1. Human Genetics

Learning Objectives

By the end of this section, you will be able to:

- Explain the basic principles of the theory of evolution by natural selection
- Describe the differences between genotype and phenotype
- Discuss how gene-environment interactions are critical for expression of physical and psychological characteristics

Psychological researchers study genetics in order to better understand the biological factors that contribute to certain behaviors. While all humans share certain biological mechanisms, we are each unique. And while our bodies have many of the same parts—brains and hormones and cells with genetic codes—these are expressed in a wide variety of behaviors, thoughts, and reactions.

Why do two people infected by the same disease have different outcomes: one surviving and one succumbing to the ailment? How are genetic diseases passed through family lines? Are there genetic components to psychological disorders, such as depression or schizophrenia? To what extent might there be a psychological basis to health conditions such as childhood obesity?

To explore these questions, let's start by focusing on a specific genetic disorder, sickle cell anemia, and how it might manifest in two affected sisters. Sickle-cell anemia is a genetic condition in which red blood cells, which are normally round, take on a crescent-like shape (Figure 3.2). The changed shape of these cells affects how they function: sickle-shaped cells can clog blood vessels and block blood flow, leading to high fever, severe pain, swelling, and tissue damage.

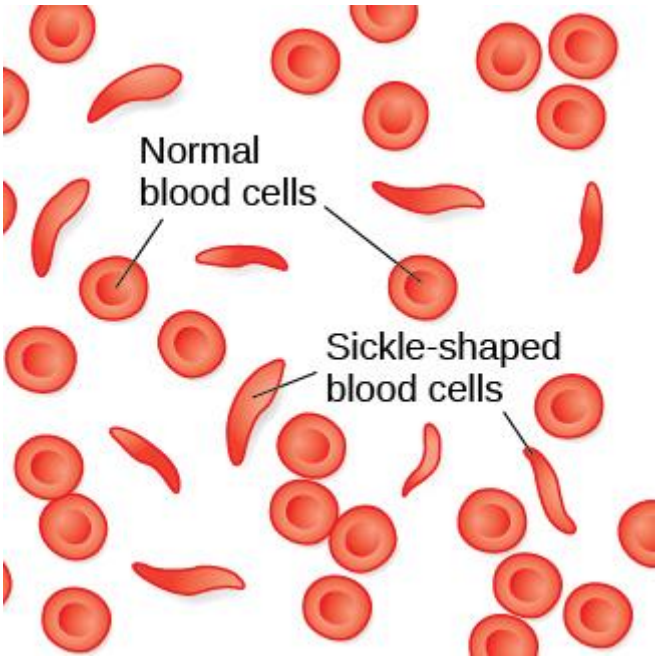


Figure 3.2 Normal blood cells travel freely through the blood vessels, while sickle-shaped cells form blockages preventing blood flow.

Many people with sickle-cell anemia—and the particular genetic mutation that causes it—die at an early age. While the notion of “survival of the fittest” may suggest that people with this disorder have a low survival rate and therefore the disorder will become less common, this is not the case. Despite the negative evolutionary effects associated with this genetic mutation, the sickle-cell gene remains relatively common among people of African descent. Why is this? The explanation is illustrated with the following scenario.

Imagine two young women—Luwi and Sena—sisters in rural Zambia, Africa. Luwi carries the gene for sickle-cell anemia; Sena does not carry the gene. Sickle-cell carriers have one copy of the sickle-cell gene but do not have full-blown sickle-cell anemia. They experience symptoms only if they are severely dehydrated or are deprived of oxygen (as in mountain climbing). Carriers are thought to be immune to malaria (an often deadly disease that is widespread in tropical climates) because changes in their blood chemistry and immune functioning prevent the malaria parasite from having its effects (Gong, Parikh, Rosenthal, & Greenhouse, 2013). However, full-blown sickle-cell anemia, with two copies of the sickle-cell gene, does not provide immunity to malaria.

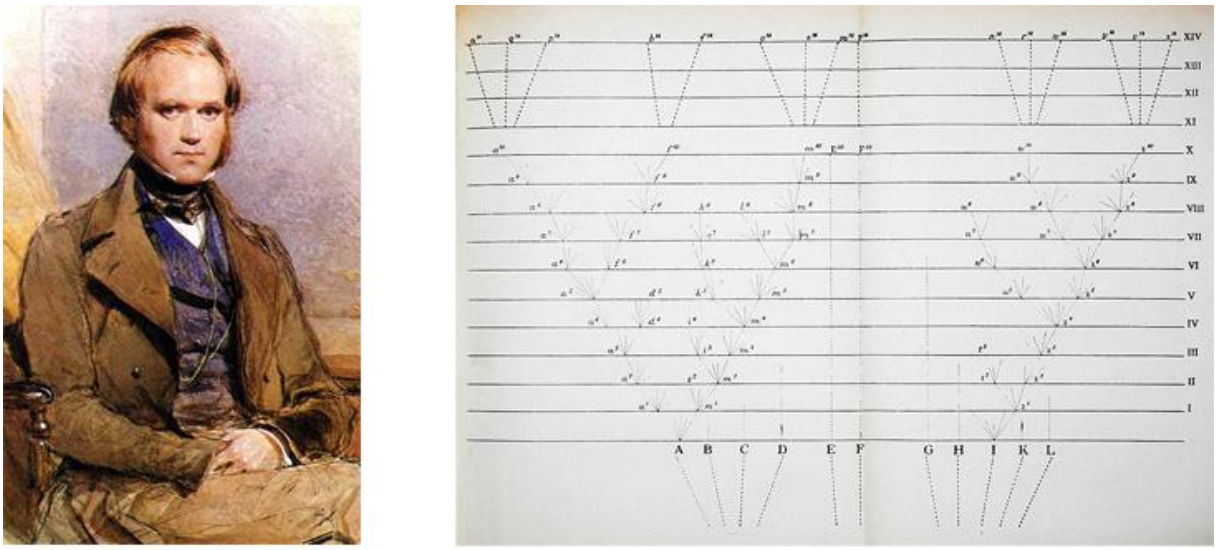
While walking home from school, both sisters are bitten by mosquitoes carrying the malaria parasite. Luwi is protected against malaria because she carries the sickle-cell mutation. Sena, on the other hand, develops malaria and dies just two weeks later. Luwi survives and eventually has children, to whom she may pass on the sickle-cell mutation.

LINK TO LEARNING

Visit this website about how a mutation in DNA leads to sickle cell anemia to learn more.

Malaria is rare in the United States, so the sickle-cell gene benefits nobody: the gene manifests primarily in minor health problems for carriers with one copy, or a severe full-blown disease with no health benefits for carriers with two copies. However, the situation is quite different in other parts of the world. In parts of Africa where malaria is prevalent, having the sickle-cell mutation does provide health benefits for carriers (protection from malaria).

The story of malaria fits with Charles Darwin's **theory of evolution by natural selection** (Figure 3.3). In simple terms, the theory states that organisms that are better suited for their environment will survive and reproduce, while those that are poorly suited for their environment will die off. In our example, we can see that, as a carrier, Luwi’s mutation is highly adaptive in her African homeland; however, if she resided in the United States (where malaria is rare), her mutation could prove costly—with a high probability of the disease in her descendants and minor health problems of her own.



(a) (b)

Figure 3.3 (a) In 1859, Charles Darwin proposed his theory of evolution by natural selection in his book, *On the Origin of Species*. (b) The book contains just one illustration: this diagram that shows how species evolve over time through natural selection.

DIG DEEPER

Two Perspectives on Genetics and Behavior

It's easy to get confused about two fields that study the interaction of genes and the environment, such as the fields of evolutionary psychology and behavioral genetics. How can we tell them apart?

In both fields, it is understood that genes not only code for particular traits, but also contribute to certain patterns of cognition and behavior. Evolutionary psychology focuses on how universal patterns of behavior and cognitive processes have evolved over time. Therefore, variations in cognition and behavior would make individuals more or less successful in reproducing and passing those genes on to their offspring. Evolutionary psychologists study a variety of psychological phenomena that may have evolved as adaptations, including fear response, food preferences, mate selection, and cooperative behaviors (Confer et al., 2010).

Whereas evolutionary psychologists focus on universal patterns that evolved over millions of years, behavioral geneticists study how individual differences arise, in the present, through the interaction of genes and the environment. When studying human behavior, behavioral geneticists often employ twin and adoption studies to research questions of interest. Twin studies compare the likelihood that a given behavioral trait is shared among identical and fraternal twins; adoption studies compare those rates among biologically related relatives and adopted relatives. Both approaches provide some insight into the relative importance of genes and environment for the expression of a given trait.

LINK TO LEARNING

Watch this interview with renowned evolutionary psychologist David Buss to learn more about how a psychologist approaches evolution and how this approach fits within the social sciences.

Genetic Variation

Genetic variation, the genetic difference between individuals, is what contributes to a species' adaptation to its environment. In humans, genetic variation begins with an egg, about 100 million sperm, and fertilization. Roughly once per month, active ovaries release an egg from follicles. During the egg's journey from the ovary through the fallopian tubes, to the uterus, a sperm may fertilize the egg.

The egg and the sperm each contain 23 chromosomes. **Chromosomes** are long strings of genetic material known as **deoxyribonucleic acid (DNA)**. DNA is a helix-shaped molecule made up of nucleotide base pairs. In each chromosome, sequences of DNA make up **genes** that control or partially control a number of visible characteristics, known as traits, such as eye color, hair color, and so on. A single gene may have multiple possible variations, or alleles. An **allele** is a specific version of a gene. So, a given gene may code for the trait of hair color, and the different alleles of that gene affect which hair color an individual has.

When a sperm and egg fuse, their 23 chromosomes combine to create a zygote with 46 chromosomes (23 pairs). Therefore, each parent contributes half the genetic information carried by the offspring; the resulting physical characteristics of the offspring (called the phenotype) are determined by the interaction of genetic material supplied by the sperm and egg (called the genotype). A person's **genotype** is the genetic makeup of that individual. **Phenotype**, on the other hand, refers to the individual's inherited physical characteristics, which are a combination of genetic and environmental influences (Figure 3.4).

(a) (b)

Figure 3.4 (a) Genotype refers to the genetic makeup of an individual based on the genetic material (DNA) inherited from one's genetic contributors. (b) Phenotype describes an individual's observable characteristics, such as hair color, skin color, height, and build. (credit a: modification of work by Caroline Davis; credit b: modification of work by Cory Zanker)

Note that, in genetics and reproduction, "parent" is often used to describe the individual organisms that contribute genetic material to offspring, usually in the form of gamete cells

(sperm and egg). The concept of a genetic parent is distinct from social and legal concepts of parenthood, and may differ from those whom people consider their parents.

Most traits are controlled by multiple genes, but some traits are controlled by one gene. A characteristic like cleft chin, for example, is influenced by a single gene from each parent. In this example, we will call the gene for cleft chin "B," and the gene for smooth chin "b." Cleft chin is a dominant trait, which means that having the **dominant allele** either from one parent (Bb) or both parents (BB) will always result in the phenotype associated with the dominant allele. When someone has two copies of the same allele, they are said to be **homozygous** for that allele. When someone has a combination of alleles for a given gene, they are said to be **heterozygous**. For example, smooth chin is a recessive trait, which means that an individual will only display the smooth chin phenotype if they are homozygous for that **recessive allele** (bb).

Imagine that a person with a cleft chin mates with a person with a smooth chin. What type of chin will their offspring have? The answer to that depends on which alleles each parent carries. If the person with a cleft is homozygous for cleft chin (BB), their offspring will always have cleft chin. It gets a little more complicated, however, if the person is heterozygous for this gene (Bb). Since the other person has a smooth chin—therefore homozygous for the recessive allele (bb)—we can expect the offspring to have a 50% chance of having a cleft chin and a 50% chance of having a smooth chin (Figure 3.5).

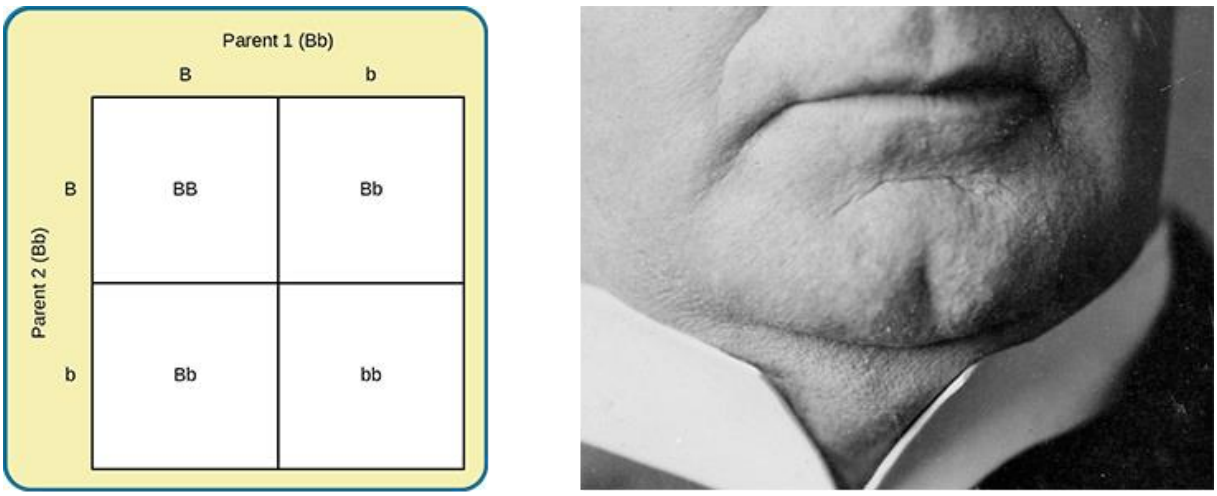


Figure 3.5 (a) A Punnett square is a tool used to predict how genes will interact in the production of offspring. The capital B represents the dominant allele, and the lowercase b represents the recessive allele. In the example of the cleft chin, where B is cleft chin (dominant allele), wherever a pair contains the dominant allele, B, you can expect a cleft chin phenotype. You can expect a smooth chin phenotype only when there are two copies of the recessive allele, bb. (b) A cleft chin, shown here, is an inherited trait.

In sickle cell anemia, heterozygous carriers (like Luwi from the example) can develop blood resistance to malaria infection while those who are homozygous (like Sena) have a potentially lethal blood disorder. Sickle-cell anemia is just one of many genetic disorders caused by the pairing of two recessive genes. For example, phenylketonuria (PKU) is a condition in which individuals lack an enzyme that normally converts harmful amino acids into harmless

byproducts. If someone with this condition goes untreated, they will experience significant deficits in cognitive function, seizures, and an increased risk of various psychiatric disorders. Because PKU is a recessive trait, each parent must have at least one copy of the recessive allele in order to produce a child with the condition (Figure 3.6).

So far, we have discussed traits that involve just one gene, but few human characteristics are controlled by a single gene. Most traits are **polygenic**: controlled by more than one gene. Height is one example of a polygenic trait, as are skin color and weight.

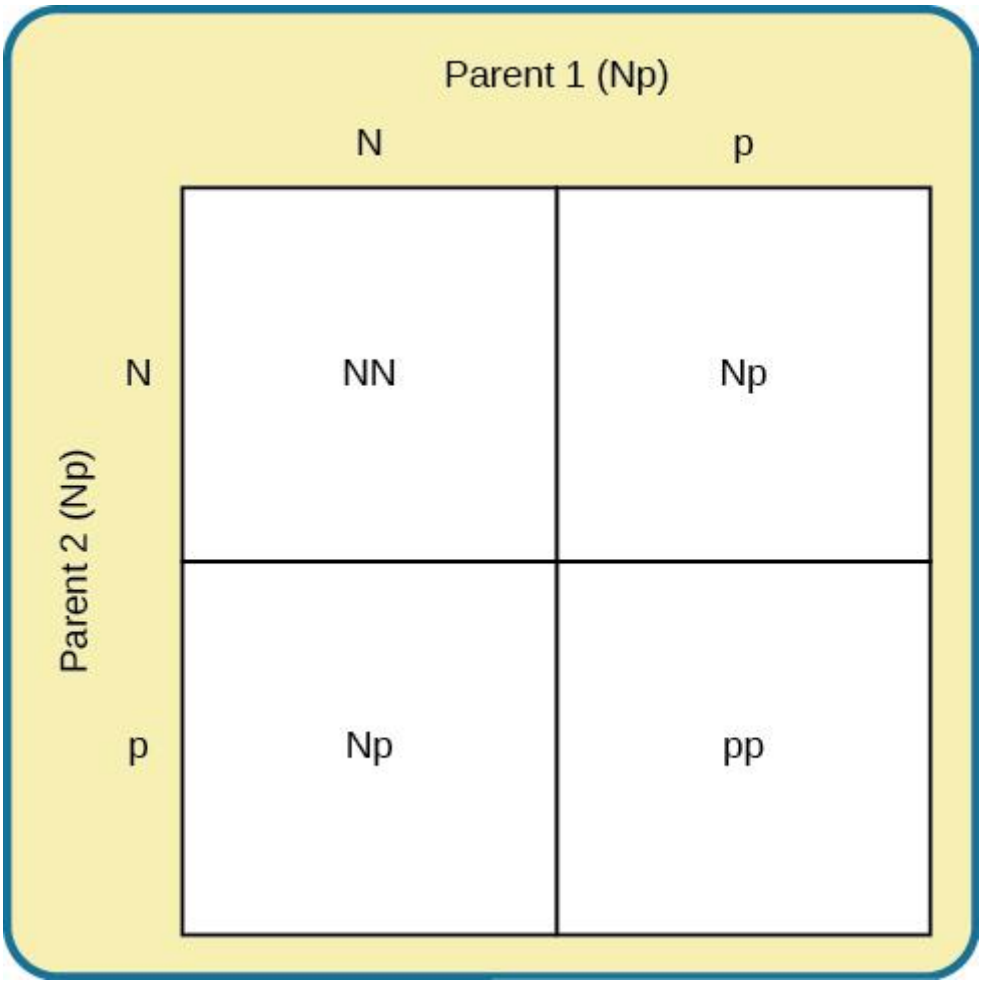


Figure 3.6 In this Punnett square, N represents the normal allele, and p represents the recessive allele that is associated with PKU. If two individuals mate who are both heterozygous for the allele associated with PKU, their offspring have a 25% chance of expressing the PKU phenotype.

Where do harmful genes that contribute to diseases like PKU come from? Gene mutations provide one source of harmful genes. A **mutation** is a sudden, permanent change in a gene. While many mutations can be harmful or lethal, once in a while, a mutation benefits an individual by giving that person an advantage over those who do not have the mutation. Recall that the theory of evolution asserts that individuals best adapted to their particular environments are more likely to reproduce and pass on their genes to future generations. In order for this process to occur, there must be competition—more technically, there must be variability in genes (and resultant traits) that allow for variation in adaptability to the environment. If a population consisted of identical individuals, then any dramatic changes in the environment would affect everyone in the same way, and there would be no variation in

selection. In contrast, diversity in genes and associated traits allows some individuals to perform slightly better than others when faced with environmental change. This creates a distinct advantage for individuals best suited for their environments in terms of successful reproduction and genetic transmission.

DIG DEEPER

Human Diversity

This chapter focuses on biology. Later in this course you will learn about social psychology and issues of race, prejudice, and discrimination. When we focus strictly on biology, race becomes a weak construct. After the sequencing of the human genome at the turn of the millennium, many scientists began to argue that race was not a useful variable in genetic research and that its continued use represents a potential source of confusion and harm. The racial categories that some believed to be helpful in studying genetic diversity in humans are largely irrelevant. A person's skin tone, eye color, and hair texture are functions of their genetic makeups, but there is actually more genetic variation within a given racial category than there is between racial categories. In some cases, focus on race has led to difficulties with misdiagnoses and/or under-diagnoses of diseases ranging from sickle cell anemia to cystic fibrosis. Some argue that we need to distinguish between ancestry and race and then focus on ancestry. This approach would facilitate greater understanding of human genetic diversity (Yudell, Roberts, DeSalle, & Tishkoff, 2016).

Gene-Environment Interactions

Genes do not exist in a vacuum. Although we are all biological organisms, we also exist in an environment that is incredibly important in determining not only when and how our genes express themselves, but also in what combination. Each of us represents a unique interaction between our genetic makeup and our environment; range of reaction is one way to describe this interaction. **Range of reaction** asserts that our genes set the boundaries within which we can operate, and our environment interacts with the genes to determine where in that range we will fall. For example, if an individual's genetic makeup predisposes them to high levels of intellectual potential and they are reared in a rich, stimulating environment, then they will be more likely to achieve full potential than if they were raised under conditions of significant deprivation. According to the concept of range of reaction, genes set definite limits on potential, and environment determines how much of that potential is achieved. Some disagree with this theory and argue that genes do not set a limit on a person's potential with reaction norms being determined by the environment. For example, when individuals experience neglect or abuse early in life, they are more likely to exhibit adverse psychological and/or physical conditions that can last throughout their lives. These conditions may develop as a function of the negative environmental experiences in individuals from dissimilar genetic backgrounds (Miguel, Pereira, Silveira, & Meaney, 2019; Short & Baram, 2019).

Another perspective on the interaction between genes and the environment is the concept of **genetic environmental correlation**. Stated simply, our genes influence our environment, and our environment influences the expression of our genes (Figure 3.7). Not only do our genes and environment interact, as in range of reaction, but they also influence one another bidirectionally. For example, the child of an NBA player would probably be exposed to basketball from an early age. Such exposure might allow the child to realize their full genetic, athletic potential. Thus, the parents' genes, which the child shares, influence the child's

environment, and that environment, in turn, is well suited to support the child's genetic potential.

Figure 3.7 Nature and nurture work together like complex pieces of a human puzzle. The interaction of our environment and genes makes us the individuals we are. (credit "puzzle": modification of work by Cory Zanker)

In another approach to gene-environment interactions, the field of **epigenetics** looks beyond the genotype itself and studies how the same genotype can be expressed in different ways. In other words, researchers study how the same genotype can lead to very different phenotypes. As mentioned earlier, gene expression is often influenced by environmental context in ways that are not entirely obvious. For instance, identical twins share the same genetic information (**identical twins** develop from a single fertilized egg that split, so the genetic material is exactly the same in each; in contrast, **fraternal twins** usually result from two different eggs fertilized by different sperm, so the genetic material varies as with non-twin siblings). But even with identical genes, there remains an incredible amount of variability in how gene expression can unfold over the course of each twin's life. Sometimes, one twin will develop a disease and the other will not. In one example, Aliya, an identical twin, died from cancer at age 7, but her twin, now 19 years old, has never had cancer. Although these individuals share an identical genotype, their phenotypes differ as a result of how that genetic information is expressed over time and through their unique environmental interactions. The epigenetic perspective is very different from range of reaction, because here the genotype is not fixed and limited.

LINK TO LEARNING

Watch this video about the epigenetics of twin studies to learn more.

Genes affect more than our physical characteristics. Indeed, scientists have found genetic linkages to a number of behavioral characteristics, ranging from basic personality traits to sexual orientation to spirituality (for examples, see Mustanski et al., 2005; Comings,

Gonzales, Saucier, Johnson, & MacMurray, 2000). Genes are also associated with temperament and a number of psychological disorders, such as depression and schizophrenia. So while it is true that genes provide the biological blueprints for our cells, tissues, organs, and body, they also have a significant impact on our experiences and our behaviors.

Let's look at the following findings regarding schizophrenia in light of our three views of gene-environment interactions. Which view do you think best explains this evidence?

In a 2004 study by Tienari and colleagues, adoptees whose biological mothers had schizophrenia *and* who had been raised in a disturbed family environment were much more likely to develop schizophrenia or another psychotic disorder than were any of the other groups in the study:

- Of adoptees whose biological mothers had schizophrenia (high genetic risk) and who were raised in disturbed family environments, 36.8% were likely to develop schizophrenia.
- Of adoptees whose biological mothers had schizophrenia (high genetic risk) and who were raised in healthy family environments, 5.8% were likely to develop schizophrenia.
- Of adoptees with a low genetic risk (whose mothers did not have schizophrenia) and who were raised in disturbed family environments, 5.3% were likely to develop schizophrenia.
- Of adoptees with a low genetic risk (whose mothers did not have schizophrenia) and who were raised in healthy family environments, 4.8% were likely to develop schizophrenia.

The study shows that adoptees with high genetic risk were most likely to develop schizophrenia if they were raised in disturbed home environments. This research lends credibility to the notion that both genetic vulnerability and environmental stress are necessary for schizophrenia to develop, and that genes alone do not tell the full tale.

2. Cells of the nervous system

Learning Objectives

By the end of this section, you will be able to:

- Identify the basic parts of a neuron
- Describe how neurons communicate with each other
- Explain how drugs act as agonists or antagonists for a given neurotransmitter system

Psychologists striving to understand the human mind may study the nervous system. Learning how the body's cells and organs function can help us understand the biological basis of human psychology. The **nervous system** is composed of two basic cell types: glial cells (also known as glia) and neurons. Glial cells are traditionally thought to play a supportive role to neurons, both physically and metabolically. **Glial cells** provide scaffolding on which the nervous system is built, help neurons line up closely with each other to allow neuronal communication, provide insulation to neurons, transport nutrients and waste products, and mediate immune responses. For years, researchers believed that there were many more glial cells than neurons; however, more recent work from Suzanna Herculano-Houzel's laboratory has called this long-standing assumption into question and has provided important evidence that there may be a nearly 1:1 ratio of glia cells to neurons. This is important because it suggests that human brains are more similar to other primate brains than previously thought (Azevedo et al, 2009; Herculano-Houzel, 2012;

Herculano-Houzel, 2009). **Neurons**, on the other hand, serve as interconnected information processors that are essential for all of the tasks of the nervous system. This section briefly describes the structure and function of neurons.

Neuron Structure

Neurons are the central building blocks of the nervous system, 100 billion strong at birth. Like all cells, neurons consist of several different parts, each serving a specialized function (Figure 3.8). A neuron's outer surface is made up of a **semipermeable membrane**. This membrane allows smaller molecules and molecules without an electrical charge to pass through it, while stopping larger or highly charged molecules.

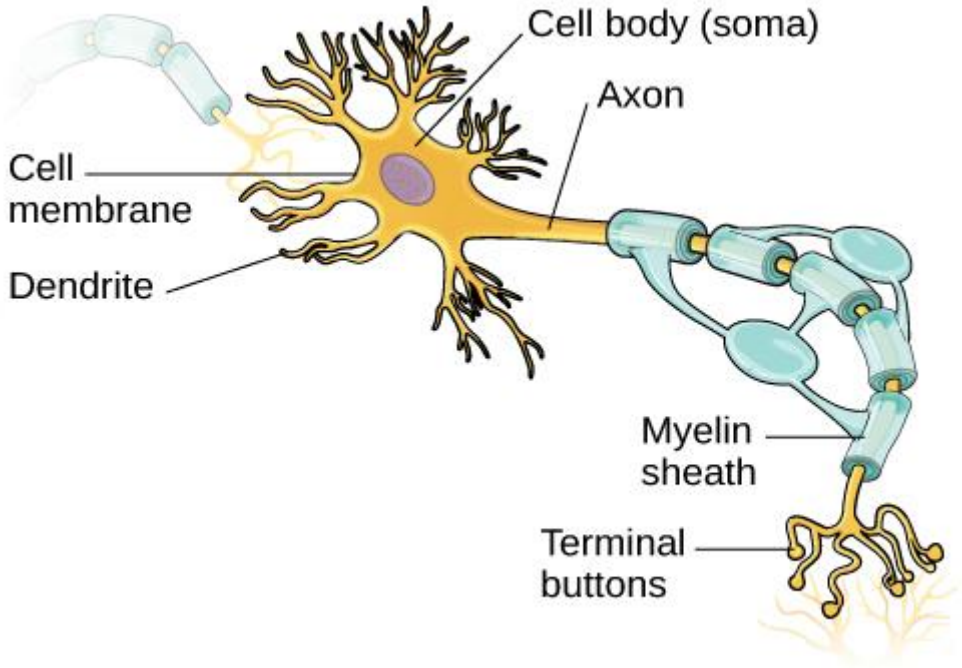


Figure 3.8 This illustration shows a prototypical neuron, which is being myelinated by a glial cell.

The nucleus of the neuron is located in the **soma**, or cell body. The soma has branching extensions known as **dendrites**. The neuron is a small information processor, and dendrites serve as input sites where signals are received from other neurons. These signals are transmitted electrically across the soma and down a major extension from the soma known as the **axon**, which ends at multiple **terminal buttons**. The terminal buttons contain **synaptic vesicles** that house **neurotransmitters**, the chemical messengers of the nervous system.

Axons range in length from a fraction of an inch to several feet. In some axons, glial cells form a fatty substance known as the **myelin sheath**, which coats the axon and acts as an insulator, increasing the speed at which the signal travels. The myelin sheath is not continuous and there are small gaps that occur down the length of the axon. These gaps in the myelin sheath are known as the **Nodes of Ranvier**. The myelin sheath is crucial for the normal operation of the neurons within the nervous system: the loss of the insulation it provides can be detrimental to normal function. To understand how this works, let's consider an example. PKU, a genetic disorder discussed earlier, causes a reduction in myelin and abnormalities in white matter cortical and subcortical structures. The disorder is associated with a variety of issues including severe cognitive deficits, exaggerated reflexes, and seizures (Anderson &

Leuzzi, 2010; Huttenlocher, 2000). Another disorder, multiple sclerosis (MS), an autoimmune disorder, involves a large-scale loss of the myelin sheath on axons throughout the nervous system. The resulting interference in the electrical signal prevents the quick transmittal of information by neurons and can lead to a number of symptoms, such as dizziness, fatigue, loss of motor control, and sexual dysfunction. While some treatments may help to modify the course of the disease and manage certain symptoms, there is currently no known cure for multiple sclerosis.

In healthy individuals, the neuronal signal moves rapidly down the axon to the terminal buttons, where synaptic vesicles release neurotransmitters into the synaptic cleft (Figure 3.9). The **synaptic cleft** is a very small space between two neurons and is an important site where communication between neurons occurs. Once neurotransmitters are released into the synaptic cleft, they travel across it and bind with corresponding receptors on the dendrite of an adjacent neuron. **Receptors**, proteins on the cell surface where neurotransmitters attach, vary in shape, with different shapes "matching" different neurotransmitters.

How does a neurotransmitter "know" which receptor to bind to? The neurotransmitter and the receptor have what is referred to as a lock-and-key relationship—specific neurotransmitters fit specific receptors similar to how a key fits a lock. The neurotransmitter binds to any receptor that it fits.

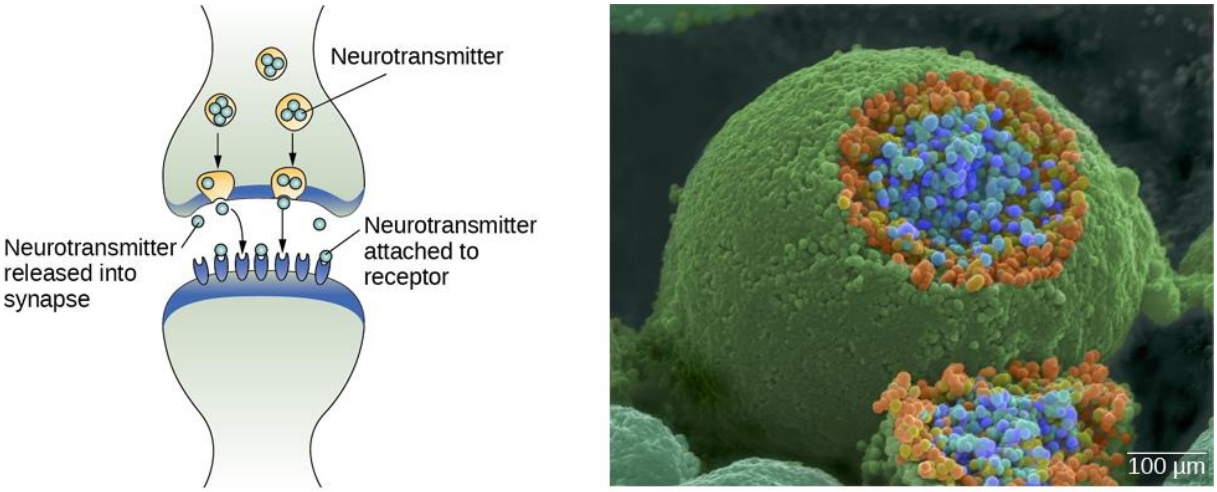


Figure 3.9 (a) The synaptic cleft is the space between the terminal button of one neuron and the dendrite of another neuron. (b) In this pseudo-colored image from a scanning electron microscope, a terminal button (green) has been opened to reveal the synaptic vesicles (orange and blue) inside. Each vesicle contains about 10,000 neurotransmitter molecules. (credit b: modification of work by Tina Carvalho, NIH-NIGMS; scale-bar data from Matt Russell)

Neuronal Communication

Now that we have learned about the basic structures of the neuron and the role that these structures play in neuronal communication, let's take a closer look at the signal itself—how it moves through the neuron and then jumps to the next neuron, where the process is repeated.

We begin at the neuronal membrane. The neuron exists in a fluid environment—it is surrounded by extracellular fluid and contains intracellular fluid (i.e., cytoplasm). The

neuronal membrane keeps these two fluids separate—a critical role because the electrical signal that passes through the neuron depends on the intra- and extracellular fluids being electrically different. This difference in charge across the membrane, called the **membrane potential**, provides energy for the signal.

The electrical charge of the fluids is caused by charged molecules (ions) dissolved in the fluid. The semipermeable nature of the neuronal membrane somewhat restricts the movement of these charged molecules, and, as a result, some of the charged particles tend to become more concentrated either inside or outside the cell.

Between signals, the neuron membrane's potential is held in a state of readiness, called the **resting potential**. Like a rubber band stretched out and waiting to spring into action, ions line up on either side of the cell membrane, ready to rush across the membrane when the neuron goes active and the membrane opens its gates. Ions in high-concentration areas are ready to move to low-concentration areas, and positive ions are ready to move to areas with a negative charge.

In the resting state, sodium (Na^+) is at higher concentrations outside the cell, so it will tend to move into the cell. Potassium (K^+), on the other hand, is more concentrated inside the cell, and will tend to move out of the cell (Figure 3.10). In addition, the inside of the cell is slightly negatively charged compared to the outside, due to the activity of the sodium-potassium pump. This pump actively transports three sodium ions out of the cell for every two potassium ions in, creating a net negative charge inside the cell. This provides an additional force on sodium, causing it to move into the cell.

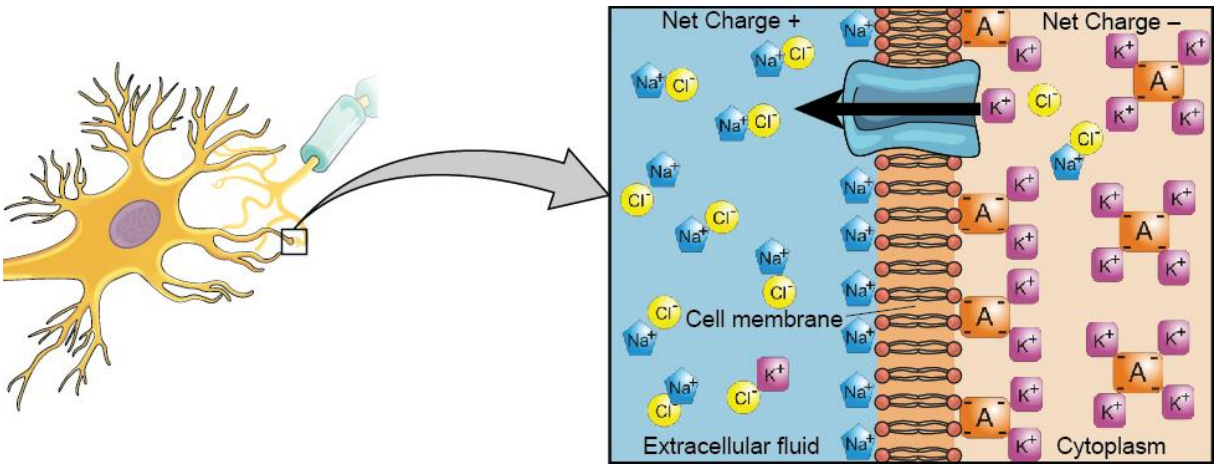


Figure 3.10 At resting potential, Na^+ (blue pentagons) is more highly concentrated outside the cell in the extracellular fluid (shown in blue), whereas K^+ (purple squares) is more highly concentrated near the membrane in the cytoplasm or intracellular fluid. Other molecules, such as chloride ions (yellow circles) and negatively charged proteins (brown squares), help contribute to a positive net charge in the extracellular fluid and a negative net charge in the intracellular fluid.

From this resting potential state, the neuron receives a signal and its state changes abruptly (Figure 3.11). When a neuron receives signals at the dendrites—due to neurotransmitters from an adjacent neuron binding to its receptors—small pores, or gates, open on the neuronal membrane, allowing Na^+ ions, propelled by both charge and concentration differences, to move into the cell. With this influx of positive ions, the internal charge of the cell becomes

more positive. If that charge reaches a certain level, called the **threshold of excitation**, the neuron becomes active and the action potential begins.

Many additional pores open, causing a massive influx of Na^+ ions and a huge positive spike in the membrane potential, the peak action potential. At the peak of the spike, the sodium gates close and the potassium gates open. As positively charged potassium ions leave, the cell quickly begins repolarization. At first, it hyperpolarizes, becoming slightly more negative than the resting potential, and then it levels off, returning to the resting potential.

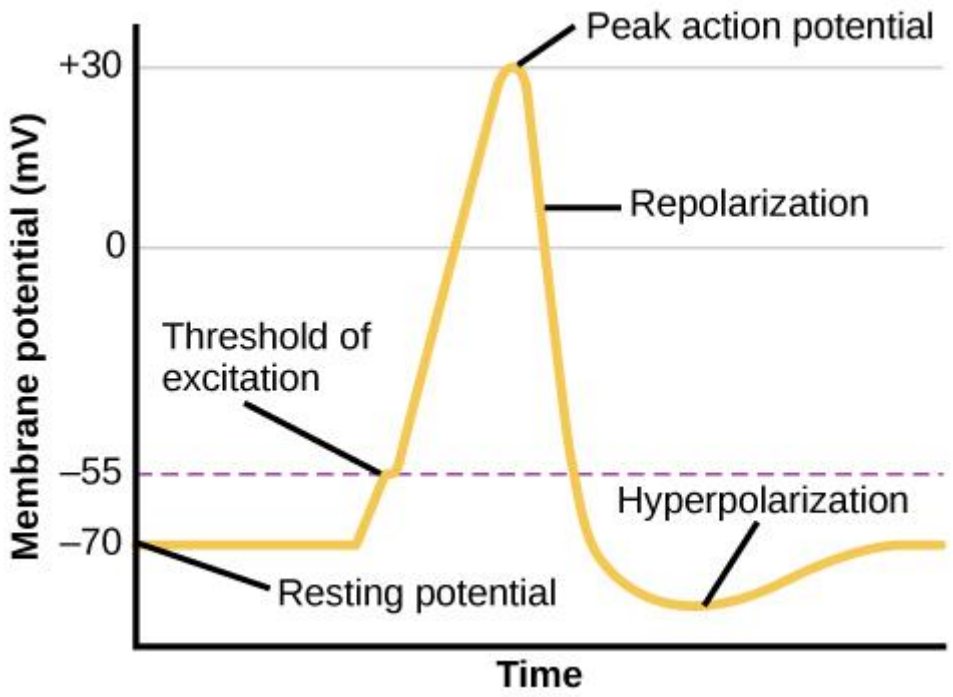


Figure 3.11 During the action potential, the electrical charge across the membrane changes dramatically.

This positive spike constitutes the **action potential**: the electrical signal that typically moves from the cell body down the axon to the axon terminals. The electrical signal moves down the axon with the impulses jumping in a leapfrog fashion between the Nodes of Ranvier. The Nodes of Ranvier are natural gaps in the myelin sheath. At each point, some of the sodium ions that enter the cell diffuse to the next section of the axon, raising the charge past the threshold of excitation and triggering a new influx of sodium ions. The action potential moves all the way down the axon in this fashion until reaching the terminal buttons.

The action potential is an **all-or-none** phenomenon. In simple terms, this means that an incoming signal from another neuron is either sufficient or insufficient to reach the threshold of excitation. There is no in-between, and there is no turning off an action potential once it starts. Think of it like sending an email or a text message. You can think about sending it all you want, but the message is not sent until you hit the send button. Furthermore, once you send the message, there is no stopping it.

Because it is all or none, the action potential is recreated, or propagated, at its full strength at every point along the axon. Much like the lit fuse of a firecracker, it does not fade away as it

travels down the axon. It is this all-or-none property that explains the fact that your brain perceives an injury to a distant body part like your toe as equally painful as one to your nose.

As noted earlier, when the action potential arrives at the terminal button, the synaptic vesicles release their neurotransmitters into the synaptic cleft. The neurotransmitters travel across the synapse and bind to receptors on the dendrites of the adjacent neuron, and the process repeats itself in the new neuron (assuming the signal is sufficiently strong to trigger an action potential). Once the signal is delivered, excess neurotransmitters in the synaptic cleft drift away, are broken down into inactive fragments, or are reabsorbed in a process known as **reuptake**. Reuptake involves the neurotransmitter being pumped back into the neuron that released it, in order to clear the synapse (Figure 3.12). Clearing the synapse serves both to provide a clear "on" and "off" state between signals and to regulate the production of neurotransmitter (full synaptic vesicles provide signals that no additional neurotransmitters need to be produced).

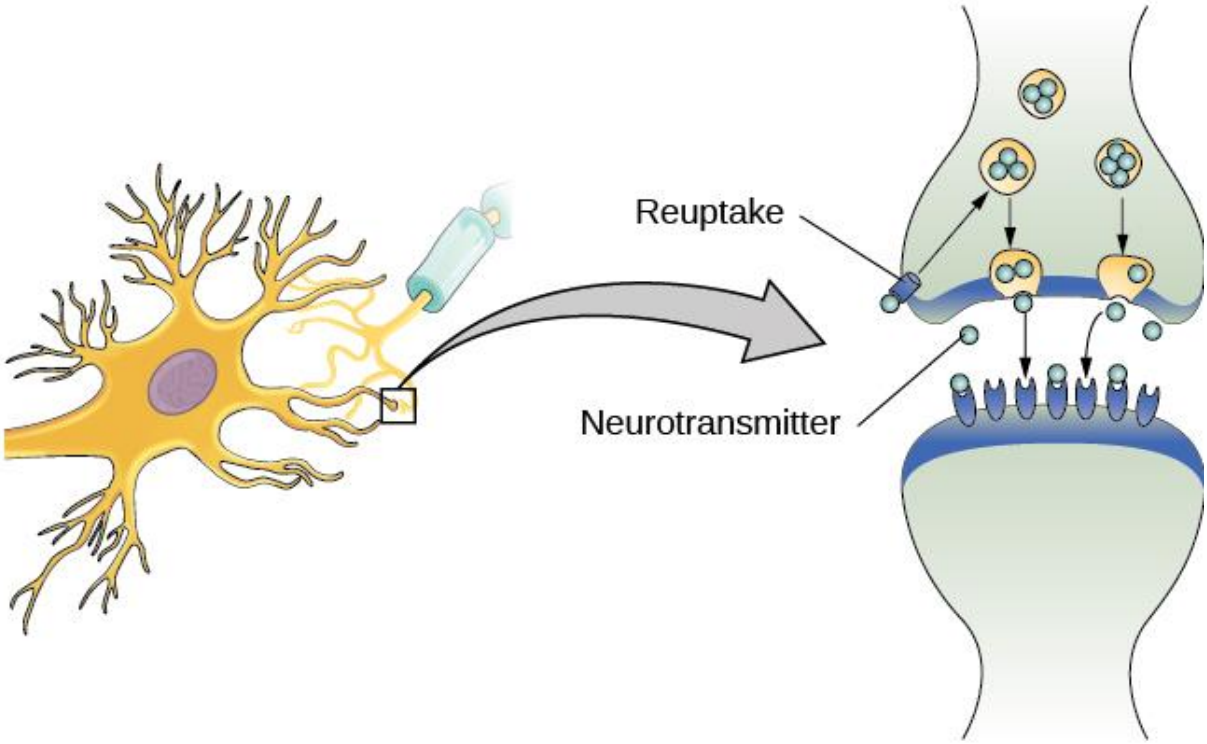


Figure 3.12 Reuptake involves moving a neurotransmitter from the synapse back into the axon terminal from which it was released.

Neuronal communication is often referred to as an electrochemical event. The movement of the action potential down the length of the axon is an electrical event, and movement of the neurotransmitter across the synaptic space represents the chemical portion of the process. However, there are some specialized connections between neurons that are entirely electrical. In such cases, the neurons are said to communicate via an electrical synapse. In these cases, two neurons physically connect to one another via gap junctions, which allows the current from one cell to pass into the next. There are far fewer electrical synapses in the brain, but those that do exist are much faster than the chemical synapses that have been described above (Connors & Long, 2004).

LINK TO LEARNING

Watch this video about neuronal communication to learn more.

Neurotransmitters and Drugs

There are several different types of neurotransmitters released by different neurons, and we can speak in broad terms about the kinds of functions associated with different neurotransmitters (Table 3.1). Much of what psychologists know about the functions of neurotransmitters comes from research on the effects of drugs in psychological disorders. Psychologists who take a **biological perspective** and focus on the physiological causes of behavior assert that psychological disorders like depression and schizophrenia are associated with imbalances in one or more neurotransmitter systems. In this perspective, psychotropic medications can help improve the symptoms associated with these disorders. **Psychotropic medications** are drugs that treat psychiatric symptoms by restoring neurotransmitter balance.

Major Neurotransmitters and How They Affect Behavior

Neurotransmitter	Involved in	Potential Effect on Behavior
Acetylcholine	Muscle action, memory	Increased arousal, enhanced cognition
Beta-endorphin	Pain, pleasure	Decreased anxiety, decreased tension
Dopamine	Mood, sleep, learning	Increased pleasure, suppressed appeti
Gamma-aminobutyric acid (GABA)	Brain function, sleep	Decreased anxiety, decreased tension
Glutamate	Memory, learning	Increased learning, enhanced memory
Norepinephrine	Heart, intestines, alertness	Increased arousal, suppressed appetite
Serotonin	Mood, sleep	Modulated mood, suppressed appetite

Table 3.1

Psychoactive drugs can act as agonists or antagonists for a given neurotransmitter system. **Agonists** are chemicals that mimic a neurotransmitter at the receptor site. An **antagonist**, on the other hand, blocks or impedes the normal activity of a neurotransmitter at the receptor. Agonists and antagonists represent drugs that are prescribed to correct the specific neurotransmitter imbalances underlying a person's condition. For example, Parkinson's disease, a progressive nervous system disorder, is associated with low levels of dopamine. Therefore, a common treatment strategy for Parkinson's disease involves using dopamine agonists, which mimic the effects of dopamine by binding to dopamine receptors.

Certain symptoms of schizophrenia are associated with overactive dopamine neurotransmission. The antipsychotics used to treat these symptoms are antagonists for dopamine—they block dopamine's effects by binding its receptors without activating them. Thus, they prevent dopamine released by one neuron from signaling information to adjacent neurons.

In contrast to agonists and antagonists, which both operate by binding to receptor sites, reuptake inhibitors prevent unused neurotransmitters from being transported back to the neuron. This allows neurotransmitters to remain active in the synaptic cleft for longer durations, increasing their effectiveness. Depression, which has been consistently linked with reduced serotonin levels, is commonly treated with selective serotonin reuptake inhibitors (SSRIs). By preventing reuptake, SSRIs strengthen the effect of serotonin, giving it more time to interact with serotonin receptors on dendrites. Common SSRIs on the market today include Prozac, Paxil, and Zoloft. The drug LSD is structurally very similar to serotonin, and it affects the same neurons and receptors as serotonin. Psychotropic drugs are not instant solutions for people suffering from psychological disorders. Often, an individual must take a drug for several weeks before seeing improvement, and many psychoactive drugs have significant negative side effects. Furthermore, individuals vary dramatically in how they respond to the drugs. To improve chances for success, it is not uncommon for people receiving pharmacotherapy to undergo psychological and/or behavioral therapies as well. Some research suggests that combining drug therapy with other forms of therapy tends to be more effective than any one treatment alone (for one such example, see March et al., 2007).

3. Parts of the Nervous System

Learning Objectives

By the end of this section, you will be able to:

- Describe the difference between the central and peripheral nervous systems
- Explain the difference between the somatic and autonomic nervous systems
- Differentiate between the sympathetic and parasympathetic divisions of the autonomic nervous system

The nervous system can be divided into two major subdivisions: the **central nervous system (CNS)** and the **peripheral nervous system (PNS)**, shown in Figure 3.13. The CNS is comprised of the brain and spinal cord; the PNS connects the CNS to the rest of the body. In this section, we focus on the peripheral nervous system; later, we look at the brain and spinal cord.

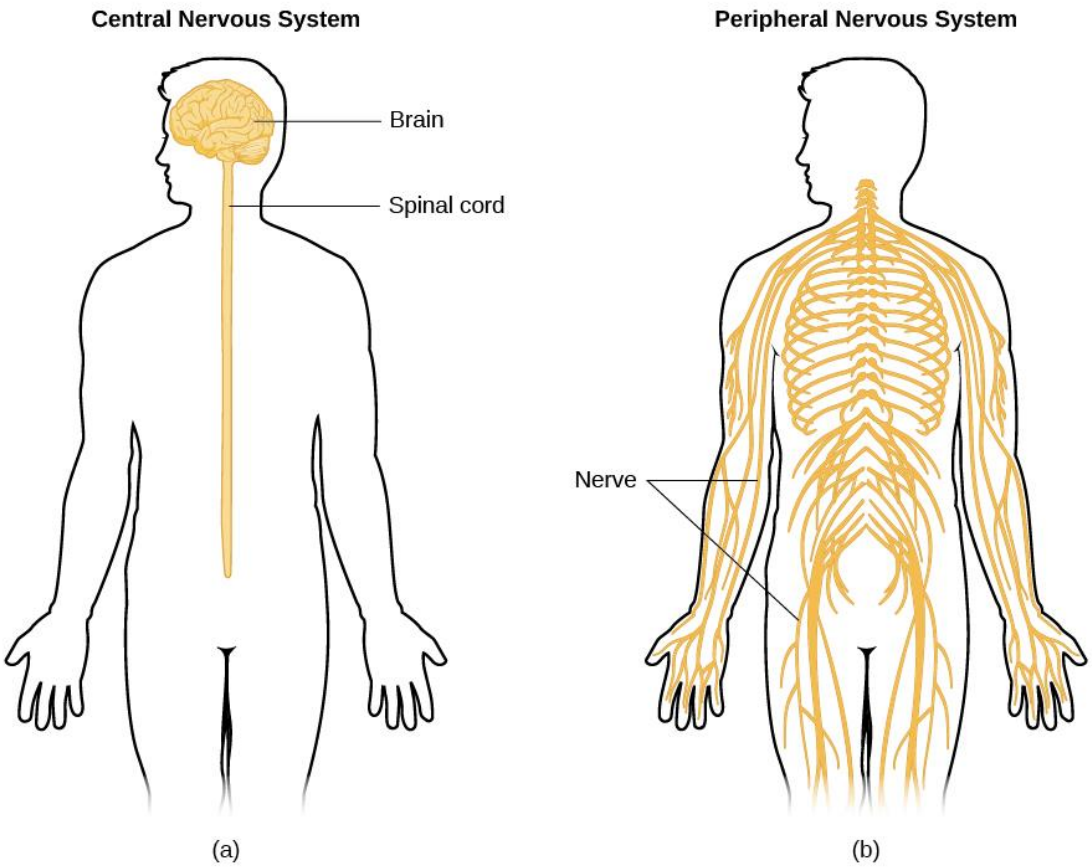


Figure 3.13 The nervous system is divided into two major parts: (a) the Central Nervous System and (b) the Peripheral Nervous System.

Peripheral Nervous System

The peripheral nervous system is made up of thick bundles of axons, called nerves, carrying messages back and forth between the CNS and the muscles, organs, and senses in the periphery of the body (i.e., everything outside the CNS). The PNS has two major subdivisions: the somatic nervous system and the autonomic nervous system.

The **somatic nervous system** is associated with activities traditionally thought of as conscious or voluntary. It is involved in the relay of sensory and motor information to and from the CNS; therefore, it consists of motor neurons and sensory neurons. Motor neurons, carrying instructions from the CNS to the muscles, are efferent fibers (efferent means "moving away from"). Sensory neurons, carrying sensory information to the CNS, are afferent fibers (afferent means "moving toward"). A helpful way to remember this is that **e**fferent = **e**xit and **a**fferent = **a**rrive. Each nerve is basically a bundle of neurons forming a two-way superhighway, containing thousands of axons, both efferent and afferent.

The **autonomic nervous system** controls our internal organs and glands and is generally considered to be outside the realm of voluntary control. It can be further subdivided into the sympathetic and parasympathetic divisions (Figure 3.14). The **sympathetic nervous system** is involved in preparing the body for stress-related activities; the **parasympathetic nervous system** is associated with returning the body to routine, day-to-day operations. The two systems have complementary functions, operating in tandem to maintain the body's

homeostasis. **Homeostasis** is a state of equilibrium, or balance, in which biological conditions (such as body temperature) are maintained at optimal levels.

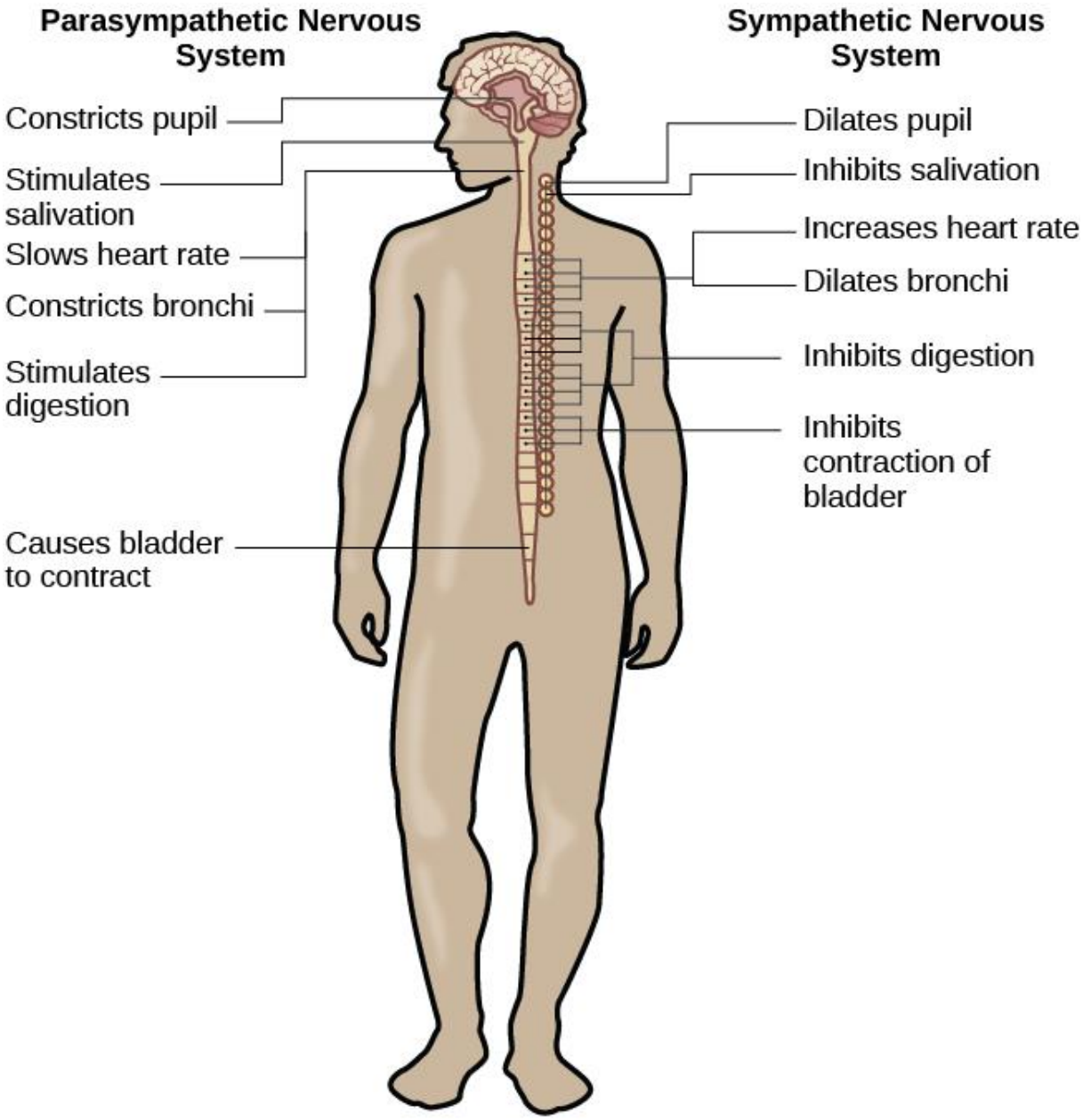


Figure 3.14 The sympathetic and parasympathetic divisions of the autonomic nervous system have the opposite effects on various systems.

The sympathetic nervous system is activated when we are faced with stressful or high-arousal situations. The activity of this system was adaptive for our ancestors, increasing their chances of survival. Imagine, for example, that one of our early ancestors, out hunting small game, suddenly disturbs a large bear with her cubs. At that moment, the hunter's body undergoes a series of changes—a direct function of sympathetic activation—preparing them to face the threat. The pupils dilate, the heart rate and blood pressure increase, the bladder relaxes, and the liver releases glucose; adrenaline surges into the bloodstream. This constellation of physiological changes, known as the **fight or flight response**, allows the body access to

energy reserves and heightened sensory capacity so that it might fight off a threat or run away to safety.

LINK TO LEARNING

Watch this video about the Fight Flight Freeze response to learn more.

While it is clear that such a response would be critical for survival for our ancestors, who lived in a world full of real physical threats, many of the high-arousal situations we face in the modern world are more psychological in nature. For example, think about how you feel when you have to stand up and give a presentation in front of a roomful of people, or right before taking a big test. You are in no real physical danger in those situations, and yet you have evolved to respond to a perceived threat with the fight or flight response. This kind of response is not nearly as adaptive in the modern world; in fact, we suffer negative health consequences when faced constantly with psychological threats that we can neither fight nor flee. Recent research suggests that an increase in susceptibility to heart disease (Chandola, Brunner, & Marmot, 2006) and impaired function of the immune system (Glaser & Kiecolt-Glaser, 2005) are among the many negative consequences of persistent and repeated exposure to stressful situations. Some of this tendency for stress reactivity can be wired by early experiences of trauma.

Once the threat has been resolved, the parasympathetic nervous system takes over and returns bodily functions to a relaxed state. Our hunter's heart rate and blood pressure return to normal, the pupils constrict, bladder control is restored, and the liver begins to store glucose in the form of glycogen for future use. These restorative processes are associated with activation of the parasympathetic nervous system.