

# WHO ARE YOU?

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WHO ARE YOU—WHO WHO, WHO WHO?

—The Who



Figuring out how the brain works is a daunting task. For that reason, neuroscientists usually work only on pieces of the puzzle—like aspects of cognition, emotion, or motivation—rather than on the whole organ and its systems at once. But if we want to understand how our brains make us who we are, we have to figure out how these individual processes blend together to cause a person to emerge effortlessly from the electrochemical activities of the protoplasmic mass that is his or her brain. It's time for me to bite the bullet and explain how I think the brain, specifically its synapses, makes us who we are.

## BRAINS AND OTHER PARALLEL COMPUTERS

I once had a friend who supported a bohemian life in downtown Manhattan with a job as a computer programmer in a neuroscience lab. He was extremely talented, too talented for such work, and eventually got a position more suited to his abilities. He was hired by a company in Boston that made powerful computers called “connection machines.” It was through him that I first heard about parallel computers.

Parallel computers work differently from the standard model with which we're familiar. Rather than doing computations one at a time in sequence (in

other words, carrying out the steps of a program line by line in serial order), they process many steps simultaneously. Parallel computers can function this way because, in contrast to your desktop PC or Mac, they have many processing units that can be devoted to the execution of a given task. By distributing the workload across the various processors, they can perform the task much faster than serial computers. (Don't plan on getting one for your home or office, however: they cost millions of dollars.)

The brain is also sometimes described as a parallel computer, but it actually functions differently from an off-the-shelf connection machine. The brain is organized into processors' (neural systems) that function independently of one another (at least to some extent). Since each of these systems has a specific job assignment, several types of tasks can be done by the brain simultaneously, that is, in parallel. This architecture enables you to chew gum and walk down the street, guiding yourself toward your destination while feeling happy and rehearsing the phone number your friend gave you a block back, all at the same time as your posture is maintained upright, your blood pressure is kept at a safe level, and your rate of breathing is paced to the oxygen needs imposed by all the activities in which you're engaged.

Connection machines can, like brains, be divided up in such a way that different groups of processors are responsible for particular tasks.<sup>2</sup> Although each task is then performed less efficiently than it would be if all the processors were devoted to it, overall this can be a more efficient use of the machine since multiple tasks can be worked on at the same time. Reversing the logic, if we had fewer neural systems using up the same overall computing power in our brains the systems would each be more powerful. However, because we have to do lots of different things each day to stay alive and well (eat, sleep, walk, avoid danger and pain, hear, see, smell, taste, talk, and think, to name some), with fewer brain systems we would almost certainly be less capable, even if the remaining systems were each more proficient at their particular tasks.

Over the long, slow evolutionary process of building the brains of vertebrates, and then of mammals, and eventually of our own species, the neural systems we have were specifically designed to take care of important jobs. We don't have extra ones that we can easily give up and continue to lead life as before. Similarly, new systems aren't easily acquired. For example, the addition of language and related aspects of cognition to the primate brain was not a trivial process—the brain was by that point in its evolution already fully booked. Adding a whole new set of functions, therefore, required either a loss

of space devoted to some functions or an increase in brain size. In fact, both seem to have occurred. The human brain is bigger than that of other animals (relative to our body size)<sup>3</sup> and also seems to have undergone some reorganization. For example, the neural mechanism underlying the perception of spatial relations is present in both hemispheres of other primates; it is mainly on the right side in humans. This implies that spatial perception was forced from the left during the language invasion of human synaptic territory.<sup>4</sup>

Life requires many brain functions, functions require systems, and systems are made of synaptically connected neurons. We all have the same brain systems, and the number of neurons in each brain system is more or less the same in each of us as well. However, the particular way those neurons are connected is distinct, and that uniqueness, in short, is what makes us who we are.

## THE PARADOX OF PARALLEL PLASTICITY

We've met a number of the brain's neural systems throughout this book. Included are networks involved in sensory function, motor control, emotion, motivation, arousal, visceral regulation, and thinking, reasoning, and decision-making. What is remarkable is that synapses in all of these systems are capable of being modified by experience. Consider a few examples.

Emotion systems, as we've seen, are programmed by evolution to respond to some stimuli, so-called innate or unconditioned stimuli, like predators or pain. However, many of the things that elicit emotions in us or motivate us to act in certain ways are not preprogrammed into our brains as part of our species heritage but have to be learned by each of us. Emotion systems learn by association—when an emotionally arousing stimulus is present, other stimuli that are also present acquire emotion-arousing qualities (classical conditioning), and actions that bring you in contact with emotionally desirable stimuli or protect you from harmful or unpleasant ones are learned (instrumental conditioning). As in all other types of learning, emotional associations are formed by synaptic changes in the brain system involved in processing the stimuli. Some of the brain's plastic emotional processors include systems involved in detecting and responding to danger, finding and consuming food, identifying potential mates and having sex.

Sensory systems are likewise plastic. Until very recently, it was believed that perceptions are stable from day to day and year to year because sensory systems are immutable after childhood. But studies by Norman Weinberger, Charles Gilbert, Mike Merzenich, and others have shown that these systems

are remarkably susceptible to modification in response to external stimulation.<sup>5</sup> Merzenich, for example, has shown in a variety of ways how experience with a particular stimulus alters the net area, and thus the underlying synaptic territory, devoted to processing that stimulus. Following amputation of a finger, for example, the area in the somatosensory cortex devoted to that finger shrinks, while extensive stimulation of a particular finger expands the area of cortex devoted to it.

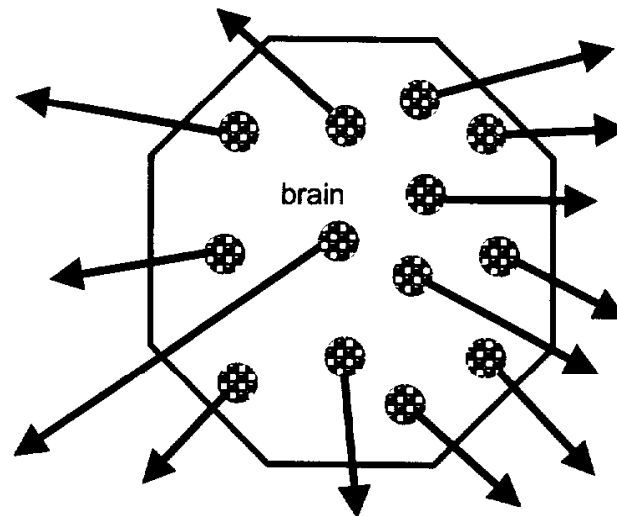
Motor systems are also plastic. This is obvious from the fact that we can learn skills and can improve our ability to perform certain movements with practice. As we saw in chapter 5, changes in synaptic transmission in the cerebellum are important for some forms of motor skill learning. Synaptic plasticity also occurs in the motor cortex, basal ganglia, and other brain regions involved in motor control.<sup>6</sup>

Because synaptic plasticity occurs in most if not all brain systems, one might be tempted to conclude that the majority of brain systems are memory systems. But as I argued in chapter 5, a better way of thinking about this is that the ability to be modified by experience is a characteristic of many brain systems, regardless of their specific function. Brain systems, in other words, were for the most part not designed as storage devices—plasticity is not their main job assignment. They were instead designed to perform particular tasks, like processing sounds or sights, detecting food or danger or mates, controlling actions, and so on. Plasticity is simply a feature that helps them do their job better.

The fact that plasticity does occur in so many brain systems, however, raises interesting questions. How does a person with a coherent personality—a fairly stable set of thoughts, emotions, and motivations—ever emerge? Why don't the systems learn different things and pull our thoughts, emotions, and motivations in different directions? What makes them work together, rather than as an unruly mob?<sup>7</sup>

## LESSONS FROM DISCONNECTION

Before we examine what holds the self together, let's consider how fragile a patch job it is. The bottom line is simple: Functions depend on connections; break the connections, and you lose the functions. This is true of the function of a single system (a lesion of the visual thalamus, for example, will prevent information from the eyes from reaching the cortex, and thus will prevent the cortex from being able to perceive the visual world) as well as of interactions



**FIGURE 11.1 BRAIN SYSTEMS AS AN UNRULY MOB**

Given that we have so many brain systems capable of learning and storing information about who we are, how is it that we develop distinct personalities? How can we function as an individual, a person, with goals and aspirations, with an identity, rather than as an unruly collection of systems that learn and store information on their own?

between systems (a lesion in a certain part of the temporal lobe will prevent information about visual objects from reaching the prefrontal cortex and thus will prevent that stimulus from being held in working memory, and hence from being used as the basis for thinking and decision-making).

The most striking instance of a broken connection I've ever personally witnessed involved a teenage boy who underwent split-brain surgery for the control of epilepsy.<sup>8</sup> In this operation, the nerves between the two sides of the brain are severed to prevent the seizures from moving between the left and right hemispheres. One of its unwanted consequences is that the two hemispheres become somewhat independent. Several days after the surgery, the boy was observed pulling his pants down with his right hand and up with his left. Since the right hand is under the control of the left hemisphere, and the left hand of the right hemisphere, the normal integration of motor control that exists so effortlessly in each of us broke down in this boy because the brain systems that had final say over what the two hands do were no longer connected. (This sort of conflicted behavior has been described for many such patients but was amazing to see firsthand.)

Another example involves conduction aphasia. Patients who suffer from this condition can speak without trouble and can understand spoken words (for example, they can point to the picture of an object named by someone

else), but cannot, upon hearing a spoken word or sentence, repeat it, or answer a question posed to them. The reason for this, according to the late Norman Geschwind, one of the great neurologists of the twentieth century, is that the neural pathway that transmits information between the areas of the brain responsible for the comprehension and production of speech is cut.<sup>9</sup> Conduction between the brain areas is thus disrupted.

Geschwind used the phrase *disconnection syndromes* to characterize conditions in which specific behavioral or mental consequences result from disrupting communication between brain regions. What is striking about these disorders is that the deficit results not from the loss of a particular function, but from the inability to exchange information between brain areas. Disconnection syndromes illustrate how critical internal coordination between brain systems is in maintaining the unity of mind and behavior.

While the effects of brain lesions are not invariably interpreted as disconnection syndromes, disconnections always occur when the brain is damaged. For example, lesions of the prefrontal cortex, especially the ventral or orbital frontal cortex, have, since the nineteenth century, been known to drastically alter personality.<sup>10</sup> After an iron rod passed through the head of Phineas Gage in a railroad accident, this upstanding citizen suddenly became fitful, irreverent, and unrestrained. As one contemporary observer put it, "The equilibrium between his intellectual faculties and animal urges seems to have been destroyed."<sup>11</sup> Building on this case and numerous others, Antonio Damasio has suggested that damage to the ventral prefrontal cortex can lead to a loss of social control, and in the extreme can cause sociopathic behavior.<sup>12</sup> But it would be wrong to think of the ventral prefrontal region as the center of social grace. According to Damasio, the consequences of damage to the ventral prefrontal cortex can be conceived of as a breakdown in the ability to use emotional information to guide thoughts and actions. This seems reasonable given that some of the key connections of the ventral prefrontal cortex include areas involved in higher cognitive processes (other prefrontal regions such as the lateral prefrontal cortex and anterior cingulate cortex, as well as the hippocampus) and emotional and motivational functions (the amygdala and nucleus accumbens). Ventral prefrontal damage thus does more than just create a hole in this part of the brain. It removes that brain region from the circuits in which it participated. Regardless of whether it is obvious or not, brain lesions always produce disconnections.

Connectivity changes also take place in psychiatric disorders, but they are typically more subtle than those in neurological patients with overt brain le-

sions. As a result, psychiatric disorders might be best thought of as malconnection rather than disconnection syndromes. For example, as described in the previous chapter, a growing body of evidence suggests that certain forms of depression appear to involve alterations in the way circuits in the hippocampus, as well as in the prefrontal cortex and amygdala, adapt to the consequences of long-term elevations of stress hormones. And just as a brain lesion in one area can affect the functions mediated by other regions or systems with which it is connected, so, too, can alterations in the synaptic operation of a region. The only thing one brain area knows about another is the state of its synapses. Change the synapses in one area, and like dominoes in a line, synapses in others will be altered as well.

Most of the time the brain holds the self together pretty well. But when connections change, personality, too, can change. That the self is so fragile an entity is disconcerting. At the same time, if the self can be disassembled by experiences that alter connections, presumably it also can be reassembled by experiences that establish, change, or renew connections. An important challenge for the field of neuroscience is to figure out how to manipulate the brain in a way that patients with mental disorders can, either alone or with the help of a therapist, try to put the self's synapses back together.

## SELF-ASSEMBLY

As described in chapter 4, your brain was assembled during childhood by a combination of genetic and environmental influences. Genes dictated that your brain was a human one and that your synaptic connections, though more similar to those of members of your family than to those of members of other families, were nevertheless distinct. Then, through experiences with the world, your synaptic connections were adjusted (by selection and/or instruction and construction), further distinguishing you from everyone else.

Synaptic connections are adjusted by environmentally driven neural activity in specific neural systems. When these changes occur during early life, they are said to involve developmental plasticity; when they occur later, they are considered as learning. But the line between developmental plasticity and learning is a fine one and perhaps nonexistent. I will therefore ignore this distinction, and plunge right into the question of how synaptic plasticity occurring in multiple neural systems is coordinated in the process of assembling, and maintaining, the self. The manner in which this occurs can, I think, be understood in terms of seven principles.

**PRINCIPLE 1** DIFFERENT SYSTEMS EXPERIENCE THE SAME WORLD.

Although the different neural systems have different functions, because they are part of the same brain they will be involved in encoding the same life events. One system processes the sights, another the sounds, and still another the smells in a given scene. Additional systems will determine whether those sights, sounds, or smells indicate that danger is present, or that there might be something tasty to eat out there. From the point of view of the organism, and the world with which it is interacting, these are not different experiences, but rather different aspects of a single experience. And although each system is plastic, and can thus learn and store information, each is learning and storing information about the same experience. Just as people living in different towns of a country who never meet face-to-face can share a culture because they have similar environmental influences (similar climate, similar geography, similar myths and legends, similar political histories, similar current political situation, similar social institutions), within the brain, a kind of shared culture develops between the various systems because they are exposed to similar environmental circumstances.

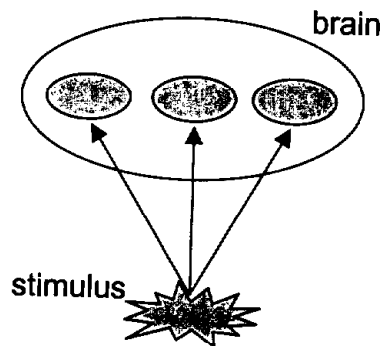
To make this principle more explicit, I've put it in pictorial form in figure 11.2. Part A shows a hypothetical brain with three neural systems. Each system receives inputs from the outside world. In this model brain, the systems do not communicate directly with one another. But because they have the same inputs, they come to know exactly the same things about the world. They are thus completely redundant processors.

Part B adds a bit of complexity. Now the three systems are distinct and nonredundant—each encodes the world differently (as represented by the different fills in the shapes). These can be thought of, for example, as different sensory systems—visual, auditory, and olfactory, if you like. In spite of the fact that each processes qualitatively different information, because they experience the same events, there is still a strong degree of overlap in what they represent about the world. The sounds, sights, and smells that occur during an experience are different pieces of information from the point of view of each of the three systems, but are all part of the same experience from the point of view of the brain and person.

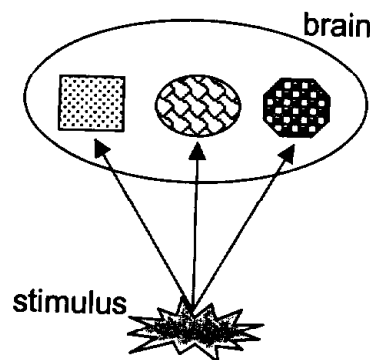
Now, imagine that our hypothetical brain visits three different places where it has three different experiences. Each experience will be encoded in parallel by the three systems—as sights, sounds, and smells. Contrast this to what would take place if, during each of the three experiences, only one of the neural systems was active, and the one that was active differed in each situation.



### A Processing of Common Input by Identical Brain Systems



### B Processing of Common Input by Unique Brain Systems



**FIGURE 11.2 SHARED INPUTS TO DISTINCT SYSTEMS COORDINATE PARALLEL PLASTICITY**

Although we have many brain systems that are capable of learning and storing information, because they experience the same events, they all learn and store information about the same things rather than about different things. However, each system is different. As a result, although they learn and store information about the same events, they process different aspects of these events.

In this case, although the organism and its brain had three unique experiences, each system had only one. As a result, this brain has information about one environment in the form of sights, the second in sounds, and the third in smells, but because no parallel encoding took place, there is no shared information about the three experiences across the three systems.

Normally, this condition does not occur. The various systems of your brain share the same experiences. They encode them differently, but they encode

the same external events. They will not always focus on the same details, and each may not always participate in every experience. But to the extent that a neural system encodes an experience, it is likely that some other systems of the brain are encoding the same experience. As a result of parallel encoding by, and parallel plasticity within, neural systems, a shared culture develops and persists among the systems, even if they never communicate directly.

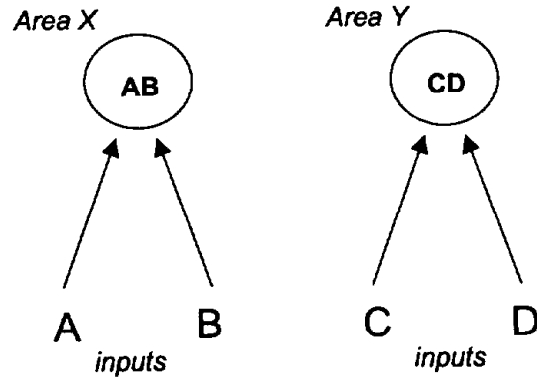
**PRINCIPLE 2** SYNCHRONY COORDINATES PARALLEL PLASTICITY.

In real brains neural networks do not exist in isolation. They communicate with other networks by way of synaptic transmission. For example, in order to see an apple, instead of a roundish, reddish blob, the various features of the stimulus, each processed by different visual subsystems, have to be integrated. As we saw in chapter 7, the problem of understanding the manner in which this occurs is called *the binding problem*. One popular solution to this problem is based on the notion of neuronal synchrony.<sup>13</sup> Synchronous (simultaneous) firing, and thus binding, has been proposed as an explanation of consciousness (chap. 7), but our interest here is more in the ability of synchronous firing between cells in different interconnected regions to coordinate plasticity across the regions.

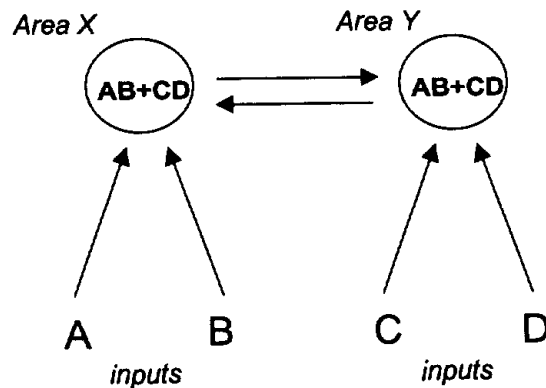
Wolf Singer is one of the major proponents of synchrony as a means of integrating plasticity across regions, especially within the visual system.<sup>14</sup> In brief, his basic idea is that information processing across different interconnected regions is coordinated when cells in the individual regions fire action potentials synchronously—that is, at the same time. Form and color, for example, are brought together for an immediately present object by the fact that the cells processing the particular form and particular color are active at the same time. By way of the synaptic interconnections between cells in the color and form regions, Hebbian plasticity occurs (since the cells will be activating each other at the same time they are being activated by the external visual stimulus). Hebbian plasticity thus binds simultaneously active cells together so that the next time the same or similar stimulus occurs, the same cells and connections will be activated (fig. 11.3). That synchronous firing can lead to Hebbian plasticity (as opposed to conscious perception) is incontrovertible. In fact, simultaneous (or near simultaneous) activation of inputs is what accounts for Hebbian plasticity (chap. 6).

Unfortunately, little is known about whether changes of this type actually take place between networks in the brain (as opposed to within individual

### Co-active inputs create associations in each area



### Interactions between areas also create associations



**FIGURE 11.3 PROPAGATION OF PLASTICITY ACROSS NETWORKS COORDINATES PARALLEL PLASTICITY**

In order for Hebbian plasticity to be useful in integration across networks, as opposed to simply inducing plasticity within a region, the plasticity would need to propagate between networks. Two areas (X and Y) that receive nonoverlapping inputs (X receives A and B, and Y receives C and D) are shown. If inputs A, B, C, and D are all active at the same time, the co-activity of A and B will induce Hebbian plasticity in X, and co-activity of C and D will produce plasticity in Y. But because X and Y are connected, activation of X (produced by activity in A and B) will lead to activation of Y at roughly the same time that Y is being activated by C and D. As a result, the CD association in Y will be associated with X (that is, with the AB association). Similarly, activation of Y (produced by activity in C and D) will lead to activation of X at about the same time X is being activated by A and B, and the AB association in X will be associated with Y (that is, with the CD association).

networks). However, recent studies using computer simulations have begun to explore how plasticity occurring in individual systems alters information processing in interconnected systems.<sup>15</sup> This work provides a foundation for pursuing similar studies in real brains.

Integration across brain regions (binding) is usually discussed in the context of perception. However, understanding long-distance communication in the brain is also important for memory, emotion, motivation, and other systems. Exploration of interactions across systems is going to be especially crucial as we try to come to grips with the relation of the self to the brain. A ridiculously simple example is the fact that our perception of an apple is not just based on the integration of the shape and form and other visual features of the object, but also on the integration of these features with information stored in memory about the object and our experiences with it, and its significance for us at the moment, in the past, and in the future.

**PRINCIPLE 3** PARALLEL PLASTICITY IS ALSO COORDINATED BY MODULATORY SYSTEMS.

Parallel processing in different brain systems is further coordinated by modulators. As we've seen, these are released throughout the brain in the presence of significant stimuli, including novel, unexpected, or painful stimuli, or stimuli that otherwise signal emotional arousal. In the last chapter, we examined the role of one class of modulators, the monoamines, in mental disorders. Here we examine their role in normal brain function. These are two sides of the same coin.

In the middle of the twentieth century, researchers discovered a region of the brain stem that was required for alertness and arousal.<sup>16</sup> Damage to this region put animals and people into a comatose state. Stimulation of the same region could awaken an animal from a deep sleep, and if an animal was already awake, alertness and attention were enhanced by the stimulation. This area came to be called the "reticular activating system" or the "reticular formation." Subsequently, it was discovered that arousal functions were largely accounted for not by a single integrated system in the brain stem but by the activities of several different groups of neurons in the vicinity, with each group having a unique chemical signature (different neurotransmitter molecules are present in the different groups).<sup>17</sup> The chemicals in question are all amines, and specifically include the monoamines—dopamine, norepinephrine, epinephrine, serotonin—and acetylcholine.

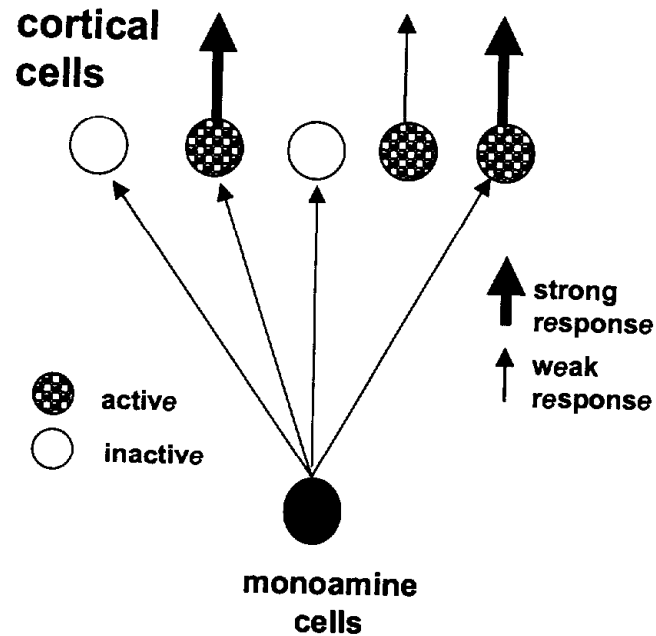
The cells that produce modulators are located primarily in the brain stem,

but their axons are distributed throughout the brain. Consequently, when these cells are activated, many brain areas are affected. The widespread action of modulators makes them especially useful in broadcasting that something significant has happened, but they are less suited to identifying exactly what it is that's happening. The modulatory system functions somewhat like an alarm sounded by the firehouse in the center of a small town. The alarm is very effective in alerting all the town's firemen to the fire, and in summoning them to the station, but it doesn't tell them whose house is on fire. This they have to learn by other means, just as brain areas have to determine precisely what it is that's causing the arousal by other means.

The main job of modulators is to regulate neurotransmission between neurons, but they don't work at all the many synapses that they bathe. They are mainly effective in modulating transmission at synapses that are already active when the modulator arrives (fig. 11.4).

Because modulatory systems are activated during significant experiences, modulators can selectively facilitate transmission at the synapses actively processing information about such experiences across widely distributed neural systems. Emotional or otherwise significant experiences are the ones we tend to form memories about, and as described in chapter 8, it is well established that modulators like norepinephrine are involved in the enhancement of memory that occurs during emotional events. Norepinephrine has also been implicated in the induction of long-term potentiation (LTP),<sup>18</sup> which, as we've seen, is a laboratory procedure for studying synaptic plasticity. Thus, LTP is facilitated when this chemical is present, and disrupted when it is absent. So not only can modulators produce a momentary facilitation of transmission in circuits actively involved in processing significant events, they can also promote synaptic plasticity, and thus learning and memory, in those circuits. Recall that one of the most prominent current theories supporting monoamine treatment of mental disorders is that these drugs make more serotonin and/or norepinephrine available at synapses and thereby trigger intracellular molecular cascades that promote synaptic plasticity. Modulation of plasticity across brain systems is thus important in both normal and pathological mental conditions.

One of the most important features of modulators is that, once released, they have a prolonged action, at least with respect to transmitters like glutamate or GABA. The primary action of glutamate or GABA is typically concluded within a matter of milliseconds, whereas modulators can have effects that last for seconds. Given that not all brain systems operate at exactly the same rate (some involve more distant pathways with more connections, and,



**FIGURE 11.4 MODULATORY CHEMICALS COORDINATE PARALLEL PLASTICITY**

Monoamine cells in the brain stem send connections to widespread brain regions (cells in different regions of the cortex are illustrated) and release monoamines during significant events. Although cells in many regions will simultaneously be bathed by monoamine release, only active cells (cells actively involved in processing current events) will be affected. For example, three active cortical cells are shown. The two active cells that receive monoamine inputs produce a stronger response than the one cell that does not receive the monoamine inputs. One effect of monoamines is to facilitate plasticity. Thus, learning is facilitated in those cells in areas actively processing the event. In this way, plasticity is coordinated across widespread regions during significant events, increasing the likelihood that cells actively engaged in processing the event will store information about the event. Because different brain regions store different aspects of an experience, such coordination is important to the unity of our memories (explicit and implicit) of an experience.

in general, the more complex the process, the longer it takes to occur because more connections are involved), the slow recovery time of modulators allows them to affect a wide range of processes, from the earliest and simplest in an episode to the last and most complex, promoting learning independent of information extracted during different components of an experience.

Although different systems learn about different aspects of an experience, the widespread action of modulators increases the likelihood that when something significant takes place, plasticity will occur in parallel at active synapses in all these systems. As a result, the learning of multiple elements of an expe-

rience (its sights, sounds, and smells, its emotional and motivational significance, its movement patterns, and so on) is facilitated, allowing the whole experience to be stored at once, albeit across multiple systems. Included, of course, would be systems that process information both implicitly and explicitly, but more about that later.

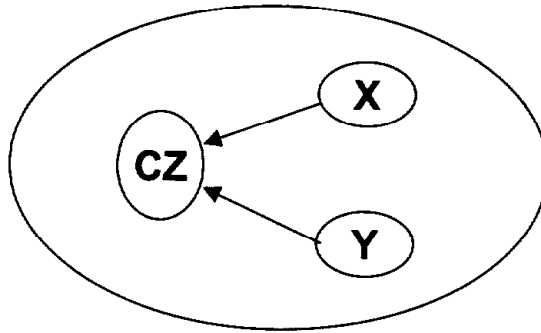
Not all modulators have the same effect (some inhibit rather than enhance plasticity), and the same modulator can have different effects depending on the particular postsynaptic receptor with which it interacts. Moreover, the same interaction between a modulator and its receptor can be different depending on the other cells in the circuit. For example, when serotonin interacts with one of its receptors, it produces inhibition, and when it interacts with another receptor, it produces excitation. But the net effect (that is, whether excitation or inhibition is the outcome) will depend on the kind of neurons on which the serotonin receptors are located. Serotonin, for instance, inhibits the activity of amygdala projection cells (excitatory cells that transfer information from one region of the amygdala to another). But it achieves this inhibition by way of an excitatory serotonin receptor located on GABA cells.<sup>19</sup> Thus, serotonin excites GABA inhibitory cells, and these inhibit amygdala projection cells. While the outcome of the interaction between serotonin and its receptor is excitatory, the outcome for the overall circuit is inhibition. Much more work is needed to better understand the contribution of modulators to routine transmission in specific circuits, and to the induction of plasticity (learning) and its maintenance (memory). But the gaps in our knowledge do not take away from the established role of some modulators in enhancing plasticity, and thus potentially serving as regulators of plasticity across neural systems.

#### **PRINCIPLE 4** CONVERGENCE ZONES INTEGRATE PARALLEL PLASTICITY.

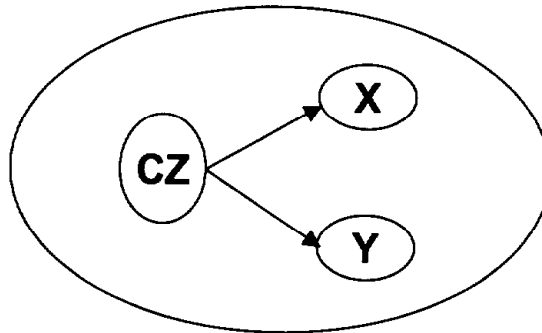
In the examples cited so far, brain systems learn in parallel. While parallel learning is surely an important part of the complex process by which the self is assembled, parallel learning, on its own (even when buttressed by synchrony and modulatory chemicals), is not sufficient to account for the coherent personality of a human being.

Another important mechanism in self-assembly, especially in humans and other primates, is the existence of convergence zones, regions where information from diverse systems can be integrated. Figure 11.5 shows two independent processing units, the outputs of which meet up in a third, a convergence

**Convergence zones integrate processing  
in independent systems**



**Convergence zones can influence  
processing in independent systems**



**FIGURE 11.5 CONVERGENCE ZONES (CZ) INTEGRATE PARALLEL PLASTICITY**

A convergence zone (CZ) is a region that receives inputs from other brain regions and that integrates the information separately processed by the other regions. Important convergence zones are located in the prefrontal cortex. Once information is integrated, it can then be used to influence the activity of the input regions. These are examples of bottom-up and top-down processing. The ability of working memory to integrate information from various systems and hold that information temporarily for the purpose of performing mental operations (comparing, contrasting, recognizing) is a typical bottom-up process, and the ability of working memory to use the outcome of this processing to regulate what we attend to is a typical top-down or executive function.

zone. Many kinds of animals have multiple independent learning systems that can be coerced into learning simultaneously by modulatory chemicals and synchronous firing, but only some animals have convergence zones in their cortex.<sup>20</sup> The cognitive sophistication of a mammalian species, in fact, is nicely predicted by the extent of convergence that occurs in its cortex—more is present in humans than in monkeys, for example, and more in monkeys



than in rats. When plasticity occurs simultaneously in two regions that feed into a convergence zone, plasticity is also likely to occur in the convergence zone since it will be the recipient of the high level of activity that occurs when plasticity is being established in the individual regions. Obviously, synchrony and modulation also influence convergence zones, further increasing their potential to integrate information across systems.

Convergence takes place within systems before it takes place between systems. This can be illustrated by considering object recognition in the “what” stream of the visual cortex (chap. 7). The object recognition system, like other cortical processing systems, is hierarchically organized.<sup>21</sup> The later stages depend on the earlier ones, and information representation becomes increasingly complex as the stages are passed through. In the earliest stage, for example, each cell primarily responds to the orientation of a contour or edge of a small part of the stimulus. Across many cells, all the contours that make up the shape of the stimulus are represented. In the next stage, cells receive convergent inputs from the earlier-stage cells. As a result of getting inputs from cells that represent different parts of that object, each cell in the second stage can represent a larger part of the object. This sort of convergence continues through the hierarchy until at the final stage individual cells represent much of the entire object. These latter cells were once affectionately known as “grandmother cells,” since they were supposed to be able to receive all the information needed to represent a stimulus as complex as your grandmother’s face. While it is no longer believed that grandmother cells exist, many scientists do believe that small sets of synaptically connected cells, called ensembles, receive convergent inputs from lower levels in their processing hierarchy, and represent faces, complex scenes, and other objects of perception.<sup>22</sup> This difference is sometimes described as one between “pontifical” cells, which would make final decisions alone about the way things are, and “cardinal” cells, which do things as a small group.<sup>23</sup> Although single cells have been shown to have remarkable capacities,<sup>24</sup> most researchers accept that ensembles rather than single cells underlie mental and behavioral functions.<sup>25</sup>

Once convergence is completed within systems, it begins to occur across systems. In 1970, E. G. Jones and T. P. S. Powell published a landmark study in which they identified several regions in the monkey cortex that receive convergent inputs from the last processing stage of two or more sensory systems in the cortex.<sup>26</sup> Some of the key convergence zones identified were the posterior parietal cortex, the parahippocampal region,<sup>27</sup> and areas of the prefrontal cortex. The ability of these regions to integrate diverse kinds of infor-

mation explains why they are involved in the most sophisticated cognitive functions of the brain. As we've seen, areas of the prefrontal cortex are involved in working memory functions, which underlie many aspects of thinking, planning, and decision-making. The posterior parietal area plays an important role in the cognitive control of movement in space in nonhuman primates,<sup>28</sup> and in humans is crucially involved in language comprehension in the left hemisphere and spatial cognition in the right.<sup>29</sup> Rhinal cortical areas are part of the medial temporal lobe memory system (chap. 5). They establish critical links between sensory areas of the cortex and the hippocampus, and thus provide the hippocampus with the raw materials needed to form relations between external stimuli in the process of establishing long-term explicit memories. The hippocampus, too, is a convergence zone; rather than integrating inputs from different sensory systems per se, it receives inputs from other convergence zones, and is thus something of a super-convergence zone.<sup>30</sup>

In convergence zones like the hippocampus, it is possible for completely independent sensory representations to be synthesized into memory representations that transcend the individual systems involved in the initial processing. Thus, while different systems may form independent memories of separate aspects of an experience, memories formed in, or by way of, a convergence zone are multifaceted—they include information extracted from different systems. Such memories reflect the whole experience of the organism, rather than bits and pieces of an experience recorded by other systems. But because the bits and pieces are the raw materials, there is a kind of unity of experience between the memory established by a convergence zone and by its lower connections. And because the hippocampus and other convergence zones receive inputs from modulatory systems, during significant states of arousal plasticity in these networks is coordinated with the plasticity occurring in other systems throughout the brain.

One important consequence of this arrangement is that though memories are formed by systems that function both implicitly and explicitly during significant experiences, the memories are coordinated to some degree.<sup>31</sup> That is, the elements you are able to consciously remember about an experience overlap often with some of the elements that were also being separately stored implicitly in other systems. Convergence zones such as those in the medial temporal lobe make possible the creation of consciously accessible memories that integrate elements being encoded separately and implicitly in the other systems. But, remember, while the medial temporal lobe system forms mem-

ories in a way that allows them to be consciously accessible, these memories only enter consciousness when they are placed in working memory. And once in working memory, memories and thoughts can, as we see next, influence activity back down the processing hierarchy.

**PRINCIPLE 5** DOWNWARDLY MOBILE THOUGHTS COORDINATE  
PARALLEL PLASTICITY.

So far, I've placed much of the burden of assembling the self on processes that work more or less automatically, from the bottom up. But this is only part of the story. Convergent representations built from the bottom up are also used to direct activity back down the processing hierarchies. Thoughts and memories placed in working memory, for example, can influence what we attend to, the way we see things, and the way we act. These executive control functions of working memory, which were discussed in chapter 7, are possible because the prefrontal cortex, like other convergence zones, reciprocates projections. That is, connections are sent back to the regions that provide the convergent inputs. By pulling the right strings (activating the right axons), working memory can direct traffic in the areas with which it is connected, enhancing the processing of stimuli that are relevant to the task on which it is engaged and suppressing the processing of other stimuli.<sup>32</sup>

The process by which a thought can cause the brain to issue certain orders is known as downward causation.<sup>33</sup> We prove that downward causation exists every time we carry out an intention. Downward causation is only mind-boggling if you believe that thoughts are one phenomenon and brain activities another. It's still a difficult problem even if you view thinking as a form of brain activity, but the nature of the solution is far more obvious in this case.

If a thought is embodied as a pattern of synaptic transmission within a network of brain cells, as must be the case, then it stands to reason that the brain activity that is a thought can influence activity in other brain systems involved in perception, motivation, movement, and the like. But there's one more connection to make. If a thought is a pattern of neural activity in a network, not only can it cause another network to be active, it can also cause another network to change, to be plastic.

All that is required to induce plasticity at a synapse is the right kind of synaptic activity. If cells processing sensory events can undergo plasticity as a result of the kind of activity those events trigger in sensory systems, then why can't cells processing a thought change the connections of the cells with which

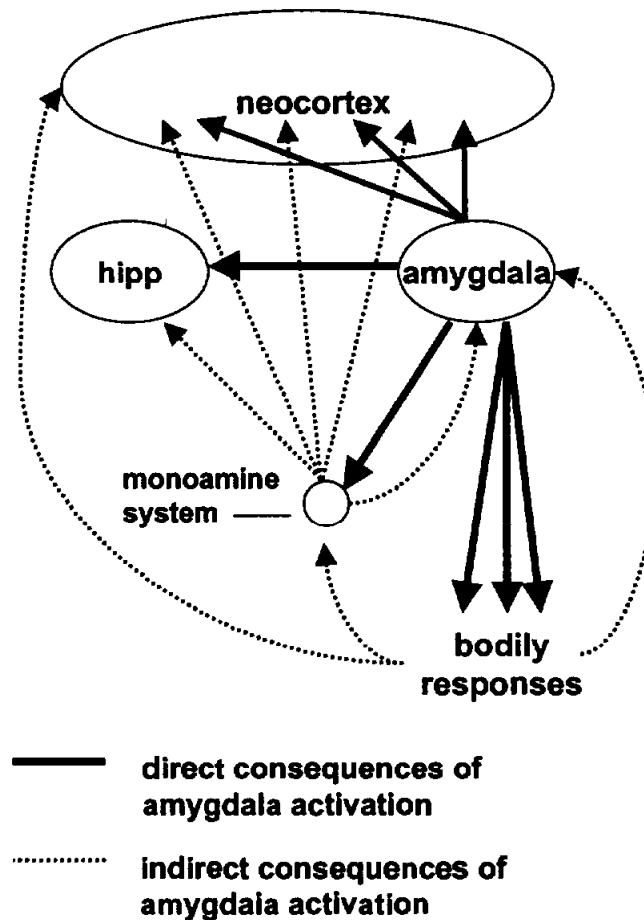
they communicate? Obviously, they do; we simply need to learn more about precisely how this happens.

The downward mobility of thought provides a powerful means by which parallel plasticity in neural systems is coordinated. The more elaborate the convergence zones present in a species, the more elaborate will be the cognitive capacity of the species and the more sophisticated will be the ability of information convergence to coordinate plasticity in that species. With thoughts empowered this way, we can begin to see how the way we think about ourselves can have powerful influences on the way we are, and who we become. One's self-image is self-perpetuating.

**PRINCIPLE 6** EMOTIONAL STATES MONOPOLIZE BRAIN RESOURCES.

Emotions, too, play a key role in organizing brain activity.<sup>34</sup> We've already had a hint of this when we discussed the function of modulators in coordinating parallel plasticity, since emotional stimuli are some of the most potent activators of modulatory systems. But the influence of emotions is much broader than simply activating modulatory systems, and can be illustrated by reconsidering the various ways that the amygdala affects other brain systems when it detects danger (fig. II.6).

In the presence of a threatening stimulus, the amygdala sends direct feedback by way of neural connections to sensory areas of the cortex, encouraging these areas to stay focused on those aspects of the stimulus world that are critical. Amygdala feedback also reaches other cortical areas engaged in thinking and explicit memory formation, encouraging them to think certain thoughts and to form certain memories about the current situation. In addition, the amygdala sends connections to arousal networks, causing them to release their modulatory chemicals throughout the brain. The synapses that are actively involved in processing the external world, in thinking about the world, in forming memories about it, and in receiving the amygdala's feedback will thus be enhanced. In addition, plasticity will be facilitated at these active synapses. And interconnections between active cells in different regions and systems that fire synchronously will be linked by the plasticity that is induced. At the same time, bodily responses controlled by the amygdala will be expressed, and these will provide additional feedback to the brain, not only in the form of bodily sensations that are part of the "felt" response of the emotion, but also in the form of hormones that further affect synaptic activity, over a longer time scale than even modulators. The net result is that emotional arousal penetrates the brain widely, and perpetuates itself.



**FIGURE 11.6 EMOTIONAL MONOPOLY**

Once the amygdala determines that danger is present, it activates a variety of other brain networks. The net result is that the various systems affected are coordinated in their response to the threatening situation. Thus, while many brain systems are active during danger, they are activated in a coordinated way because of the extensive connectivity of the amygdala. Abbreviation: hipp, hippocampus.

As we've seen, the brain has a number of emotion systems, including networks involved in identifying sexual partners and food sources, as well as detecting and defending against danger. When one of these is active, the others tend to be inhibited. For example, other things being equal, animals will tend to spend time in areas where they are safe. So when it comes time to search for food, their fear of certain places, like wide-open spaces or places where they've previously encountered a predator, has to be overcome if that's where food is likely to be found. The hungrier the animal is, the more it will tolerate fear and anxiety and take risks to obtain nourishment. Similarly, both eating and sexual arousal are decreased by activation of systems involved in fear and stress.<sup>35</sup> But, once aroused, sexual desire can override many other brain

systems—people risk all sorts of adverse consequences for an adulterous fling. Not only does the arousal of an emotional state bring many of the brain's cognitive resources to bear on that state, it also shuts down other emotion systems. As a result, learning is coordinated across systems in a very specific manner, ensuring that the learning that does occur is relevant to the current emotional situation.

Because emotion systems coordinate learning, the broader the range of emotions that a child experiences the broader will be the emotional range of the self that develops. This is why childhood abuse is so devastating. If a significant proportion of the early emotional experiences one has are due to activation of the fear system rather than positive systems, then the characteristic personality that begins to build up from the parallel learning processes coordinated by the emotional state is one characterized by negativity and hopelessness rather than affection and optimism.

The wide influence of emotional arousal results in many brain systems being activated simultaneously, many more than if one is engaged in quiet cognitive activity, like lying back musing about something, or even when vigorously thinking about the solution to a problem. And because more brain systems are typically active during emotional than during nonemotional states, and the intensity of arousal is greater, the opportunity for coordinated learning across brain systems is greater during emotional states. By coordinating parallel plasticity throughout the brain, emotional states promote the development and unification of the self.

**PRINCIPLE 7** IMPLICIT AND EXPLICIT ASPECTS OF THE SELF OVERLAP, BUT NOT COMPLETELY.

In spite of the multiplicity of checks and balances that help keep the various systems of the brain on one track, learning about the same experiences and about the same things in each experience, the job performed is not always performed perfectly. Sometimes, the things learned explicitly are not the things that were focused on by the implicit systems, especially emotional systems—recall the ability of the amygdala to learn independent of the cortex (chap. 8). Although there are probably many reasons why this is so, the most obvious one is that there is an imperfect set of connections between cognitive and emotional systems in the current stage of evolution of the human brain. This state of affairs is part of the price we pay for having newly evolved cognitive capacities that are not yet fully integrated into our brains. Although this is also a problem for other primates, it is particularly acute for humans,

since the brain of our species, especially our cortex, was extensively rewired in the process of acquiring natural language functions.

Language both required additional cognitive capacities and made new ones possible, and these changes took space and connections to achieve. The space problem was solved, as we saw earlier, by moving some things around in existing cortical space, and also by adding more space. But the connection problem was only partially solved. The part that was solved, connectivity within cortical processing networks, made the enhanced cognitive capacities of the hominid brain possible. But the part that hasn't been fully solved is connectivity between cognitive systems and other parts of the mental trilogy—emotional and motivational systems. This is why a brilliant mathematician or artist, or a successful entrepreneur, can like anyone else fall victim to sexual seduction, road rage, or jealousy, or be a child abuser or rapist, or can have crippling depression or anxiety. Our brain has not evolved to the point where the new systems that make complex thinking possible can easily control the old systems that give rise to our base needs and motives, and emotional reactions. This doesn't mean that we're simply victims of our brains and should just give in to our urges. It means that downward causation is sometimes hard work. *Doing* the right thing doesn't always flow naturally from *knowing* what the right thing to do is.

In the end, then, the self is maintained by systems that function both explicitly and implicitly. Through explicit systems, we try to willfully dictate who we are, and how we will behave. But we are only partially effective in doing so, since we have imperfect conscious access to emotional systems, which play such a crucial role in coordinating learning by other systems. In spite of their importance, though, emotion systems are not always active and have only episodic influence on what other brain systems learn and store. Furthermore, because there are multiple independent emotion systems, the episodic influence of any one system is itself but a component of the total impact of emotions on self-development.



## YOU ARE YOUR SYNAPSES

Synaptic connections hold the self together in most of us most of the time. Sometimes, though, thoughts, emotions, and motivations come uncoupled. If the mental trilogy breaks down, the self is likely to begin to disintegrate and

mental health to deteriorate. When thoughts are radically dissociated from emotions and motivations, as in schizophrenia, personality can, in fact, change drastically. When emotions run wild, as in anxiety disorders or depression, a person is no longer the person he or she once was. And when motivations are subjugated by drug addiction, the emotional and intellectual aspects of life suffer.

That the self is synaptic can be a curse—it doesn't take much to break it apart. But it is also a blessing, as there are always new connections waiting to be made. You are your synapses. They are who you are.



102. Gottesman 1991.
103. This paragraph is based on Barondes 1993.
104. This paragraph is based on Barondes 1993.
105. Butcher 2000.
106. DePaulo 2000.
107. Kandel 1998; Kandel 1999.
108. Gorman 1996.

#### CHAPTER ELEVEN WHO ARE YOU?

1. The brain's processors can be described at a variety of levels, from neurons to synapses to circuits to systems. I'll roughly equate the various processors of a connection machine to the various functional systems of the brain.
2. This is multiparallelism, since several tasks are performed in parallel and each is performed using multiple processors.
3. Jerison 1973.
4. LeDoux 1982.
5. Weinberger 1998; Weinberger 1995; Gilbert 1998; Merzenich et al. 1996.
6. Sanes and Donoghue 2000.
7. The artificial intelligence pioneer Marvin Minsky wrote an interesting book called *The Society of Mind* that also discusses the multiplicity of systems that contribute to mental function. He seems to have been mainly interested in pointing out the diversity and complexity of mental life, rather than in explaining how the diversity is coped with in the process of keeping one's self together.
8. This is an effort of last resort when all other methods fail. For more information on split-brain patients, see Gazzaniga 1970; Gazzaniga and LeDoux 1978.
9. Geschwind 1965.
10. Damasio 1994.
11. Harlow 1868.
12. Damasio 1994.
13. Damasio 1989; Llinas et al. 1994; von der Malsburg 1995; Roskies 1999; Reynolds and Desimone 1999; Singer 2001; for a critique of binding and synchrony as explanations of perceptual phenomena, see Shadlen and Movshon 1999.
14. Engel and Singer 2001; Gray et al. 1989; Singer 2001; Engel et al. 1992; Phillips and Singer 1997.
15. Nargeot 2001; O'Reilly and Munakata 2000; Grossberg 2000.
16. Moruzzi and Magoun 1949.
17. Nieuwenhuys 1985.
18. Izumi and Zorumski 1999; Kobayashi et al. 1997; Katsuki et al. 1997; Brocher et al. 1992; Harley 1991.
19. Stutzmann et al. 1998; Stutzmann and LeDoux 1999.
20. Many animals have subcortical convergence zones that are involved in the regulation of simple behaviors in response to sensory events, but it is in the mammalian cortex that these come to have their full significance.
21. This summary is based on Albright et al. 2000. Some of the key findings over the

years have come from David Hubel and Torsten Weisel, Horace Barlow, Semir Zeki, Charles Gross, Mortimer Mishkin, Leslie Ungerleider, David Van Essen, and many others.

22. For example, see: Rolls 1992; Gochin et al. 1994; Nicolelis and Chapin 1994; Deadwyler and Hampson 1995; Buzsaki and Chrobak 1995; Wilson and McNaughton 1993; Young and Yamane 1992; Shadlen et al. 1996.
23. Martin 1994.
24. Shadlen et al. 1996.
25. Shadlen et al. 1996; Parker and Newsome 1998; Shadlen and Newsome 1994; Zohary et al. 1994.
26. Jones and Powell 1970.
27. As defined in chapter 5, this includes the perirhinal, entorhinal, and parahippocampal cortices.
28. Platt and Glimcher 1999; Colby and Goldberg 1999; Xing and Andersen 2000; Pare and Wurtz 1997.
29. Geschwind 1965; Bhatnagar et al. 2000; Vicari et al. 2000.
30. Mesulam et al. 1977.
31. Kim and Baxter 2001.
32. Reynolds and Desimone 1999; Desimone and Duncan 1995; Kastner et al. 1999; Kastner et al. 1998; Tomita et al. 1999; D'Esposito et al. 1995; Smith and Jonides 1999.
33. Szentagothai 1984.
34. Scherer 2000; Maturana and Varela 1987.
35. Gray 1987.

# SYNAPTIC SELF

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HOW OUR BRAINS BECOME  
WHO WE ARE



JOSEPH LeDOUX

V I K I N G

VIKING

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