, and attachment? Judging by the recent upsurge in research focusing on the microlevel analysis of attachment, the answer is—a lot. Employing the knowledge base and methods developed within cognitive psychology, neuroscience, psychophysiology, genetics, endocrinology, and immunology, researchers provide a new and exciting set of answers to fundamental questions related to attachment theory and research. Questions such as: "How do attachment bonds develop?", "Why do people have a specific attachment style?", "What is attachment security?," and "Is attachment an emotion or a motivation?" are being revisited with renewed interest. These questions are now being tackled from new angles, focusing on the neural systems and processes that underlie attachment. Neuroscience can provide a lens on these issues that other methods cannot.

NEUROSCIENCE IN THE SERVICE OF ATTACHMENT

The majority of research on attachment has dealt with macrolevel processes (Levinger, 1994). Research and analysis at the macrolevel focus on the associations or effects that environment, context, and experience (eg, dyad, family, society, culture) can have on attachment processes and outcomes. For example, research from a macro perspective may tackle questions such as "How does growing up in a poor, dangerous neighborhood predict one's attachment style?" (Del Giudice, 2009a, and chapter: What Are the Effects of Context on Attachment?). Conversely, research and analysis at the microlevel focus on the associations that neurons, hormones, genes, neurotransmitters, and so on, have with attachment processes and outcomes. For example, researchers taking the micro level perspective may ask, "How does hippocampus size or brain activation within the hippocampus correlate with people's attachment style scores?" To more fully understand attachment and its underlying mechanisms, one must look beyond (or below) macrolevel processes and effects, and into microlevel processes and effects (Levinger, 1994).

To investigate the microlevel of attachment, researchers have relied on the knowledge base and methods developed within cognitive psychology (to study processes such as attention, memory, control, and inhibition, to name but a few), psychophysiology (including animal models), and social/affective/

developmental neuroscience. One of the central questions in attachment neuroscience is whether attachment processes and constructs (such as bonds, style, and figures) are based on a unique neural system (parallel to the theoretical notion of the attachment behavioral system) or a combination of other systems, such as thought control and emotion-regulation. A related question is whether attachment is one system/mechanism or a set of modules/systems. For example, there might be one system underlying insecurity and a different one underlying security. To answer these questions, researchers have used a diverse set of methods and techniques ranging from brain activation to levels of oxytocin in one's blood or saliva.

WHAT ARE THE TOOLS, METHODS, AND TECHNIQUES USED TO STUDY THE NEUROSCIENCE OF ATTACHMENT?

There are different ways to study brain functioning, including functional magnetic resonance imaging (fMRI), electroencephalography (EEG), near infrared spectroscopy (NIRS), positron emission tomography (PET), computerized tomography (CT)/computerized axial tomography (CAT), and transcranial magnetic stimulation (TMS). To date, researchers have mainly used fMRI and EEG to study the neural mechanisms underlying attachment (eg, Canterberry & Gillath, 2012; Zhang, Li, & Zhou, 2008). Both of these noninvasive methods allow researchers to assess brain activation. fMRI relies on the fact that cerebral blood flow and neuronal activation are coupled. When a brain area is in use, blood flow to-and-from that region increases. These changes can be captured using an fMRI scanner. EEG is a method to record electrical activity (ionic current within the neurons) of the brain, as measured along the scalp. fMRI is thought to provide better spatial resolution of brain activity, whereas EEG provides better temporal resolution.

Before delving into specific questions regarding the neuroscience of attachment an extensive mapping of brain regions and processes involved in attachment needs to occur. This type of research will help to identify which brain regions or neural systems/processes are particularly relevant to the study of attachment. Based on such mapping, researchers can understand how general processes, such as consolidation of memories, or shifts of attention, take place and clarify their contribution to attachment. For example, understanding how people form new social ties and which brain processes are involved, can potentially help us better understand how people form attachment bonds (as well as affiliation bonds). Once this understanding is achieved, researchers can search for ways to change or improve bonding (eg, Johnson, 2002; Perry, 2001). For instance, by using drugs or other chemical interventions researchers may be able to affect people's brains in a way that enables people to feel more secure. Knowing which brain regions or processes are active during certain attachment-related behaviors can also allow researchers to compare people with different attachment styles, thereby facilitating a better understanding of the

neuroscientific bases of the differences between such individuals. Next we provide a few examples of research that has focused on central topics in the attachment literature and have used neuroscientific methods.

WHAT HAS ALREADY BEEN DONE? EXAMPLES OF ATTACHMENT NEUROSCIENCE

fMRI

As reviewed in depth in chapter: What Are Attachment Working Models? Bowlby (1969/1982) coined the term internal working models (IWMs) to capture the different attachment-related mental representations that people have. According to Bowlby, IWMs allow people to understand the past, act in the present, and plan/prepare for the future (eg, Brumbaugh & Fraley, 2006). This conceptualization suggests that attachment includes a top-down regulation process that modulates people's emotions, thoughts, and behaviors (top-down is a cognitive process where existing knowledge affects the perception and processing of incoming new knowledge. This is in contrast to bottom-up processing in which perception and processing of new information serve to build knowledge). Despite ample work on IWMs (eg, Bretherton & Munholland, 2008), our understanding of them is still far from complete. For example, it is still unclear what mechanisms allow the formation of IWMs and their updating over time. Likewise it is unclear how the top-down cognitive process involved in IWMs differs from general top-down processes—that is, do working models function just like schemas and similar cognitive structures, or is there a unique mechanism only for IWMs? The use of neuroimaging can help provide a novel approach to answer these questions that may help us better understand how IWMs function.

In one of the first studies to examine the neural correlates of attachment style, Gillath and colleagues (Gillath, Bunge, Shaver, Wendelken, & Mikulincer, 2005) used fMRI to scan 20 women and found that the regulation of attachment-related thoughts was associated with activation in the prefrontal cortex (PFC)—an area involved in various cognitive processes that are not necessarily related to attachment (eg, Miller & Cohen, 2001). Specifically, Gillath and his colleagues found that when women were trying to stop thinking about rejection and separation from a romantic partner, there was greater brain activation in areas associated with attention, conflict-monitoring, and working memory [ie, the medial PFC, the anterior cingulate cortex (ACC), and the dorsolateral PFC; see also Anderson et al., 2004; Fig. 10.1]. These patterns of activation are similar to those identified when people suppress nonattachment-related thoughts, suggesting that IWMs and their associated top-down regulatory mechanisms are manifestations of general regulatory processes used to cope with attachment-related material (for a fuller discussion, see Gillath, Giesbrecht, & Shaver, 2009).

Neuroimaging studies not only shed light on general attachment processes, they also allow these processes to be studied across people with different attachment styles. For example, in the same study discussed earlier, Gillath and

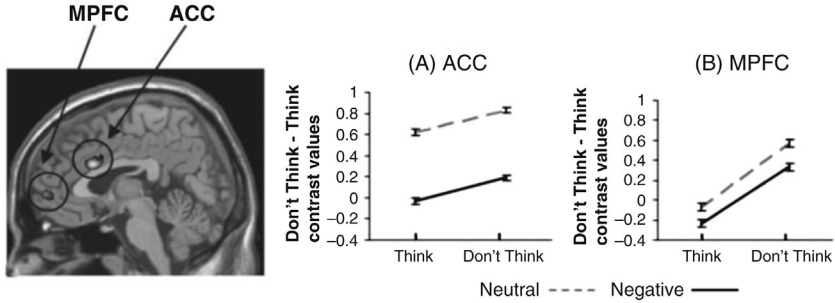

FIGURE 10.1 Neural correlates of thought suppression.

his colleagues found that, although most participants exhibited activation in the medial PFC and ACC when suppressing attachment-related thoughts (which is similar to the activation pattern viewed when suppressing other general thoughts, such as thoughts of white bears), avoidantly attached people showed a different pattern of activation. Whereas less avoidant people deactivated various brain regions when suppressing attachment-related thoughts such as the subgenual cingulate cortex (SCC; known to be associated with the regulation of emotion; Anderson et al., 2004; Drevets, 2000), avoidantly attached people did not. Gillath and his colleagues interpreted this lack of deactivation as related to the constant suppression that highly avoidant people engage in—suppression of emotions and relationship-related thoughts. This suppression (of emotions and attachment-related thoughts) is potentially being done in a way that involves brain activation in the same areas that other people, low on avoidance, deactivated during the task.

Neuroimaging methods have also provided insight into another central component of attachment theory: the formation of attachment bonds and identification of attachment figures (see chapter: What Is an Attachment Relationship?). Specifically, two brain areas appear to be involved in these processes—the amygdala and hippocampus (eg, Gillath et al., 2005a). The attachment system is activated when people feel threatened. When the system is activated, people look for help and for someone who can provide safety and security, such as an attachment figure. For this to occur, people need to quickly process information, identify the risk and a potential solution (enlisting help or support), and learn to associate a specific person with this solution.

The amygdala underlies many of these processes. Activation in the amygdala is associated with processing of emotional or salient material, paying attention to novel stimuli, and the consolidation of new memories through tagging (ie, labeling something as important or meaningful; see Phelps & LeDoux, 2005). For example, when a child experiences stress and then receives help, these events are likely to be associated with heightened activation in the amygdala. The amygdala is thought to tag such events as meaningful and the people who provided help as important, making recall of these people more likely in the future

(Lemche et al., 2006). Lemche et al., 2006 demonstrated that when people were exposed to cues of insecurity, the amygdala was indeed active—presumably as people processed the risk and retrieved images to help them cope. Other studies have identified neighboring brain regions, such as the anterior temporal pole (ATP)—known to be associated with emotion perception and response—to be activated when people are exposed to attachment-insecurity-related cues. This activation is thought to represent the recollection of attachment-related memories (eg, Gillath et al., 2005a; Vrticka & Vuilleumier, 2012).

Retrieval of images or scenarios is thought to take place in the hippocampus, which is also involved in creating associations between internal states (eg, feeling secure or distressed) and cues in the environment (eg, having a caregiver around; Kennedy & Shapiro, 2004), and with the consolidation of memories. Together, the amygdala, ATP, and hippocampus appear to allow the formation of an association between close others and meaningful events and experiences, which contributes to the perception of these others as attachment figures (eg, Buchheim et al., 2006; Lemche et al., 2006; Vrticka, Andersson, Grandjean, Sander, & Vuilleumier, 2008).

We suggest that the attachment system relies on such general abilities to generate lifelong associations regarding the roles of others in one's life (eg, provide love and care) and to tag specific people who are more important than others as attachment figures. By better understanding the mechanisms involved in the conditioning and processing of emotional information in the amygdala, the ATP, and the hippocampus, we might be able to help people form better attachment bonds and potentially help those who have issues creating such bonds (eg, Romanian orphans; Chisholm, 1998). For example, knowing that the amygdala and other brain regions are active during bond formation, it might be possible to help people form bonds by manipulating their brain chemistry (Hurlemann et al., 2010) or stimulating their brain using methods such as transcranial magnetic stimulation (Camprodon et al., 2015) or deep brain stimulation (Bewernick et al., 2010).

A third example of how neuroscience sheds light on attachment involves emotion regulation. People with different attachment styles cope differently and exhibit different emotion-regulation strategies (eg, suppression vs. enhancement). For example, anxiously attached people tend to be highly emotional and overwhelmed by their emotions, whereas avoidantly attached people have a weaker emotional reaction to distressing information (eg, Nash, Prentice, Hirsh, McGregor, & Inzlicht, 2013). A number of explanations have been suggested for these behaviors, but it remains unclear why anxiously attached people manifest emotions so intensely. Is it due to higher sensitivity to environmental cues? Lower ability to control emotions? Or both? Using neuroimaging, Gillath et al. (2005a) have found that when people are asked to suppress their negative thoughts and emotions during an emotion-regulation task, anxiously attached people exhibit lower activation in the orbitofrontal cortex (OFC). The OFC is associated with emotion regulation skills—the lower activation found in anxiously

attached people could be interpreted as lesser engagement of this brain area among anxiously attached people. This, in turn, suggests that the extreme emotional reactions of anxiously attached people are at least in part due to their lack of ability to regulate emotions (Gillath et al., 2005a; Warren et al., 2010).

A final example to the contribution of neuroimaging involves attachment security priming. Whereas most of the research on attachment in general, and attachment neuroscience in particular, has focused on attachment styles (anxiety and avoidance), less is known about the enhancement of attachment security and especially its underlying neural mechanisms. To address this gap, Canterberry and Gillath (2012) exposed people to attachment security-related primes or control primes and examined the activation of various brain regions. Behavioral studies have provided ample evidence that the enhancement of attachment security has a host of beneficial outcomes for personal and relational well-being (see Gillath et al., 2008b; Mikulincer & Shaver, 2007a, for reviews). Canterberry and Gillath suggested that the benefits associated with security are the result of cognitive, affective, and behavioral processes. Indeed, they found that security priming led to distributed, cooccurring activation in brain areas reflective of cognitive, affective, and behavioral processes (eg, the PFC, parahippocampus, and temporal and parietal gyri). These patterns of activation related to security priming were moderated by attachment styles. For example, avoidance was associated with activation in areas related to encoding and retrieval (parahippocampal gyrus), suggesting that avoidantly attached people were making increased memory retrieval attempts, perhaps reflecting a difficulty in accessing secure working models.

These findings, although consistent with the existing attachment literature, go beyond behavioral findings to show that all three components (cognitive, affective, and behavioral) operate simultaneously. Thus, security seems to act as a mental resource derived from multiple sources that facilitates prorelational and prosocial tendencies. Furthermore, the findings provide support for the idea that security priming is not merely a shift in the cognitive accessibility of security-related concepts. Rather, it seems to activate a system of emotions, cognitions, and behaviors (or behavioral tendencies) that contribute to growth and well-being (see also Eisenberger et al., 2011; Karremans, Heslenfeld, van Dillen, & Van Lange, 2011).

These are only a few examples within the rapidly growing literature on brain regions and mechanisms involved in bonding and attachment processes (see also Coan, 2008). These studies reveal that there are additional regions involved in attachment processes, such as the nucleus accumbens (eg, Aron et al., 2005), the ACC (dorsal ACC; eg, DeWall et al., 2012; Warren et al., 2010, and rostral ACC; Eisenberger & Lieberman, 2004), the dorsolateral PFC (eg, Gillath et al., 2005a; Warren et al., 2010), and the insula (eg, DeWall et al., 2012). These areas are thought to be involved in emotions related to attachment and bonding, such as love and desire (reward) or rejection and fear (punishment), and their regulation. Knowing which brain regions are involved in each of these processes

and how they work together can improve the design of attachment-related interventions. For example, one reason that anxiously attached people show lower activation in the OFC when trying to suppress thoughts may be that they have fewer/more specific neurotransmitters and receptors in the OFC. If this is the case, neurotransmitters could be modulated with chemical or pharmaceutical interventions. This, in turn, could potentially assist anxiously attached people to cope better with emotions and feel less insecure.

EEG

Another way to study the neural correlates of attachment is via EEG, which unlike fMRI, provides high temporal resolution. In EEG studies participants are often exposed to various events or cues and their event-related potential (ERPs) components are monitored. These components often have labels, such as P3 or N1, which represent whether the signal has a negative (N) or positive (P) polarity; and the number represents the latency in hundreds of milliseconds from the event (eg, P300 or in its short form P3 represents a positive signal that manifests approximately 300 ms after an event). ERPs are caused by cognitive processes that involve memory, expectation, attention, and other changes in mental states. Correlating attachment style with the amplitudes of ERP components can help us understand the timing with which various cognitive processes unfold for people who vary in attachment style.

For example, Zhang et al. (2008) examined people's brain activity following exposure to facial expressions. They found that as people were exposed to facial expressions, attachment style was associated with differences in ERP components (N1, N2, P2, and N4). These differences suggest that attachment styles are associated with both early automatic encoding as well as late elaborative retrieval of emotional content. Specifically, avoidant participants showed a less negative N1 compared to anxious and secure participants. N1 is thought to represent level of attention to cues (Hillyard, Teder-Sälejärvi, & Münte, 1998). Based on these results one might conclude that avoidant individuals devote less attention to emotional stimuli than secure or anxious people.

In a similar manner, Dan and Raz (2012) found differences on C1 and P1 mean amplitudes at occipital and posterior-parietal channels in response to angry faces versus neutral faces, but only among people high on avoidance (C1, or Component 1, can be either positive or negative; it is the first visual ERP component, which peaks between 50 and 100 ms). The processing biases toward angry faces (in the P1 component) and toward neutral faces (in the C1 component) among avoidant people suggest that only avoidant participants have the capacity to identify cues at such early stages of information processing, which allows them to rapidly apply their deactivating strategies (also see Niedenthal, Brauer, Robin, & Innes-Ker, 2002).

Focusing on anxious individuals, Zayas and colleagues (Zayas, Shoda, Mischel, Osterhout, & Takahashi, 2009) and Zilber, Goldstein, and Mikulincer

(2007) demonstrated attachment anxiety to be associated with later ERP components, such as N4 (reflecting the amount of semantic processing elicited by a stimulus) and late positive potential (LPP; an index of the emotional salience of a stimulus). For example, Zayas et al. found that when exposing participants to attachment-related cues, rejection-related words (eg, dismissing) elicited greater N4 amplitudes than acceptance-related words (eg, supporting) among women high on anxiety and low on avoidance. People tend to process more when the stimulus is unexpected or has a greater personal significance. Zayas and her colleagues concluded that anxiously attached women perceive rejection cues as more personally significant, posing greater threat to the self, and requiring more processing.

Laterality

A different way electrophysiology can help us understand attachment dynamics is by providing information on where in the brain activation takes place, and specifically in which side—what scholars refer to as brain laterality. For example, using EEG, Dawson et al. (2001) found that insecurely attached infants, as compared with secure ones, exhibited reduced left frontal brain activity. Whereas left frontal brain activity is often associated with positive emotions and approach tendencies (Hellige, 1993), reduced activity in the left frontal brain is associated with depression. Dawson and colleagues suggested that the reduced left frontal brain activity they found among insecure infants represents a greater tendency to use withdrawal-type emotion regulation strategies (turning away from the external environment) and a failure to use appropriate approach regulation strategies (eg, approaching an attachment figure when stressed). These findings that tie attachment insecurity with laterality, suggest more broadly that attachment insecurity is associated with alterations in infants' psychophysiological responses.

In a similar vein, Cohen and Shaver (2004), using a divided visual field task, found that avoidantly attached adults, as compared with nonavoidants, made more errors when judging positive attachment-related words presented to the right hemisphere (which is often involved in the processing of negative emotions; eg, Ahern & Schwartz, 1985). The findings further support the idea that people's attachment history and attachment style—levels of avoidance—are correlated with the way they represent and process attachment-related information. Cohen and Shaver suggested that because avoidantly attached people have less experience with positive attachment-related information, they are more likely to make more errors, especially in the hemisphere that has less to do with processing of positive information.

Brain Volume

In addition to looking at brain activation, either per region (fMRI), at a specific time-point (ERPs), or per hemisphere (in laterality studies), researchers have

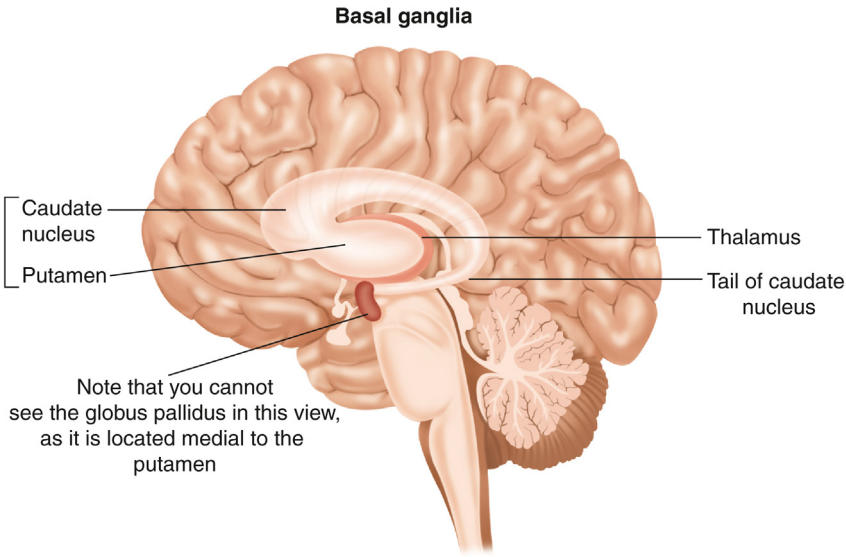

FIGURE 10.2 Depiction of the basal ganglia.

also investigated brain structure or volume. For instance, Quirin et al., 2010 found that attachment insecurity was associated with a smaller hippocampal cell density. This finding is compatible with a neurotoxical model of stress-induced cell reduction in the hippocampus. According to this model, unresponsive and insensitive caregiving promotes insecure attachment and simultaneously induces high stress for long periods of time. In turn, chronic high stress and high levels of cortisol (stress-related hormone) result in smaller hippocampus size. Benetti et al. (2010) found similar results, such that attachment anxiety was associated with a decrease in gray matter in the ATP. Activations in this area and the adjacent hippocampus were associated with greater attachment anxiety by Gillath et al. (2005a), providing convergent validity for the relevance of these brain areas (hippocampus and ATP) for attachment anxiety.

Tharner et al. (2011) also examined brain volume. However, they did so using a longitudinal rather than a cross-sectional design. Utilizing ultrasound imaging, they assessed infants' brain volume when they were 6 weeks old, followed them until they were 14 months old, and then used the strange situation (see Ainsworth, Blehar, Waters, & Wal, 1978) to assess individual differences in attachment. They found that infants who had a larger gangliothalamic ovoid, which is comprised of the basal ganglia (including the caudate, putamen, nucleus accumbens; Fig. 10.2) and the thalamus, were at a lower risk of developing attachment disorganization—regardless of their general brain development/maturity. The basal ganglia are thought to connect higher cortical regions, such as the PFC, with lower motor areas, and are believed to be involved in voluntary

motor action and learning (eg, Redgrave, Prescott, & Gurney, 1999). To achieve the set goals of the attachment system (proximity to caregiver and a sense of safety/security), specific behaviors such as crawling, reaching out, and crying must take place. The inability to select and execute such goal-directed attachment behaviors is a salient characteristic of people with insecure or disorganized attachment (Main & Solomon, 1990). Smaller volume of the basal ganglia structures may contribute to this inability and, in turn, to the development of disorganized attachment. Tharner and colleagues suggest that either intrauterine influences (eg, stress) or genetics (eg, a dopamine receptor gene, DRD4) may underlie the subcortical volume differences they identified in their study.

Together, the findings described earlier emphasize the importance of investigating brain volume either on its own or in conjunction with other methods (eg, brain functioning and genetics) to fully understand how attachment functions and develops over time. The existing findings suggest that attachment styles are associated with differences in brain volume, and that smaller volume in specific areas is related to disorganized (basal ganglia) or anxious attachment (hippocampus and ATP). More research is needed to understand how these structural differences come to be, and to what extent individual differences in brain volume are shaped by environmental cues. As suggested by Tharner et al. (2011), structural differences are likely to be the outcomes of both genes and environmental cues; however, no study to date has examined this.

Physiological Correlates

Physiological indices such as heart rate, blood pressure, skin conductance, and glucocorticoid levels can shed further light on the neuroscience of attachment (eg, Powers, Pietromonaco, Gunlicks, & Sayer, 2006; see Diamond & Fagundes, 2010, for a review). For example, Quirin et al. (2010) have made claims, based on their findings of brain volume differences regarding the association between attachment insecurity and the hypothalamic–pituitary–adrenocortical (HPA) axis system. These claims have received ample support from studies using physiological markers (eg, heart rate, blood pressure), which have repeatedly found associations between attachment insecurity and stronger physiological reaction (eg, higher HPA activity as an index of stress), especially following relational stressors (eg, Powers et al., 2006).

These findings, which demonstrate regulation failures or deficits among insecurely attached people, can be explained based on the decreased volume or increased activity in specific brain areas. To tie these bodies of research together, studies that combine neural and physiological indexes should be carried out. Such studies will allow scholars to tie the relatively new and sometimes unclear neural findings, with the broad knowledge base on human physiology, and the literature about attachment in a comprehensive explanatory model. As suggested by Tharner et al. (2011), an additional step will be to integrate neural and physiological findings with genetics.

GENES, NEUROTRANSMITTERS, AND HORMONES

There are different ways to utilize the knowledge about genes, neurotransmitters, and hormones to investigate attachment. First, researchers can use *behavioral* or *molecular genetic* methods to estimate the contribution of genetic and environmental factors to attachment style. *Behavioral genetic* methods partition the variation among individuals into genetic and environmental components (shared vs. unique environment). A common way to do so is by examining differences among identical twins (who share 100% of their genetic material) and fraternal twins (who share an average of 50% of their genetic material). Conversely, *molecular genetic* methods focus on the structure and function of genes at the molecular level. A common research methodology is to examine associations of a given trait or behavior and various *polymorphisms.*[2] These polymorphisms are often on genes that regulate either the release, reuptake, or degradation of hormones and neurotransmitters or the density of receptors of these hormones and neurotransmitters in the brain. With regard to attachment, scholars can examine the contribution of unique and shared environment and genetics to the development of a specific attachment style, or examine the correlations between attachment style or attachment behavior and specific polymorphisms.

Second, researchers can examine the correlation between the *blood or saliva levels* of neurotransmitters or hormones and people's attachment-related behaviors or style (eg, Edelstein, Stanton, Henderson, & Sanders, 2010). For example, one can measure levels of cortisol in the blood, or oxytocin in the blood or saliva, and correlate these with people's attachment style. Finally, going back to brain structure and functioning discussed earlier, researchers can use the distribution of receptors for neurotransmitters such as dopamine, oxytocin, and vasopressin in the brain to identify brain regions most likely to be associated with attachment processes and outcomes. For example, the nucleus accumbens, which is rich in neurotransmitter receptors related to dopamine, plays a role in various processes associated with attachment and bonding (eg, Young & Wang, 2004). We briefly provide a few examples of research focusing on behavioral and molecular genetics below.

Behavioral Genetics

Early studies using *behavioral genetics* found little consistent evidence for heredity or genetic influence, and more support for shared environment influence on infant attachment (eg, O'Connor & Croft, 2001). More recently, researchers focusing on adults have started to provide evidence to support the influence of genetics on attachment styles. For example, Crawford et al. (2007) found that 40% of the variance in adult attachment anxiety was accounted for by genetic

2. Polymorphisms can be homozygous (having identical alleles at corresponding chromosomal loci) or heterozygous (having dissimilar alleles).

influences, and Donnellan and colleagues (Donnellan, Burt, Levendosky, & Klump, 2008) found that additive genetic effects accounted for 45% of the variability in attachment anxiety and 39% of the variability in avoidance. These findings suggest that part of the variation in adult attachment styles can be accounted for by genetic differences among individuals. Similar findings were obtained recently with regard to adolescents (Fearon, Shmueli-Goetz, Viding, Fonagy, & Plomin, 2014).

Molecular Genetics

Turning to *molecular genetics*, the three main genetic candidates that scholars have been studying with regard to attachment are dopamine, serotonin, and oxytocin (but see Troisi et al., 2012, for findings on μ-opioid). Dopamine is involved in the motivation/reward system and in goal-related behavior (eg, Berridge, 2007) as well as in social and relational behaviors (eg, Schneier et al., 2000). Gillath et al. (2008c) found that attachment anxiety was associated with polymorphisms of dopamine (DRD2), and Lakatos and colleagues (Lakatos et al., 2002) found an association between dopamine (DRD4, the 7-repeat allele) and the likelihood of disorganized attachment. Bakermans-Kranenburg and van IJzendoorn (2011) highlight the interactions of dopamine (receptor DRD2, DRD4, and transporter DAT) with environmental conditions to affect attachment outcomes. For example, children who have less efficient dopamine-related genes do worse in poor environments (eg, insensitive parenting) than those without "genetic risk," and they are more likely to be insecurely attached, with a particular predisposition toward disorganized attachment. However, children who have these genes also profited more from nurturing environmental conditions, such as high parental involvement, enrichment programs, and alike.

Serotonin, the second gene candidate, is also known to be related with affect and affective disorders (eg, Gross et al., 2002) and social behavior (Raleigh, Brammer, & McGuire, 1983). In line with this research, serotonin was associated with greater attachment avoidance by Gillath et al. (2008c) and with greater anxiety by Salo, Jokela, Lehtimäki, and Keltikangas-Järvinen (2011) and Fraley, Roisman, Booth-LaForce, Owen, and Holland (2013). Both Salo et al. and Fraley et al. found that this association was moderated by environmental factors (defined as either maternal nurturance or maternal sensitivity). Caspers et al. (2009) found an association between the serotonin short 5-HTTLPR allele and increased risk for disorganized attachment. They interpreted this as being consistent with the role of serotonin in modulating the frontal-amygdala circuitry (see also Cicchetti, Rogosch, & Toth, 2011).

Oxytocin also plays a central role in social behavior and specifically in attachment. Costa et al. (2009) found associations between the GG genotype of OXTR single-nucleotide polymorphisms (SNPs; 6930G > A or 9073G > A) and attachment scores, such that it was negatively associated with "confidence" (an aspect of attachment security) and positively associated with "need for

approval" (a facet of attachment anxiety) and "relationship as secondary" (a facet of attachment avoidance). In contrast, Chen and Johnson (2012) found (only among females) that those who had at least one copy of the A allele of OXTR rs2254298 reported greater attachment anxiety than females who had two copies of the G allele. However, neither Gillath et al. (2008c) nor Fraley et al. (2013b) found an association between attachment and oxytocin OXTR (see also Bakermans-Kranenburg & van IJzendoorn, 2014).

Together, these findings suggest that, rather than conceptualizing attachment style as a blank slate at birth (ie, people having an equal or similar potential to develop a secure or insecure attachment style based on their interactions and the environment), some people might be more predisposed than others to develop (in)secure attachment styles. In apprising the research reviewed in this chapter on neuroimaging, we suggest that specific polymorphisms may affect the development and functioning of specific brain areas, which in turn, are associated with certain attachment behaviors, and more broadly people's predispositions for specific attachment styles (Fig. 10.1).

Recently researchers have started to use experimental methods to study the links between neurotransmitters and attachment variables, with a focus on oxytocin. Researchers have done so by investigating the effects of intranasal oxytocin (compared with placebo), which is thought to bypass the blood brain barrier (Talegaonkar & Mishra, 2004), on attachment-related behaviors. For example, Bartz et al. (2010) found that oxytocin affected attachment cognitions (eg, remembering one's mother as being more caring and close), but that these effects were moderated by attachment styles. Thus, people low on attachment anxiety remembered their mothers as more close and caring after oxytocin induction (vs. placebo), whereas people high on attachment anxiety remembered their mothers as less caring and close after the same manipulation.

Similarly, while oxytocin induction increased the ease of imagining a secure-script scenario (someone else being deeply compassionate to the self), this was moderated by attachment styles, with insecure individuals having less positive experiences (had a harder time to imagine another person being deeply compassionate to them) after the induction (Rockliff et al., 2011). De Dreu (2012) also found that oxytocin interacted with attachment styles; however, it specifically interacted with avoidance. That is, among people who scored higher on avoidance, oxytocin reduced betrayal aversion, and increased trust and cooperation compared to the placebo group.

Animal Models

There is a long research tradition in using animal models to study bonding, attachment, and close relationships (eg, Carter et al., 2005) —a research tradition that we will only briefly touch upon. Animal models are a powerful method to study the social brain and the neurobiological mechanisms underlying social relationships, attachment included (eg, Bales, Maninger, & Hinde, 2012). For

instance, oxytocin, which is thought to be a central player in human attachment and bonding, was first examined in animal models (see Carter et al., 2005; Insel & Young, 2001). Importantly attachment theory was partially developed on the basis of ethology (the science of animal behavior), which guided Bowlby's thinking regarding universal behavioral systems and bonding.

In studies using animal models, researchers use observational methods to identify bonding (social or pair-bonding) behaviors such as separation distress and soothing, or relationship/attachment styles. Animal models of attachment and pair bonding created by Michael Meaney and others are crucial in our understanding of the role that epigenetics and neural mechanisms play in these systems and behaviors (see Bagot et al., 2009; Bales et al., 2012; Carter et al., 2005; Lim & Young, 2006). Meaney's work demonstrated that parental behavior affects gene expression in the rat pup, which in turn affects the future parenting behavior of the pup when it reaches adulthood. The major advantages of this approach over work based on humans are the abilities to: (1) study intergenerational effects in much shorter timeframes and by moving pups from the care of their biological parents to genetically different rat caretakers; (2) use genetic or chemical manipulations that would be hard or impossible to use in humans; (3) inflict lesions; and (4) perform postmortem analysis. All of these methods are either impossible or more difficult to perform with humans. Utilizing these methods, animal models permit a better and deeper understanding of the structures, mechanisms, and functions involved in attachment processes and outcomes in ways that typically are not possible with human participants or with correlational research designs.

THEORETICAL MODELS

Although research in the domain of attachment neuroscience is relatively young, important findings have started to accumulate, and researchers have developed preliminary conceptual models to organize these findings. For example, Fonagy, Luyten, and Strathearn (2011) suggest a developmental, biobehavioral switch-model, not focused on attachment per se, but rather on the association between attachment with mentalization (ie, the ability to understand the mental state of oneself and others) and stress. The model is based on early work of Panksepp (1998) and Insel (eg, Insel & Young, 2001) on animals. The work links attachment bonds with substance dependence and opioids, suggesting that attachment bonds might be based on the same mechanisms as addictive disorders (Burkett & Young, 2012). These mechanisms involve two neural systems, which are the same systems that Fonagy and his colleagues focus on in their model: (1) the dopaminergic system (Ferris et al., 2005; Strathearn, Fonagy, Amico, & Montague, 2009), and (2) the oxytocinergic system (Bartels & Zeki, 2004; Champagne, Diorio, Sharma, & Meaney, 2001; Feldman, Weller, Zagoory-Sharon, & Levine, 2007). The dopaminergic system is associated with sensitivity to cues, and both the dopaminergic and oxytocinergic systems are associated with responding to social cues and with rewarding social and relational behaviors.

Tying their model to personality disorders, Fonagy et al. (2011) suggest that a complex set of interactions among environmental, biological, and psychosocial factors affect the two neural systems (dopaminergic, oxytocinergic), which in turn shape the attachment system, and more specifically its threshold of activation. These interactions also affect people's ability to differentiate the mental states of self and others. This, in turn, decreases the sensitivity to and susceptibility of being influenced from other people's mental states, reduces integration of cognitive and affective aspects of mentalization, and increases dysfunctions in stress-regulation systems. These, then, affect the ability of people to regulate their behavior. Together, the changes in threshold level and regulation or control can lead to the development of insecure or even disorganized attachment.

Fonagy et al.'s model focuses on attachment and its association with mental disorders. It draws a lot of its evidence from findings relevant to mothers' behaviors in response to their offspring, which are more closely related to the activation of the caregiving system than that of the attachment system (for similar models, see Atzil, Hendler, & Feldman, 2011; Galynker et al., 2012). Given this emphasis on the caregiving system, we turn next to Vrticka and Vuilleumier's (2012) model, which focuses less on mental disorders and the caregiving system, and more on the attachment behavioral system.

Vrticka and Vuilleumier (2012) suggest that individual differences in attachment styles correlate with various affective and cognitive processes, particularly in attachment-relevant or social contexts. Their model, on the influence of adult attachment on social processing [which incorporates Fonagy et al.'s (2011) model] involves two core networks: one network associated with affective evaluation processes (such as threat or reward and includes approach and avoidance components); and another network associated with cognitive control and mentalizing abilities (and includes emotion-regulation and mental state representation components). Their model is similar to the attachment model suggested by Pietromonaco and Barrett (2000) in terms of its affective and emotion-regulation components, and to more general models of social cognition and emotion processing (eg, Lieberman, 2007).

When describing the neuroscientific aspect of their model, Vrticka and Vuilleumier (2012) add the serotonergic and cortisol systems to the dopaminergic and oxytocinergic systems suggested by Fonagy et al. (2011). They discuss a set of specific brain regions for each network's component. *Approach* is associated with the ventral tegmental, hypothalamus, striatum, and ventral medial orbitofrontal cortex (OFC). *Avoidance* is associated with the amygdala, hippocampus, insula, anterior ACC, and ATP. *Emotion-regulation* is associated with the dorsolateral PFC and lateral OFC, and *mental state representation* is associated with the medial PFC, posterior cingulate cortex, precuneus, posterior superior temporal sulcus, temporoparietal junction, and anterior superior temporal gyrus.

Vrticka and Vuilleumier (2012) further suggest that there is a dynamic balance between the threat-sensitive system motivating social aversion and the

attachment system that promotes a sense of safety via close relationships and approach behavior (MacDonald & MacDonald, 2011). According to this explanation, attachment bonds serve as social rewards in the approach system. Both approach and aversion are thought to be shaped by genes and the environment, and modulated by attachment avoidance and anxiety. Thus, people high on attachment avoidance are thought to have weaker brain activation in areas related to both the approach and the avoidance systems—in line with their use of deactivating strategies. Conversely people high on anxiety have higher brain activation, but mainly with regard to the aversion system, and the processing of negative social cues—in line with their use of hyperactivating strategies. People who are low on both dimensions are thought to also have weaker reactions as compared with anxiously attached individuals, but due to their effective regulation rather than their deactivation of the attachment system (for a similar model and findings, see Warren et al., 2010).

Coan (2010) proposed a different model, one that focuses on the regulatory role of the attachment system via overt behavior associated with emotional and social functioning. His model describes the neural systems involved in the formation and maintenance of adult attachment relationships and the way the brain supports attachment behaviors. Similar to Vrticka and Vuilleumier (2012), Coan (2010) builds on research done on the neural systems that support the experience of emotion, emotion-regulation, motivation, and social behavior. He also introduces the social baseline model of social affect regulation. The model integrates existing models of attachment with a neuroscientific principle—economy of action—in the management of metabolic resources devoted to emotional and social behavior. According to the model, adult attachment relationships conserve brain metabolic resources, especially those of the PFC.

Coan's (2010) model, which tries to bridge the gap between the broad animal literature on bonding and the extended work on human attachment behavior, depicts the attachment behavioral system as a higher-order construct. This construct includes basic behaviors, such as recognition and familiarity, proximity-seeking, separation distress, soothing behaviors, and maternal caregiving. Like Vrticka and Vuilleumier (2012) and Fonagy et al. (2011), Coan discusses the emotion and emotion-regulation systems used for attachment behaviors, the relevance of threat- and reward-related systems, and associations between attachment and cognitive processes, such as attention and memory. However, he adds an economic aspect above and beyond these other models. According to this aspect, attachment is tied to the brain's management of energy expenditure. Being together with other people, or feeling securely attached, "saves" brain energy. Interacting with others—the default of human existence according to Coan—is less effortful. Being with others allows people to spend fewer resources on activities such as threat detection and emotion-regulation. People can share or distribute the load of these activities via familiarity, interdependence, and interpersonal conditioning. Conversely, being alone is straining and costly—there is no one to share the burden with and no one who can

provide energy or resources (Beckes & Coan, 2011). Attachment security is therefore conceptualized as a sign that less energy is needed, allowing people to save energy.

SUMMARY AND FUTURE DIRECTIONS

The three theoretical models reviewed earlier share a few things in common. They all discuss two aspects or systems underlying attachment styles, which broadly represent (1) threshold or sensitivity and (2) regulation. Furthermore, these models also incorporate automatic and controlled processes. This is in line with both nonneuroscientific models of attachment (eg, Pietromonaco & Barrett, 2000), and nonattachment-related models in neuroscience (eg, Lieberman, 2007). All three models reviewed also connect attachment with broader literatures, be it the temperament or personality literature, or the cognitive literature on affect regulation and thought control. The models use findings from these broader literatures to explain attachment-related processes, and identify brain systems or genes relevant to attachment. Finally, all the models highlight similar neurotransmitters (eg, dopamine, serotonin, and oxytocin) and their role in animal and human attachment [although this is less central in Coan's (2010) model].

There are a few things missing in the current models of attachment neuroscience. First, there is a need for an integrative explanation that describes how the various components reviewed earlier (eg, brain structure and function, genes, neurotransmitters) fit together to generate a comprehensive model of attachment. Second, existing neuroscientific models focus on the microlevel of attachment (intraindividual factors) without connecting it to the macrolevel (eg, context, culture). Third, most models (and the attachment literature more broadly) focus on explaining attachment insecurity, and less attention is given to the underlying mechanisms of attachment security. We suggest some new directions to fill these gaps later in the chapter.

A model of attachment neuroscience should integrate all the components reviewed earlier (and potentially others not reviewed here) into a comprehensive explanation that takes advantage of the unique contributions of each method or approach and integrates them into an overall picture. This idea is not unique to the neuroscience of attachment, and is related to data fusion and analytical approaches that deal with data fusion (Calhoun, Liu, & Adali, 2009). For example, in many recent studies, researchers collect multiple types of imaging data from the same participants (fMRI, ERPs, etc.). Each imaging method focuses on a limited domain (eg, near scalp electrical activity) and provides both common and unique information about the issues being studied. For instance, ERPs reveal the *when*, whereas fMRIs reveal the *where* of a phenomenon. Combining them in the same study with the same participants can provide a more complete picture than having them in separate studies using different samples and different designs.

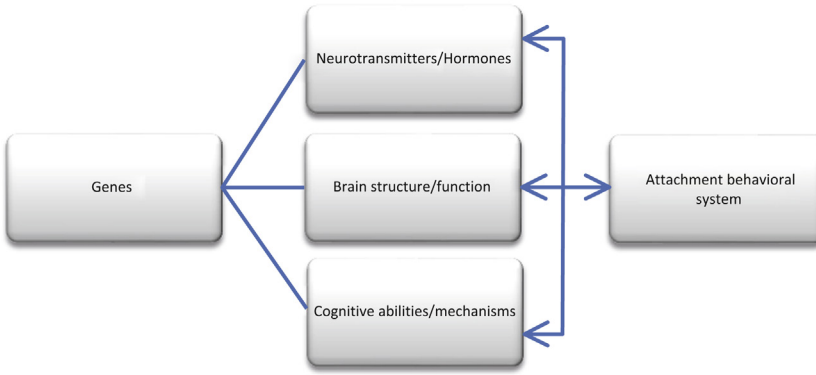

FIGURE 10.3 Attachment as the outcome of genes and brain structure and function. Genes include dopamine (DRD2, DRD4, DAT), serotonin (5HT), oxytocin (OXTR), and catechol-O-methyltransferase (COMT), among others. Brain structure/function includes volume, connectivity, and mechanisms in areas such as the hippocampus, amygdala, dACC, SCC, and OFC. Cognitive abilities and mechanisms include attention, emotion-regulation, thought control, self-regulation, working models, etc.

Statistical approaches such as independent component analysis (ICA) allow one to put these pieces (brain imaging, electrophysiology, genetics, etc.) together. Going beyond the mathematical or statistical level represented by ICA, there is also a need to provide a theoretical framework that connects all the informational dots. Gillath, Canterberry, and Collins (2012) have started this task (Fig. 10.3), connecting genetics, specific brain structure/volume and functioning, connectivity between the areas of activation, and attachment behaviors. For example, attachment anxiety is associated with polymorphisms of dopamine (fewer D2 receptors), decreased hippocampal volume, higher activation of the hippocampus, ATP, dorsal ACC (and a few other areas), lower activation of the OFC (and negative correlations between these activations), and higher sensitivity to attachment-related information. Conversely, avoidant attachment is associated with polymorphisms of serotonin (fewer 5HT receptors), increases in early brain waves (C1 and P1), higher activation in the dorsolateral PFC, and higher ability to suppress attachment-related cues. Future research should further test the associations among the components of the framework suggested by Gillath and colleagues, including different methodologies in the *same* study, and by adding more components (or pieces of the puzzle) as the evidence for their role accumulates.

Although neuroscience provides researchers with a preview of the microlevel of attachment, combining microlevel research with the macrolevel is necessary to better understand the attachment system (see Fig. 10.4, and Gillath et al., 2012). For instance, adopting a cultural perspective can allow researchers to grasp how the brain adapts to better fit with specific contexts or environmental demands (eg, Wilson, 2010). Understanding the functions of attachment in the culture-ready brain (Whitehead, 2010) can position attachment at

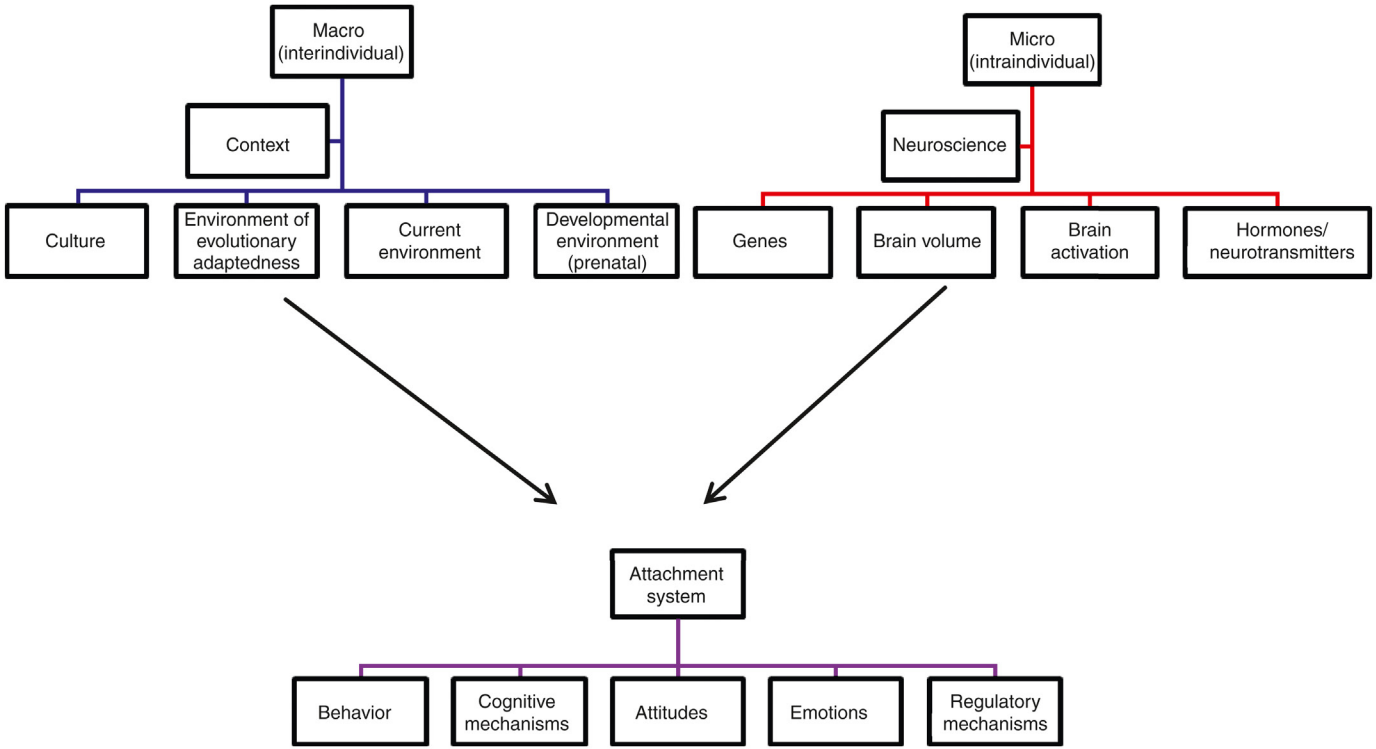

FIGURE 10.4 Combining the micro- and macrolevels to gain a better understanding of attachment.

the forefront of the new domain of cultural neuroscience (Chiao, 2010). Some preliminary work in this direction already exists. For example, Eisenberg et al. (2010) describe the role of D4 dopamine receptors in pair-bonding processes across different cultures/contexts, and Ray et al. (2010) describe differences in neural representations of self and other (specifically, the mother) as a function of a specific cultural context—interdependent self-construal.

Any model that seeks to explain the neuroscience of attachment should also deal with the construct of attachment security and its underlying mechanisms. As mentioned earlier, Canterberry and Gillath (2012) have conducted a study focusing on this aspect, showing that security involves affective (increased positive mood and relaxation), cognitive (increased self- and emotion regulation), and behavioral (prorelational and prosocial tendencies) components. In a different study, Gillath, Atchley, Imran, and El-Hodiri (2016a), using cognitive methods and ERPs, showed that priming attachment security increased the tendency to behave generously, and affected the reactions people had to their generosity being reciprocated or not. Examining feedback negativity (FN) and P3 ERP components, they found that security priming buffers emotional reactions to loss, especially among insecurely attached people, potentially making them focus on the importance of social cues (other people) rather than financial ones (possessions). In yet another study, exposing people to an attachment security prime resulted in increased glucose levels, supporting the idea that security provides resources to people, which in turn allows them to deal with stress and react more efficiently and flexibly to threats (Gillath, Pressman, Stetler, & Moskovitz, 2016). This extends Coan's (2010) model, showing that security not only helps to save energy, but actually provides energy, that could potentially help a person to cope better with the threats that activate the attachment system.

While providing initial information on security, these studies do not deal with the relations between security and insecurity. Currently, for example, it is unclear whether the two represent two different systems (similar to approach and avoidance systems or to threat-oriented vs. growth-oriented systems), or two sides/poles of the same system/dimension (see Fig. 10.5). More work is needed to answer questions such as "What happens when people are exposed to an insecurity prime?" We know that the attachment system is activated (Mikulincer, Gillath, & Shaver, 2002), and that people seek proximity to attachment figures to regain security, but what is the end result of this process with regard to the system? Is it "returning to baseline" (its zero or default state)? Or, because security is achieved or regained, are people reaching a state that is "above" baseline, which is closer to how they would feel (or what they would have experienced) when primed with a security prime? Using neuroscience techniques and comparing activation when security versus insecurity is primed can help answer these important questions. Based on our own findings, it seems that security priming brings people into a higher state of growth or flow (Csikszentmihalyi, 2014), which are associated with different brain mechanisms compared with insecurity.

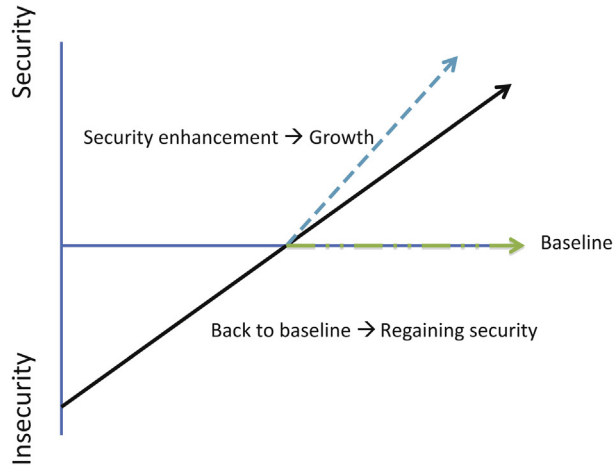

FIGURE 10.5 The relations between attachment security and insecurity. The black line represents a continuum from insecurity to security; the *dash-dotted line* (*green* in the web version) and *dash line* (*blue* in the web version) represents two alternatives: one is that security can only be increased up to the baseline, the other that security can be elevated beyond that to some growth or flow state, but both of them suggest a nonlinear move/growth/trend from insecurity to security.

Another question has to do with state versus trait differences in attachment (eg, Gillath, Hart, Noftle, Stockdale, 2009). For example, what happens when a dispositionally insecure person is primed with security? Or primed repeatedly over time? Do temporary changes in state attachment accumulate to yield some kind of change in trait attachment over time? And if so, how do these changes reflect in brain structure and functioning or gene expression? These issues should be tested and integrated into the suggested framework of attachment, while keeping other models of attachment in mind [eg, how does security fit into models depicting attachment (and love) as an addiction; Burkett & Young, 2012].

In summary, the domain of human attachment neuroscience, although young, is mushrooming and continuously contributing to our understanding of attachment. Although adult attachment has been studied for almost 30 years, and attachment in general has been studied for more than 50 years, there is still much to learn and many questions remain open. Neuroscience is an essential approach to finding answers for these questions. In this chapter, we have reviewed some of the key findings obtained using various methods of neuroscience, have described some of the models suggested to explain the neuroscience of attachment, and have provided a few directions for future investigations. Despite all we have covered in this chapter, this is merely the tip of the iceberg when it comes to understanding attachment neuroscience. Finally, modern psychology is increasingly interested in understanding the relationship between psychological outcomes and brain function and structure. Both neuroscience and relationship science stand to benefit from this relatively new, yet successful integration represented here in the neuroscience of attachment.