CHAPTER 2

Brain Development, Language, and Literacy

MAIN IDEAS OF THE CHAPTER

1. Scientists have discovered that the brain of human adults contains several different kinds of cells that are arranged into a characteristic organization (its *cytoarchitecture*); this organization has been described in terms of an *areal aspect* (different areas perform different functions) and a *laminar aspect* (layers of the brain contain different kinds of cells).
2. There are seven main processes of brain development that eventually produce the characteristic cytoarchictecture of the adult brain: *proliferation* (creating the right total number of cells), *migration* (movement of the created cells to the right location), *differentiation* (transforming cells into the right types), *growth* (each cell increasing in length and width), *synaptogenesis* (cells creating connections with each other), *regressive processes* (eliminating connections and cell death), and *myelination* (coating cells with a fatty acid).
3. There are five factors that can affect the manner in which the aforementioned seven processes of brain development are carried out: (a) genetics, (b) environmental stimulation, (c) nutrition, (d) steroids, and (e) teratogens. These factors can either help or hinder the operation of the seven processes of brain development. When problems arise in brain organization, one or more of these factors could be responsible.
4. Although genes play an important role in shaping brain development, plasticity and environmental input show that children are not destined to have a particular brain—in other words, biology is not destiny.

WHY STUDY BRAIN DEVELOPMENT?

As the second installment in the discussion of foundational issues, we turn next to the topic of brain development. We include a chapter on this topic for several reasons. First, all aspects of human cognition (including language and literacy) are products of brain activity. When people are processing spoken language or expressing ideas themselves, their brains are active. Given that the brain is clearly involved in acts of communication, it seems reasonable to examine what we know about the neural basis of language and literacy to see whether this information provides any clues to the functioning of these skills. Second, for over 100 years, researchers have documented the serious communication problems faced by individuals when particular regions of their brains are damaged or poorly developed. By examining brain development as it pertains to language and literacy skills, readers of this book may gain insight into the possible differences between individuals who acquire these skills in normative ways and individuals who lag behind their peers in one or more ways.

It is important to note, however, that there is a strong tendency among some school personnel, the media, and curriculum publishers to overinterpret neuroscientific research (Byrnes & Eaton, in press). There is no meaningful difference between instructional approaches that "teach to the brain" than those that do not since all approaches have some effect on brain functioning. *Brain-based education* as a term is as meaningless as *stomach-based nutrition*. That said, there are four primary reasons why neuroscientific studies could have relevance to educational theories and practice. The first is that *certain kinds* of neuroscientific findings could help decide among competing claims of psychological researchers. For example, psychological researchers used to strongly debate the question of whether it was necessary to subdivide working memory into visuospatial and verbal components. After neuroscientific studies showed that different brain regions were active when visuospatial and verbal working memory tasks were performed, the debate was resolved. The second reason why neuroscience could be informative has to do with helping us understand why disorders of language and literacy emerge. When we know which brain regions work together to perform some task, we can develop hypotheses as to the possible reasons for disorders. The third reason is that brain development could be the reason why certain language skills emerge when they do at particular ages and what could go wrong to produce brain systems that are not functioning properly. The fourth reason is that there have been some surprising serendipitous findings in neuroscience that have led to new studies that would never have been considered using psychological research alone. For example, one part of the brain called the *angular gyrus* (take your left hand and point to a spot a little above and to the rear of your left ear) participates in both reading and math performance (Byrnes & Eaton,

in press). Why might that be? Could such common recruitment of brain areas explain the very high correlation between reading and math skills? There are many neuroscientific findings that have little bearing on these four reasons and are, then, potentially interesting but uninformative with respect to psychological theory and educational practice.

THE GOAL OF BRAIN DEVELOPMENT

All of our brains are produced by a combination of genetically determined and environmentally determined processes. The goal of these two sets of processes is *to produce a brain that has the right number and right type of brain cells that are located in the right brain areas and connected to other brain cells in the right way*—that is, neuroscientists have revealed that learning problems can ensue from having either too many or too few brain cells in particular areas of the brain. In addition, problems also seem to arise from there being too many local synaptic connections between neurons in specific brain areas and from aberrant long-distance synaptic connections between brain areas (Byrnes & Eaton, in press). Such anatomical descriptions are referring to the *cytoarchitecture* of each person's brain. Although neuroscientists still do not have all of the details, it must clearly be the case that certain cytoarchitectures support normal and even high levels of language and literacy skills, while others may lie at the basis of language and literacy disorders.

In the first section of this chapter, the notion of cytoarchitecture is explored further as a means of laying the groundwork for subsequent sections. Then, consideration is given to the processes by which this cytoarchitecture is constructed during prenatal and postnatal development. Finally, the factors that facilitate or hinder optimal brain development are discussed.

FURTHER EXPLORATIONS OF CYTOARCHITECTURE: CELL TYPES AND BRAIN LAYERS

An interesting fact about the brain is that there is a certain degree of localization of cognitive functions in the cortex and elsewhere (Byrnes, 2001b; Kosslyn & Koenig, 1994). The evidence for the *areal organization* of the cortex comes from a variety of sources. With animal brains, for example, researchers can insert probes into an animal's cortex and show that a small cluster of cells are selectively active only when the animal is engaged in a particular kind of task (e.g., detecting vertical lines). With human brains, researchers have relied on other kinds of evidence such as *double dissociations*. A double dissociation exists when damage to one area of the brain

causes a deficit in one aspect of a skill (e.g., semantic processing), while damage to another area causes a deficit in a different aspect (e.g., syntactic processing). Although it is important not to infer too much from such findings (because the localization is relative rather than absolute), researchers could reasonably argue that such evidence provides support for theoretical models that propose that a given skill (e.g., language) can be subdivided into specific component subskills (e.g., semantic and syntactic processing).

In addition to the areal organization of the brain into specific processing regions, the cortex also has a characteristic *laminar* organization (i.e., layers that extend deeper into the brain). A prerequisite to understanding the latter type of organization is to recognize that the brain contains a number of different types of cells that fall into two broad classes: *glial cells* and *neurons*. Glial cells are far more numerous than neurons, but they do not seem to play a role in the processing of information (as far as scientists know). Instead, they provide a number of other important functions including (1) providing firmness and structure to the brain, (2) forming the myelin sheath that surrounds the axons of long neurons and speeds up their firing, (3) providing a "scaffold" for neurons to latch on to during the process of cell migration (see below), and (4) taking up and removing some of the chemical transmitters that are released during synaptic transmission (Kandel, 1991).

Neurons, in contrast, do play a role in the processing of information and come in a variety of types that differ in terms of their shape, patterns of connectivity, and the neurotransmitters they release. The shape dimension underlies the distinction between *pyramidal cells* (see Figure 2.1) and other types of cells (e.g., star-shaped cells called *stellate cells*). The former comprise more than 80% of the neurons in the brain (Johnson, 1997; Moyer, 1980). The connectivity dimension underlies the distinction between excitatory and inhibitory neurons. The neurotransmitter dimension allows one to distinguish among neurons that excrete dopamine, neurons that excrete gamma-aminobutyric acid (GABA), and neurons that excrete serotonin.

Scientists discovered these various aspects of neurons in the midst of examining microscopic slides of brain tissue. This microscopic approach also revealed the fact that the cortex comprises six horizontal layers that differ in terms of the morphology, density, and functional properties of the neurons in them (Chenn, Braisted, McConnell, & O'Leary, 1997; Johnson, 1997). Layer 1 is the highest (most superior, closest to the scalp) layer and consists primarily of long, horizontal fibers that connect different regions of the cortex to one another. Note that when electrodes placed on the scalp record brain activity in studies using electroencephalograph (EEG) methods, these electrodes record only the horizontal neurons in the top layer. In the next layer down, layers 2 and 3 also contain horizontal fibers as well as small pyramidal cells that extend apical dendrites upward as well as collateral projections outward to neighbors. A *dendrite* is the branching

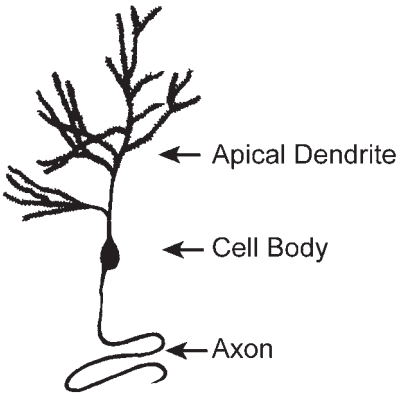

FIGURE 2.1. Basic structure of a pyramidal cell. From Byrnes (2001b). Copyright 2001 by The Guilford Press. Reprinted by permission.

portion of a neuron that receives neurotransmitters secreted by presynaptic neurons (see Figure 2.1). Layer 4 is the terminal point for many input fibers from subcortical regions (e.g., the thalamus). These inputs primarily make contact with the large number of stellate cells found in layer 4. Layers 5 and 6 are the most inferior (deepest) layers that have a high concentration of large pyramidal cells that project long-distance, output fibers to important subcortical sites as well as apical dendrites that extend upward to layers 4 and 1 (but not layers 2 and 3). Thus, all of the layers contain neurons that make either horizontal connections with neurons in other layers or vertical connections with neurons in the same layer (or both).

This laminar arrangement of cells is a defining characteristic of an adult brain. As such, it can be used to judge the relative maturity of children's brains at various ages. For example, a researcher may wish to consider whether 5-year-old children seem to have the same number of stellate cells in layer 4 as adults have (e.g., Huttenlocher, 1993). Similarly, researchers might consider whether the neurons in children's brains seem to make the same number of synaptic connections as the neurons in adults' brains. In the next section, these issues are considered further as the processes that create the laminar structure of the brain are explored.

SEVEN MAJOR PROCESSES OF BRAIN DEVELOPMENT

Prenatal development is often characterized in terms of structurally defined phases—that is, the boundaries of specific prenatal periods are set by the emergence of particular structural or anatomical features in an embryo or fetus (Purves & Lichtman, 1985). The period of the *zygote,* for example,

begins when a sperm cell fertilizes an ovum and concludes when cell divisions within the zygote create a structure called a *blastula* (a hollow sphere of cells). Soon thereafter, the cells on the surface of the blastula invaginate along an indentation and create a groove called the *primitive streak*. The latter process is called *gastrulation* because it creates a structure called a *gastrula*. Somewhat later during a process called *neurulation,* two symmetrical, protruding folds of tissue emerge on the longitudinal surface of the gastrula, move closer together (like two ocean waves moving toward each other), and eventually fuse above the primitive streak to form the *neural tube* (imagine a thin rubber tube placed on top of a ball). One end of the neural tube eventually gives rise to the structures of the forebrain and midbrain. The other end eventually gives rise to the spinal cord (Johnson, 1997; Moyer, 1980). The end that gives rise to the forebrain and midbrain structures continues to develop and expand in such a way that a characteristic pattern of five convolutions and bulges appears by 5 weeks gestational age. (Imagine a partially inflated balloon attached to a straw.) Whereas the most anterior bulge is eventually transformed into the cortex, the second-most anterior bulge is eventually transformed into structures such as the thalamus and hypothalamus (Johnson, 1997).

Early on, scientists suspected that these bulges arose because the neural tube was manufacturing brain cells somewhere inside its walls. To confirm this suspicion, they used microscopic techniques to observe the formation of bulges *in vivo*. They found two regions within the neural tube (called *proliferative zones*) out of which brain cells emerged in rapid succession. Subsequent studies revealed that precursor cells within the zones produced approximately 100 generations of clones of themselves through *mitotic division* (Rakic, 1993). Mitotic division involves creating exact duplicates and splitting into two identical "daughter" cells, each with a full complement of DNA (as opposed to *meiosis,* which creates gametes, each with half of the DNA of the parent cell). For some types of neuron, each precursor ends up producing 10,000 offspring cells. Given that the process of proliferation is largely over by the seventh prenatal month (in all areas except for the hippocampus) and that children's brains contain more than 10^{11} neurons, it follows that the two proliferative zones must produce progenitors at an explosive rate of 250,000 cells per minute (Johnson, 1997; Purves & Lichtman, 1985)!

Subsequent research revealed a second major process besides proliferation that is instrumental in determining the eventual configuration of cells in an adult brain: *migration*. To understand migration, it is helpful to imagine a (coronal) cross-section of the neural tube that has concentric circles corresponding to various layers (similar to a bull's-eye pattern). The proliferative zones lie near the innermost layer of the tube wall (close to the hollow of the tube or the target of the bull's-eye). The neural tube expands in an outward, bulging manner because newly created cells migrate away

from the proliferative zones to the outer layers of the tube wall. More specifically, as each generation of cells is produced through repeated cell division, they migrate farther and farther away from the inner wall (an inside-out, or radial, progression). Hence, the cells that end up near the outer wall of the neural tube were some of the last ones produced during the process of proliferation. This outer layer is called the *cortical plate* because it ultimately becomes part of the cortex of a mature brain.

In vivo studies have revealed that neurons migrate to outer layers in one of two ways. In the first, newly created cells emerge from the proliferative zones and push older "siblings" away from the zones as they emerge (in the same way that an advancing crowd of protesters would push a line of police backward). In the second, newly created cells traverse along glial cells that are aligned in a perpendicular direction to the concentric layers of the tube (like spokes in a wheel). The glial cells secrete a substance to which the migrating cells adhere (Rakic, 1993), and the migrating cells themselves adhere to one another using so-called adhesion molecules (Edelman, 1992).

So far, we have described two processes that could produce a brain that has the right number of cells (proliferation) that are located in the right places (migration). Next, we consider how the brain manages to produce the various types of cells that are stereotypically distributed across the six layers of the cerebral cortex (as described above in the description of the laminar organization of the cortex). There are two ways that a developing brain could make sure that the right kind of cells (e.g., stellate cells) end up in the right layers (e.g., layer 4). One way would be to transform progenitor cells into the right type (via genetic transcription processes) immediately after they are produced within the proliferative zones. According to this approach, a cell would "know" what kind of neuron it will become even before it migrates. The second way would be to withhold transforming the progenitor cell until after it migrates to a particular layer. In the latter approach, chemicals secreted by neighboring cells "inform" the migrated cell what kind of cell it should become (Chenn et al., 1997). Experimental studies conducted with animals suggest that both of these processes seem to be involved in creating various types of neurons. In the absence of chemical signals from neighbors, postmitotic progenitors differentiate into one (and only one) type of cell. When these same cells are transplanted to atypical layers, however, they differentiate into cells typical for that layer. Note that this dual approach to differentiation must have evolved as a safeguard for the possibility of migration going awry.

The correlation between the birthdate of a cell and its final laminar position suggests that a neuron's phenotype might be determined early (Chenn et al., 1997). As noted above, there are two main classes of neurons in the cortex: pyramidal and nonpyramidal. Whereas the majority of pyramidal cells use excitatory amino acids as neurotransmitters, the majority of nonpyramidal neurons use inhibitory neurotransmitters (e.g., GABA).

Moreover, whereas nonpyramidal cells are distributed uniformly across the six layers of the cortex, excitatory pyramidal neurons are found only in particular layers. Evidence suggests that local signals from neighboring cells have differing effects on neuronal specification, depending on when a cell was produced. For example, studies of regions that contain a large number of inhibitory neurons show that cells transplanted to that region during a certain phase of their development (the "*S* phase") fail to become inhibitory. Cells transplanted after this phase, however, express inhibitory neurotransmitters (Chenn et al., 1997).

With differentiation processes added to the mix, we now have a brain that has the right number and right type of cells located in the right layers. But to create a fully mature brain, several additional things have to happen. First, each brain cell has to grow in size and send projections to other cells. Second, these cells have to form synaptic connections with some of the cells to which they project. Third, an optimal numerical correspondence has to emerge between presynaptic and postsynaptic neurons. (Note: When two neurons are connected in a one-way chain by way of a synapse, the one earlier in the chain is called "presynaptic"; the next one to which it sends neurotransmitters is called "postsynaptic"). Finally, a myelin sheath has to form along the axons of many of the longer neurons. Let's examine each of these four processes a little further.

A fascinating aspect of neuronal growth processes is that neurons seem to seek out highly specific targets during their development. Even when the targets of these projections are experimentally transplanted to atypical locations in the brains of animals, the axons of the seeking neurons still find their targets (Chenn et al., 1997; Purves & Lichtman, 1985). Other evidence of neuronal specificity is the fact that there is a highly stereotyped pattern of lamina-specific axonal projections across individuals. For example, whereas layer 5 neurons make long-distance projections to targets such as the spinal cord and superior colliculus (located in the midbrain) in most people, those in layer 6 make long-distance projections to the thalamus (Chenn et al., 1997). Unlike the synaptic connections that form in response to experience (see later in this chapter), the stereotyped, laminar patterning of connections among neurons seems to be largely determined by genes (Chenn et al., 1997; Goodman & Tessier-Lavigne, 1997).

Although the evidence is still coming in, it would appear that a combination of factors explain how it is that neurons can find their genetically determined targets. The first thing to note is that axons solve the daunting task of finding long-distance targets by proceeding in small steps (Goodman & Tessier-Lavigne, 1997)—that is, they project a small distance and leave behind a new portion of an axon. At each point, they make use of both local cues (e.g., *chemoaffinity*, or attraction to certain "guidepost" cells along the way, as well as repulsion toward other cells) and long-distance cues (e.g., a steady increase in the concentration of nerve growth

factor [NGF] as the axon gets closer to the target). Then, gradations of molecular guideposts along the surface of targets help individual axons recognize their targets.

Once the projected axons of neurons are in close proximity to other neurons, a correlation of activity patterns in these neurons promotes the formation of synapses. In other words, if two neurons are always active at the same times, they are likely to form a synapse with each other. If they are active at different times, however, they are unlikely to form synapses with each other. In the developing fetus, these activity patterns seem to be intrinsic and spontaneous (i.e., they are not caused by afferent stimulation from the environment that travels from sensory organs along pathways and registers ultimately in the brain—they fire in an unprovoked manner). Such processes are the basis of the adage of neuroscientists: "Neurons that fire together wire together."

The first sign that a synapse is forming is that the membranes of the presynaptic and postsynaptic cells thicken at the site of the synapse in response to recurrent activity. The second sign is that the tip of the axon changes in appearance from looking like a *growth cone* (i.e., a starburst) to looking like a *synaptic bouton* (i.e., an oval with a flat bottom—imagine a cloth bag of marbles tied at the top). Then, three other changes take place: (1) spherical vesicles containing neurotransmitters appear near the edge of the bouton (imagine a row of dots along the bottom of the bag of marbles), (2) the synaptic cleft between the bouton and the surface of the postsynaptic neuron's dendrite widens somewhat (i.e., forms a narrow oval shape like a parenthesis), and (3) glial cells encase the bouton. The net result of all of these anatomical changes is that the information exchange between presynaptic and postsynaptic neurons can occur in a fast and efficient manner. From the standpoint of neuroscience, however, we need to know what a mature synapse looks like before we can say anything about changes in synapses that occur with age or experience. When a scientist counts the number of fully formed synapses in a region of a child's brain and an adult's brain, for example, he or she needs to be able to recognize a fully formed synapse.

In an adult brain, each of the approximately 10^{11} neurons makes a thousand or more synaptic contacts with other neurons (Goodman & Tessier-Lavigne, 1997). In addition, these neurons tend to form contacts in a stereotypical manner. For example, some neurons form synapses only on dendritic spines, while others form synapses only on dendritic shafts. (Note: The spines are like circular leaves on a tree, and the shafts are like branches.) Early in development, however, this stereotyped patterning is not yet apparent, and neurons make many more synaptic contacts than needed to create functional circuits for processing information. So, slides of brain tissue would reveal a lot of synapses all over the neurons in young children but a stereotyped reduced pattern in slides of adult brain tissue.

One way that the overabundance of synapses is reduced with age (in many species) is through the death of neurons. A second way is through the process of *axonal retraction*. In the latter, a presynaptic neuron literally retracts its axon away from the postsynaptic neuron by shrinking in size. (Imagine a tree pulling back one of its branches away from a neighboring tree by making the branch smaller.)

What causes neurons to die, and why do surviving neurons retract their axons? Many scientists believe that both of these processes reflect the fact that neurons have to compete for substances called *trophic factors* that are secreted by activated postsynaptic neurons (Purves & Lichtman, 1985; Reichardt & Farinas, 1997). To explain cell death, scientists note that only some of the axonal projections that make contact with target neurons will succeed in obtaining enough trophic factors to survive. Those that survive and become activated at the same time as postsynaptic neurons tend to form stable synapses with these postsynaptic neurons. To explain axonal retraction, scientists note that sometimes neurons fail to get enough trophic factor from one site but succeed in getting enough from other sites. Instead of dying, these neurons simply retract their axons away from the unsupportive areas.

The net result of the competition for trophic factors is an optimum balance between a population of innervating neurons and a population of innervated neurons (i.e., a functional circuit that has the right number of each class of cells). Presumably, the initial oversupply of neurons and synapses emerged phylogenetically as an evolutionary adaptation to guard against possible problems that might arise as brains are being constructed. To see the utility of this oversupply, consider the following thought experiment. Imagine a species that had a brain that contained 300 functional circuits (one circuit for each of 300 cognitive operations). Next, assume that each circuit must have 1,000 neurons configured in a particular way to work properly (total = 300,000 neurons). Finally, assume the proliferative zones in this species produce exactly 300,000 cells during development. A little reflection shows that a properly functioning brain would be constructed in this species only if all of the following happened during development: (1) all cells managed to migrate to the right locations, (2) chemical signals from neighboring cells were detected by the DNA transcription processes of all migrated cells, and (3) the axons of all cells found their targets. In effect, a properly functioning brain would emerge only if everything went right. But things often go wrong in nature, so the biological strategy of producing exactly the number of cells needed is obviously not the best way to go.

Shortly after the regressive processes of cell death and axonal retraction were discovered, scientists wondered whether all species relied on these two processes to the same extent. Evidence suggests that lower-order species (e.g., rats) seem to rely more on cell death than higher-order species

(e.g., humans). The opposite seems to be true for axonal retraction (Huttenlocher, 1993). However, this conclusion is based on a few studies using postmortem techniques. Hence, more evidence needs to accumulate before strong statements can be made regarding interspecies differences in regressive processes.

The last two processes of brain development that should be discussed are *dendritic arborization* and *axonal myelination*. In addition to growing in size (length and width), neurons also sprout new dendrites (arborization) and acquire a myelin sheath along some of their axons. The addition of new dendrites is thought to be the primary neural basis of cognitive development (Quartz & Sejnowski, 1997). To get a vivid image of arborization, or neurons bathed in solutions that foster sprouting, imagine "Chia Pets" (those small terra-cotta animals that one covers with a seed paste and then waters)! Myelination, in contrast, is the process of adding a fatty-acid coating (myelin) to the axon of some neurons to speed up their firing. Myelin adds considerable mass to the brain beyond that produced by other types of growth. When the brain is finished maturing in late adolescence, it weighs four times as much as it did at birth (Johnson, 1997). Of course, a brain is never really finished changing in an absolute sense because there is a constant shifting of synaptic contacts with experience (see later in this chapter). In addition, the brain often shrinks in size for inactive, undernourished individuals who live beyond the age of 80. Note that whereas cell bodies of neurons comprise what we call "gray matter," the myelin along axons comprise what we call "white matter." A fairly new neuroscientific method called *diffusion tensor imaging* (DTI) allows scientists to see whether different regions of the brain are connected by long-distance fibers and whether these fibers have sufficient levels of myelin (Byrnes & Eaton, in press). Readers interested in the fascinating images that are produced by DTI should Google this term and select "images."

In a way, the foregoing discussion of brain development could be transformed into a checklist for determining the state of development in a child's brain. As noted earlier, autopsies have been used to determine the kinds of cells that normally appear in specific layers and the number of synapses that form between cells in various regions of the brain. This "final state" can serve as the reference point for developmental comparisons. For example, one could ask, "How many cells are present in layer 4 at ages 1, 4, 7, 10, and 13?" and "How many synapses per neuron are there, on average, in an adult brain and a child's brain?" Moreover, once we know how things should "look" in an average adult brain that developed in an average environment, we can consider the effects of various substances or experiences on brain development. For example, we can compare the brains of individuals who smoked cigarettes for many years to those of individuals who did not smoke to see whether there are differences in the number of neurons in particular regions, the number of synapses formed

by these neurons, and so on. In the next section, we explore the latter theme more fully.

FACTORS AFFECTING BRAIN DEVELOPMENT

In the previous two sections, the focus was on describing the general characteristics of mature brains (e.g., the laminar organization of the cortex) as well as the processes that produce these general characteristics (e.g., migration). This reveals how all of our brains are similar at a basic level. In the present section, the primary goal is to elucidate the factors that produce *individual differences* in brain structure. Five such factors are described in turn: (1) genetics, (2) environmental stimulation, (3) nutrition, (4) steroids, and (5) teratogens.

Genetics

Long before scientists discovered genes, they knew that some intrinsic (i.e., nonenvironmental) factor was responsible for producing the large-scale physical differences that can be observed among species (Edelman, 1992). Today, we know that this intuition was clearly correct. The human brain looks very different from other mammalian brains (including our closest ape cousins) chiefly because of a difference in genetic instructions. But what about the more subtle differences in brain structure that arise between individuals of the same species (Goldman-Rakic, 1994; Talairach & Tournoux, 1988)? Everyone's brain looks a little different. Relatedly, the precise location of particular areas of the brain (e.g., Broca's area) differs slightly among individuals. Are these within-species differences also caused by genetic differences? In order to answer this question, we need to expand upon our earlier descriptions of proliferation, migration, and differentiation. Then, we can consider the implications of this expanded analysis for two related lines of genetic research.

The most important variable that explains between-species differences in brain size is the length of the proliferation phase. As Finlay and Darlington (1995) note, an additional 17 doublings of precursor cells can yield 131,000 times the final number of neurons (roughly the difference in the number of neurons found between the brains of humans and shrews). How does a developing brain know when to stop proliferating cells? One possibility is that the proper number of mitotic divisions is encoded somewhere in the DNA of precursor cells. Another possibility is that precursor cells continue to double until they receive signals that enough cells have migrated to various locations in the brain (Chenn et al., 1997). Regardless of which of these possibilities turns out to be the case, it is clear that proliferation is largely under the control of genetic instructions.

The same could be said for migration and differentiation. Earlier in this chapter, we saw how proliferated cells seem to be genetically destined to become particular types of cells and migrate to particular levels as soon as they leave the proliferative zones. However, we also saw that this predestination is not set in stone. Once again, signals from neighboring cells seem to play a role in determining the ultimate fate of particular cells. Thus, one could summarize the role of genetics so far by saying that genes largely constrain how things turn out, but there is a certain degree of protective flexibility built into the system.

One other point worth noting pertains to the probabilistic nature of migration. As cells migrate, they overlap, pass by, make contact with, and adhere to one another in a complex way (Edelman, 1992; Rakic, 1993). As a result, there is no way to know for sure where a given cell will end up when it migrates (Chenn et al., 1997). The stochastic, "bump-and-grind" quality of the migration process means that genetic instructions are not really analogous to blueprints. Whereas two houses built from the same blueprint would turn out to be identical (in terms of their size, location of rooms, etc.), two brains built from the same set of genetic instructions could be rather different.

The latter point provides a nice segue to a second line of work that provides further insight into the role of genetics in brain development. Researchers who have conducted neuroscientific studies of twins have revealed two important findings. First, people who have exactly the same genetic instructions (i.e., monozygotic [MZ] twins) sometimes develop brains that are structurally different (Edelman, 1992; Segal, 1989; Steinmetz, Herzog, Schlaug, Huang, & Lanke, 1995). In fact, MZ twins have been found to develop brains that are mirror images of each other (e.g., one has a dominant left hemisphere, while the other has a dominant right hemisphere).

However, researchers have also found that there can be a relatively high concordance rate between MZ twins for disorders that are alleged to have a neurological basis. By "concordance rate," it is meant that both of the twins have the same phenotype (i.e., both have the disorder, or both lack the disorder). In a study of the genetic basis of reading disability, for example, DeFries, Gillis, and Wadsworth (1993) found that 53.5% of MZ twins were concordant for reading problems, compared to just 31.5% of dizygotic (DZ) twins. Note, however, that both of these concordance rates are considerably lower than 100%, which suggests that there is not a deterministic, one-to-one correspondence between genes and brain structure. Also, when DeFries et al. estimated the heritability of reading disability from their data, they found that genetics accounted for 44% of the variance in reading profiles (which means that 56% is explained by nongenetic factors). Later studies produced somewhat higher concordance rates and heritability estimates (e.g., Knopik, Alarcon, & DeFries, 1997), but the

overall findings are essentially the same. Since MZ twins are highly similar in terms of their height and weight (r = .97), it would appear that reading disability is probably not a product of proliferation problems. Rather, it would seem that reading problems arise from other aspects of brain development (e.g., problems of migration, long-distance connections, formation of synapses, pruning of axons, cell death).

A final way to assess the relative importance of genes for brain development is to examine disorders such as *Down syndrome* (DS) in which there is a known linkage between specific genes and neurological pathology. DS, or trisomy 21, is the leading cause of intellectual disability in the United States. It results from the nondisjunction of a portion of the 21st chromosome during meiotic division of gametes (usually the ovum), so that three copies of that portion are present in the cells of the affected individual instead of the usual two (Coyle, Oster-Granite, Reeves, & Gearhart, 1988; Hassold, Sherman, & Hunt, 1995). The presence of this extra genetic material produces a number of anatomical and health-related differences between individuals with DS and unimpaired individuals (Coyle et al., 1988; Kemper, 1988). Of particular interest here are the differences related to brain structure. Individuals with DS tend to have brains that are smaller and less developed than those of typically developing children and adults. In addition, individuals with DS tend to have 33% fewer cortical neurons, less complex patterns of connectivity, reduced levels of myelin, and apical dendrites that are abnormal in appearance (e.g., fewer spines and elongated necks). Taken together, these findings suggest that the extra genetic material probably interferes with the processes of proliferation, synaptogenesis, and myelination.

Although much more can be said about the role of genes in brain development, the preceding discussion of developmental processes (e.g., proliferation), twin research, and genetic disorders is sufficient for drawing several broad conclusions. First, it seems clear that certain changes in genetic instructions can lead to large-scale changes in brain volume and patterns of connectivity. For example, a 2% change in the amount of genetic material (e.g., shifting from 46 to 47 chromosomes) can produce a 33% difference in brain structure (e.g., the number of cortical neurons in the brains of individuals with DS vs. typically developing individuals). But even when people have the same genes, there is still a chance that they could have brains that differ somewhat in terms of their size, shape, and areal organization. The lack of one-to-one correspondence between genes and brain structure means that researchers should not assume that two individuals definitely have different genes simply because their brains are different in size or in organizational structure. Perhaps more to the point, researchers should not argue with certainty that two people have different brains because they have different genes (consider the case of identical twins), nor argue with certainty that two people must have the same genes because their brains

seem to be anatomically identical (two unrelated people who share few genes could have identical brains).

Environmental Stimulation

In essence, then, we see that genes only partly explain why we have the brains that we do. The second factor that is instrumental in sculpting the brain is environmental stimulation. In order for animals to respond adaptively to their environment, they have to be able to form mental representations that match their experiences (Greenough, Black, & Wallace, 1987; Johnson, 1997). For example, they need to be able to recognize conspecifics (e.g., their mothers and their siblings) as well as store new, adaptation-relevant experiences in memory (e.g., the fact that fire can burn). Many animals come into the world ready to learn such things, using a prewired circuitry that is closely aligned with their external sense organs.

The prewired circuitry consists of (1) cortical neurons that receive, process, and store input signals from the environment and (2) afferent neurons that bring these input signals to the brain from the various sensory organs. Postmortem studies reveal that the afferent neurons from particular sensory organs terminate in the same general regions for all people (e.g., area V1 in the occipital lobes in the case of afferents coming from the eyes). By necessity, then, it would be expected that all people would process and store input signals from particular organs in roughly the same regions of the cortex (Johnson, 1997). But it is important to note that this areal organization is a joint function of the preset location of afferent projections and environmental stimulation. Take away the afferents, and we would not develop particular types of representations (e.g., visual representations of people we know) in particular regions of the cortex (e.g., area V1). Similarly, we would obviously not develop representations of events in the world without experiencing these events. Each of these counterfactual claims has been tested empirically.

For example, surgical studies with animals have shown that atypical cortical maps can be created by redirecting the input fibers that extend from the thalamus to new areas. Normally, a neural tract that carries visual information from the eyes projects upward and backward from the thalamus (located in the middle of the brain) to area V1 of the occipital lobe in the cortex (in the back of the head). Scientists have redirected this tract such that it projects to, for instance, the frontal lobe in the front of the head. They have found that cells in the frontal lobe then process visual information the same way cells in the occipital lobe normally do! In addition, studies have shown that animals will create new projections and new representations in a cortical area even after some of the thalamic projections that normally project to that area have been severed (e.g., Recanzone, Schreiner, & Merzenich, 1993). A similar type of plasticity has been observed in

human infants who have suffered brain injury or had portions of their brains removed to relieve epilepsy (Johnson, 1997). Normally, the neural regions responsible for language skills are in the left hemisphere for most right-handed people. Infants who have had their left hemispheres removed to relieve constant seizures develop language areas in their remaining right hemispheres. Thus, whereas the laminar organization of the cortex seems to be largely intrinsically determined (i.e., preprogrammed by genes and unrelated to afferent stimulation that comes into the cortex), the areal organization seems to be jointly determined by preexisting projections and environmental input (Chenn et al., 1997; Johnson, 1997). To understand the latter point by analogy, imagine that the neural assemblies in the cortex that process sounds or letters are like employees of a credit card company who operate in their own cubicles. Imagine further that outside phone calls from their customers are analogous to afferent stimulation from the environment. The company could not do its business if someone (e.g., the boss) had not placed the workers there and hooked them up to phone lines and one another via a computer network. But their computer databases would all be empty if their customers never called in. Thus, a functioning company requires both preset architectures ready to receive input and the input itself.

However, studies show that environmental stimulation can have different effects on brain structure depending on when it occurs in development (Greenough et al., 1987). For example, animals can be permanently blinded if they are reared in total darkness for 2 weeks right after birth. However, if the deprivation occurs somewhat later in the postnatal period, their visual skills develop normally. To explain such time-dependent results, Greenough et al. proposed that mammalian brain development involves two types of neural plasticity: experience expectant and experience dependent.

Experience-expectant plasticity exploits regularities in the environment to shape developing neural systems. Appropriate circuits develop if the animal experiences these regularities (e.g., contrast borders, movement). The mechanism of change in experience-expectant plasticity appears to be an early overproduction of synapses followed by a pruning of exuberant projections in response to experience. As noted earlier, the overproduction of synapses is said to take place in order to compensate for possible problems that arise during proliferation and migration (to make sure that there are enough neurons to form a functional circuitry). The pruning takes place because the neurons must compete for a limited supply of trophic factors (as described earlier).

The second type of plasticity, *experience dependent,* is thought to have evolved to allow the animal to form representations of unique features of its environment (e.g., characteristics of its own mother, sources of food and haven, native language properties). The mechanism of change here is

not the elimination of excessive synapses as much as the creation of new synapses (Greenough et al., 1987; Quartz & Sejnowski, 1997), or, more accurately, new learning is probably best conceived of as the reorganization of synapses (elimination of some combined with the addition of others).

In sum, then, we can attribute the fact that most of us can see (or hear) correctly to the fact that we had appropriate visual (or auditory) experiences when we were young. In contrast, we can attribute the fact that we represent a particular sensory experience in a particular region of the cortex to the fact that (1) afferents project to that region and (2) we had the experience. It is in this way that experience can sculpt our brains and create a dynamic type of circuitry. What is clear in any case with the human brain is that the "wiring" of the brain is not finished at birth—rather, we need environmental stimulation to finish the job.

Nutrition

Numerous experimental studies with animals have shown that malnutrition can have different effects on brain development, depending on when it occurs (Winick, 1984). Scientists explain such time-dependent outcomes by arguing that early (i.e., prenatal) malnutrition slows the rate at which cells are proliferated, thereby reducing the total number of neurons and glial cells in an animal's brain. Later malnutrition, in contrast, slows the rate at which the already proliferated cells grow in size or acquire a myelin sheath. Whereas the latter problems can be ameliorated by providing enriched diets to malnourished animals, the former problem of too few cells cannot be corrected in this way. Such findings suggest, then, that prenatal malnutrition would cause more permanent harm to developing human brains than postnatal malnutrition (because proliferation largely occurs during the prenatal months in humans).

In support of this claim are various correlational studies of malnourished and normally fed children around the world (e.g., Streissguth, Barr, Sampson, Darby, & Martin, 1989; Winick, 1984), as well as a quasi-experimental study conducted by Pollitt, Gorman, Engle, Martorell, and Rivera (1993). These researchers gave either a high-protein, high-calorie supplement ("Atole") or a low-protein, lower-calorie supplement ("Fresco") to poor Guatemalan pregnant women and their children. Some children received the supplement postnatally, while others received it both prenatally and postnatally. Children were followed longitudinally and given various assessments when they were preschoolers and adolescents. At the preschool assessment, Pollitt et al. found that children given the Atole supplement performed significantly better than children given the Fresco supplement, even after controlling for gender, age, and socioeconomic status. However, the findings were largely limited to motor skills. At the adolescent assessment, the Atole supplement was associated with higher cognitive skills, but

it explained only 1 to 5% of the variance in these abilities. Factors such as gender, socioeconomic status, and schooling explained much more of the variance. In line with the findings with animals, however, Pollitt et al. found that children who started supplemental feeding after 24 months showed less benefit than children who received the supplement before and after birth. A more recent review (Georgieff, Brunette, & Tran, 2015) also suggests that randomized experiments with supplements often do not have a significant effect on cognition, but the authors do point out that different brain regions have different sensitive periods and also have different nutritional needs (e.g., some iron, some amino acids). Failure to take regional differences into account could explain the noneffects.

Taken together, such studies suggest that nutrition has the potential to affect two important aspects of brain development: proliferation and myelination. Studies have also shown that brain development can be enhanced in most children by making sure that they have adequate levels of protein and fatty acids in their diets (Winick, 1984). For children who have the condition known as phenylketonuria (PKU), however, a high-protein diet could prove disastrous if it is left unchecked. Children with PKU are unable to convert the amino acid phenylalanine into the amino acid tyrosine (Diamond, Prevor, Callender, & Druin, 1997). As a result, they experience two main problems. First, high levels of phenylalanine in the bloodstream cause progressive brain damage and intellectual disability. Second, tyrosine is a precursor to dopamine. Circuits comprising dopaminergic neurons cannot work properly when the level of dopamine is too low.

Whereas the first problem can be alleviated by having children with PKU avoid foods that contain high levels of phenylalanine (a strategy that has been in place for many years), researchers have not yet figured out how to solve the second problem (Diamond et al., 1997). As a result, many children with PKU still experience subtle cognitive deficits.

Steroids

Scientists believe that steroids affect brain development for four reasons. First, the brain is one of several organs in the body that contain receptors for estrogens and related substances (e.g., cortisol and other stress hormones). As such, there is reason to think that it would be transformed during prenatal development in the same way that other so-called steroid target tissues (e.g., genitalia) are transformed (Kelley, 1993). Also, excessive amounts of stress hormones could promote the death of neurons in certain key areas of the brain. The second reason relates to the consistent patterns of gender differences that have been found in areas such as cognitive skills, psychological disorders, and violent behavior. Many scientists believe that the consistency of these differences argues in favor of inborn structural differences in the brains of men and women (Halpern, 1992).

The third reason derives from various experimental studies of animals. In one line of research, scientists uncovered gender differences in brain structure that are visible to the naked eye (Breedlove, 1994). Other studies have shown that sex hormones can alter the brains and behaviors of animals. For example, female rats exposed to androgens have been found to engage in sexual behaviors that are characteristic of males (e.g., mounting).

The fourth reason derives from several recent studies that have compared the brains of three groups: homosexual men (group 1), heterosexual men (group 2), and heterosexual women (group 3). The logic of this comparison is as follows: If sexual attraction to men is brain based, then the brains of people in group 1 should look more like the brains of people in group 3 than the brains of people in group 2 (Breedlove, 1994). Researchers recently demonstrated this expected pattern for a region of the hypothalamus that has been implicated in sexual functioning. The region was significantly larger in group 2 than in either group 1 or 3 (which did not differ).

While the results of all of these studies are certainly intriguing, it cannot be said that they convincingly demonstrate that the human brain is sexually dimorphic. The first problem relates to a high level of inconsistency in the evidence reported by researchers who have used either magnetic resonance imaging (MRI) or postmortem methodologies within human brains. Some have found structural differences that are consistent with the behavioral evidence (e.g., larger spatial areas in men), others report differences that are opposite to what would be predicted (e.g., larger spatial areas in women), and a third group has found no structural differences at all (Beaton, 1997; Breedlove, 1994; Driesen & Raz, 1995; Giedd et al., 1996). The second problem is that human behavior is far more flexible and context sensitive than animal behavior (Breedlove, 1994; Byrnes & Fox, 1998). As such, there is little reason to think that humans and animals would respond in the same way to some experimental intervention (e.g., an injection of androgens). Third, the brain regions targeted by experimental interventions have not been consistently related to sexual behaviors in either animals or humans. Finally, it is not at all clear why the size of a particular brain region would necessarily relate to behaviors in a meaningful way (Beaton, 1997; Breedlove, 1994).

Thus, even though there is reason to think that steroids could alter the brains of men and women, scientists have found little hard evidence of this transformation. This lack of evidence means that either sexual dimorphisms do not exist or that scientists have not been looking in the right places (using the right metrics). With respect to the latter possibility, note that few researchers have investigated whether steroids alter (1) the distribution of particular types of cells in given regions or (2) patterns of connectivity. Such differences could not be detected with MRI technology, but they could be detected in postmortem studies.

Teratogens

Any foreign substance that causes abnormalities in a developing embryo or fetus is called a *teratogen*. Researchers identify teratogens through retrospective analyses, prospective longitudinal studies, and prospective experimental investigations. Using the retrospective technique, mothers of children who are born with birth defects are interviewed to determine whether they may have ingested something (e.g., alcohol) or been exposed to something (e.g., a virus) that could have altered the course of their child's development in utero. Using the prospective longitudinal technique, pregnant women are interviewed regarding their behaviors during their pregnancy and followed for years after they give birth. Of interest is the association between their exposure to teratogens and developmental outcomes in their children. Using the prospective experimental technique, pregnant animals are exposed to various dosages of suspected teratogens. Their offspring are then analyzed for the presence of physical or behavioral anomalies.

Scientists consider a substance to be a teratogen only if both of the following are true: (1) a sufficient level of evidence has accumulated from retrospective or prospective studies to show a consistent linkage between the substances and birth defects and (2) the dosages utilized in prospective experimental studies are not unrealistically high. Many common substances (e.g., caffeine and nicotine) have been found to produce birth defects when given at extremely high and unrealistic dosages but not when given at more realistic levels. Such substances are generally not considered teratogens by scientists, but they may nevertheless appear on lists of to-be-avoided substances issued by the federal government or on the warning labels of packages. The government takes a more cautious approach since it often takes time to determine whether a substance really is dangerous. Hence, government officials think it is better to be safe than sorry.

Generally speaking, two types of teratogens have been the subject of numerous investigations: viruses and drugs. Viruses reproduce themselves by invading a host cell (e.g., a neuron), releasing their nucleic acids into the surrounding tissue, and co-opting the host cell's metabolic machinery. In mature organisms, this invasion usually results in transient symptoms, such as lethargy and fever. In developing organisms, however, a viral infection can have more permanent effects if it occurs when the organism's cells are in the midst of proliferating, migrating, or differentiating. Viruses such as rubella have been linked to a range of birth defects, including *microencephaly* (i.e., a small head and brain). It is not yet clear whether other viruses are also linked to abnormal brain development in the same way, but the key seems to be whether the virus targets particular kinds of tissues. Cold viruses and flu viruses target tissues in the nose and lungs, respectively, and do not target developing embryonic tissue (such as developing neurons).

As for drugs, numerous prospective and retrospective studies have been conducted to determine whether substances such as lead, alcohol, marijuana (cannabis), cocaine, caffeine, nicotine, aspirin, acetaminophen, and antihistamine are teratogens. Maternal exposure to high levels of lead has been found to be associated with higher rates of fetal loss (i.e., spontaneous abortion), but despite popular reports in the media, lower levels do not appear to produce large-scale cognitive deficits or physical abnormalities in children (Bellinger & Needleman, 1994). Maternal consumption of alcohol, however, has been consistently linked to a range of cognitive and motor deficits (Barr, Streissguth, Darby, & Sampson, 1990; Streissguth et al., 1989; Streissguth, Sampson, Barr, Bookstein, & Olson, 1994). In heavy drinkers and alcoholics, prenatal exposure to alcohol occasionally leads to a disorder called *fetal alcohol syndrome*, which has an incidence rate of about 3 per 1,000 births. In addition, later in adolescence when brains are still maturing, teens who drink and smoke marijuana have detectable differences in their brains, such as thinner cortices and frontal lobe abnormalities (Silveri, Dager, Cohen-Gilbert, & Sneider, 2016). As for marijuana, cocaine, caffeine, nicotine, aspirin, acetaminophen, and antihistamine, the collective evidence from prospective and retrospective studies with humans suggests that these substances do not appear to be consistently related to long-term cognitive or motoric deficits (Barr & Streissguth, 1991; Hinds, West, Knight, & Harland, 1996; Streissguth et al., 1994). Experimental studies with animals, however, have found teratogenic effects for all of these substances. In each case, there is evidence that the substance has the potential to interfere with the processes of proliferation, migration, and differentiation. In the final section of this chapter, we explore possible reasons why these drugs seem to affect the offspring of animals more than the offspring of humans.

Summary

The preceding discussion focused on five factors that have been found to produce individual differences in brain structure: genes, environmental stimulation, nutrition, steroids, and teratogens. This analysis revealed that two people could develop different brains because they (1) had different genes, (2) had differing levels or types of environmental stimulation, (3) ingested differing levels or types of food, (4) were exposed to differing levels or types of steroids, or (5) were exposed to differing levels or types of teratogens. The evidence as a whole, however, suggests that most of the structural differences that might arise among people would tend to be rather small and subtle. Large-scale differences in brains might arise only when several of these factors work in concert, or when extreme values of the individual factors are involved (e.g., shifting from 46 to 47

chromosomes, reducing diets by 60%, rearing animals in the dark, drinking large quantities of alcohol daily).

CONCLUSIONS AND CAVEATS

In a certain sense, this chapter represents a "how-to" manual for building a human brain. The standard model of this brain is clearly the default option that is likely to emerge in all but the most adverse environments. The underlying principles behind this high degree of adaptive success appear to be two notions:

1. *Overproduction:* Build more brain cells and synaptic connections than most people will need; if proliferation, migration, differentiation, and synaptogenesis are somehow slowed or altered, there may still be enough cells around to create functional circuits.
2. *Flexibility and plasticity:* Augment genetic instructions with cellular feedback loops, make use of both experience-expectant and experience-dependent learning processes, and make use of alternative brain regions if the typical brain region lacks functional circuits (the latter applies mostly to young children).

These two principles, combined with physical aspects of the intrauterine environment (e.g., crowding, passive migration), also mean that individual differences in brain structure will be the norm rather than the exception (even in identical twins). However, in most cases, the differences that emerge in brain structure will tend to be rather subtle. Whether any of these smaller differences are responsible for either individual differences in behavior or phenotypic similarity in twins is currently a matter of controversy.

Moreover, it is important to point out that much of what we know (or rather, believe) about brain development is still tentative and fairly controversial. To a large extent, the lack of certainty is due to the fact that researchers have had to resort to experimental studies with animals to determine the possible role of certain factors in development (e.g., environmental stimulation, hormones). Interspecies differences clearly cloud the conclusions that can be drawn from such studies. Moreover, studies with humans are, by necessity, correlational rather than experimental. Any linkages between background variables (e.g., nutrition) and outcomes (e.g., brain size) could be spurious.

What, then, are the implications of the research on brain development for psychological theory, educational practice, and public policy? We have seen that the adult form of a person's brain is jointly a function of (1) genetic instructions that specify the length of the proliferative phase

and the kinds and locations of neurons that are produced; (2) mechanical processes involved in the movement of cells and progressive lengthening of axons; (3) chemical signals between neurons (neurotransmitters that are sent across synapses as well as other signals that help guide axonal projections and inform the differentiation process); (4) environmental stimulation that causes clusters of neurons to fire together, form synapses with one another, and create functional areas of the cortex (e.g., for vision or math); and (5) other factors that interfere with the normal processes of sculpting (e.g., teratogens and diseases). We have also seen that there is not a one-to-one relationship between genes and the final cytoarchitecture of someone's brain and that the human brain is highly plastic in the sense that it can reorganize itself and overcome obstacles imposed by the environment. However, the ability of the brain to overcome problems varies over time. For example, whereas the effects of prenatal malnutrition on the brain seem to be relatively permanent, the effects of postnatal malnutrition seem to be reversible. Similarly, whereas young children can often overcome brain injuries, adults often lose functions permanently.

When confronted with these tentative conclusions, a variety of reactions seem possible. Some have used the findings in this chapter and related findings to argue in favor of the *constructivist orientation* to cognitive development (e.g., Quartz & Sejnowski, 1997). The constructivist orientation lies midway between a *nativist orientation* (espouses the idea that mental representations of such things as faces, math skills, and grammatical categories exist at birth prior to environmental input) and an *empiricist orientation* (espouses the idea that the mind is a blank slate that is entirely shaped by environmental input). Among developmental psychologists, there is an ongoing, vigorous debate between the nativist and constructivist camps, so presumably the findings can be used to bolster the position of the constructivists. Relatedly, most mathematics and science educators espouse the constructivist philosophy these days (Byrnes, 2008), so these educators may use the findings to their advantage as well. However, there is a large gap between finding support for the metatheoretical belief system of constructivism and finding support for a particular theory or instructional technique that is consistent with this paradigm (e.g., Piaget's [1983] theory or the instructional approach advocated by the National Council of Teachers of Mathematics [NCTM, 1989]). There are many ways to conceptually and behaviorally implement constructivism (in the same way that there are many ideas, behaviors, and rituals consistent with the religious beliefs of a particular type of religion, such as Christianity).

Relatedly, some have used the time-dependent relation between environmental input and brain sculpting to argue in favor of starting foreign language and music instruction in the preschool period (before a presumed critical period is over), for example, rather than later in development. Others, in contrast, have argued that nothing at all can be concluded from the

time-dependent effects of the environment because they apply only to cases of extreme deprivation (Bruer, 1998). We tend to agree more with the latter than the former reaction, but we add that the findings are nevertheless important for the attitudes we take toward children. There is an unfortunate tendency for scientists, educators, and ultimately parents to take a deterministic, pessimistic view of abilities and disabilities. The findings regarding plasticity and environmental input show that children are not destined to particular outcomes due to their genes—in other words, there is much we can do to improve skills in children. The sooner we start, the sooner the sculpting and plasticity can begin. In the same way, the findings show that gender and ethnic differences in abilities may be relatively easy to eliminate and that parental guilt over "giving" their children a malady with a presumed genetic basis (e.g., reading disability, autism) may be misguided. The stochastic, mechanical quality of brain maturation may be the culprit in many cases (i.e., things just did not go as planned by the genes).

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