- Physical attractiveness best predicts which women will bond with men of high occupational status.
- The major mate-attraction strategy of women is increasing their physical attractiveness; in men, it is displaying their power and resources.
- Men are more likely than women to commit adultery.

Fundamental Genetics

Darwin did not understand two of the key facts on which his theory of evolution was based. He did not understand why conspecifics differ from one another, and he did not understand how anatomical, physiological, and behavioral characteristics are passed from parent to offspring. While Darwin puzzled over these questions, an unread manuscript in his files contained the answers. It had been sent to him by an unknown Augustinian monk, Gregor Mendel. Unfortunately for Darwin (1809–1882) and for Mendel (1822–1884), the significance of Mendel's research was not recognized until the early part of the 20th century, well after both of their deaths.

Mendelian Genetics

LO 2.9 Describe what Mendel's work with pea plants tells us about the mechanisms of inheritance.

Mendel studied inheritance in pea plants. In designing his experiments, he made two wise decisions. He decided to study dichotomous traits, and he decided to begin his experiments by crossing the offspring of true-breeding lines. **Dichotomous traits** occur in one form or the other, never in combination. For example, seed color is a dichotomous pea plant trait: Every pea plant has either brown seeds or white seeds. **True-breeding lines** are breeding lines in which interbred members always produce offspring with the same trait (e.g., brown seeds), generation after generation.

In one of his early experiments, Mendel studied the inheritance of seed color: brown or white. He began by crossbreeding the offspring of a line of pea plants that had bred true for brown seeds with the offspring of a line of pea plants that had bred true for white seeds. The offspring of this cross all had brown seeds. Then, Mendel bred these first-generation offspring with one another, and he found that about three-quarters of the resulting second-generation offspring had brown seeds and about one-quarter had white seeds. Mendel repeated this experiment many times with various pairs of dichotomous pea plant traits, and each time the result was the same. One trait, which Mendel called the **dominant trait**, appeared in all of the first-generation offspring; the other trait, which he called the **recessive trait**, appeared in about one-quarter of the second-generation offspring. Mendel would have obtained a similar result if he had conducted an experiment with true-breeding lines of brown-eyed (dominant) and blue-eyed (recessive) humans.

The results of Mendel's experiment challenged the central premise on which all previous ideas about inheritance had rested: that offspring inherit the traits of their parents. Somehow, the recessive trait (white seeds) was passed on to one-quarter of the second-generation pea plants by first-generation pea plants that did not themselves possess it. An organism's observable traits are referred to as its **phenotype**; the traits that it can pass on to its offspring through its genetic material are referred to as its **genotype**.

Mendel devised a theory to explain his results. It comprised four ideas. First, Mendel proposed that there are two kinds of inherited factors for each dichotomous trait—for example, that a brown-seed factor and a white-seed factor control seed color. Today, we call each inherited factor a **gene**. Second, Mendel proposed that each organism possesses two genes for each of its dichotomous traits; for example, each pea plant possesses either two brown-seed genes, two white-seed genes, or one of each. The two genes that control the same trait are called **alleles** (pronounced "a-LEELZ"). Organisms that possess two identical genes for a trait are said to be **homozygous** for that trait; those that possess two different genes for a trait are said to be **heterozygous for** that trait. Third, Mendel proposed that one of the two kinds of genes for each dichotomous trait dominates the other in heterozygous organisms. For example, pea plants with a brown-seed gene and a white-seed gene always have brown seeds because the brown-seed gene always dominates the white-seed gene. And fourth, Mendel proposed that for each dichotomous trait, each organism randomly inherits one of its "father's" two factors and one of its "mother's" two factors. Figure 2.15 illustrates how Mendel's theory accounts for the result of his experiment on the inheritance of seed color in pea plants.

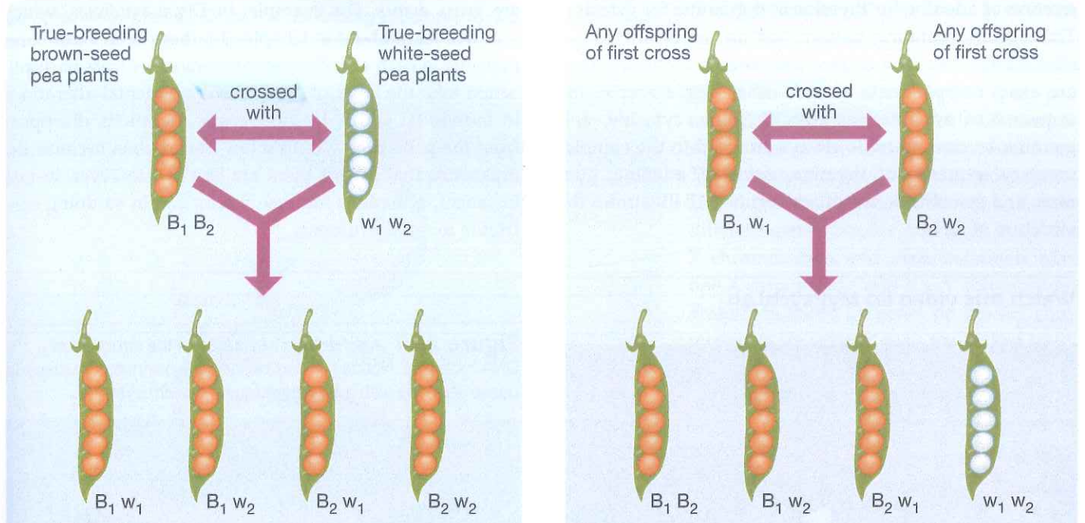
Chromosomes

LO 2.10 Understand the structure and function of chromosomes.

REPRODUCTION AND RECOMBINATION. It was not until the early 20th century that genes were found to be located on **chromosomes**—the threadlike structures in the nucleus of each cell (see Brenner, 2012). Chromosomes occur in matched pairs, and each species has a characteristic number of pairs in each of its body cells; humans have 23 pairs. The two genes (alleles) that control each trait are situated at the same location, one on each chromosome of a particular pair.

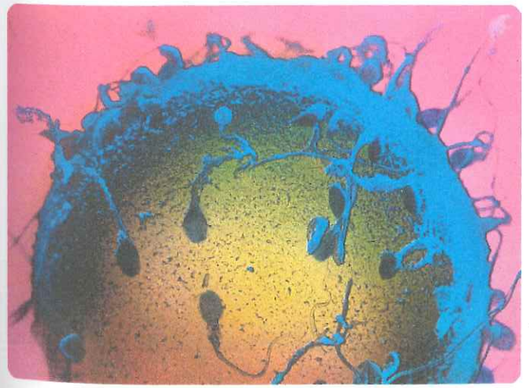
The process of cell division that produces **gametes** (egg cells and sperm cells) is called **meiosis** (pronounced "my-OH-sis")—see Sluder and McCollum (2000). In meiosis, the chromosomes divide, and one chromosome of each

Figure 2.15 How Mendel's theory accounts for the results of his experiment on the inheritance of seed color in pea plants.



pair goes to each of the two gametes that results from the cell division. As a result, each gamete has only half the usual number of chromosomes (23 in humans); and when a sperm cell and an egg cell combine during fertilization (see Figure 2.16), a **zygote** (a fertilized egg cell) with the full complement of chromosomes is produced.

Figure 2.16 During fertilization, sperm cells attach themselves to the surface of an egg cell; one will enter the egg cell and fertilize it.



The random division of the pairs of chromosomes into two gametes is not the only way meiosis contributes to genetic diversity. Let us explain. During the first stage of meiosis, the chromosomes line up in their pairs. Then, the members of each pair cross over one another at random points, break apart at the points of contact, and exchange sections. As a result of this **genetic recombination**, each of the gametes that formed the zygote that developed into you contained chromosomes that were unique, spliced-together recombinations of chromosomes from your mother and father.

In contrast to the meiotic creation of the gametes, all other cell division in the body occurs by **mitosis** (pronounced "my-TOE-sis"). Just prior to mitotic division, the number of chromosomes doubles so that, when the cell divides, both daughter cells end up with the full complement of chromosomes.

STRUCTURE AND REPLICATION. Each chromosome is a double-stranded molecule of **deoxyribonucleic acid (DNA)**. Each strand is a sequence of **nucleotide bases** attached to a chain of *phosphate* and *deoxyribose*; there are four nucleotide bases: *adenine*, *thymine*, *guanine*, and *cytosine*. It is the sequence of these bases on each chromosome that constitutes the genetic code—just as sequences of letters constitute the code of our language.

The two strands that compose each chromosome are coiled around each other and bonded together by the attraction of adenine for thymine and guanine for cytosine. This specific bonding pattern has an important consequence: The two strands that compose each chromosome are exact complements of each other. For example, the sequence of adenine, guanine, thymine, cytosine, and guanine on one strand is always attached to the complementary sequence of thymine, cytosine, adenine, guanine, and cytosine on the other. Figure 2.17 illustrates the structure of DNA.

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MEIOSIS

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MITOSIS

Replication is a critical process of the DNA molecule. Without it, mitotic cell division would not be possible. Figure 2.18 illustrates how DNA replication is thought to work. The two strands of DNA start to unwind. Then the exposed nucleotide bases on each of the two strands attract their complementary bases, which are floating in the fluid of the nucleus. Thus, when the unwinding is complete, two double-stranded DNA molecules, both of which are identical to the original, have been created.

Chromosome replication does not always go according to plan; there may be errors. Sometimes, these errors are gross errors. For example, in *Down syndrome*, which you will learn about in Chapter 10, there is an extra chromosome in each cell. But more commonly, errors in duplication take the form of **mutations**—accidental alterations in individual genes. In most cases, mutations disappear from the gene pool within a few generations because the organisms that inherit them are less fit. However, in rare instances, mutations increase fitness and in so doing contribute to rapid evolution.

Figure 2.17 A schematic illustration of the structure of a DNA molecule. Notice the complementary pairings of nucleotide bases: thymine with adenine and guanine with cytosine.

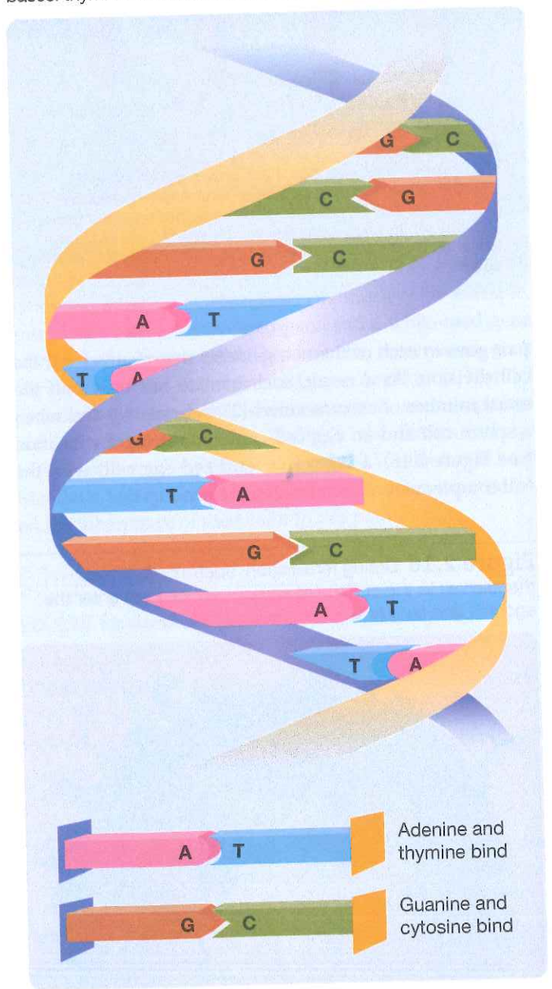
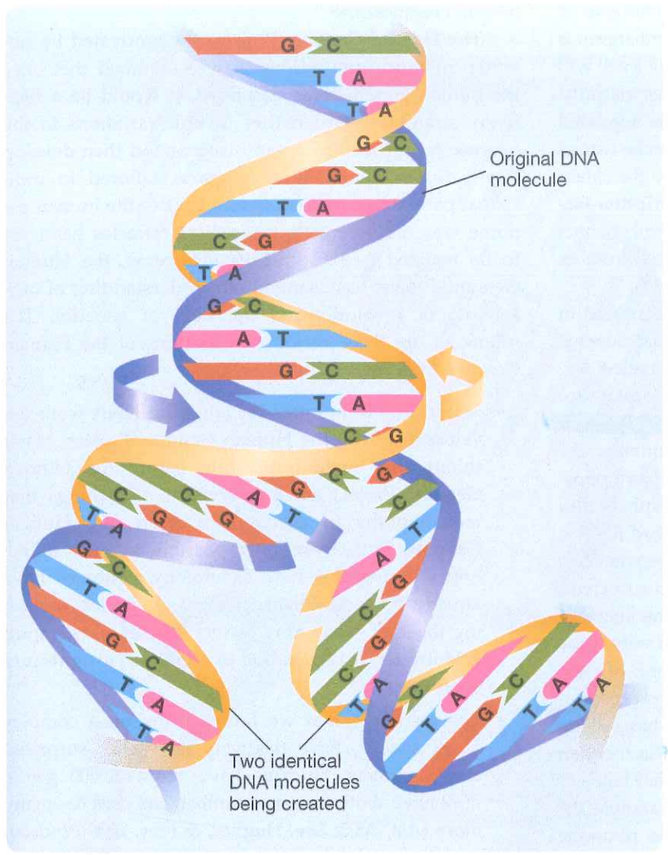


Figure 2.18 DNA replication. As the two strands of the original DNA molecule unwind, the nucleotide bases on each strand attract free-floating complementary bases. Once the unwinding is complete, two DNA molecules, each identical to the first, will have been created.



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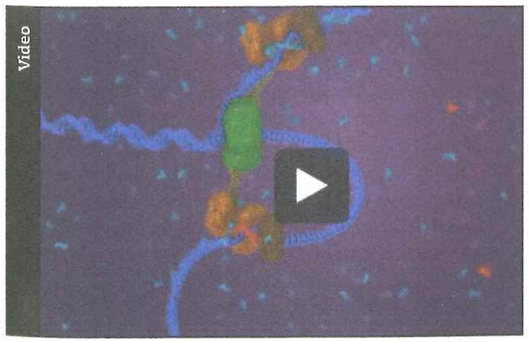
DNA REPLICATON



SEX CHROMOSOMES AND SEX-LINKED TRAITS. There is one exception to the rule that chromosomes always come in matched pairs. The typical chromosomes, which come in matched pairs, are called **autosomal chromosomes;** the one exception is the pair of **sex chromosomes**—the pair of chromosomes that determines an individual's sex. There are two types of sex chromosomes, X and Y, and the two look different and carry different genes. Female mammals have two X chromosomes, and male mammals have one X chromosome and one Y chromosome. Traits influenced by genes on the sex chromosomes are referred to as **sex-linked traits**. Virtually all sex-linked traits are controlled by genes on the X chromosome because the Y chromosome is small and carries few genes (see Maekawa et al., 2014).

Traits controlled by genes on the X chromosome occur more frequently in one sex than the other. If the trait is dominant, it occurs more frequently in females. Females have twice the chance of inheriting the dominant gene because they have twice the number of X chromosomes. In contrast, recessive sex-linked traits occur more frequently in males. The reason is that recessive sex-linked traits are manifested only in females who possess two of the recessive genes—one on each of their X chromosomes—whereas the traits are manifested in all males who possess the gene because they have only one X chromosome. The classic example of a recessive sex-linked trait is color blindness. Because the color-blindness gene is quite rare, females almost never inherit two of them and thus almost never possess the disorder; in contrast, every male who possesses one color-blindness gene is color blind.

Genetic Code and Gene Expression

LO 2.11 Outline the mechanisms of gene expression.

Structural genes contain the information necessary for the synthesis of proteins. **Proteins** are long chains of **amino acids;** they control the physiological activities of cells and are important components of cellular structure. All the cells in the body (e.g., brain cells, hair cells, and bone cells) contain exactly the same genes. How then do different kinds of cells develop? The answer lies in stretches of DNA that lack structural genes—indeed, although all genes were once assumed to be structural genes, those genes comprise only a small portion of each chromosome.

The stretches of DNA that lack structural genes are not well understood, but it is clear that they include portions called *enhancers* (or *promoters*). **Enhancers** are stretches of DNA whose function is to determine whether particular structural genes initiate the synthesis of proteins and at what rate. The control of **gene expression** by enhancers is an important process because it determines how a cell will develop and how it will function once it reaches maturity. Enhancers are like switches because they can be regulated in two ways: They can be turned up, or they can be turned down. Proteins that bind to DNA and influence the extent to which genes are expressed are called **transcription factors**. Many of the transcription factors that control enhancers are influenced by signals received by the cell from its environment (see Shibata, Gulden, & Sestan, 2015).

The expression of a structural gene is illustrated in Figure 2.19. First, the small section of the chromosome that contains the gene unravels, and the unraveled section of one of the DNA strands serves as a template for the transcription of a short strand of **ribonucleic acid (RNA)**. RNA is like DNA except that it contains the nucleotide base uracil instead of thymine and has a phosphate and ribose backbone instead of a phosphate and deoxyribose backbone. The strand of transcribed RNA is called **messenger RNA** because it carries the genetic code out of the nucleus of the cell. Once it has left the nucleus, the messenger RNA attaches itself to one of the many **ribosomes** in the cell's *cytoplasm* (the clear fluid within the cell). The ribosome then moves along the strand of messenger RNA, translating the genetic code as it proceeds.

Each group of three consecutive nucleotide bases along the messenger RNA strand is called a **codon**. Each codon instructs the ribosome to add 1 of the 20 different kinds of amino acids to the protein it is constructing; for example, the sequence guanine-guanine-adenine instructs the ribosome to add the amino acid glycine. Each kind of amino acid is carried to the ribosome by molecules of **transfer RNA**; as the ribosome reads a codon, it attracts a transfer RNA molecule that is attached to the appropriate amino acid. The ribosome reads codon after codon and adds amino acid after amino acid until it reaches a codon that tells it the protein is complete, whereupon the completed protein is released into the cytoplasm. Thus, the process of gene expression involves two phases: the *transcription* of the DNA base-sequence code to an RNA base-sequence code and the *translation* of the RNA base-sequence code into a sequence of amino acids.

Human Genome Project

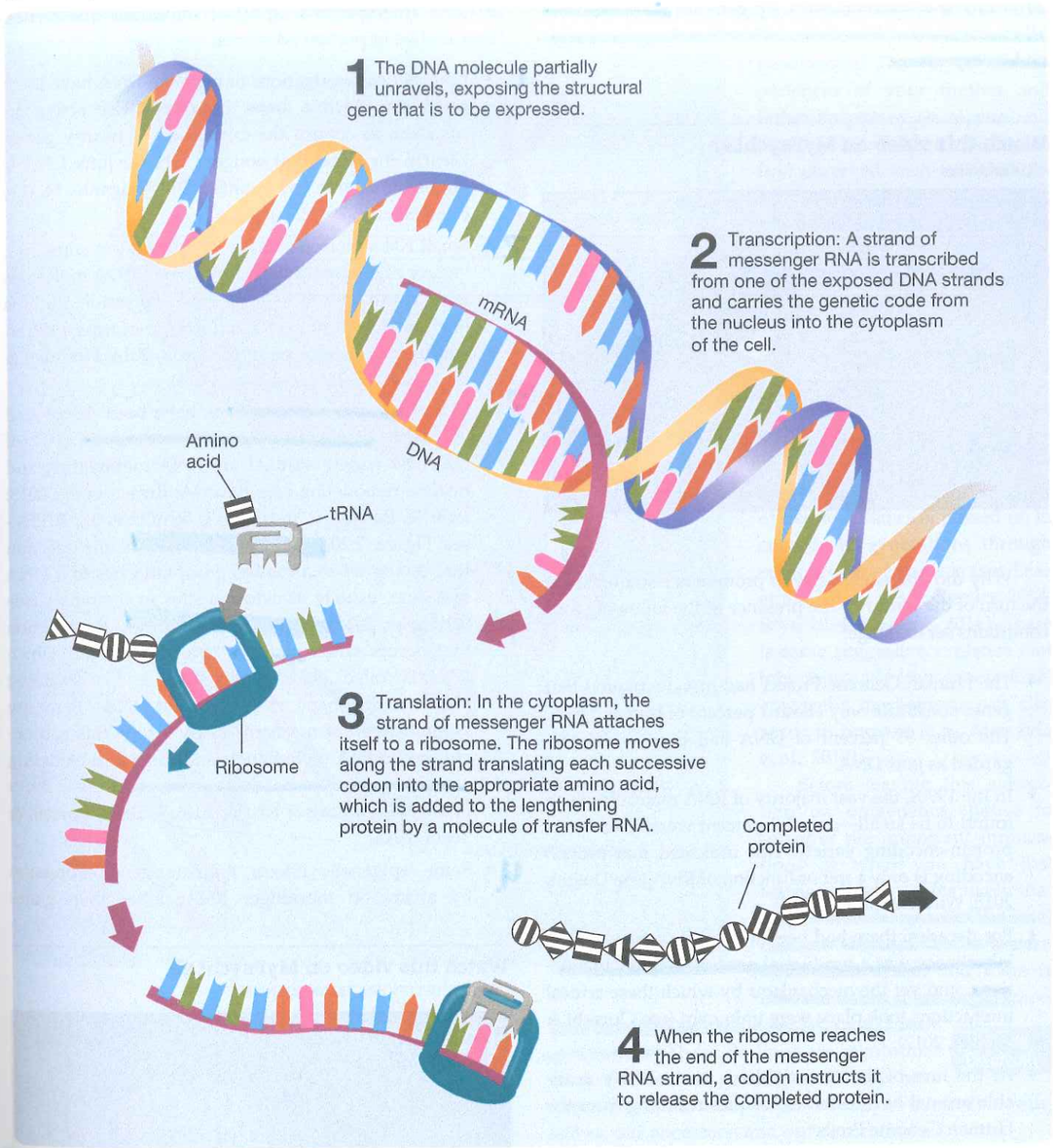
LO 2.12 Discuss several ways in which modern advances have changed our understanding of genetic processes.

One of the most ambitious scientific projects of all time began in 1990. Known as the **Human Genome Project**, it was a loosely knit collaboration of major research institutions and individual research teams in several countries. The purpose of this collaboration was to compile a map of the sequence of all 3 billion bases that compose human chromosomes.

The Human Genome Project was motivated by potential medical applications. It was assumed that once the human genome was described, it would be a relatively straightforward matter to link variations in the genome to particular human diseases and then develop treatment and prevention programs tailored to individual patients. More than a decade after the human genome was described, these medical miracles have yet to be realized (see Hall, 2010). However, the Human Genome Project has changed our understanding of ourselves and revolutionized the field of genetics. The following are three major contributions of the Human Genome Project:

- Many new techniques for studying DNA were developed during the Human Genome Project. Many things that were impossible before the Human Genome Project are now routine, and things that took months to accomplish before the Human Genome Project are now possible in only a few hours. Using this new technology, genomes have already been established for many species, including those of many long-extinct species (see Shapiro & Hofreiter, 2014), leading to important insights into evolution.
- The discovery that we humans, the most complex of all species, have relatively few genes surprised many scholars. Humans have about 20,000 genes; mice have about the same number, and corn has many more (Ast, 2005; Lee, Hughes, & Frey, 2006). Indeed, protein-encoding genes constitute only about 1 percent of human DNA. Researchers have now generated a nearly complete map of the entire set of proteins encoded for by our genes: the **human proteome** (Kim et al., 2014).
- Many variations in the human genome related to particular diseases have been identified. However, this has proven to be less useful than anticipated: So many genes have been linked to each disease that it has proven difficult to sort out the interactions among the numerous genes and experience (Hall, 2010). Compounding the problem is that even when many genes have been linked to a disease, all of them together often account for only a small portion of its heritability (Manolio et al., 2009). For example, 18 different gene variants have been linked to adult-onset diabetes, but these 18 variants account for only 6 percent of the heritability of the disease (see Stumvoll, Goldstein, & Haeften, 2005).

Figure 2.19 Gene expression. Transcription of a section of DNA into a complementary strand of messenger RNA is followed by the translation of the messenger RNA strand into a protein.



Modern Genetics: Growth of Epigenetics

LO 2.13 Define epigenetics, and explain how it is transforming our understanding of genetics.

Around the turn of the century, the field of genetics changed. Interest shifted away from protein-encoding genes and their expression to other possible mechanisms of inheritance. In particular, interest shifted to the mechanisms by which experience exerts its effects on development. This led to an explosion of interest in an area of genetics research that had been lingering in the background since 1942: epigenetics.

Epigenetics is often defined by what it is not: It is not what genetics had been prior to epigenetics' rise to

prominence. Since the discovery of genes in the 1960s, the structure and expression of genes had been the focus of genetics research and thinking (see Franklin & Mansuy, 2010; Zhang & Meaner, 2010). **Epigenetics** is the study of all mechanisms of inheritance other than the genetic code and its expression.

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EPIGENETICS

Why did epigenetics rise to prominence so quickly at the turn of the century? The presence of the following four conditions set the stage:

- The Human Genome Project had just discovered that genes constitute only about 1 percent of human DNA. The other 99 percent of DNA had been widely regarded as *junk DNA*.
- In the 1990s, the vast majority of RNA molecules were found to be small—only 1.2 percent were of the large protein-encoding variety. This indicated that protein encoding is only a minor function of RNA (see Dolgin, 2015; Wilusz & Sharp, 2013).
- For decades, there had been a general consensus that inheritance was a product of gene-experience interactions, and yet the mechanisms by which these critical interactions took place were unknown (see Oureshi & Mehler, 2012).
- At the turn of the century, there was a newly available arsenal of research techniques resulting from the Human Genome Project.

Stimulated by these four factors, it was not long before the wave of research into epigenetics began to produce important discoveries, which further fanned the flames of enthusiasm for epigenetic research. Genetics had just spent half a century focused exclusively on the genetic code as the mechanism of inheritance, and the new epigenetic research led to discoveries that challenged this narrow view.

Despite its youth, epigenetic research has already amassed an impressive array of important discoveries. Here are five important advances:

1. Epigenetic investigations of nongene DNA have identified many active areas. Many of these active areas seem to control the expression of nearby genes. Clearly, the belief that nongene DNA is junk DNA is no longer tenable (see Pennisi, 2014; Tragante, Moore, & Asselbergs, 2014).
2. Small RNA molecules have been found to come in a variety of different types. Some small RNA molecules have been found to regulate gene expression, but it is likely that each type of small RNA performs a different function (see Gorman & Maron, 2014; Hoffman & Pilpel, 2015; Schmiedel et al., 2015).
3. Many epigenetic mechanisms have been discovered by which gene expression can be regulated. Two of the most widely studied are DNA methylation and histone remodeling (see Baker-Andresen et al., 2013; LaSalle, Powell, & Yasui, 2013; Schultz et al., 2015)—see Figure 2.20. **DNA methylation** is the reaction that occurs when a methyl group attaches to a DNA molecule, usually at cytosine sites in mammals (see Schübeler, 2012). **Histone remodeling** is the reaction that occurs when **histones** (proteins around which DNA is coiled) change their shape and in so doing influence the shape of the adjacent DNA—there are several different mechanisms by which this can occur. Both DNA methylation and histone remodeling can either decrease or increase expression (see Bintu et al., 2016; Keung & Khalil, 2016; LaSalle, Powell, & Yasui, 2013).
4. Some epigenetic effects regulate gene expression by acting on messenger RNA rather than genes (see Izaurralde, 2015 Mattick, Mehler, 2008); this is called **RNA editing**. Small RNA molecules and other proteins have been shown to cleave messenger RNA apart at precise points and sometimes to splice sections of new RNA to create a new sequence of bases.
5. Remarkably, epigenetic changes such as DNA methylation and histone remodeling can be induced by particular experiences (e.g., neural activity, hormonal state, changes to the environment) that can last a lifetime (Handel & Ramagoplan, 2010; Nadeau, 2009; Nelson & Nadeau, 2010; Riccio, 2010; Sweatt, 2013).

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CHALK IT UP! EPIGENETIC MECHANISMS

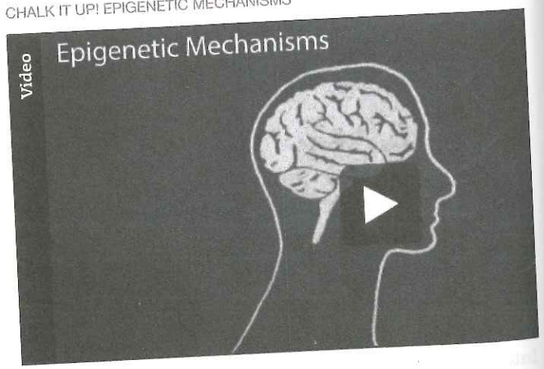
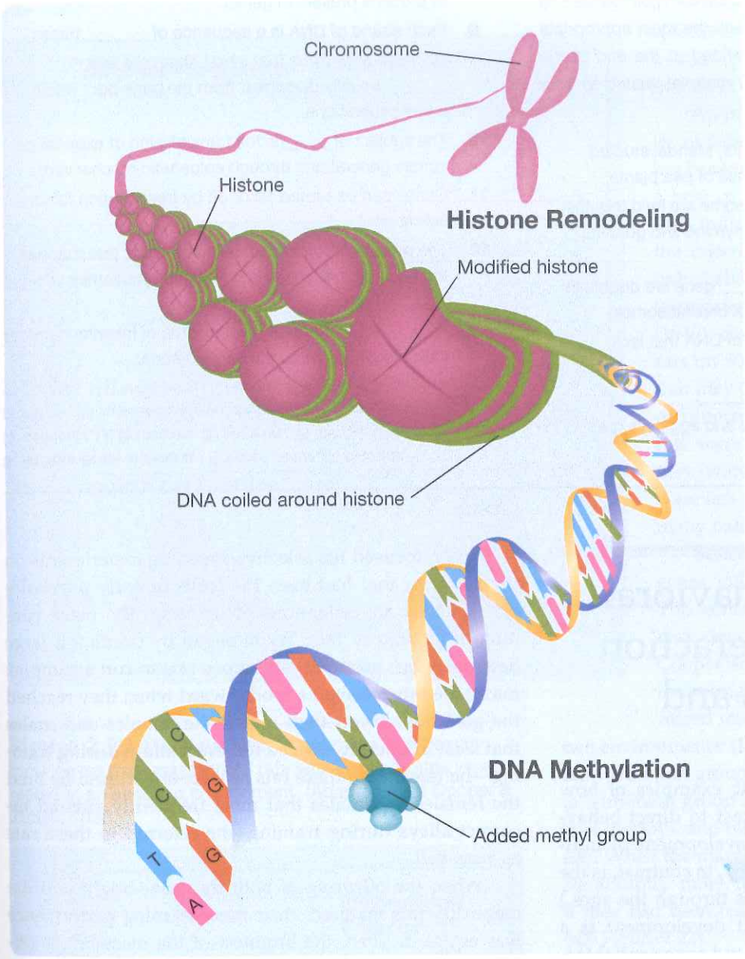


Figure 2.20 Two epigenetic mechanisms. Histone remodeling involves modifications to a histone protein (around which DNA is coiled). DNA methylation involves the attachment of a methyl group to DNA. Both DNA methylation and histone remodeling can either decrease or increase gene expression.



Epigenetic mechanisms are known to produce enduring changes in an individual. But can those experience-induced changes be passed on to future generations? That is, can the experiences of your mother and father be passed on to you and on to your children? Biologists first observed such transgenerational epigenetic effects in plants, but such effects have now been observed in mammals as well. **Transgenerational epigenetics** is a subfield of epigenetics that examines the transmission of experiences via epigenetic mechanisms across generations (see Hughes, 2014). For example, it has been shown that when mice experience an odor associated with a painful shock, the memory of that experience is passed on to subsequent generations through epigenetic mechanisms (see Dias et al., 2015; Dias & Ressler, 2014; Szyf, 2014; Welberg, 2014). There is some suggestive evidence that inheritance via transgenerational epigenetic mechanisms can also occur in humans (e.g., Marczylo et al., 2012).

Before leaving this subsection on epigenetics, pause to consider the important implications of what you have just learned. It now seems likely that each person's genetic material changes through life as experiences accumulate, and there is evidence that these experience-induced changes can be passed on to future generations. These findings are revolutionizing the field of genetics, and they have major implications for how we humans think about ourselves, our ancestors, and our descendants.

Thinking Creatively What implications does the study of epigenetics have for researchers who are trying to determine the genetic bases of a particular disorder, like schizophrenia?

Scan Your Brain

Do you remember what you have just read about genetics so that you can move on to the next module with confidence? To find out, fill in the following blanks with the most appropriate terms. The correct answers are provided at the end of the exercise. Before proceeding, review material related to your errors and omissions.

1. In his groundbreaking experiments, Mendel studied _____ traits in true-breeding lines of pea plants.
2. The double strands of a chromosome are held together by the attraction of adenine for thymine and guanine for _____.
3. The chances of inheriting the _____ gene are double in females because they have two X chromosomes.
4. _____ are found in the stretches of DNA that lack structural genes.
5. Egg cells and sperm cells are _____.
6. All body cells except sperm cells and egg cells are created by _____.
7. The _____ gives complete information about the sets of proteins present in genes.
8. Each strand of DNA is a sequence of _____ bases.
9. Because organisms that inherit them are less fit, _____ usually disappear from the gene pool within a few generations.
10. The subject of _____ is the transmission of experiences across generations through epigenetic mechanisms.
11. Genes can be turned off or on by transcription factors acting on _____.
12. The massive international research effort that mapped the sequence of bases in human chromosomes was the _____ Project.
13. _____ is the study of mechanisms of inheritance other than modifications to the genetic code.

Scan Your Brain answers: (1) dichotomous, (2) cytosine, (3) dominant, (4) Enhancers, (5) gametes, (6) mitosis, (7) human proteome, (8) nucleotide, (9) mutations, (10) transgenerational epigenetics, (11) enhancers, (12) Human Genome, (13) Epigenetics.

Epigenetics of Behavioral Development: Interaction of Genetic Factors and Experience

This module comprises three classic examples of how genetic factors and experience interact to direct behavioral ontogeny. (**Ontogeny** is the development of individuals over their life span; **phylogeny**, in contrast, is the evolutionary development of species through the ages.) In each example, you will see that development is a product of the interaction of genetic and experiential factors, which we now know is likely mediated by epigenetic mechanisms (see Sweatt, 2013).

Selective Breeding of "Maze-Bright" and "Maze-Dull" Rats

LO 2.14 Discuss what insight into the genetics of behavior was gained from early research on selective breeding.

You have already learned in this chapter that most early psychologists assumed that behavior develops largely through learning. Tryon (1934) undermined this assumption by showing that behavioral traits can be selectively bred.

Tryon focused his selective-breeding experiments on the behavior that had been the focus of early psychologists in their investigations of learning: the maze running of laboratory rats. Tryon began by training a large heterogeneous group of laboratory rats to run a complex maze; the rats received a food reward when they reached the goal box. Tryon then mated the females and males that least frequently entered incorrect alleys during training—he referred to these rats as *maze-bright*. And he bred the females and males that most frequently entered incorrect alleys during training—he referred to these rats as *maze-dull*.

When the offspring of both the maze-bright and the maze-dull rats matured, their maze-learning performance was assessed. Then, the brightest of the maze-bright offspring were mated with one another, as were the dullest of the maze-dull offspring. This selective breeding procedure was continued for 21 generations (and the descendants of Tryon's original strains are still available today). By the eighth generation, there was almost no overlap in the maze-learning performance of the two strains. With a few exceptions, the worst of the maze-bright strain made fewer errors than the best of the maze-dull strain (see Figure 2.21).

To control for the possibility that good maze-running performance was somehow being passed from parent to offspring through learning, Tryon used a *cross-fostering control procedure*: He tested maze-bright offspring that had been reared by maze-dull parents and maze-dull offspring that had been reared by maze-bright parents. However, the offspring of maze-bright rats made few errors even when they were reared by maze-dull rats, and the offspring of maze-dull rats made many errors even when they were reared by maze-bright rats.

Figure 2.21 Selective breeding of maze-bright and maze-dull strains of rats by Tryon (1934). (Data from Cooper, R. M., & Zubek, J. P. (1958). Effects of enriched and restricted early environments on the learning ability of bright and dull rats. *Canadian Journal of Psychology, 12*, 159–164.)

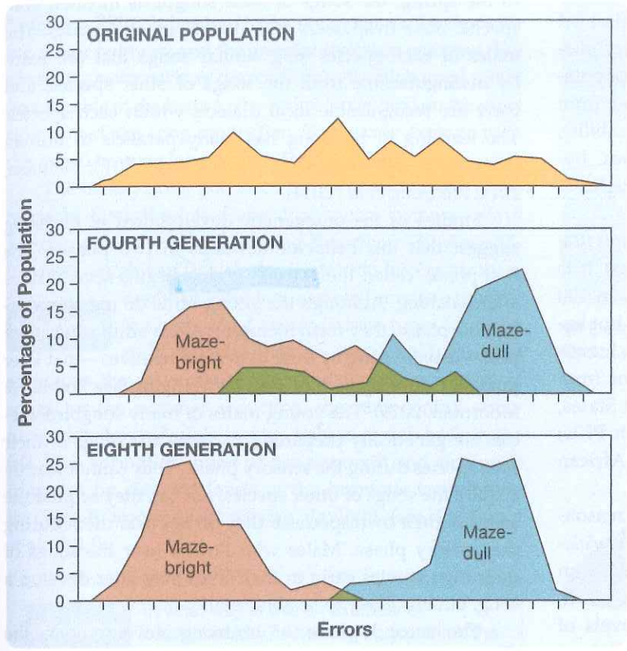
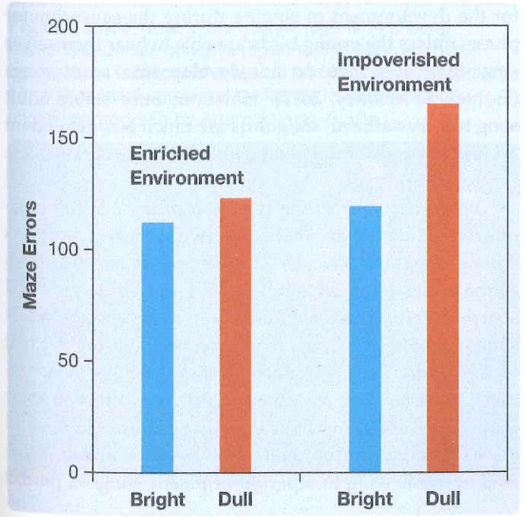


Figure 2.22 Maze-dull rats did not make significantly more errors than maze-bright rats when both groups were reared in an enriched environment. (Adapted from Cooper & Zubek, 1958.)



Since Tryon's seminal selective-breeding experiments, many behavioral traits have been selectively bred. Indeed, it appears that any measurable behavioral trait that varies among members of a species can be selectively bred.

An important general point made by studies of selective breeding is that selective breeding based on one behavioral trait usually brings a host of other behavioral traits along with it. This indicates that the behavioral trait used as the criterion for selective breeding is not the only behavioral trait influenced by the genes segregated by the breeding. Indeed, Searle (1949) compared maze-dull and maze-bright rats on 30 different behavioral tests and found that they differed on many of them. The pattern of differences suggested that the maze-bright rats were superior maze learners not because they were more intelligent but because they were less fearful—a trait that is not adaptive in many natural environments.

Selective-breeding studies have proved that genes influence the development of behavior. This conclusion in no way implies that experience does not. This point was driven home by Cooper and Zubek (1958) in a classic study of maze-bright and maze-dull rats. The researchers reared maze-bright and maze-dull rats in one of two environments: (1) an impoverished environment (a barren wire-mesh group cage) or (2) an enriched environment (a wire-mesh group cage that contained tunnels, ramps, visual displays, and other objects designed to stimulate interest). When the maze-dull rats reached maturity, they made significantly more errors than the maze-bright rats only if they had been reared in the impoverished environment (see Figure 2.22).

Phenylketonuria: A Single-Gene Metabolic Disorder

LO 2.15 Explain how classic research on phenylketonuria (PKU) has informed our understanding of the genetics of behavior.

It is often easier to understand the genetics of a behavioral disorder than it is to understand the genetics of normal behavior. The reason is that many genes influence the development of a normal behavioral trait, but it sometimes takes only one abnormal gene to screw it up.

A good example of this point is the neurological disorder **phenylketonuria (PKU)**.

Clinical Implications

PKU was discovered in 1934 when a Norwegian dentist, Asbjörn Fölling, noticed a peculiar odor in the urine of his two intellectually disabled children. He correctly assumed that the odor was related to their disorder, and he had their urine analyzed. High levels of **phenylpyruvic acid** were found in both samples. Spurred on by his discovery, Fölling identified other intellectually disabled children who had abnormally high levels of urinary phenylpyruvic acid, and he concluded that this subpopulation of intellectually disabled children was suffering from the same disorder. In addition to intellectually disability, the symptoms of PKU include vomiting, seizures, hyperactivity, irritability, and brain damage (Strisciuglio & Concolino, 2014).

The pattern of transmission of PKU through the family trees of afflicted individuals indicates that it is transmitted by a single gene mutation. About 1 in 100 people of European descent carry the PKU gene; but because the gene is recessive, PKU develops only in homozygous individuals (those who inherit a PKU gene from both their mother and their father). In the United States, about 1 in 10,000 Caucasian infants is born with PKU; the incidence is much lower among infants of African Americans.

The biochemistry of PKU turned out to be reasonably straightforward. PKU homozygotes lack *phenylalanine hydroxylase*, an enzyme required for the conversion of the amino acid *phenylalanine* to *tyrosine*. As a result, phenylalanine accumulates in the body; and levels of *dopamine*, a neurotransmitter normally synthesized from tyrosine, are low. The consequence is abnormal brain development.

Like other behavioral traits, the behavioral symptoms of PKU result from an interaction between genetic and environmental factors: between the PKU gene and diet (see Rohde et al., 2014). Accordingly, in most modern hospitals, the blood of newborn infants is routinely screened for high phenylalanine levels (see Casey, 2013). If the level is high, the infant is immediately placed on a special phenylalanine-restricted diet; this diet reduces both the amount of phenylalanine in the blood and the development of intellectual disability—however, it does not prevent the development of subtle cognitive deficits (Moyle et al., 2007; Simon et al., 2008). The timing of this treatment is extremely important. The phenylalanine-restricted diet does not significantly reduce the development of intellectual disability in PKU homozygotes unless it is initiated within the first few weeks of life; conversely, the restriction of phenylalanine in the diet is usually relaxed in late childhood, with few obvious adverse consequences to the patient. The period, usually early in life, during which a particular experience must occur to have a major effect on the development of a trait is the **sensitive period** for that trait.

Development of Birdsong

LO 2.16 Describe how research on the ontogenetic development of birdsong has provided insight into the development of human language.

In the spring, the songs of male songbirds threaten conspecific male trespassers and attract potential mates. The males of each species sing similar songs that are readily distinguishable from the songs of other species, and there are recognizable local dialects within each species. The learning of birdsong has many parallels to human language learning (see Brainard & Doupe, 2013; Elemans, 2014; Pfenning et al., 2014).

Studies of the ontogenetic development of birdsong suggest that this behavior develops in two phases. The first phase, called the **sensory phase**, begins several days after hatching. Although the young birds do not sing during this phase, they form memories of the adult songs they hear—usually sung by their own male relatives—that later guide the development of their own singing (see Bolhuis & Moorman, 2015). The young males of many songbird species are genetically prepared to acquire the songs of their own species during the sensory phase. They cannot readily acquire the songs of other species, nor can they acquire the songs of their own species if they do not hear them during the sensory phase. Males who do not hear the songs of their own species early in their lives may later develop a song, but it is likely to be abnormal.

The second phase of birdsong development, the **sensorimotor phase**, begins when the juvenile males begin to twitter *subsongs* (the immature songs of young birds), usually when they are several months old. During this phase, the rambling vocalizations of subsongs are gradually refined until they resemble the songs of the birds' earlier adult tutors. Auditory feedback is necessary for the development of singing during the sensorimotor phase; unless the young birds are able to hear themselves sing, their subsongs do not develop into adult songs (Tschida & Mooney, 2012). However, once stable adult song has crystallized, songbirds are much less dependent on hearing for normal song production (see Brainard & Doupe, 2013).

When it comes to the retention of their initial crystallized adult songs, there are two common patterns among songbird species. Most songbird species, such as the widely studied zebra finches and white-crowned sparrows, are *age-limited learners*; in these species, adult songs, once crystallized, remain unchanged for the rest of the birds' lives. In contrast, some species are *open-ended learners*; they are able to add new songs to their repertoire throughout their lives. For example, at the end of each mating season, male canaries return from a period of stable song to a period of plastic song—a period during which they can add new songs for the next mating season. Male zebra finches (age-limited learners) and male canaries (open-ended learners) are shown in Figure 2.23.

Figure 2.24 is a simplified version of the neural circuit that controls birdsong in the canary (see Hahnloser & Kotowicz, 2010). It has two major components: the descending motor pathway and the anterior forebrain pathway. The *descending motor pathway* descends from the high vocal center on each side of the brain to the syrinx (voice box) on the same side; it mediates song production. The *anterior forebrain pathway* mediates song learning (see Prather, 2013).

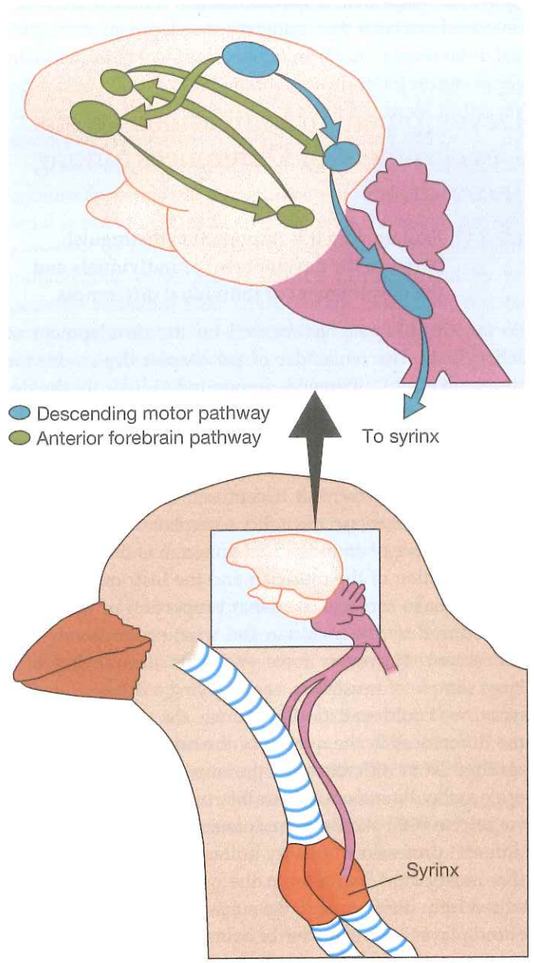
The canary song neural circuit is remarkable in three respects. First, the left descending motor pathway plays a more important role in singing than the right descending motor pathway (which duplicates the left-hemisphere dominance for language in humans). Second, the high vocal center is four times larger in male canaries than in females. Third, each spring, as the male canary prepares its new repertoire of songs for the summer seduction, the song-control structures of its brain double in size, only to shrink back in the fall. This springtime burst of brain growth and singing is triggered by elevated levels of the hormone testosterone that result from the increasing daylight (see De Groof et al., 2013).

Neuroplasticity

Neuroplasticity In what way is this an example of neuroplasticity? And why do you think the song-control structures wouldn't just stay consistently enlarged, rather than demonstrate seasonal variation in their size?

Figure 2.23 Male zebra finches (age-limited song learners) and male canaries (open-ended song learners) are common subjects of research on birdsong development. (Illustration kindly provided by Trends in Neuroscience; original photograph by Arturo Alvarez-Buylla.)

Figure 2.24 The neural pathways responsible for the production and learning of song in the male canary.



Genetics of Human Psychological Differences

This chapter has focused on three topics: human evolution, genetics, and the interaction of genetics and experience through epigenetic mechanisms. All three topics converge on one fundamental question: Why are we the way we are?

You have learned that each of us is a product of gene–experience interactions and that the effects of genes and experience on individual development are inseparable. This

final module of the chapter continues to look at the effects of gene–experience interactions, but it focuses on a developmental issue that is fundamentally different from the ones we have been discussing: the development of individual differences rather than the development of individuals.

Development of Individuals versus Development of Differences among Individuals

LO 2.17 Explain why it is important to distinguish between the development of individuals and the development of individual differences.

So far, this chapter has focused on the development of individuals. The remainder of the chapter deals with the development of differences among individuals. In the development of individuals, the effects of genes and experience are inseparable. In the development of differences among individuals, they are separable. This distinction is extremely important, but it confuses many people. Let's return to the musician metaphor to explain it.

The music of an individual musician is the product of the interaction of the musician and the instrument, and it doesn't make sense to ask what proportion of the music is produced by the musician and what proportion by the instrument. However, if we evaluated the playing of a large sample of musicians, each playing a different instrument, we could statistically estimate the degree to which the differences in the quality of the music they produced resulted from differences in the musicians themselves as opposed to differences in their instruments. For example, if we selected 100 people at random and had each one play a different professional-quality guitar, we would likely find that most of the variation in the quality of the music resulted from differences in the subjects, some being experienced players and some never having played before. In the same way, researchers can select a group of volunteers and ask what proportion of the variation among them in some attribute (e.g., intelligence) results from genetic differences as opposed to experiential differences.

To assess the relative contributions of genes and experience to the development of differences in psychological attributes, behavioral geneticists study individuals of known genetic similarity. For example, they often compare **monozygotic twins**, who developed from the same zygote and thus are genetically similar, with **dizygotic twins**, who developed from two zygotes and thus are no more similar than any pair of *siblings* (brothers and sisters). Studies of pairs of monozygotic and dizygotic twins who have been separated at infancy by adoption are particularly informative about the relative contributions of genetics and experience to differences in human psychological development. The most extensive of such adoption studies is the Minnesota Study of Twins Reared Apart (see Bouchard & Pedersen, 1998).

Watch this video on MyPsychLab

TWINS AND PERSONALITY

Heritability Estimates: Minnesota Study of Twins Reared Apart

LO 2.18 Explain heritability estimates and how they are commonly misinterpreted.

The Minnesota Study of Twins Reared Apart involved 59 pairs of monozygotic twins and 47 pairs of dizygotic twins who had been reared apart as well as many pairs of monozygotic and dizygotic twins who had been reared together. Their ages ranged from 19 to 68. Each twin was brought to the University of Minnesota for approximately 50 hours of testing, which focused on the assessment of intelligence and personality. Would the adult monozygotic twins reared apart prove to be similar because they were genetically similar, or would they prove to be different because they had been brought up in different environments?

The results of the Minnesota Study of Twins Reared Apart proved to be remarkably consistent—both internally, between the various cognitive and personality dimensions that were studied, and externally, with the findings of other studies. In general, adult monozygotic twins were substantially more similar to one another on all psychological dimensions than were adult dizygotic twins, whether or not both twins of a pair were raised in the same family environment (see Turkheimer, 2000).

In order to quantify the contributions of genetic variations in a particular study, researchers calculate heritability estimates. A **heritability estimate** is not about individual development; it is a numerical estimate of the proportion of variability that occurred in a particular trait in a particular study as a result of the genetic variation in that study (see Turkheimer, Pettersson, & Horn, 2014). Heritability estimates tell us about the contribution of genetic differences to phenotypic differences among the participants in a study; they have nothing to say about the relative contributions of genes and experience to the development of individuals.

The concept of heritability estimates can be quite confusing. We suggest that you pause here and carefully think about the definition before proceeding. The musician metaphor may help you.

Thinking Creatively

The magnitude of a study's heritability estimate depends on the amount of genetic and environmental variation from which it was calculated, and it cannot be applied to other kinds of situations. For example, in the Minnesota study, there was relatively little environmental variation. All participants were raised in industrialized countries (Great Britain, Canada, and the United States) by parents who met the strict standards required for adoption. Accordingly, most of the variation in the subjects' intelligence and personality resulted from genetic variation. If the twins had been separately adopted by European royalty, Los Angeles rap stars, London advertising executives, and Inuit, the resulting heritability estimates for IQ and personality would likely have been much lower.

Now that you understand the meaning of heritability estimates, let us tell you how big they tend to be for a variety of complex human traits and behaviors: for example, for intelligence, personality traits, aggression, divorce, religious beliefs, sports participation, psychiatric disorders, and television watching. The answer is simple because heritability estimates tend to be about the same regardless of the particular trait or behavior under consideration and regardless of the particular basis used to calculate them (i.e., twin, adoption, or family-tree studies). In the representative Western samples that have been studied, all complex traits and behaviors have substantial heritability estimates—most between 40 and 80 percent.

The discovery that genetic variability contributes substantially to individual differences in virtually all human traits and behaviors has led several eminent geneticists to argue that no more heritability estimate studies should be conducted (e.g., Johnson et al., 2009; Petronis, 2010). What could more heritability estimate studies possibly add? These geneticists are, however, excited about the potential of two other types of twin studies that have recently been reported. The chapter ends with them.

A Look into the Future: Two Kinds of Twin Studies

LO 2.19 Describe two ways that twin studies can be used to study the interaction of genes and experience (i.e., nature and nurture).

Two lines of research on twins have recently created considerable excitement among geneticists and other scholars. We hope you share their enthusiasm.

TWIN STUDIES OF EPIGENETIC EFFECTS. Most studies of epigenetic effects have focused on nonhuman species. In plants and nonhuman animals, it is quite clear that epigenetic changes can be triggered by experience, can last a lifetime, and can be passed on to future generations (see Szyf, 2014). To what extent do these amazing results apply to humans? Twin studies may provide a route to the answers (see Aguilera et al., 2010; Feil & Fraga, 2012).

The study of epigenetic effects in humans is difficult because experimental manipulation of human genetic material is not ethical. Monozygotic twins, however, provide a method of circumventing this difficulty. At conception monozygotic twins are genetically identical, and by repeatedly assessing their DNA one can document the development and survival of the many epigenetic differences that develop between them (see Bell & Saffery, 2012; Bell & Spector, 2011; Chatterjee & Morison, 2011; Silva et al., 2011). Moreover, by comparing monozygotic and dizygotic twins, it is possible to get a sense of the degree to which changes are caused by experiential as opposed to genetic factors—if epigenetic changes developed under genetic control, one would expect that the pattern of epigenetic changes would be more similar in monozygotic than dizygotic pairs.

The first systematic demonstration of epigenetic differences in human twins was published by Fraga and colleagues (2005). They took tissue samples (blood, skin, muscle) from 40 pairs of monozygotic twins, ranging in age from 3 to 74. Then, they screened the tissues for DNA methylation and histone modifications. They found that the twins were epigenetically indistinguishable early in life, but differences accumulated as they aged, each type of tissue displaying a different epigenetic profile (see Zong et al., 2012). As a result, the former assumption that monozygotic twins are genetically identical was disproven, and the common practice of referring to monozygotic twins as *identical twins* should be curtailed.

In another study of epigenetic changes in twins, Wong and colleagues (2010) examined DNA methylation in *buccal cells* (cells of the lining of the mouth) scraped from 46 pairs of monozygotic twins and 45 pairs of dizygotic twins. They took samples from the twins at age 5 and again from the same twins at age 10. Then they assessed DNA methylation. Wong and colleagues found DNA methylation to be prominent in both groups of twins at both ages. Because the concordance rates of DNA methylation were the same between monozygotic and between dizygotic twins, they concluded that differences in DNA methylation are mainly a consequence of experiential factors.

The discovery of epigenetic differences in monozygotic twins raises the possibility that epigenetic differences may explain why one twin develops a disease and the other doesn't

Figure 2.25 Epigenetic research suggests that the common practice of referring to monozygotic twins as 'identical twins' should be curtailed.

(Bell & Spector, 2011; Haque, Gottesman, & Wong, 2009). Once identified, such epigenetic differences would provide important clues to the cause and mechanisms of the disease. Bell and Spector (2011) suggest that *disease-discordant monozygotic twin studies* are a particularly powerful approach (see also Czyz et al., 2012). This kind of study begins with the identification of monozygotic twins who are discordant for the disease of interest. Then one searches each pair for epigenetic differences focusing on those areas of DNA that are thought to be involved in the disorder. Large-scale studies in monozygotic twins across different ages, tissues, and epigenetic effects could greatly improve our understanding of human disease (see Bell & Spector, 2011; Tan et al., 2014).

Clinical Implications

TWIN STUDIES OF THE EFFECTS OF EXPERIENCE ON HERITABILITY. In thinking about heritability estimates, it is paramount to remember that heritability estimates depend on the particular conditions and subjects of a particular study. This point was driven home by the influential study of Turkheimer and colleagues (2003). Before the Turkheimer et al. study, all published studies of the heritability of intelligence were conducted on middle- to upper-class families, and the heritability estimates for intelligence tended to be about 75 percent.

Turkheimer and colleagues assessed heritability of intelligence in two samples of 7-year-old twins: those from families of low socioeconomic status (SES) and those from families of middle to high SES. The heritability estimates for intelligence in the middle- to high-SES twins was, as expected, about 70 percent. However, the heritability estimate for intelligence in the twins from low-SES families was only 10 percent. This effect was subsequently replicated and extended to other age groups: babies (Tucker-Drob et al., 2010) and adolescents (Harden, Turkheimer, & Loehlin, 2007).

One major implication of this finding is that it forces one to think of intelligence as developing from the interaction of genes and experience, not from one or the other. It seems that one can inherit the potential to be of superior intelligence, but this potential is rarely realized in a poverty-stricken environment (see Loehllin, Harden, & Turkheimer, 2009; Nisbett et al., 2012).

Thinking Creatively

This finding also has important implications for the development of programs to help the poor. Many politicians have argued against special programs for the poor because most heritability estimates of intelligence are high. They incorrectly argue that because intelligence is inherited, special programs for the poor are a waste of money. However, the findings of Turkheimer and colleagues suggest otherwise: Reducing poverty would permit many of the poor to develop their genetic potential.

Thinking Creatively

> Thinking Creatively — Do you think that reducing poverty would improve educational achievements? Why or why not?

Themes Revisited

This chapter introduced the topics of evolution, genetics, and development, but its unifying focus was thinking creatively about the biology of behavior. Not surprisingly, then, of this text's four major themes, the thinking creatively theme received the most attention. This chapter challenged you to think about important biopsychological phenomena in new ways. Thinking creatively tabs marked points in the chapter where you were encouraged to sharpen your thinking about the nature–nurture issue, the physiological-or-psychological dichotomy, human evolution, the biopsychological implications of the Human Genome Project, the implications of epigenetics, the genetics of human psychological differences, the meaning of heritability estimates, and the important study of Turkheimer and colleagues.

Thinking Creatively

The other three themes also received coverage in this chapter, and each case was marked by the appropriate tab. The evolutionary perspective was illustrated by comparative research on self-awareness in chimps, by consideration of the evolutionary significance of social dominance and courtship displays, and by efforts to understand mate bonding.

Evolutionary Perspective

The clinical implications theme was illustrated by the case of the man who fell out of bed, the discussion of phenylketonuria (PKU), and the discussion of disease-discordant twin studies. The neuroplasticity theme arose when you learned that brain growth occurs in male songbirds prior to each breeding season.

Clinical Implications

Neuroplasticity

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