2 BIOLOGICAL AND BRAIN DEVELOPMENT

When Tesalia was born full-term, at nearly 40 weeks, she weighed only 5 pounds (lbs.), 15 ½ ounces (oz.), and her head circumference was only 33 cm. She was in the 10th percentile of all infants. Doctors and parents worry when a baby has a small head like Tesalia did because it might mean that there could be problems with brain development. However, over her infancy and early childhood, Tesalia's head continued to grow, alleviating any concern that she had suffered a problem with the development of her brain before birth.

In fact, as she got older, Tesalia stayed small compared to her peers—she was petite throughout infancy and childhood. Why was she relatively small? Recall when we discussed Martha in Chapter 1, we talked about the interaction between *genes* and *environment* in determining Martha's height. Just as Martha had genes to be tall, Tesalia has genes to be on the petite side. Both of her grandmothers are 5'3" or shorter, and even her mother is average height at 5'4." Thus, we think that Tesalia is relatively small because, at least in part, she has the genes for being relatively small.

This chapter is about these aspects of development—the biological foundations of development in infancy and the early development of the nervous system. We will review the topics of genetics and outline the types of cells in the brain, their function, and how they give rise to the hemispheres. These topics are crucial for understanding the biological foundations of development in infancy. And they are also topics that parents and pediatricians care a lot about.

After reading this chapter, you will be able to:

1. *Define* heredity, genotype, phenotype, genes, DNA, chromosomes, alleles, and autosomes, and explain how each of these relates to the others.
2. *Distinguish* among gene–environment correlations, gene–environment interactions, and epigenetics.
3. *Label* the function and parts of a neuron and *explain* the difference between experience-expectant and experience-dependent plasticity using an example of each to highlight the difference.

THE "NATURE" IN THE NATURE–NURTURE QUESTION

Of the six infants we are following, there are three pairs of siblings: Alison and Carter, Diego and Tesalia, and Edwin and Charlie. Each sibling pair shares a family resemblance, and they are more similar in appearance to each other than to the other four children, to whom they are unrelated.

For example, Diego and Tesalia both have curly hair that coils into ringlets, whereas the other four have fairly straight hair. Diego and Tesalia's complexions also hint at their Central American heritage, whereas Alison and Carter's complexions hint at their northern European heritage. At the same time, there are striking differences within the sibling pairs. Diego's midnight hair is so black, it shines, a contrast to Tesalia's chestnut brown hair, which reflects hues of red. Charlie is blonde while his brother Edwin has brown hair. Alison and Carter have greater similarity in their hair color, but as newborns, Carter came into the world with red hair and Alison with dark brown, and by their first birthdays shifted to strawberry blonde for Carter and blonde for Alison. Edwin and Charlie both have brown eyes, but the other sibling pairs differ in eye color; Alison and Diego have brown eyes, but each of their siblings, Carter and Tesalia, has blue-gray eyes.

The sibling pairs also share family resemblances in their *behavior*. Alison and Carter have similar senses of humor, and both can be a bit cautious. Both Edwin and Charlie love to dance. Tesalia and Diego are musical. But the sibling pairs also differ. Alison is more social, and Carter would definitely prefer to be alone. Charlie and Tesalia are highly energetic and adventurous (sometimes to the point of seeming reckless), whereas their siblings Edwin and Diego are calm and cautious. It took Diego and Edwin weeks and weeks of experience in a swimming pool to dip their faces in the water. At 11 months, Tesalia and Charlie dove right in, literally. As infants, Charlie, Diego, and Alison were quick with smiles toward *everyone*, even strangers, whereas their siblings, Edwin, Tesalia, and Carter reserved their most radiant smiles for their caregivers (much to their parents' delight).

What accounts for the physical and behavioral differences (and similarities) of our six infants? Our goal in this section is to provide a basic understanding of **heredity**, or the transmission of genetic information across generations, and how that genetic information translates to differences in physical characteristics and behavior.

How Genes Influence Development

You may already know that **genotype** refers to the unique genetic makeup of an individual (i.e., their individual collection of genes). **Phenotype** refers to the observable characteristics, or traits, of an individual, and includes any characteristic from physical ones (e.g., freckles, dimples, height) to psychological ones (e.g., personality, intelligence). Our phenotype is the expression of our genotype. We inherit our genotype, but our phenotype reflects both our particular combination of genes *and* our environment (Table 2.1). The interplay between genes and environments differs not only across genes but also across environments, adding many layers of

TABLE 2.1 ■ Genotype vs. Phenotype

	Definition	How It Is Determined
Genotype	All of a person's genes	The genes inherited from the biological parents
Phenotype	A person's traits, or observable characteristics	The particular combination of the genes, as influenced by the environment

complexity to the story of how genes and environments contribute to development. Before we tackle this complex interaction, we will first review basic information about genes, their structure, and their expression.

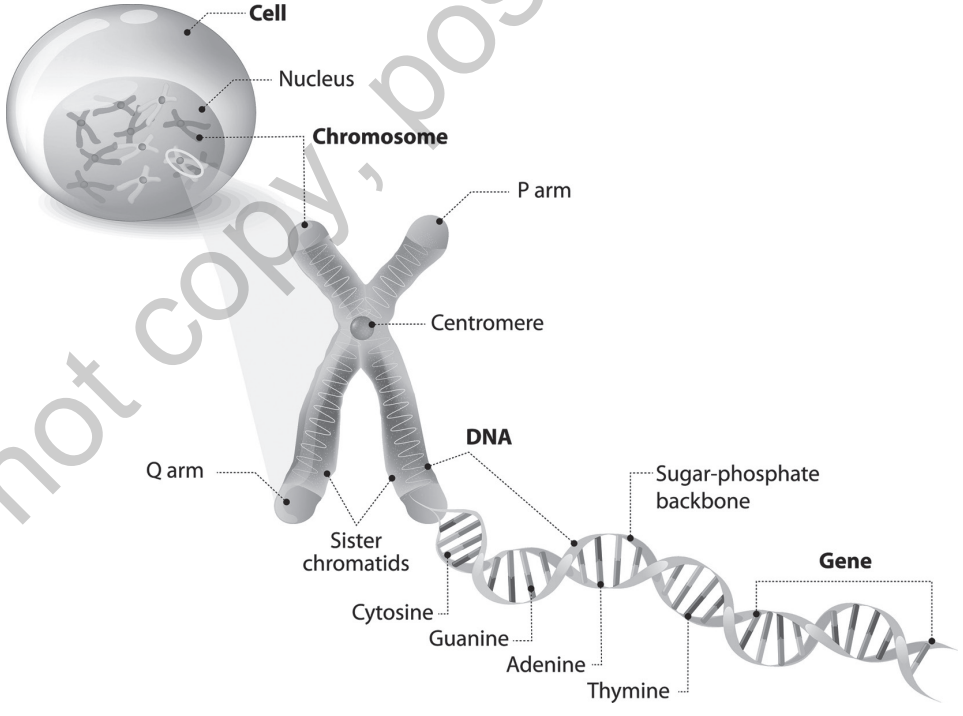
What Are Our Genetics?

Although this is not a biology or a genetics textbook, it is important to review some key points about the biology of genetics to appreciate how genes and environments interact. As a starting point, we need to understand the relation between **DNA**, **chromosomes**, and **genes**. Our bodies contain trillions of cells, which vary in structure and function. Within the nucleus of each cell is an impressively long molecule, known as deoxyribonucleic acid (DNA). DNA is tightly coiled around proteins, and this is what makes up our chromosomes. Our genes are segments of DNA, and each gene has a unique address on a chromosome (Figure 2.1).

The autosomes are 22 pairs of chromosomes. For these pairs, a gene on one chromosome in a pair will have the same gene on the other chromosome in that pair (Figure 2.2). The final pair of chromosomes are the sex chromosomes, the X and Y in humans. The sex chromosomes differ from each other in size (the X chromosome is much larger than the Y) *and* in the number and

FIGURE 2.1 ■ Chromosome

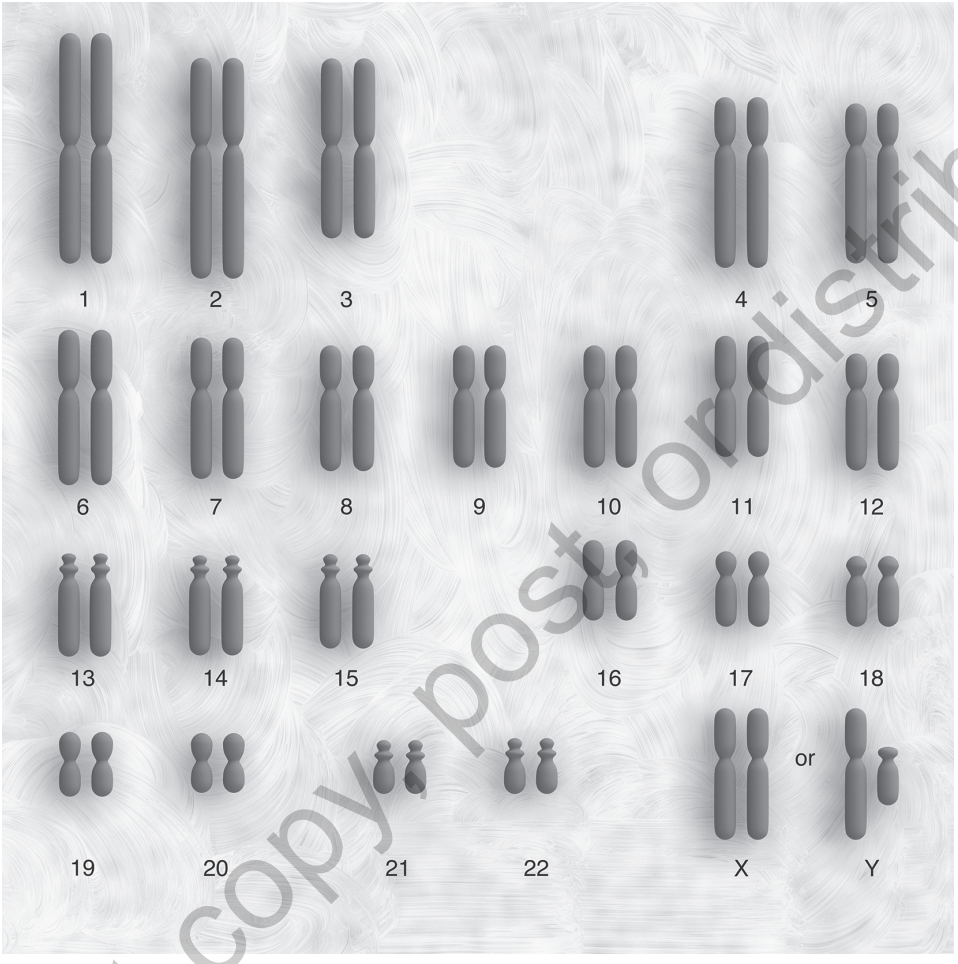
The relationship between a cell, a cell nucleus, chromosomes, DNA, and a gene.



Source: iStock/ttsz

FIGURE 2.2 ■ Karyotype

This is a karyotype, an illustration of chromosomes, arranged in their pairs and by chromosome number.



Source: iStock/somersault18:24

type of genes that each carries. So, unlike the other pairs of genes, each gene in the X chromosome does not necessarily have a pair on the Y chromosome. As we will see in Chapter 3, this fact about the sex chromosomes means that some traits are inherited differently for individuals with two X chromosomes (genetically female) versus individuals with an X and a Y chromosome (genetically male).

You may wonder where these 23 pairs of chromosomes come from. Most cells in our bodies have 23 pairs of chromosomes, summing to a total of 46 chromosomes. However, the sex cells in our bodies, known as the gametes (the egg in females and the sperm in males), have only one chromosome from each of the 23 pairs (one chromosome from pair 1, one from pair 2, and so on). Thus, these cells have half the number of chromosomes that other cells have. Importantly,

what these cells contain is one chromosome of each *pair*. So, instead of having two chromosomes at location 1, two at location 2 and so on, these cells have just one chromosome at each location. When the egg and sperm combine at conception (see Chapter 3), the chromosomes at each location become a pair. The result is a *zygote,* a single cell that divides and develops into a new human, with 23 pairs of chromosomes. Each pair contains one chromosome from the egg (i.e., the female parent) and one chromosome from the sperm (i.e., the male parent). Thus, each of our cells has chromosomes from each parent.

Patterns of Inheritance

This combining of the chromosomes (and genes) of the two parents is what determines the individual's genetic *inheritance*. You may have noticed that some children look more like one parent than the other (although family resemblance can shift as children age). For example, Edwin is nearly a miniature version of his mother (see photo). Other children are a blend of both

Edwin's mother, age 1 and Edwin, age 1.

parents. For example, Carter has blonde hair and blue-gray eyes, like his father, but his eye and face shape are more like his mother. What determines how a parent transmits their traits (e.g., extroversion, love of math, hair or eye color) to a child? Amazingly, most humans share the same DNA, with individuals differing by only about 0.1%! How can such a tiny amount of DNA explain the uniqueness of an individual?

Children's characteristics reflect, in part, the fact that although the two genes in each pair relate to the same characteristic (e.g., eye color, shyness), they may be different *forms* of those genes, or different **alleles**. The types of alleles that make up the pair of genes determines the patterns of inheritance and the traits expressed in an individual's phenotype. When both parents contribute the same allele of a particular gene, then the child is **homozygous** for that gene and will express the trait associated with that allele. For example, one of the authors and her spouse (and you would know which one immediately from our photos) both have curly hair. Each of their children inherited the same allele from each parent for hair shape, and as a result, both children have curly hair. With homozygous genes, it doesn't matter if the allele of the gene inherited from each parent is dominant or recessive (a pattern of inheritance that we describe in the next paragraph); either way, the child will express that allele.

However, when each parent contributes a different allele for a particular gene, the child is **heterozygous** for the gene. In this case, how the alleles interact to express a trait in the phenotype can follow several distinct patterns of inheritance (Table 2.2). Specifically, when a child is heterozygous for a trait, how the trait is expressed depends on the *dominance* of the allele. When a child has two different alleles for a gene, and only one of the two alleles is expressed in their phenotype, the expressed allele is considered **dominant** and the non-expressed allele is considered **recessive**. Note that when an individual has a dominant trait in their phenotype, they could have one allele that is dominant and one that is recessive, or they could have two dominant alleles (i.e., is homozygous for the dominant allele). The point is that two individuals may display the same phenotype even though they have different genotypes. If an individual has a recessive trait in their phenotype, they must have two copies of the recessive allele (one from each parent).

Several traits follow the dominant–recessive pattern of inheritance. Dimples, freckles, cleft chin, widow's peak, dark hair color, and brown eyes reflect alleles of genes that are dominant. To express these traits requires inheriting the allele from just one parent. To express the alternate trait (i.e., no dimples, no freckles, no cleft chin, blonde hair, and blue-gray eyes) requires inheriting the recessive allele from both parents. Interestingly, individuals who show a dominant trait in their phenotype could be (a) homozygous with two copies of the dominant allele, and therefore can only pass on the dominant allele to their offspring, or (b) heterozygous with one dominant and one recessive trait. In this second case, the individual is said to be a carrier of the recessive allele and can pass it to their offspring.

We can see this pattern of inheritance in our own children. For example, all three authors of this book have brown eyes, but among us only one spouse also has brown eyes. When the parents with brown eyes had their first child, it was no surprise that their son also had brown eyes. After all, the alleles that contribute to brown eye color are dominant. What *was* surprising was that their second child had blue-gray eyes. This could only happen because both parents were heterozygous and carriers of the recessive alleles that code for blue eye color. Both Alison

TABLE 2.2 ■ Patterns of Inheritance

Inheritance Pattern	Description	Notes	Example(s)
Autosomal Dominant	One allele is seen in the phenotype, the other is silent. The "seen" allele is dominant.	Only one allele is needed to express the trait in the phenotype. A child who is homozygous (i.e., has two dominant alleles) will have the same phenotype as a child who is heterozygous (i.e., has only one copy of the dominant allele).	Huntington's disease Widow's peak Freckles Right handedness Dimples
Autosomal Recessive	Two "silent" alleles are needed to express this phenotype.	Both parents must pass the recessive allele to their child to express the trait. Parents who are heterozygous are carriers of the recessive allele.	Sickle-cell anemia Cystic fibrosis Left handedness Attached earlobes
Codominance	Two distinct dominant alleles are expressed.	Phenotype of child differs from either parent.	Blood type AB Blood cell shape
Incomplete Dominance	A blend of two alleles is expressed.	Phenotype of child differs from either parent.	Hair color or shape

and Carter have freckles, as do both of their parents. Because freckles are the dominant trait, we don't know if the parents have one or two alleles for freckles. Alison and Carter may have one or two dominant alleles. The point is that when a dominant trait is expressed, we can't know if the recessive gene is "hidden" in the genotype.

The story is made more complicated by the fact that there are other types of inheritance patterns. When there is codominance between the alleles, one dominant allele cannot silence the expression of the other allele. As a result, the phenotype expresses *both* alleles. This can happen when both alleles are dominant. One example is AB blood type. The alleles for the A and B blood types are both dominant. So, for example, if a child inherits the recessive allele for blood type O from one parent and a dominant allele for blood type A or B from the other parent, the offspring's blood type will be A or B, respectively. But, if the infant inherits the allele for the A blood type from one parent and the allele for the B blood type from the other parent, the infant's phenotype will express both alleles, displaying AB blood type.

In another inheritance pattern, called incomplete dominance, we don't see a single dominant trait (suppressing the recessive trait), but the phenotype is a *blend* of the alleles. For example, Edwin's brown hair is a blend of his father's blonde hair and his mother's dark hair. Hair

shape can also display incomplete dominance. An infant can have wavy hair because they express a blend of the straight hair of one parent and the curly hair of the other parent. In both codominant and incomplete dominance patterns, the phenotype of the infant is different than the phenotype of either parent, just like the AB blood type is distinct from type A or type B blood, and wavy hair is a distinct hair shape than either curly or straight hair. In summary, how two distinct versions of an allele are expressed in a phenotype varies with the relation between the alleles of a gene.

Regardless of the particular pattern of inheritance observed, we can see how the alleles from each parent work *together* to determine the phenotype. This is nicely illustrated by single-gene traits—or traits that are determined by only one gene (or pair of genes). Free-hanging versus attached earlobes, the presence or absence of a widow's peak in the hairline, having or lacking freckles, blood type, as well as genetic disorders, like Huntington's disease, sickle-cell anemia, and Tay-Sachs disease are traits determined by a single gene. (We will talk more about these genetic disorders in Chapter 3.)

The story is much more complicated because the vast majority of human traits, including height, weight, skin and hair pigmentation, are polygenic, or influenced by many genes. As you may now guess, eye color is a polygenic trait, believed to be influenced by as many as 16 distinct genes, although there are 2 main genes associated with eye color in humans (located on chromosome 15, in case you were wondering). Behavioral traits, such as intelligence or aggression, and those linked to diseases, such as type 2 diabetes, are also polygenic. The involvement of many genes to express a trait makes it much harder to see the effects of heterozygous and homozygous pairs of alleles; you may have the dominant allele for one gene contributing to a trait, but two recessive alleles for a second gene contributing to that trait at the same time. As you can see, the story of how genes interact with each other can be complicated.

Check Your Learning

1. Define DNA, genes, alleles, and chromosomes. What is the relation among each of these?
2. What is the difference between genotype and phenotype?
3. List the patterns of inheritance for single-trait genes on autosomal chromosomes. Describe the difference among these inheritance patterns.

UNDERSTANDING HOW GENES INFLUENCE DEVELOPMENT

As we discussed in Chapter 1, understanding the relative roles of nature and nurture in human characteristics has been debated for centuries. How can we tell how much of who we are is due to our genes and how much is due to our environment? We can't just look at everyone's genes and link their genes to specific characteristics. The human genome project took many years just to describe human genes. There are methods, such as genome-wide association studies (GWAS), used to identify the parts of genes associated with a trait. In GWAS, researchers scan

the genomes of individuals within a population to identify specific single-nucleotide polymorphisms (SNPs), or a single change in a DNA base, that is associated with a particular trait. The goal is to link variations in SNPs with particular phenotypes, with a focus on linking SNPs to the occurrence of particular diseases. However, it will take decades of additional research to know how certain genes map onto particular traits, and we will probably never know the whole story. In this section we will discuss how researchers understand the role of genes in development using other methods.

Behavioral Genetics and the Study of the Heritability of Traits

The branch of science called **behavioral genetics** is the study of how an individual's genetic makeup and environment affect behavior. Instead of trying to map specific genes to specific features, behavioral geneticists compare individuals who share different genes and/or environments and observe the ways in which those individuals are similar and different. This is particularly challenging with humans because most of our traits are polygenic and because our environments are complex and difficult to control or quantify. To deal with these issues, many behavioral geneticists study laboratory mice. Mice are ideal research subjects because they can be bred to be genetically identical to each other and then scientists can randomly assign them to be raised in highly controlled environments. Of course, this same approach isn't feasible, or ethical, in humans. We can't clone humans and then randomly assign them to distinct environments to study genetic and environmental contributions in the expression of a trait. Instead, to understand the influence of genes on human behavior, the field of behavioral genetics has used twin and adoption studies to evaluate the relative contributions of genetics and environments in the expression of a specific trait (Table 2.3). Both methods are examples of quasi-experiments, a type of natural experiment in which researchers take advantage of naturally occurring conditions. As we discussed in Chapter 1, in these experiments the "manipulation" is not under the experimenter's control; experimenters can't decide who will be an identical twin or randomly select some children to be adopted. This also means that we can't draw causal conclusions (because the studies are essentially correlational). But, these methods have been important for understanding the relative roles of genes and environment in the development of traits and behaviors.

Twin Studies

Scientists interested in the effects of genes and environment have studied the similarity of traits in pairs of monozygotic and dizygotic twins. Monozygotic twins, or identical twins, result when a single zygote (i.e., a fertilized human egg) divides in two separate zygotes during the first days of a pregnancy. The spontaneous division results in two infants who have all the same genes. Because the identical twins originated from a single fertilized egg, each twin has the same 23 pairs of chromosomes. Dizygotic twins, or fraternal twins, are the result of two eggs released at ovulation and each egg fertilized by a different sperm. Each egg and each sperm will have a unique set of chromosomes because of the random assortment of chromosomes into a particular egg or sperm. Thus, these two fertilized eggs will have distinct genetic information from each parent and, on average, share about half of their genes (just as any set of siblings with the same parents).

TABLE 2.3 ■ Methods for Studying Heritability

Method	Description	Logic
Twin studies	Compare the similarities of phenotypes within pairs of monozygotic and dizygotic twins	If traits (phenotypes) are highly heritable, monozygotic twins will be more similar on that trait than will dizygotic twins.
Adoption studies	Compare the similarities of phenotypes between children and their biological parents and between children and their adoptive parents.	If traits (phenotypes) are highly heritable, children will be more similar to their biological parents than to their adoptive parents.
Twin-adoption studies	Compare the similarities of phenotypes between twins who were adopted into different households.	If traits (phenotypes) are highly heritable, identical twins will be very similar even if they have been raised in different households.

Behavioral geneticists reason that if monozygotic twins are more similar to each other on a trait than dizygotic twins, this greater similarity is the result of their greater overlap in genes. These conclusions are drawn from studies in which the children are *reared together.* They live with both their biological parents and their twin (as well as any other siblings they have in common). Thus, it is assumed that the two children in a twin set share the same environment. Both monozygotic and dizygotic twins share a womb, are born into a family at the same time, and are more likely to experience major life events (moving, birth of a sibling, poverty) at the same age. As a result, the fact that identical twins are more similar to each other (e.g., they are more likely to have the same eye color, hair color, and build) than fraternal twins is thought to reflect the fact that they share more genes.

Of course, even though dizygotic twins' environments are more similar than the environments of siblings born at different times, monozygotic twins have even more similar environments. Dizygotic twins never share a placenta, whereas monozygotic twins may or may not share a placenta. Dizygotic twins can be the same sex or different sexes, whereas monozygotic twins are always the same sex. And the fact that identical twins are physically identical might mean they are treated more similarly than fraternal twins. Thus, the similarities between identical twins may reflect both similarities in their genes and their environments.

Consider two sets of famous twins, Ashley and Mary-Kate Olsen, who are fraternal twins, and Cole and Dylan Sprouse, who are identical twins (see photos). Despite being fraternal twins, as infants, Ashley and Mary-Kate shared the role of a single character, Michelle Tanner, on the TV sitcom *Full House*. As is clear from the next photo, they looked very similar, despite being dizygotic twins. Cole and Dylan Sprouse, on the other hand, are identical twins, who began to differ in appearance as adolescents. Cole Sprouse is taller and leaner than his brother, who is stockier and has a fuller face. The brothers also have different beauty marks. Cole has

IDENTICAL TWINS: Monozygotic or identical twins share all of their genes and are always the same sex.

iStock/kali9

FRATERNAL TWINS: Dizygotic or fraternal twins share on average of only about half of their genes and can be the same sex or different sexes.

iStock/kate_sept2004

THE OLSEN TWINS: Dizygotic twins, Ashley and Mary-Kate Olsen.

David Shankbone/Flickr

one on his chin while his brother sports his beauty mark over his lip. Even though these brothers share 100% of their genes, their appearance is not "identical."

These two examples show that the same genes can vary in how they are expressed. No one trait is dictated 100% by genes—the differences between Cole and Dylan show that some amount of environmental influence must have influenced their physical appearance traits. The similarities between Ashley and Mary-Kate Olsen further suggest that similar environments might make individuals more similar to each other.

Adoption Studies

Adoption studies are a second type of study used to understand the influence of genes and environment on the developing child. These studies compare infants adopted at birth into a biologically unrelated family to their biological and adoptive relatives. When an adopted infant is more similar to their biological than to their adoptive relatives, it points to a stronger influence of genes than environment. The adopted infant shares a genetic, but not an environmental, link to their biological relatives. When adoptive children are more similar to their adoptive than their biological relatives, the expression of the trait is argued to reflect stronger environmental than genetic influence because these children share their environments but not their genes with their adoptive families.

THE SPROUSE TWINS: Monozygotic twins, Cole and Dylan Sprouse.

Piper's Picks® TV, CC BY 2.5 <https://creativecommons.org/licenses/by/2.5>, via Wikimedia Commons

However, children only share 50% of their genes with each biological parent. So, we don't expect them to be exactly the same as their parents. An even stronger test is to compare individuals who have the same genes but are raised in different environments. In a variant of the adoption method, researchers study monozygotic twins who were separated at birth and adopted into distinct families. A clear advantage of this design is the ability to assess the expression of a trait across two genetically identical individuals (i.e., the monozygotic twins) raised in distinct environments (i.e., different adoptive families). There are some famous examples, such as the triplets in the movie *Three Identical Strangers*. Often these comparisons reveal eerie similarities, like twins who prefer the same brand of cigarettes or who married women with the same first name. A challenge with these studies, however, is that adoption agencies often place infants with families that are similar to the birth family in many ways, and so identical twins adopted into different families may actually have similar environments despite being raised with different families. The practice of adopting identical twins into distinct homes has declined in favor of raising the twins together in the same family.

What Have Twin and Adoption Studies Told Us?

Twin and adoption studies have been used for decades to provide insight into how much of the variation in traits and abilities is due to genes. Both types of studies have identified a genetic link to many physical and psychological traits, including psychological disorders and medical

conditions (Plomin et al., 2016). Recently, researchers have used this approach to study very specific behaviors. John Constantino and colleagues (2017), for example, compared monozygotic and dizygotic twin infants' looking at the eyes and mouths of human faces (an indicator of social engagement). They observed that monozygotic twins were more similar to each other in their interest in these features than were dizygotic twins. Thus, this early aspect of social engagement seems to be influenced by genes.

Camille Cioffi and her colleagues (2020) studied a large cohort of infants adopted at birth in open adoptions. The families were from across the United States and, unlike many studies, the group of children was racially and ethnically diverse. Cioffi and colleagues measured children's ability to voluntarily regulate their attention, a skill known as inhibitory control. They also measured inhibitory control in their biological parents. Infants whose biological parents were low on inhibitory control were at risk for also being low on inhibitory control. From this, you might conclude that inhibitory control is determined by one's genes. But it turned out that the environment also mattered. Infants who were biologically at high risk for low inhibitory control had better inhibitory control in childhood if they had warm adoptive mothers. Thus, this adoption study shows how one trait, inhibitory control, is influenced by *both* genes and the environment.

BOX 2.1—INFANCY IN REAL LIFE: THE HANSEL AND BIJANI TWINS

There is no better example of the impact of genes and the environment on development than to look at a very special type of twins called conjoined twins. Conjoined twins start out just like typical monozygotic twins, where a single fertilized human egg begins to divide in two during the first days of a pregnancy. However, conjoined twins develop when the egg doesn't fully separate into two individuals, and the two remain physically connected, most often at the chest, abdomen, or pelvis; sometimes they even share one or more internal organs. Like monozygotic twins, because the conjoined twins originated from a single fertilized egg, each twin has the same 23 pairs of chromosomes. But in this case, they also share certain parts of their bodies, or in essence, their physical and social environments.

Conjoined twins are rare, and sadly, many don't survive pregnancy or die shortly after birth. However, advances in medical technology have improved their survival rates, and sometimes surgical interventions are possible to separate the twins physically so that they can live independent lives. The ability to separate conjoined twins depends on whether they share organs and which organs they share.

Perhaps the best known pair of conjoined twins in the mainstream media are Abby and Brittney Hensel. Abby and Brittney were born in 1990, and each has a separate head, heart, lungs, spine, stomach, and spinal cord, but they share two arms, legs, large intestine, bladder, and reproductive organs. Given that they share a body, and most importantly, a single pair of arms and legs, they have to coordinate everything they do. In fact, each twin manages only one side of their body, making all movements an amazing feat of team work. In fact, they can walk, run, swim, play basketball, and even drive a car.

What is most interesting about the Hensel twins is not that they share their genes but that they literally share an environment—they share a family, a home, and a *body*. But, even with a shared "environment," Brittney and Abby are different. They have a seamstress to

make clothes for their unique body, each outfit containing separate necklines to emphasize their individuality. One twin would prefer to live in a city, while the other would opt for the calmness of a suburb. Although they both majored in education in college, they each had a different focus. And while they sometimes share meals out of pure convenience, they like different foods (despite sharing a means by which to digest those foods), and often prepare themselves different meals of foods that they each like.

These differences are not unique to this set of conjoined twins. Ladan and Laleh Bijani were conjoined twin sisters who opted for surgical separation despite the high risk of the procedure. The sisters wished to pursue different careers. Ladan wanted to be a lawyer, but Laleh wished to pursue journalism. The sisters also differed in their preference for where to live and had distinct hobbies. One preferred to play computer games, while her sister preferred computer programming. Similar to Brittany and Abby, one sister, Ladan, described herself as more outgoing and talkative while her sister, Laleh, claimed to be quieter and more introverted. Thus, although twins can share genes and are often assumed to share an environment, even twins that literally share a body experience the world in different ways, and as a result, develop differently. The two examples of conjoined twins highlight that genes are not destiny, but instead, that experiences shape each individual, making them unique.

Heritability

Heritability is a statistical measure, referred to as h^2. It is an estimate of the proportion of the differences in the expression of a trait *in a population or group* that is due to genetic differences among those individuals. It tells you the heritability of a trait given the variability in the environment of that population or group. Heritability is a ratio (i.e., fraction), whose value ranges from 0 to 1. It is calculated by dividing the variability in the genotypes of the individuals in a population by the variability in the phenotype (i.e., the trait). Heritability estimates closer to one mean that most of the differences in the phenotype in the population come from genetic differences. Heritability estimates closer to zero mean that the environment accounts for most of the differences in the phenotype of a population.

No known trait has a heritability estimate of 1 (i.e., all the variability that trait is due to genes) or 0 (i.e., all the variability in that trait is due to the environment). Rather, human traits tend to have heritability estimates of .30 to .60, indicating that genes account for only some of the variance in those traits. In other words, 30% to 60% of the variations of a trait within a population are genetic. The rest of the variation is due to environment and can be determined by subtracting the heritability estimate from 1. So, if most human behaviors have a heritability estimate of .30 to .60, then 70% to 40% of the differences in phenotype are attributed to the environment.

What isn't obvious is how heritability estimates are influenced by variation in both the genes and the environment. Basically, if there isn't much variability in one factor, the other factor will have a bigger influence on differences between people. Consider again Cole and Dylan Sprouse. Clearly, the similarity between them is the result of their genes. But, as they got older, they became more different from one another. This presumably reflects the fact that the environment and their experiences shaped their development. If traits were completely determined by genes, they would remain identical throughout their lives.

An extreme example is illustrated by the case of the mixed up brothers of Bogotá (Dominus, 2015). This is the remarkable story of four young men who were raised as two sets of fraternal twins. However, at birth, they were two sets of identical twins. As the result of a series of hospital mix-ups, two families went home with one twin from each set, and assumed that their twins were fraternal, not identical. The four young men are pictured in following photo. It is not hard to tell which brothers are actually identical twins. Despite being raised in different families, in different environments (urban vs. rural), and with different opportunities, the men who share 100% of their genes look quite a lot alike. However, the two men in each set of identical twins look different from each other. They differ in face shapes, overall size, and other aspects of their physical appearance that we assume are due to genes. Because they share 100% of their genes, these differences are 100% due to differences in experience or environment (nutrition, physical activity, disease, and so on).

Heritability estimates refer to the contribution of genes to the expression of a trait within a particular population of individuals living in a particular environment at a particular time. The heritability estimate of a trait is an average for that population given the amount of variation in the environment of that population. The more similar the environment is, the more that variation in traits will be due to genes. As illustrated by the brothers of Bogotá, when there is little variation in genes, differences in traits will be due to variation in the environment; the two men with the same genes were different from each other because of differences in their environment. When a heritability estimate is calculated, it is based on a specific population (with a particular amount of environmental variation). Therefore, the heritability estimate for *that same trait* will differ for a different population, especially if the population has greater variability in the phenotype or environment. For this reason, when interpreting a heritability estimate, consider who is in that population, how similar the individuals in the population are to each other, and the similarity of their environments. If the individuals live in a fairly uniform environment, then heritability estimates will tend to be higher. If instead the individuals come from highly varied or challenging environments, heritability estimates will be lower.

We can see this better by considering some examples. Take height. Physical height is highly heritable, with a heritability estimate as high as .90 in some studies (Macgregor et al., 2006; Perola et al., 2007). This isn't surprising. Tall parents tend to have tall children and shorter parents tend to have shorter children. Remember Tesalia? Once we considered the size of her grandmothers, her petite stature didn't seem so surprising. But the environment influences the expression of height (Dubois et al., 2012; Jelenkovic et al., 2016). Heritability of height is higher in highly resourced countries, where access to food is fairly uniform across the population, than it is for populations in other parts of the world, particularly developing countries in which access to food is not as uniform across the population (Hur et al., 2008; Jelenkovic et al., 2016).

Just like for physical traits, heritability estimates for behavioral traits also differ in different groups of people. For example, heritability estimates for intelligence differ drastically across distinct populations (Gottschling et al., 2019; Nisbett et al., 2012; Scarr-Salapatek, 1971). Intelligence reflects both our genes and our environment, so it is no surprise that its heritability estimate shifts with the environment of a population. For a population that is affluent, with access to resources and enriched environments, the heritability estimate of intelligence is on the higher end, around .72. For such populations the environment supports and facilitates

BROTHERS OF BOGOTÁ: William Cañas Velasco, Wilber Cañas Velasco, Jorge Enrique Bernal Castro, Carlos Alberto Bernal Castro, identical twins who were double switched at birth.

Courtesy of Dr. Nancy L. Segal

intellectual development, and differences between individuals reflect genes. In contrast, for populations raised in impoverished environments, such as children living in poverty, heritability estimates for intelligence are close to zero (Turkheimer et al., 2003). In those environments, differences in access to food, exposure to stress, the quality of schooling, and so on, mean that children are raised in environments that differentially support their intellectual development. In other words, in challenging environments, environmental and not genetic differences account for differences in intelligence among the individuals in the population.

How Do Genes and the Environment Interact?

In the previous section we described how traits can reflect both nature and nurture, but we haven't talked much about how genes and the environment actually interact to shape development. In this section, we will talk about several different ways that genes and the environment work together to shape development.

Gene–Environment Correlations

One way genes and the environment can together shape development is how they are *correlated* (Table 2.4). At first **gene–environment correlations** might seem hard to understand—genes are part of your biology and the environment is your home, your culture, and your society. How can they be correlated? However, the environment is not wholly independent of your genes. The people who gave you your genes, your parents, are also the people who decide your environment, particularly at young ages. If you have a genetic predisposition to dislike spicy and bitter foods, your parents likely share that dislike, and so they are not going to offer you spicy and bitter foods. Alison and Carter's mother loves tomatoes, but their father does not. During their childhood, fresh tomatoes rarely were served, and Alison and Carter dislike tomatoes. They likely inherited something from their father that caused them to dislike tomatoes, but because they were rarely offered tomatoes, they did not have the opportunity to develop a liking for tomatoes. The point is that some aspects of your environment are not completely random with respect to your genes. Rather, genes and environment can be correlated with each other. Genes not only create differences in individuals but also may be responsible for different environments, as in the case of disliking tomatoes and being raised in a tomato-reduced environment. This is an example of a *passive gene–environment correlation*. In such cases, the parents transmit both the genes and the environment, and as a result, the environment may be well suited to promote the expression of a particular trait. Another

TABLE 2.4 ■ Gene–Environment Correlations

	Description	Mechanism	Examples
Passive gene–environment correlations	Characteristics of the parent and child are similar; as a result, the parent's preferences support the child's genetic traits.	The (biological) parent provides both the genes that the child inherits and the home environment.	Children who are high on effortful control, a type of self-regulation, have parents who also are high on effortful control. At the same time, these parents provide structured home environments that are low on chaos and conducive to building effortful control (Lemery-Chalfant et al., 2013).
Evocative gene–environment correlations	Characteristics of the child elicit, or evoke, environments that support genetic traits.	Genetically determined traits are reinforced by how others respond to the child.	Children at genetic risk for aggressive behaviors are more likely to have peers respond aggressively to them, even when the peers are unfamiliar and randomly paired (DiLalla, 2002).
Active gene–environment correlations	The child seeks out environments or experiences that support genetic traits, a process called "niche picking."	As children become older, they select environments that match their traits.	Children who are extroverted seek out different social environments than those who are shy and withdrawn (Jaffee & Price, 2008)

example is parents who love to read. The parents not only give their child these bibliophile genes but also expose their children to experiences that foster an enjoyment of books and reading. Thus, the child's genotype is reinforced, supported, and enhanced by the environment created by the parents who almost certainly share this same genotype.

Other gene–environment correlations are *evocative*. In these cases, a child's trait elicits a nonrandom response or environment. Imagine seeing an infant who breaks into a smile when your eyes happen to meet. Try not smiling back! It is hard to resist breaking into your own smile when confronted with such a happy face. A child who is genetically predisposed to smile more—perhaps a temperamentally easy child (who we'll talk more about in Chapter 10)—will evoke more positive interactions from a parent and other people than a child who is less prone to smile. In contrast, a child who is aggressive with a sibling or peer will elicit a very different response, one likely to include directives to cease the unwanted behavior (e.g., "no yelling"). In *evocative gene–environment correlations*, the child's genetic characteristics shape the responses of others, which influences the child's experiences.

Finally, *active gene–environment correlations* are those that result from the individual actively seeking particular experiences. A child who enjoys sports may seek activities that allow them to be active; a child who loves music may participate in the school musical or play in the marching band. When he was 3 years old, Itzhak Perlman heard a violin playing on the radio and begged his parents for a violin. Eventually, his parents relented and Itzhak began learning the instrument at age 4. Itzhak persisted in his passion for learning the violin and now is one of the best known classical violinists. His story is an example of genetic propensities that inspire an individual to seek particular experiences, even when those may not be present in their home environments.

Gene–Environment Interactions (G×E)

In many cases, the genes and the environment interact to determine development. One way to think about these **gene–environment interactions** (G×E) is that one's genes make one especially susceptible, vulnerable, or sensitive to specific environmental influences. For example, genetic differences cause some children to be more vulnerable to the effects of stress. Children with a higher genetic susceptibility to environmental experiences, sometimes called *orchid children*, thrive only when the conditions are optimal. They have difficulty adapting to stressful events (e.g., poverty, family discord, an illness), and their development is negatively shaped by those experiences. However, when the environment is supportive and reduces stress, these children thrive. Other children thrive regardless of stressors or traumas that they may experience; these children are sometimes called *dandelion children*. The botanical references allude to the distinction between nurturing an orchid so that it not only survives but blooms (a gardening feat that many novice plant lovers never achieve) and the dandelion, which is a weed that will sprout and duplicate at an alarming rate with no care and minimal growing conditions (and despite intense efforts to eradicate it). Akin to these plants, environmentally sensitive children are disproportionately more impacted by their contexts, whether positive or negative, than children who are less sensitive to their environment (Lionetti et al., 2018, 2019).

We can see how these G×E interactions shape development in a study by Grayzna Kochanska and her colleagues (2011). These researchers compared children who differed in their genotype for a particular gene, the serotonin transporter linked polymorphic region, or in the parlance of

geneticists, *5-HTTLPR*. Individuals with the short allele of this gene are more likely to develop depression, anxiety, aggression, risk taking, and alcohol abuse than are individuals with the long allele of the gene (Carhart-Harris & Nutt, 2017; Caspi et al., 2003; Martínez et al., 2020).

Kochanska and her colleagues asked whether variation in these genotypes made children more or less susceptible to maternal responsiveness (mother's attunement and engagement with their child). In other words, are children who have the short allele more susceptible to their environments (i.e., are more orchid-like) than children who have the long allele? To find out, they followed children from 1 to 5 years of age and they measured maternal responsiveness, social competence, and rule following (or moral internalization). They found that children with the long form of the allele were dandelion children; their development was minimally related to maternal responsiveness. Regardless of the level of maternal responsiveness they experienced, these children became socially competent and followed the rules. Children who had the short form of the allele, in contrast, were orchid children; their social competence and moral development depended on whether their mother was high or low in responsiveness. When maternal responsiveness was very low, these children were low on social competence and moral internalization. As maternal responsiveness increased, children's moral competence and moral internalization increased. In fact, these orchid children actually developed *better* moral internalization than did children with long alleles if they had highly responsive mothers. The main point is that unlike children with long alleles, development in children with short alleles depends on their environment. Children with a short allele developed optimally if their mother was responsive but not if their mother was unresponsive.

Epigenetics

Epigenetics is another way the *expression* of genes is influenced by experience (Alegría-Torres et al., 2011; John & Rougeulle, 2018). Epigenetics involves changes in how the genes are expressed, *without changes in the DNA itself*. Many factors can cause epigenetic changes, such as diet, physical activity, working habits, smoking and alcohol consumption, psychological stress, or environmental pollutants. Epigenetic changes can alter gene expression through several processes, but the most commonly studied is a chemical process called *methylation*, in which a methyl group is added to the DNA. This changes how cells read the DNA instructions. Importantly, these changes are passed on as cells divide (creating new genes) and can even be passed onto the offspring of the individual.

Epigenetics is often discussed in terms of how specific experiences negatively alter how genes are expressed. For example, there was significant discussion about the effects of the Dutch famine of 1944–1945 on the genes of the people who experienced it. During this period of World War II, the Nazis blocked all food supplies to the Netherlands, and everyone in the country experienced famine. To understand the effect of malnourishment during prenatal development, researchers followed the individuals whose mothers were pregnant during this time. What was fascinating was that it was during adulthood that these individuals began to look different from their peers whose mothers did not experience famine during pregnancy. Individuals that had been in utero during the Dutch famine were as adults more likely to be obese, have higher cholesterol, and develop diabetes. This seems like a mystery—how can something that happened during prenatal development show effects in adulthood? The answer is epigenetics. Because the pregnant mothers were starving, a methyl group was added to some of the fetuses' genes as an adaptation to their mother's undernourishment, silencing those genes. This change may have

aided the fetus in surviving, but once the famine had ended, this change in the functioning of the genes created health problems later in adulthood. They entered life with methylated DNA sequences that would be interpreted differently by cells throughout life.

One definition of epigenetic changes is that they can be inherited. This is clearly seen when cells divide and create new cells that contain the altered functioning of DNA. But, epigenetic changes in germ cells can be transmitted to the next generation. In fact, the epigenetic effects on pregnancies during the WWII Dutch famine continued to future generations. Adults whose paternal grandmothers were pregnant with their fathers during the famine are heavier and more obese than adults whose fathers had not experienced undernourishment during their prenatal development (Veenendaal et al., 2013). Thus, the epigenetic changes in the father as a fetus were passed on to his own offspring.

It's important to point out that epigenetics is a part of normal development. Not all epigenetic effects are negative. Physical exercise can create epigenetic modifications that yield health benefits, such as promoting the expression of genes that suppress tumors. Similarly, diet has been shown to reduce or increase the risk of particular cancers (Nystrom & Mutanen, 2009).

BOX 2.2—INFANCY IN REAL LIFE: NICU STRESS, PARENTAL SENSITIVITY, AND EPIGENETICS

One way we can see the effect of epigenetics in infant development is by carefully studying children who spend the first weeks or months after birth in the neonatal intensive care unit (NICU). The NICU is a very stressful place for an infant to be. Infants in the NICU frequently experience painful procedures (e.g., heel sticks to draw blood), they do not have the physical

BABY IN NICU: A newborn in the neonatal intensive care unit.

iStock/andresr

contact with a caregiver that is optimal for development, and they are in the NICU because they are sick and fragile.

Rosario Montirosso and her colleagues conducted work to understand exactly *how* the NICU experiences alter development. This work focused on the epigenetic effects of pain-related stress in the NICU. Specifically, in one study they observed that preterm infants who experienced more pain-inducing procedures (e.g., blood draws) had more methylation of genes in the serotonin transport system—a system in the brain that is involved in stress regulation (Provenzi et al., 2015). As we have discussed, this methylation can silence the gene. Thus, we may expect that methylation of these genes would create problems for how children cope with stress in the future.

In fact, Montirosso and her colleagues followed these children over time and found that the methylation that occurred as a result of their pain-induced stress while in the NICU was related to later emotion and stress regulation. They observed that infants' temperament (see Chapter 10) at 3 months was related to methylation of these genes (Montirosso et al., 2016), and methylation in premature newborns was related to expressions of anger at 4 years (Provenzi et al., 2020). This shows how epigenetic changes as a result of stress very early in life can have a long-lasting effect on emotion and stress regulation.

But, across development children do not have experiences at just one time that determine outcomes. Development is a *cascade*. The early experiences influence gene expression, which in turn both determine future experience and how children will respond to future experience. In another study, Provenzi and Montirossso (Provenzi et al., 2017) found that the level of methylation at birth and maternal sensitivity together contributed to how full-term 3-month-old infants responded to the stress of a face-to-face interaction with their mothers in which their mother maintained a still face (see Chapter 10). Specifically, when mothers were less sensitive in general, infants with more methylation had a more negative response to the still face. This is important because it shows that experience, in this case interactions with the mother, continue to shape the way genes are expressed.

Check Your Learning

1. What is behavior genetics?
2. List the methods researchers use to study the heritability of traits.
3. Why do researchers compare monozygotic and dizygotic twins to understand the role of genes in the expression of traits?
4. What is a heritability estimate?
5. What are the three types of gene–environment correlations?
6. Describe how genes and environments can mutually influence each other.

BRAIN AND NERVOUS SYSTEM DEVELOPMENT

Besides genetics, one of the most remarkable things about human biology is how our brains work. You may have heard that humans are born too early because of their big brains (see Chapter 3). And, in Chapter 1, we talked about how programs like WIC focus on the first 1,000

days of development because that is a time that is critically important for brain development. In this section of this chapter, we will discuss the remarkable development that happens during those first 1,000 days.

Development at the Cellular Level

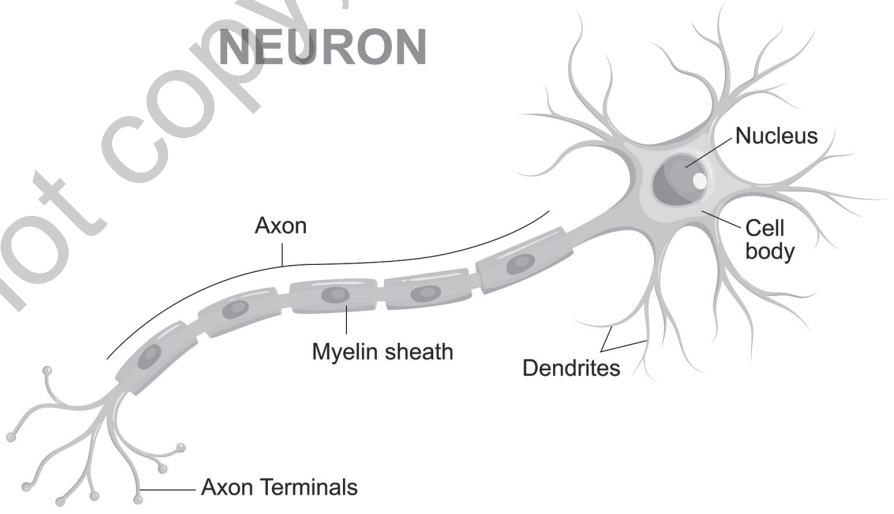
As we will see, the structure of the brain is a product of development. As you will learn in more detail in Chapter 3, during the prenatal period, the development of all body systems, including the nervous system, emerge from a single cell. Thus, the brain and nervous system develop as a function of development that happens at the cellular level.

You likely have learned in other classes that there are two types of cells in the nervous system, **neurons** and **glia**. These cells have different functions within the nervous system. The neurons are the basic units of the nervous system. They create networks that allow information to be passed throughout the brain. The glia play important support roles, making it possible for neurons to survive, make networks, and send information efficiently.

Look at the picture of the neuron in Figure 2.3. It has several parts, each of which is important for the transmission of information. As is true of any cell, neurons have a cell body with a nucleus. In addition, neurons have dendrites that branch from the cell body. The dendrites pick up information in the form of chemical signals from other neurons. This is the main way that the neuron receives messages. Each neuron also has an axon extending from the cell body toward a set of axon terminals. This is the main way neurons send messages. Once a signal is

FIGURE 2.3 ■ Neuron

This shows the parts of a neuron, including its cell body, nucleus, axon (which includes the myelin sheath) and axon terminal. Extending from the cell body are the dendrites.



Source: iStock/Vitalii Dumma

detected by the dendrites, the message is passed down through the axon in the form of an electrical signal. The information is then passed on through the axon terminals in the form of chemical messages (neurotransmitters), to be picked up by another neuron. In Figure 2.3 you can also see that the axon has myelin sheaths. These are areas of a fatty insulation layer surrounding the axon, which allows the electrical signal to move more quickly along the axons. Although some axons are as short as one millimeter—and thus information will travel pretty quickly over that short distance—other axons are as long as one meter! Myelin helps to make sure that electrical signals are transmitted rapidly even over these very long distances.

The other cells of the nervous system—the glia—are more numerous than neurons. The glia do not transmit information. Instead, the many different types of glial cells hold neurons in place, supply oxygen and nutrients to neurons, provide the myelin, and remove debris. The glial cells also play an important role in the migration of neurons. Thus, the glia are critical for normal development of the nervous system.

The neurons and glial cells are created through neurogenesis and gliogenesis (Table 2.5). Another process that contributes to the development of the nervous system is neuronal death; even during the period of neurogenesis, both neurons and glia die. Although it may seem silly to create neurons and then have them die, cell death is an important part of the developmental process. It is so important to the development of the nervous system that a lack of cell death is thought to play a role in developmental disorders (Kolb, 1989). Even though some neurons die even before birth, human newborn infants have about 100 billion neurons, whereas as adults they will have about 85 billion. Interestingly, unlike other cells in the body, neurons themselves don't divide and replicate. This means that nearly all the neurons that an individual will ever have are created during the early period of prenatal development.

TABLE 2.5 ■ Process of Brain Development

Process	Definition	Timing
Neurogenesis	Creation of new neurons from stem cells	Mostly between 3rd and 15th week of prenatal development but occurs to a limited level even into adulthood
Cell migration	Migration of newly formed neurons from the ventricular or subventricular zone to their final home	About 4 weeks after conception to about 6 months after conception
Synaptogenesis	Creation of connections between neurons	Begins at about 20 weeks after conception and continues into early adulthood
Synaptic pruning	The loss of synapses due to lack of use or cell death	From about age 2 until adolescence
Myelination	The formation of a fatty sheath on neuronal axons to facilitation conduction of electrical signals	From about 5 months postconception into adulthood

When neurons are first created, they are not fully formed with dendrites and axons; they start out as a cell body. As you will learn in Chapter 3, the nervous system develops from the creation of the neural tube. Once the neural tube is complete, its cells begin to proliferate, or to create new cells. These new cells are special stem cells; some of those stem cells become neurons and some become glial cells. The region where these cells are created is called the ventricular zone. This is a temporary layer of tissue that is involved in neurogenesis, or the creation of new neurons, and gliogenesis, the creation of new glia. As the neuronal stem cells that make up this ventricular zone are depleted (from the creation of new neurons and glia), this region disappears. Thus, neurogenesis happens in the ventricular zone only prenatally.

Neurogenesis also occurs in the subventricular zone (SVZ), and this region persists into adulthood. Although it was once believed that neurogenesis occurred only prenatally, we now know that in some animals neurogenesis can occur in the SVZ as well as the hippocampus in adulthood, although neurogenesis is not as prolific beyond the prenatal period. Neurogenesis is mostly complete by 4 months postconception. During this period of prenatal development, there is an overproduction of neurons; that is, the system creates more neurons than it will need.

While all neurons are created in the ventricular or subventricular zone, the mature brain has neurons in many regions. How does the structure of the brain emerge from cells that are all created in only a couple of locations? The answer is that the brain develops from the inside out. Neurons and glial cells are created in the ventricular zone and then **migrate** to other regions of the brain. That is, they move toward different regions of the brain that will become their final location. This is accomplished, in part, by the glial cells acting as guides for the migration. The glial cells create a radial pattern from the ventricular zone to the outer layers of the developing brain. The newly formed neurons travel along these paths to form those outer layers. The guides are temporary, however, and after migration the glial cells that provided these pathways either degenerate or become part of other supporting structures in the brain. This system helps to create the laminar structure of the cortex, or the fact that it contains six layers of cells. As each new set of cells migrates, the glial guides help them migrate *past* the previously migrated cells. Thus, the older cells remain in the inner layers, and the newer cells migrate to create the outer layers. This active migration shows how this layered pattern of the cortex is a product of development.

Although this is the main way that cells migrate, it is not the only kind of migration that happens in brain development. Some cells do not use the glial pathways and instead migrate perpendicular to them. Some regions of the brain are the product of both kinds of migration. These other forms of migration yield different organization and structures than the more common form of migration.

Once neurons have clustered in a region, they begin to form **synapses**, or connections between them. To do this, the dendrites and axons begin to grow, as synapses typically involve the dendrites of one neuron connecting with the axon of another neuron (although synapses can form between axons of two neurons or between dendrites of two neurons). During this phase of **synaptogenesis**, or creation of synapses, neurons make synapses in every direction, apparently preparing for any contingency. During this process, neurons grow, and their axons extend toward other neurons, apparently randomly. Unlike the other processes of neuronal development discussed so far, this begins prenatally (after migration), but it continues for many

years. The number of synapses increases exponentially, reaching a peak between 1 and 4 years, depending on the area of the brain. As was true for the creation of neurons, there is an overproduction of synapses during this period; that is, more synapses are created than are needed, or than will be used. During this period of development, the focus is on creating as many connections between neurons as possible.

Thus, these early processes of neurogenesis and synaptogenesis result in a large number of neurons making a large number of connections. You would think that this was good—big, connected brains should make us really smart. But, it turns out that having many neurons and connections does not allow the brain to operate *efficiently*. Because information travels through too many connections, processing is slow, and it is difficult for the resources available to maintain the neurons to support the system. Thus, development also includes **pruning** of synapses, or eliminating some.

Some pruning results from neuronal death that occurs as a normal part of brain development. Other pruning results from the loss of synapses (axons and dendrites) as the networks are used. Although it might seem like this is a waste of energy (to create connections that will be lost), this system creates more robust processing networks than if development involved creating fewer, more efficient networks from fewer synapses right from the start. As connections are stimulated, those that are used are maintained and become more stable, and those that are redundant or not used as often are eliminated. In this way, the brain adapts to experience, keeping the connections that are useful and efficient given the kinds of experience and input that the infant has.

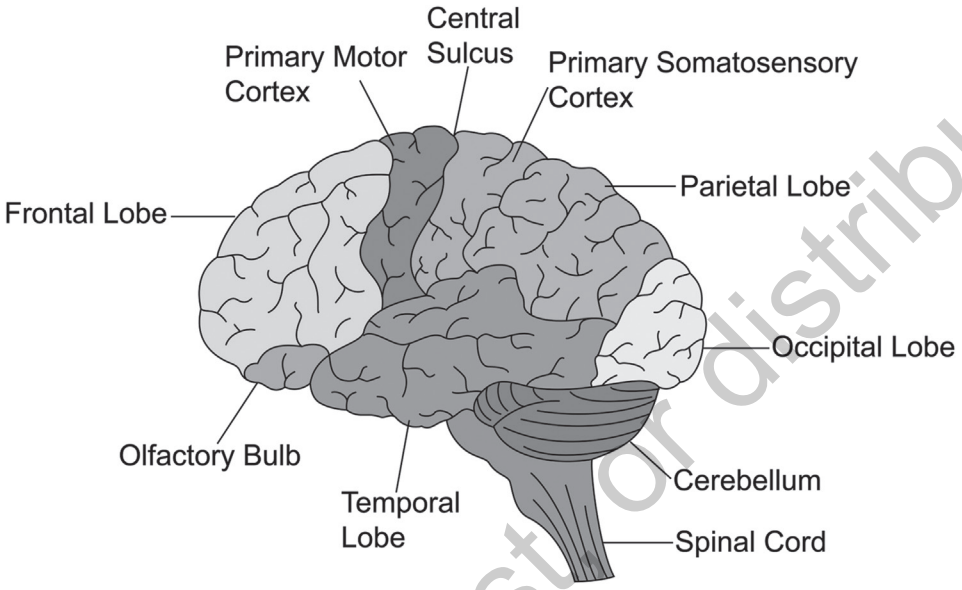
Finally, the process of **myelination**, or the formation of the myelin sheaths on the axons of neurons, occurs. Myelination begins prenatally but continues for many years, with this process happening in some brain regions into the 20s! Myelination occurs as a particular kind of glial cells, oligodendrocytes, develop and create the fatty substance that is wrapped around neuronal axons. Once myelinated, the electrical signal travels more quickly down the axon, allowing for more efficient communication in the nervous system. For example, before the neurons of the motor system are myelinated, infants have jerky and uncoordinated movements. Myelination in these brain regions allow for smoother, more controlled movement. The importance of myelin can be seen in the effect of demyelinating disorders, such as multiple sclerosis (MS). In these disorders, the existing myelin is destroyed. For example, in MS, the immune system attacks myelin. As these diseases progress, patients experience muscle weakness, visual and speech impairments, and motor problems. Although many people live with MS for many years, experiencing periods where their symptoms are worse and then better, for some people the symptoms are severe and can lead to confinement to a wheelchair.

Brain Structures as Developmental Products

The previous section was about how the cells of the nervous system are created and develop. But what about the *structure* of the brain? The brain is not a single, uniform thing; it is made up of different regions (Figure 2.4). You likely know that the brain is made up of two hemispheres, each with different functions. In fact, you may have heard people talking about being "left-brained" or "right-brained," which usually means that they are saying that they are more or less creative. This is a fun way of thinking about the two hemispheres, but in reality we are

FIGURE 2.4 ■ Brain Organization

The structures of a mature human brain.



Source: iStock/mrhighsky

all "whole-brained" and everyone uses both halves of the brain. However, it is the case that the two hemispheres are specialized for different skills and functions. For example, in most adults, language is mostly represented in the left hemisphere, and emotions are mostly represented in the right hemisphere (which supports the left-brain/right-brain distinction). But the regions of the brain are even more specialized. For example, the occipital lobe, which is located in the back of the brain, is where vision is represented and controlled. The frontal lobe, which is at the front of the brain, is the region involved in planning, inhibitory control, and other high-level functions. The question is how does this structure develop? These structures and their specialization are well established in children and adults. When do they become established?

The structure emerges, in part, from precisely the processes described in the previous section. Brain structure is a product of the creation and migration of cells, synaptogenesis, and pruning. Recall that from the start there is structure in the nervous system. The nervous system emerges from the formation of the neural tube (see Chapter 3). This neural tube already shows specialization, with the proliferation of neurons occurring on one side of the tube and the spinal cord developing from the other side of the tube. Remember also that the ventricular zone, where the neurons are created, is *transient*—it disappears when the neuronal stem cells that make up this region are depleted (because they have all become neurons). The prenatal development of the brain involves other temporary structural changes. At the same time that there are the transient ventricular and subventricular zones, there are other transient zones, such as a transient

cortical plate, which is the future cortex. The cortical plate is densely packed with post-migration neurons. This temporary structure develops rapidly during the fetal period, growing in size and eventually creating the folds (gyri) and indentations (sulci) that characterize the adult brain. So even early in development the brain has structure, and the different structures serve different functions. But, some of the structures present during development disappear, and many structures present in the adult brain are not present during the early phases of brain development. Thus, the structure of the brain is a *product* of development.

So how do brain structures develop and become specialized, as they are in adults? When does the left hemisphere become specialized for language and the right temporal lobe become specialized to process faces? This is a very complicated question with no easy answers. Consider how we know about brain specialization in older children and adults. To determine which brain regions are involved in a particular task or type of processing, researchers use functional magnetic resonance imaging (fMRI). In an MRI, a part of the body is scanned using a magnetic field and radio waves. When you are in the scanner, the magnetic field causes the water molecules in your body to realign, and the radio waves are used to create a faint signal from this realignment. This is used to create an image of the structures of the brain.

In fMRI, scans are taken when a person is doing a task. For example, a researcher might present a person with pictures of faces and houses while that person is being scanned. This procedure would show that different parts of the right temporal lobe of the brain were active when the person was looking at the faces and the houses; in adults, studies like this have shown that there is a part of the brain specialized for processing images of faces and a different part of the brain specialized for processing images of places (Cohen Kadosh & Johnson, 2007).

Clearly, this procedure cannot be used easily with preverbal infants. It is difficult to imagine how you would do fMRI with infants and examine differences in brain activity in response to different tasks. Some people have done it, though. For example, Ghislaine Dehane-Lambertz and her colleagues (2002) conducted MRI scans of 3-month-old French infants as they listened to 20 seconds of speech played forward and 20 seconds of speech played backward. The infants' "task" was to recognize that forward speech, but not backward speech, was language. Like adults, the left (or language) regions of these 3-month-old infants' brains showed more activation during forward speech. This shows that some of the structure in the brain related to how speech and language is processed is specialized by the time infants are 3 months old. Other studies have shown that there is some specialization of brain structures related to language processing even in newborns (May et al., 2011). As you will see in Chapters 3 and 9, however, the fact that newborns already have brain areas specialized for speech processing likely shows how their prenatal experience shapes their language and brain development.

Mark Johnson (2011) has suggested that specialization develops across infancy and is the result of the initial connections formed in the brain and of experience. As the brain develops, different regions are connected. For example, the occipital lobe gets input from the optic nerve, which is connected to the retina in the eye. The temporal lobe gets input from the cochlea in the ear. These connections mean that when the retina or cochlea detect information, the signal is sent to different parts of the brain. However, according to Johnson, specialization for particular types of inputs comes from experience. So, as infants look at and process faces, input about faces

connects to the future "face region" because of how the brain is initially connected. With experience looking at faces, this face region becomes specialized for processing faces.

Plasticity

An important consequence of the way the nervous system develops is that it is characterized by **plasticity**. That is, development is not fixed by some biological program; rather, it adapts in response to variations in the environment and experience. Children have different experiences: Some children hear only one language and other children hear more than one language, some children are exposed to music from early on, some children are read to, some children spend many hours outdoors with different types of plants and animals. These differences in experience shape how the brain develops. Remember, synapses are formed based on experience, and synapses are pruned based on experience.

Classic work revealed that laboratory rats raised in environmentally complex environments (e.g., with other rats, with toys and other experiences) developed bigger brains than laboratory rats raised in a typical environment (e.g., individual cages without extra toys or experiences) (Greenough et al., 1987). This work suggested that the number of synapses that were formed and maintained depended on experience. In other words, the development described earlier is not fixed but rather is plastic and depends on experience.

What about human children? We obviously can't manipulate children's experiences, but we can compare the brains of children with very different experiences.

Let's return to the Romanian orphans we discussed in Chapter 1. Compared to their counterparts who were not raised in institutions, children from Romanian orphanages showed abnormal brain activity (Chugani et al., 2001). These differences likely reflect the effect of neglect and impoverished early experience on brain development. But these comparisons, and those described earlier, reflect correlational findings. Because it is not ethical to randomly assign children to be in institutions or to experience neglect, these studies can't tell us that the brain differences were *caused* by experience.

But, the Bucharest Early Intervention Project (BEIP) discussed in Chapter 1 did randomly assign children to remain in institutions or to be put in foster care. This study then provided an opportunity to ask how different experiences cause differences in brain development. In general, this research showed that children who were randomly assigned to foster care had better brain development than did the children who remained in the institutions (Bick et al., 2015). In addition, the earlier children were put in foster care, the better their outcomes (Nelson et al., 2009). Although we would never want to have the opportunity to do this study again, there are important things we can learn from this work about plasticity and brain development. Specifically, brain development *adapts* to the kinds of experiences the child has. If the child is well nourished, stimulated, and provided with many different experiences, brain development is optimal. If the child is neglected and raised in an impoverished environment, brain development is not optimal.

These examples are extremes. It does not mean that children in poverty always experience poor brain development. Children who are fed, nurtured, and given interesting experiences,

whether with expensive toys or sticks and cardboard boxes, will have the opportunity to adapt to those experiences and develop. It is when children are neglected and the environment is severely impoverished that damage occurs. Even when there is adequate nutrition, social and emotional support, and stimulation, the brain adapts to differences in experience. The brains of infants exposed to two languages develop differently than the brains of infants exposed to only one language. The brains of children who learn to read using vision will differ from the brains of children who learn to read by touch using Braille. The brains of infants who spend a lot of time building with blocks and puzzles will develop differently than the brains of infants who never engage in spatial play. The point is that plasticity is a part of *normal* brain development, and our brains, even during infancy, reflect differences in our experiences.

Remember, however, that we said that there is similarity in how individual people's brains are structured. Most adults have language in the left hemisphere and have an area of the right temporal lobe specialized for processing faces. How can brains both be so similar and reflect this adaptive process?

Greenough and his colleagues (1987) described two types of plasticity, experience-expectant and experience-dependent, that help to explain how the brain adapts to experience and yet we see similarities across different brains (Table 2.6). Specifically, **experience-expectant plasticity** is a way of describing the commonalities across people in how the brain is organized and specialized. In this case, plasticity reflects the brain adapting to experiences that are common to virtually all members of a species. For example, virtually all human infants see with two eyes, are exposed to a caregiver, and hear (or see) human language. Because those experiences are common to all members of the species, it isn't immediately obvious how brain development reflects that experience—our experiences are all the same and our brain structures (that reflect that experience) are all the

TABLE 2.6 ■ Experience-Expectant and Experience Dependent Plasticity

Type of Plasticity	Definition	Characteristics	Examples
Experience-expectant	The brain adapts to the presence or absence of an experience that is typical of human experience	— Experience occurs during a *critical period* in development. — Experience is typical of virtually all members of the species.	— Binocular vision depends on coordinated input from two eyes. — A region in the right temporal lobe specific for face processing depends on early visual experience.
Experience-dependent	Individual differences in brain organization and structure develop from idiosyncratic differences in experience.	— Can occur throughout development. — Reflects individual differences in experience.	— Formation and maintenance of synapses in networks that are stimulated by experience.

same. But, when we find people who didn't have this typical experience, we see that their brains adapted in a different way, giving us insight into how the common structure and specialization of the brain across individuals is related to common experience. Specifically, we can see the effect of this experience by looking at the brains of individuals whose two eyes don't work together, as in amblyopia or strabismus; or who do not have a caregiver, as in the Romanian orphans; or who are not exposed to any language, such as deaf infants whose parents don't sign.

Consider the case of strabismus. For decades, children with crossed-eyes (strabismus) or a lazy eye (amblyopia) would have their eyes surgically corrected in early childhood. The thought was that it was best to have the surgery when children were a bit older, and that the main reason for the surgery was cosmetic. However, normal vision requires good vision in both eyes and alignment between the two eyes. The normal or expected visual experience is coordinated, good visual information from both eyes. When a child has or develops a lazy eye or crossed-eyes, the brain does not receive coordinated, good visual information from both eyes. For example, it is often the case in amblyopia that the brain receives clearer visual input from one eye than the other. In addition, because the child can't easily keep the two eyes in alignment, the brain can't reconcile the differences between the input from the two eyes. Without any treatment, the brain will rely more heavily on the clearer input from the "good" eye and will suppress the less clear input from the other eye. The brain will "see" by relying on the information from just one eye, and it will be as if the individual is blind in the other eye. Importantly, as we will see in Chapter 4, depth perception depends on binocular vision, or the ability to coordinate the input from the two eyes. If strabismus is left uncorrected, normal binocular vision and depth perception will not develop. Now doctors carefully examine how infants use their eyes together. Each of us remembers our pediatrician carefully examining how our infants moved their eyes and telling us what to look for so we could spot the emergence of a lazy eye early on. We now know that this experience is so important for vision development that it is not unusual to see an infant with glasses or an eye patch, which is the way doctors treat these early vision problems.

This is an example of experience-expectant plasticity because it shows how the brain develops "normally" when it receives the ubiquitous experience in human experience. Virtually all children have two functioning eyes that work together; only about 4% of children have some form of strabismus. We only see evidence of plasticity—or the brain adapting to differences in experience—in the rare cases when the typical experience is not present. And we see plasticity in how the infant brain responds to treatment. When strabismus and amblyopia are treated in infancy, by strengthening a weak eye or surgically adjusting how tight the muscles are around the eye, the effects of these conditions are minor. But, if these conditions are not "fixed" until the child is several years old, the visual parts of the brain will never be like those of a child with "normal" visual experience. This is why Greenough et al. called this experience-expectant; normal brain development occurs when the individual has the expected experience. When the individual does not have that experience, atypical or abnormal brain development occurs, for example, resulting in the inability to use the two eyes together to perceive depth.

Another characteristic of experience-expectant plasticity is that it occurs early in development and requires that the experience happens during a specific critical period. In the case of strabismus, the infant brain develops rapidly as visual input is detected, perceived, and

processed. With each day, the parts of the brain dedicated to processing and representing visual information become organized in response to visual experience. The longer the system experiences uncoordinated input and a weak signal from one eye, the longer the brain will suppress the input from that weak eye and the brain organization will be focused only on the input from the good eye. At some point in development, the organization of those brain areas will stabilize, and it will be impossible to undo that organization. However, if the strabismus is treated early in infancy—either by patching the good eye (forcing the brain to use the information from the weak eye) or by surgically correcting the problem—and children become able to use the two eyes together, binocular depth perception can develop. The point is that the experience of using the two eyes together must occur at a specific point in development for the brain to be able to effectively use the coordinated information to perceive depth.

The other kind of plasticity, **experience-dependent plasticity**, refers to how brain development adapts to idiosyncratic experiences. In this case, there is no "normal" or expected experience. Some children are first born, and others are born with older siblings. Some infants are raised in a noisy city, and other infants are raised in the quiet countryside. Some children live in small communities and see hundreds of people and other children live in big cities of millions. None of these experiences are common to virtually all members of the species, and yet they all have the potential to influence brain development.

Consider the effect of your hometown size on your ability to process faces. Ben Balas and Alyson Saville (2015) measured face memory in two groups of adults. One group was raised in small hometowns (fewer than 1,000 people). The other group was raised in towns of at least 30,000 people. Although these subjects were adults (between 18 and 24 years of age), they had spent their whole lives (until college) in either a very small or medium sized hometown. Balas and Saville wondered if the parts of their brain that are involved in face processing would be shaped by this difference in experience.

The results were striking. First, the adults who were raised around few people had poorer memory for faces than did the adults who were raised in larger towns. But, Balas and Saville also measured brain responses to faces in these adults. Many studies have shown that adults have a very specific brain response, or ERP (event-related potential), to faces. The ERP is a measure of the electrical activity that can be recorded on the scalp. By linking the electrical activity that is recorded to the presentation of specific images, researchers can get insight into how those images are processed. Specifically, in adults, the ERPs to face stimuli are different from the ERPs to other stimuli, such as chairs. Balas and Saville found that their adults who were raised in larger towns showed the pattern that has been observed in many studies, whereas adults who were raised in small towns did not show this effect. The way in which the brains responded to faces was different between these groups, presumably because of their different early life experience with faces. This is an example of experience-expectant plasticity because both groups of adults had experience with faces. But, the kind of experience they had was different. In addition, all of the people in the study could see and remember faces; it's just that their ability to do so differed as a function of their experience with faces during the time when their brain's ability to process faces was developing.

What both of these kinds of plasticity show is how brain development is adaptive and that brain structure and organization are a product of development. We have similarities in our

brains because of the similarities in our experiences. The differences we observe reflect differences in our experiences. The overproduction of neurons and synapses and the processes of cell death and synaptic pruning allow this adaptability.

BOX 2.3—INFANCY IN REAL LIFE: SES AND BRAIN DEVELOPMENT

One way we see plasticity in brain development is in the relationship between socioeconomic status (SES) and brain development. SES refers to one's position in society, and it has implications for many aspects of development. Children from low SES households—households with fewer financial resources, lower levels of parental education, and that are in less affluent neighborhoods—likely experience poorer nutrition, more stress, and less time dedicated to parent–child interactions than do children from higher SES households. Thus, differences in SES are not a single thing, but rather SES is a proxy for a number of different variables that may contribute to brain development.

Many studies have revealed differences in high and low SES infants in their cognitive and language abilities (e.g., Clearfield & Niman, 2012; Fernald et al., 2013). Indeed, in a large study of mostly White midwestern U.S. children, Kimberly Noble and her colleagues (2015) found effects of SES on memory and language scores in toddlers. The question is whether we can see differences in the brain itself. One MRI study of African American 5-week-old female infants in Philadelphia from poor and middle-class families revealed that lower SES infants had smaller brain structures than their higher SES peers (Betancourt et al., 2016). Another study of 6- to 10-month-old infants in London (mostly White) showed that the electrical signal recorded from the scalp (EEG) differed as a function of SES (Tomalski et al., 2013). Because other work has shown that across childhood cortical thickness is related to SES (higher SES children have thicker cortices), and that cortical thickness is related to language and other cognitive abilities (Brito et al., 2017), it seems likely that the observed cognitive differences in high and low SES infants reflect brain differences. Thus, there is increasing evidence that across studies conducted in different regions of the world with children from different racial and ethnic backgrounds, SES is associated with brain development.

The question is *why* and *how* these relations exist. We have already pointed out that SES is not one thing. Because effects are often observed for low SES, it is likely that some of these effects reflect the kind of epigenetic changes on gene expression we discussed earlier. Poor children are more likely to experience food insecurity or undernutrition; family stress, such as from the loss of a job, is likely higher; and other environmental factors associated with poorer neighborhoods might contribute to how genes are expressed (Hackman et al., 2010; Hackman & Farah, 2009). There are some more direct effects too. Poor children are less likely to have access to good medical care, so it is possible that some of the effects reflect poorer health.

In fact, the differences in care are evident even before birth. Low SES women have more pregnancy-related complications, including premature birth, stress, and poor nutrition (Hackman et al., 2010). Given how much brain development happens during prenatal development, these factors likely have an impact on brain development. But as we have seen, the brain continues to develop after birth. Low SES parents are more likely to experience stress after the birth of their child, which may affect their parenting and sensitivity to their infant.

Low SES households also tend to have lower levels of cognitive stimulation, for example, fewer books, fewer trips, and fewer words spoken to the infants. These are all correlational, however, making it difficult to know how each of these factors *causes* differences in brain growth and development. Some understanding can be found in studies with animals, for example, by raising some animals in a more stressful environment than other animals. We also can see from the work with the Romanian orphans who were randomly assigned to foster care that rearing conditions can cause differences in brain development. Of course, we don't know what it was exactly about the foster care that caused the effect, but this work suggests that we should be designing interventions to help children from lower SES households.

In fact, in 2014, pediatricians recommended that all parents read to their babies, which led to a campaign to encourage parents, especially parents with low SES, to read more to even the youngest infants. This was accompanied by the observation that there existed a "word gap"; children from low SES families simply hear fewer words each day than do children from higher SES families (Rowe, 2008, 2012). This led to campaigns to encourage families to talk to their babies. In 2013, the Clinton Foundation partnered with Next Generation, a California nonprofit organization, to create "Too Small to Fail" (http://toosmall.org/). The initial mission of this organization was to promote brain and language development by giving parents and caregivers information and tools to talk, sing, and read to children from birth. This is a great example of how research translates to action that is taken in the service of helping all children develop.

MOM READING WITH BABY: Mom reading to her infant. This may be one way that mothers with more education provide enriched experience to their infants from an early age.

iStock/fizkes

Check Your Learning

1. What are the main processes of cellular brain development? Define each one.
2. What do we know about how brain specialization develops in infancy?
3. What are the primary differences between experience-expectant and experience-dependent plasticity?

SUMMARY

This chapter provided an overview of the biological foundations of development in infancy and the early development of the nervous system. Our biology and the environment come together in various (and sometimes complicated) ways to produce our physical and behavioral characteristics. Individuals' genetic makeup and their environments dynamically influence each other throughout infancy. This chapter also highlighted the various ways we can study the relative contributions of genetics and the environment in the lab, and how twin and adoption studies can be particularly useful in helping scientists quantify the relative contributions of genes and the environment on behavior. Finally, we discussed the types of cells that are found in the brain, their genesis, and their function, which all set the stage for infant development across the various domains we will explore in the coming chapters.

KEY TERMS

alleles
behavioral genetics
chromosomes
DNA
dominant
epigenetics
experience-dependent plasticity
experience-expectant plasticity
gene–environment correlations
gene–environment interactions
genes
genotype
glia
heredity
heterozygous
homozygous
migration
myelination
neuron
phenotype
plasticity
pruning
recessive
synapse
synaptogenesis

REVIEW QUESTIONS

1. Define the basic structures that determine our genetics. How do these structures determine the traits that are observed in people?
2. What is heritability? How do we study heritability?

3. What are gene–environment correlations? Give an example of each of the three types.
4. What are gene–environment interactions?
5. Describe an example of epigenetics in development.
6. What are the parts of the neuron? What is the function of each of these parts?
7. Give examples of experience-expectant and experience-dependent plasticity in development.

CRITICAL THINKING QUESTIONS

1. The study of genetics in development is often thought of as the study of "nature" in development. How is genetics the study of nature? In what ways is the study of genetics actually the study of the relative roles of nature and nurture in development?
2. Why do adoption studies focus on studying infants adopted at birth rather than those adopted at older ages?
3. In the text, we described an example in which every member of a family had a dominant trait (freckles). Why is it impossible to know the underlying genotypes of these individuals? How would it be different if every member of the family had a recessive trait (e.g., light colored eyes)?
4. Why are twin and adoption studies *quasi-experimental*?
5. Heritability estimates are typically used to describe the level to which a trait is genetic. How are heritability estimates influenced by the environment? What does this mean for estimating how much a trait is due to genes?
6. The case of the "mixed up brothers of Bogotá" has fascinated psychologists as the "perfect experiment." How is this case similar to and different from other twin studies, and why are those similarities and differences important for the conclusions we can draw from this case?
7. How are passive, evocative, and active gene–environment correlations different? What does each mean for the way genes and the environment together influence development?
8. One characteristic of development of the nervous system is that neurons and connections between neurons (synapses) are overproduced, and as a result young children have more neurons and more synapses than they will ever have. Why is this characteristic of the development of the nervous system important?
9. Explain how experience-expectant and experience-dependent plasticity are each examples of how the brain adapts to differences in experience across development.